PIG-HELL (ENTERITIS NECROTICANS).

THE RECOGNITION AND STUDY OF A GANGRENOUS ENTERITIS

IN THE HIGHLANDS OF NEW GUINEA

THESIS

SUBMITTED FOR THE

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BY

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I declare that the composition of this thesis is entirely my own and that it is a true record of an original study of the disease "Fip-bel" in the Highlands of New Guinea. The material has not been submitted to any other University for an award of any other degree or diploma.
CHAPTER I

INTRODUCTION
Enteric disease is one of the greatest problems facing public health authorities in underdeveloped countries (Ordway, World Health Organisation Study Group on Diarrhoeal Diseases, Geneva, 1958). It is the diarrhoeal diseases which are chiefly responsible for the high infant and child mortality rates in these countries (Hardy, 1959) and such illnesses are related to low standards of sanitation and personal hygiene.

In Papua and New Guinea, particularly in the Highlands region, these problems exist in communities which are primitive and generally unaffected by modern living. Although there are no data on the incidence of the diarrhoeal diseases in New Guinea, a number of studies have been conducted on the nutritional status of these peoples (Hippley and Clements, 1947; Oomen and Malcolm, 1958; Venkatasahlan, 1962; Bailey, 1963a, 1963b). A pattern of widespread protein under-nutrition was shown by these surveys, which were mainly conducted in the Chimbu area of the Highlands.

Although the relationship between infectious diarrhoea and protein malnutrition was not investigated, in other countries where infant mortality is high, it has been shown that infection and protein deficiency act together to produce an effect far more severe than would result from either alone (Brook and Autret, 1952; Williams, 1952; Jelliffe, 1955; Sarimshaw, Wilson and Bressani, 1960).
Ward (1958) conducted a preliminary survey of the infectious diseases in the Western Highlands of New Guinea and concluded that, with the exception of the tubercle bacillus, the majority of the common human pathogens were present. He also considered that the factors favouring the prevalence of infectious disease were severe protein deficiency and low standards of hygiene and sanitation.

The present study describes a severe and probably unique diarrhoeal disease in the Highlands which appears to be related to the pork eating customs of the people. The condition, which commences as an acute enteritis and later develops the features of acute gangrenous enteritis, has been called "Pig-bel" (Figure 1).

This name is suggested because the disease appears to be peculiar to New Guinea and because "pig" is also the Pidgin English word for the animal believed to have an aetiological relationship to the disease. "Bel" literally means abdomen or belly in Pidgin, but the word is also used in many delightful paraphrases to describe the emotions. An example is the translation of "he is angry" to "en e gat krea long bel". "Pig-bel", as such, is an expression used by the native health workers in the Highlands to describe the abdominal discomfort associated with pig feasting. It is proposed therefore to use this new specific term in the text of this thesis for a disease now familiar to health workers in New Guinea as "enteritis necroticans", a term introduced following its discovery in 1961 (Murrell and Roth,
Necropsy appearances of "pig-bel" (enteritis necroticans). The autopsy was performed on a seven year old boy four hours after death. His illness had lasted six days (Case 150).
1963). By defining the disease as pig-bel, the author hopes to delineate the disease from other related terms used to describe a non-specific group of diseases having the pathological features of a necrotizing enteritis.

"Enteritis necroticans" was the name adopted by German writers for a similar condition which appeared in epidemic proportions in Northern Germany immediately following World War II (Hansen, Jeckeln, Jochims, Lesius, Mayer-Burgdorff and Schütz, 1949). Believing that the disease in Germany was *necroticans* a new disease in men, one group of writers led by Jeckeln (1947) specifically named the disease "Darmbrand". The literal English translation of this is "burnt bowel", and the name aptly describes the appearance of the small intestine in a severe case. The term has the clinical meaning of "bowel gangrene". In 1949, Zeissler of Hamburg attributed the disease to infection with *Clostridium perfringens* type F (*Clostridium welchii* type F, Bacillus enterotoxicus) (Zeissler and Rassfeld-Sternberg, 1949a, 1949b).

During 1961 the author and a colleague (Murrell and Roth, 1963) found in a number of patients at laparotomy, a severe patchy gangrenous condition of the upper small intestine. No mechanical or vascular cause for the gangrene was evident. The commoner intestinal pathogens were looked for and not found, but large Gram positive rods predominated in the smears taken from the duodenal
and jejunal contents of cases at operation or autopsy. Anaerobic
culture yielded *Clostridium perfringens* (*Cl. welchii*) and this led to the
supposition that the disease was a spontaneous gas gangrene of the
bowel. Because the process involved mainly the jejunum and
because the aetiology remained obscure, the syndrome in New Guinea
was initially referred to as "necrotising jejunitis". *Cl. perfringens*
type C was identified from intestinal contents of these patients
independently by Rowan (1963) at the School of Public Health and
Tropical Medicine in Sydney, and by Egerton and Walker (1964) at
the Veterinary Laboratory, Port Moresby, and the Wellcome Research
Laboratories, Kent, United Kingdom. The diagnosis of pig-bell was
firmly established by Samels at the latter laboratory during 1963-
1964. She was able to demonstrate a rise and fall of antitoxin
levels to the beta toxin produced by *Cl. perfringens* type C in sera
collected by the author from patients with the more protracted form
of the illness.

In New Guinea, enteritis necroticans was not recognised
clinically until 1961 and was regarded then as a new syndrome in
that country. Since the initial report of the disease, several
workers have reported to the author, in retrospect, their familiarity
with it. Thus Territory Medical Officers stationed at Chinsu in
1955, Mend in 1957 and Tari in 1960 saw cases of this disease at
autopsy (Ivinskis, Malcolm and Roderigue, personal communications).
A common antecedent history of pork consumption led the author to suspect that the disease was a food-borne infection (Hurrell and Beth, 1963). This view necessitated a study of the ecology of the disease, as well as an epidemiological, aetiological and clinical appraisal of the condition as it appeared in various Highland centres during the years 1961 to 1964 inclusive.

General medical duties of the author precluded a more thorough investigation of each individual case. The general conditions under which the present study evolved were conducted in hospitals providing minimal laboratory facilities and only semi-trained or illiterate local medical staff. It was not feasible for all cases to undergo complete bacteriological or serological investigation. Selected groups, therefore, were studied in order to define specific aspects of the disease. The author relied upon clinical information in some instances from existing hospital records which, at times, proved inadequate. A plan of treatment evolved by trial and error. Initial mistakes were made because there were no standard references available in the treatment of such a rare and fatal disease.

A continuing study of cases was conducted in order to define and classify the different stages of the disease. Initially, the diagnosis was established on pathological and clinical grounds alone. Investigations were then extended by further bacteriological and serological methods in an attempt to clarify the epidemiology.
In this context it was essential to study the cultural and public health aspects of pig-feasting in relation to pig-bel and other gastro-enteric infections. An examination of morbidity and mortality data from Highland hospitals reinforced certain of the epidemiological conclusions.

The pathology and etiology were correlated with the clinical aspects of the disease. Selected cases were included in follow-up studies to assess the short and long term effects of the disease. It was of interest that various components of the malabsorption syndrome were observed in cases from this group.

A study of the beliefs and customs of the people relevant to the subject of enteric disease as a whole and pig-bel in particular was considered necessary if control was to be attempted. Schofield and Parkinson (1963) in a study on the social aspects of health in two population groups of the Sepik District of New Guinea summed up this type of situation as follows:

"A knowledge of the living conditions of the people is a necessity for the doctor trying to improve the health of a community. In addition to understanding the effects of the physical environment he must possess information on the customs by which social behaviour is regulated and on the beliefs by which the problems of life are explained."
This thesis therefore covers the recognition and description of a new disease "Pig-bel", having the pathology of a necrotizing enteritis, in the Highlands of New Guinea. It attempts to relate the disease to pork feasting. \textit{C. perfringens} type C probably plays some part in the etiology, but many questions concerning the disease and its ramifications remain to be answered.
CHAPTER II

GENERAL REVIEW
Table I: The pathogenicity of Cl. perfringens in man and animals
(After McLeman, 1956)

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<td>Type A</td>
<td>Gas gangrene and food poisoning in man. Enterotoxaemia in calves.</td>
</tr>
<tr>
<td>Type B</td>
<td>Lamb dysentery of sheep. Enterotoxaemia of foals and calves.</td>
</tr>
<tr>
<td>Type C</td>
<td>&quot;Struck&quot; in sheep. Haemorrhagic enteritis of calves and lambs. Enterotoxaemia of piglets.</td>
</tr>
<tr>
<td>Type D</td>
<td>Infectious enterotoxaemia of lambs and goats. Pulpy kidney disease of sheep.</td>
</tr>
<tr>
<td>Type E</td>
<td>Enterotoxaemia of calves.</td>
</tr>
<tr>
<td>Type F (sub-type C)</td>
<td>Enteritis necroticans of man (&quot;Darmbrant&quot;).</td>
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The etiology of pig-bel (enteritis necroticans) is discussed in Chapter V, but at this point it is relevant to review other enteric diseases attributed to infection by *Clostridium perfringens*. This is especially pertinent because some of these diseases have features similar to enteritis necroticans. These are briefly outlined prior to a more complete review of the disease as it appeared in Europe soon after the close of World War II.

The disease in New Guinea was inseparably associated with the customs of the people. Some of these sociological and anthropological aspects therefore are examined as an introduction to the ecology of the disease.

1. Enteric diseases attributed to *Clostridium perfringens*

*Clostridia*, being widely distributed in nature and common inhabitants of the intestinal tract, have been implicated in a number of diseases in both human and veterinary medicine. While the role of the *Clostridia* in wound infections and abortal sepsis was established early, evidence for their involvement in enteric infections has only recently been presented. Macfadden (1956) described six types of *Clostridium perfringens* capable of causing disease in man and animals (Table I). It is evident that *Clostridium perfringens* might well be regarded as primarily an intestinal pathogen and that it is more important in veterinary than human medicine.
(a) *Clostridium perfringens* type A

Little attention was paid by earlier workers to food poisoning caused by anaerobic bacteria other than *Clostridium botulinum*. *Clostridium perfringens* type A strains are frequently isolated from normal stools and from a variety of food-stuffs. Klein (1895) first drew attention to this organism as a possible enteric pathogen when he isolated it from faeces of patients in two outbreaks of diarrhoea in hospital. During World War II when mass food preparation in canteens became necessary, it was shown that anaerobic spore-bearing bacteria were a potent cause of food poisoning. McLung (1945) first described *Clostridium perfringens* food poisoning as such. It was not until Hobbs and her colleagues published in 1953, epidemiological and bacteriological data on twenty-three outbreaks of food poisoning in the London area, that the disease became widely recognised (Hobbs, Smith, Oakley, Warrack and Cruickshank, 1953). During 1963 *Clostridium perfringens* accounted for 30 to 40 per cent of the large outbreaks of food poisoning in the United Kingdom (Robertson, 1963).

The vehicle of infection is almost always reheated or cold precooked meat. The incubation period in these epidemics has ranged from three hours to more than twenty-four hours, in contrast to the shorter and more uniform period associated with staphylococcal food poisoning.

An outbreak of necrotising enteritis caused by an atypical strain of *Clostridium perfringens* type A was recently reported from Indonesia.
by Can, Biran, Sukeno, Satri, Sujud, Waraa and Mjoe (1962). The epidemic occurred near Djakarta but the origin of the infection was not discovered.

(b) \textit{Clostridium perfringens} types \textit{B}, \textit{D} and \textit{E}

These organisms, together with type \textit{C}, have always been regarded as specific pathogens of animals, each having a wide host range. Thus type \textit{B} is associated with lamb dysentery (Dalling, 1928), enterotoxaemia in foals (Montgomery and Rowlandis, 1937) and calves (Hepple, 1952); type \textit{D} with pulpy kidney disease of sheep (Bennetta, 1932) and type \textit{E} with an enterotoxaemia of calves (Bosworth, 1943).

Gleeson-White and Bullen (1955) and Kahn and Warrack (1955) reported two cases of the type \textit{D} organism occurring in humans. One case had intestinal obstruction and the other a severe diarrhoea complicating amoebic colitis. \textit{Clostridium perfringens} type \textit{D} also causes enterotoxaemia in goats (Cher, 1956) and cows (Keast and McBarren, 1954). Both of these diseases were reported from Australia.

Further reference is made later to the pathogenetic association of this disease in animals and enteritis necroticans in man in which a dietary change is believed to play a significant role in the development of the disease.

(c) \textit{Clostridium perfringens} type \textit{C}

McEwen (1930) first isolated strains of \textit{Clostridium perfringens} type \textit{C} from cases of "struck" or "strike" in adult sheep. In "struck" the pathological feature is a necrotising enteritis. The condition was
called "enterotoxaemia" when it occurred in lambs (Griner and Baldwin, 1954). A disease of similar pathology was reported earlier in calves by Griner and Brecken (1953) from which Cl. perfringens type C was recovered. Two years later an enterotoxaemia of piglets was attributed to strains of this organism (Field and Gibson, 1955).

Only recently in the United States, Barnes and Moon (1964) recovered the organism from pigs with this disease. The clostridial origin of these veterinary diseases makes it a possibility that pig-bel in man in New Guinea has a similar etiology.

Brooks, Sterne and Warreak (1957) and Sterne and van Heyningen (1958) considered that the type F strains of Cl. perfringens isolated during the German outbreaks of enteritis necroticans were in fact type C strains, differentiated by their abnormal heat resistance. These strains have now officially been classified as type C varieties following the discovery of the New Guinea strains (Egerton and Walker, 1964; Sterne and Warreak, 1964).

2. Enteritis necroticans and allied syndromes

(a) Pseudomembranous enterocolitis or necrotizing enteritis

Pseudomembranous enterocolitis, also known as necrotizing enteritis, is occasionally a complication of abdominal surgery, particularly after partial gastrectomy. The formation of a pseudo-membrane in the bowel is known to occur in a variety of otherwise unrelated diseases including uraemia, heavy metal intoxication,
bacillary dysentery and paratyphoid enterocolitis. The post-
operative and post-antibiotic conditions are usually presumed to be
of staphylococcal origin.

Pseudomembranous enterocolitis was reported as early as 1875 by
Belfrage and Medesius. Finney (1893) then described the condition
as "diphtheritic colitis". The gross appearances of the bowel were
described by Riedel (1902) as "diphtheria of the bowel not due to
Loeffler's bacillus but to necrosis of the mucous membranes". Since
these early reports the condition after operation has been frequently
recognized (Penner and Bernheim, 1939; Dixon and Weismann, 1948;
Pettit, Baggenstoss, Deering and Judd, 1954; Dawson-Edwards and
Morrisey, 1955; Killingbeck and Lloyd, 1961). No specific cause
has been found although Killingbeck and Lloyd (1961) considered that
Cl. perfringens might be a pathogenic agent. Penner and Bernheim
(1939) concluded that the condition was a sequel to intense vaso-
constriction of the intestinal vessels because of shock.
Experimental studies on dogs by McKay, Hardaway, Wahle and Hall
(1955) have also supported this hypothesis.

Kleckner, Barger and Baggenstoss (1952) described 14 cases in
which the condition was not a sequel to operation. They stated
that the prevalence of the condition had not increased since antibi-
otics came into use and that it was not usually associated with
the staphylococcus or any other pathogen. Kay, Richards and Watson
(1958) preferred the term "acute necrotising enterocolitis". They stressed the importance of distinguishing the disease from staphylococcal enteritis because the latter condition is milder and potentially reversible. Wilson and Qualheim (1954) used the term "acute hemorrhagic enterocolitis" to describe the condition sometimes seen in chronically ill elderly individuals. Ming and Levitan (1960) reported the disease to be an acute hemorrhagic necrosis of the bowel but offered no explanation as to its cause.

A similar condition, "enteritis gravis", was described more recently by Buckley, Seiden, Jimenez and Kaufman (1962), the etiology being unknown.

In a review of these conditions Harston (1962) believed that, with the exception of staphylococcal enteritis, the final pathological state was bowel ischaemia. He also attributed the German outbreaks of enteritis necroticans to this cause.

In Australia, Bloomfield and Walters (1960) reported cases of pseudomembranous enterocolitis for which no adequate cause was found. Rose (1946, 1952) had earlier described the condition as acute regional enterocolitis in reporting four cases. Woodward (1960) and Webb (1960) independently documented cases of necrotising enteritis associated with and without abdominal surgery. Recently Coulston, Skyring and McGovern (1965) described four cases of ulcerative jejunitis associated with malabsorption for which no
cause could be found. Colman and Miald (1965) then followed this report from Sydney by describing two cases of necrotizing enteritis in which haemorrhage was the dominant clinical feature. In both of these reports adequate bacteriological studies were not made.

Recently Wiklander (1964) promoted the suggestion that the endotoxins of the Gram-negative bacteria may be associated with these ill-defined conditions. He considered that the clinical observations and experimental studies of tissue as well as of general reactions to endotoxins correspond so closely with the clinical features of phlegmonous or necrotizing enterocolitis that endotoxins must be seriously considered as an etiologic factor.

It seems probable that pseudomembranous enterocolitis and acute necrotizing enteritis represent a final pathological state for several disturbances. Penman and Druckerman (1946) reached this conclusion nearly 20 years ago and very little has been added to an understanding of the problem since.

(b) Enteritis necroticans and Darmbrand

Enteritis necroticans is regarded as an uncommon disease. However, during and shortly after the second World War large numbers of people were afflicted with this disease in Norway and Germany, especially in Hamburg, Lübeck and Kiel. Many publications reporting these outbreaks appeared at this time. After the epidemic occurrence in Norway and Germany, the disease seemed to subside and was
recorded only sporadically in the literature. Jockeln (1957), who had experience with a large number of patients in Lübeck expressed the view that this intestinal disease, the most alarming he knew, had abated.

From Hamburg, in 1946, Beckermann and Leas reported seven cases of a severe, often fatal illness due to a necrotic inflammation of several areas of the small intestine. The first case in their series developed the disease some years earlier in 1935. Ernst (1948a) observed a sporadic case as early as 1934, although the condition "enteritis necroticans ulcerosa" had been described much earlier than this in Russian prisoners of war during World War I by Jaffe (1918). Jockeln (1947) and Ruppert (1947) in Lübeck subsequently described a similar apparently new disease and called it "Darmbrannt". Klein (1947) and Ernst (1948b) described the pathology as a "necrotizing enteritis". Pick (1947) and Pick and Holben (1949) referred to the condition as "necrotic jejunitis" and recalled personal cases as early as 1944. From Leipzig, Wigand (1947) described similar cases of necrotizing jejunitis and Haschke (1948) referred to the condition as "jejunitis acuta phlegmonosa".

Reports showing that the disease had reached epidemic proportions appeared in both the German and Scandinavian literature, all with varying nomenclature (Beckermann, 1947; Hermann, 1947; Klee, 1947; Mannel and Boving, 1947; Bardeleben, 1948; Schilen, 1948; Singmann,
More confusion in defining the disease was then provided by Kloos and Brooman (1949) who thought that the title "Enteritis Grevis" should be given to the disease. No one specific cause could be agreed upon for the disease by these workers. When Zoissler in 1948 and then in association with Bassefiel-Danenberg (1949a, 1949b) found what they believed to be the cause, the condition was called "enteritis necroticans due to Clostridium perfringens type F".

Epidemics were established in July and August of 1946 and again in July, August and September of 1947 in the Kiel-Lübeck-Hamburg triangle of Northern Germany (Herrmann, 1947; Ernst, 1948a; Hansen, 1949). The disease was described in detail in a monograph "Darmbrand - Enteritis Necroticans" published in 1949 (Hansen et alii, 1949).

The etiology remained clouded. One view was promoted that the population in Germany, subjected to poor and unbalanced diets in the latter war years, was predisposed to the disease (Siegwund, 1948a). It was certain that anaerobic invasion of the bowel occurred but the conditions under which this change occurred and the events leading to the gangrene were not understood. Derrmann (1948) thought that a one-sided bacterial flora became manifest when richer diets were eaten. Stasis, distension and stony led to a vascular shut down of the terminal collateral arterioles resulting in the ischaemic necrosis. Baniecke (1947), Siegwund (1948) and
Schoen (1947) went further and believed that it was the toxins of the fermentative bacteria, particularly the anaerobic clostridial organisms, which caused spasm and thrombosis of the submucosal vessels, so evident in the micro-pathology. Siegmund (1948b) also associated the disease with undernourished subjects, although all classes of persons were equally affected. Views diverged as to which bacterium or bacteria were the direct cause.

Shigella and Salmonella organisms were looked for and not found. Acute arsenical poisoning was also excluded by Hansen (1948). Schildt (1949a, 1949b) first considered that an unusually virulent type of Cl. perfringens related to the Welch-Frankel bacillus (gas gangrene bacillus) was the cause. This was borne out by the toxic effect and rapid course of the disease. Zeissler and Emsfeld-Starnberg (1949a, 1949b) isolated an organism closely resembling Cl. perfringens from the bowel contents of 12 cases of enteritis necroticans. Definitive differences between this organism and Cl. perfringens type A were the high thermal resistance of its spores and the production of beta toxin. These characteristics differentiated the new bacillus from other types of clostridia and the organism was called "Cl. welchii type F or "Bacillus enterotoxicanus". Oakley (1949a, 1949b) confirmed that the German strains consistently produced beta toxin which was quantitatively neutralised by Cl. welchii antitoxin. This was the principal toxin produced by the type B organism responsible for
lamb dysentery. Some years later Oakley (1963) corrected his classification of this organism and considered it to belong to the type C group.

Hain (1949a) in Hamburg then demonstrated the presence of _Clostridium perfringens_ type F in 16 per cent of faeces collected from 108 control subjects. He also clearly identified the source of this organism from a can of home tinned rabbit which had caused a family outbreak (Hain, 1949a, 1949b).

The disease was reproduced in animals according to Koch's postulate by Schults (1949), Zeissler (1948) and Krause (1949, 1950) but only under certain optimal conditions of pH and diet. The first of these workers described an animal inoculation test which he considered was diagnostic. A bacterium free filtrate of duodenal contents, when injected intraperitoneally into a guinea pig, caused death within minutes from overwhelming toxemia.

The view that this organism was the primary cause of the disease has been adopted by several recent authors (Cattan, Carasso, Antissier, Ray, Hivet, Verley, 1961; Hobbs et alii, 1953; Jarkowski and Wolf, 1962; Patterson and Rosenbaum, 1952; MacLennan, 1956 and 1962).

During 1948 in central Germany, 150 cases of enteritis necroticans were reported to the Department of Health, Berlin, and a "Bowel Gangrene Committee" was set up to investigate the disease (Marcusse and Konig, 1950). The basis of forming this committee was
to gain the co-operation of physicians, surgeons, pathologists and bacteriologists. The clinical features of 147 cases in the Berlin area had been reported earlier by Anders (1949). At the Central Health Institute, these workers confirmed Zaisler's bacteriological findings isolating \textit{Clostridium perfringens} type F from 22 of the 41 cases of known disease. The specific organism was not recovered from 31 control subjects. Dischmann (1949) examined sixty-three cultures of \textit{Clostridium perfringens} from wounds of soldiers serving on the Russian front and found that only two were type F strains. Further bacteriological confirmation of the aetiology was provided by a study of 42 cases by Pletsenka and Rosenfeld-Sternberg (1950). \textit{Clostridium perfringens} type F was isolated from all of these cases. (Twenty-four were from the Dithmarschen area near Kiel and eighteen from Hamburg.) A double infection with typhoid or paratyphoid and \textit{Clostridium perfringens} type F occurred in five of this series. The latter organism was recovered up to four to six weeks after the illness in seven cases and up to four months later in three cases.

However, these authors were still baffled by the sporadic nature of the disease if a food poisoning epidemiology was to be contemplated. In dietary histories taken from several patients in this series, engargement by rich and bad food was common but normally only one member of a family at risk suffered the severe form of the disease. They concluded that enteritis necroticans occupied a mid-position between an exogenous poisonous disease like botulism and an
endogenous enteric illness like paratyphoid.

In a review of 124 cases, Grissman (1950) was not convinced that a specific infective agent was the cause of Darmsbrand. There was a wide range of variables between the mild and severe forms of the disease which he maintained were explained by local disturbances in circulation. Secondary electrical invasion inevitably occurred when this happened. Curtius and Ahrens (1948) also believed that there was not one pathognomonic symptom of Darmsbrand and that the disease had no specific etiology.

Löckenkämper (1948) and Brandt (1949) postulated that a neurotropic virus was the cause because the intramural nerves of the bowel wall showed degeneration of the ganglion cells. Even earlier, a virus etiology had been suggested by Herms (1948). This writer thought that the behavioural pattern of the disease simulated that of polysymalitis. Kloes and Brummond (1951) made a comprehensive study of the epidemiology of the disease in the northern districts of Germany excluding Lübeck. They called their 335 case series "Enteritis Gravis" and came to the conclusion that there was a genuine bowel infection. This, however, was not the result of food poisoning. A number of factors indicated that the epidemic was widespread with many mild and sub-clinical cases. Diabetes mellitus seemed to be a predisposing factor because there were four times as many diabetics in the group as normals. They concluded that the disease was probably of viral origin, although
me virus was isolated, and that bacteria of the Welch-Frankel group
as well as other facultative pathogenic bacteria became involved
only by secondary invasion. They did not consider the disease to
be new in Germany and this view was reaffirmed by Kloos and Nissen
three years later in 1954.

Initially therefore, it may be said that there were two schools
of thought in Germany concerning the etiology of enteritis
macericans. One, led by workers in Lübeck and Hamburg (Zeaal,er,
Schütz, Schützen, Krauspe) believed that Cl. perfringens type F was
the specific causal agent. The other, led by workers in Kiel (Kloos,
Nissen, Griesmann, Hornmann) were of the opinion that a secondary
invasion by facultative anaerobic bacteria took place, the primary
cause remaining unknown.

A third major etiological cause was proposed by Kulpe (1948)
and Koelwski (1951). Their hypothesis was that the necrosis had
an allergic basis described as a Schwartzman phenomena (Schwartzman,
1928). Koelwski fed guinea pigs with a meat diet for three weeks,
and after a meat free interval of two weeks, reintroduced large meat
meals. Anaphylactic shock caused death in the animals. They
developed asthmatic symptoms and oedema, eosinophilic infiltration
and fibrinoid swelling in the terminal mucosal vessels of the upper
intestine. He considered that the animals developed a sensitivity
to beef protein and that a similar stage of affairs occurred in the
early lesions of Darmbränd, promoted by a dietary change from
carbohydrate to protein.

(c) Enteritis necroticans outside Germany

Cases described by the term "enteritis necroticans" have been reported elsewhere although the bacteriology was incomplete. The first record outside Germany was in Argentina (DeFilippe, 1948). de Almeida and Hungria Filho (1949) reported the condition in Brazil and von Heyenberg and Bosch-Gwalter in Switzerland (1953). These latter writers also noted that the disease had occurred in Denmark, Austria and Italy. Egedy, Szanto and Sotonyi (1950) also described cases in Hungary. Although Scandinavian writers in Norway and Sweden (Brynjulfson, 1948; Berg, 1949; Hartsberg, 1954) used the term "jejunitis acuta" to describe large case series, they considered the disease to be the same as the "enteritis necroticans" of the German reports. Patterson and Rosenbaum (1952) reported the first case in the United States in a woman with idiopathic steatorrhea. Greville Young (1949) and Calnan (1950) reported cases in the United Kingdom. A case complicating pregnancy was reported by Telford, Govan and MacIntosh (1957). Blesova and Novotny (1952) associated the presence of Cl. perfringens type F with the so-called infant dyspepsias. Except for this last reference, the bacteriological investigations of cases outside of Germany have been inadequate and no specific cause for the disease found.

The disease seems to have attracted little attention in France, Holland and Belgium; most publications from these countries are of
recent date (Olivier, 1949; Hollander and Schwingt, 1953; Houtsmuller and Koster, 1956; Koster, 1957; Cattan et alii, 1961).

It can be appreciated, then, that the disease has been described under a variety of names, for example, Darmbrand, enteritis necroticans, jejunitis acuta, enteritis phlegmonosa, enterocolitis necroticans, enteritis gravis, phlegmonous and necrotising enterocolitis (Wiklander, 1964). But the syndrome known as "Darmbrand" has been the only entity in this group of synonymous conditions which appeared in similar epidemic members apart from New Guinea. Because of this similarity and because of a causal relationship with pork consumption, it is suggested that the New Guinea condition should be known as "Pig-bel".

The People of the Highlands

(a) Origins

The evidence from unearthed implements resembling Palaeolithic south-east Asian materials indicates that the high valleys have long been populated (Bulmer, 1960). The present highlander shares some of the cultural and racial characteristics of the Melanesian. In Australian New Guinea the people can be divided into two main cultural groups. The village people east of Chiabu (population 200 to 400 per clan) have two broad language families. West of the Chiabu the people live in scattered hamlets (population 20 to 40 per lineage) in three further language families (Wurm, 1962).
Europeans first ventured into the highlands in 1927. These were German missionary expeditions. In 1933, Taylor, Spinks and the Leabys entered the Bena, Asaro, Chimbu and Waghi valleys in successive patrols (Souter, 1964). Roman Catholic and Lutheran missions were established a year later at Chimbu and Mount Hagen. Since that time Administration and Mission personnel have brought law, order and the Christian religion to most highland areas. It is interesting to record that the external staff of the two largest missions, namely, Roman Catholic and Lutheran, is made up predominantly of European immigrants from Germany.

(b) Social Structure

The highlanders have been accustomed to wearing little clothing. For warmth at night they rely solely on fires in the centre of the floor of their squat thatched houses. The social structure consists chiefly of a subsistence economy, the recognition of bonds of kinship with obligations extending beyond the family group, and a strong attachment to their land. Aside from the tribe, the important racial units are conceived in terms of common agnostic descent. The New Guinea highland societies have all been shown to have a hierarchy of patrilineal groups (Brookfield, 1961). There are no formal political institutions. Any emphasis on the acquisition of material goods is not primarily for personal use or the creation of a differential living standard, but rather as a means of establishing
individual prestige and status within the community. This is achieved by displays at feasts and the performance of a complex sequence of gift exchanges.

(c) Beliefs and Practices

Magico-religious beliefs and practices are an integral part of the indigenous cultures. They are numerous and diverse in character and are largely based on ancestor and spirit worship. General initiation, fertility and mourning rites are important to the people. The placation of ancestral ghosts is paramount in rules governing agriculture, hunting, marriage and pig killing ceremonies. The people have not built up a consistent theology or magicology as in the great religious denominations of the world, but continue their magical practices because tradition has given these a validity. Sickness and misfortune are normally ascribed to sorcery, the breaking of taboos, or to malevolent spirits (Murrell, 1961; Schofield and Parkinson, 1963). Pigs occupy a unique position in these beliefs. Faeces, blood and meat from the animal are used by medicine men to drive away evil spirits. Eating pork is considered to make a man strong, and any illness resulting from the eating of pig meat is blamed upon witchcraft worked on the individual by a known or unknown sorcerer. It is believed that meat from the pig cannot cause illness on its own accord.

Ninety-five per cent of the Highlands people are engaged in agriculture. Food is grown to meet their own needs, and occasionally
for sale or barter. The basis of their subsistence is shifting
cultivation of a staple food, the sweet potato (*Ipomoea batatas*).
The principal subsidiary crops in lower altitude areas are sugar
cane (*Saccharum officinarum*) and bananas (*Musa spp.*) of which many
species are grown. Other crops form only a small part of the
diet and are interplanted in mixed gardens. These include tare
(*Colocasia, Aleoasia*, and *Zanthosoma spp.*), maize (*Zea mays*),
beans (*Phaseolus spp.*), cucumbers, squash, ginger, yams (*Dioscorea
spp.*) and manioc (*Manihot spp.*). The winged bean, *Saccharum adule,*
and other green vegetables collectively known as "kumu" in Pidgin,
are relished on festive occasions. The wild lima bean (*Phaseolus
lunatus Linn.*) is sometimes mistaken for the winged bean, and being
cyaniogenic, causes acute poisoning and rapid death. Leaves of a
coleus plant (*Amaranthus tricolor Linn.*) when boiled give a red dye
and when improperly cooked can give rise to enteric poisoning.
During seasonal bearing pandanus nuts (*Pandanus julianettii* and *P.
prosimae*) are stored and eaten, being associated with magico-
religious beliefs. *P. conoides* has a red fruit, is used to
garnish food and in excess can give rise to gastritis and diarrhoea.
Groundnut (*Arachis hypogaea*) has been introduced to the diet on the
recommendation of Bailey (1963a) and is well accepted. Highland
breadfruit (*Ficus insipida*) is of value and the leaves are
widely used as mat and coverings at pig feasts.
Division of labour between the sexes involves the extension of a woman's work beyond domestic duties. She maintains the garden and cares for the domestic animals (pigs, dogs and fowls). However, men perform the heavy work of clearing, logging, fencing and building.

In the last 30 years in the Chimbu, Asaro and Naghi valleys and in the last 5 to 10 years in other areas, the people have learnt to use steel tools and to grow and process agricultural products such as coffee, tobacco, groundnuts and pyrethrum. Some young men have worked on plantations elsewhere in the Territory. The Highlanders have seen the introduction of rudimentary health services, schools, supplementary agriculture, a police and judicial system and local government administration. Tribal warfare has, in the main, been controlled.

Despite these changes, there are still no accurate demographic, fertility or mortality data available.

(4) The Pig in Highland Culture

In spite of this progress, one practice remains common to all highland cultures, that of pig raising and feasting. Pig ownership remains the highest status symbol in these societies. The Enga men when questioned about their obvious preoccupation with pigs, reply briefly "pigs are our hearts" (Meggitt, 1958). Anthropologists have stressed the importance of pigs is in the economic and social structure of the Highlander (Nilles, 1950, 1953; Read, 1952; Bermit, 1954; Meggitt, 1958; Brookfield and Brown, 1958). The
pig is the central figure in all social events - part of the bride price in weddings, currency in compensations, and the main menu dish in feasts celebrating the epochal events in the life of the Highlander. It is offered as a sacrifice to placate the spirits of the deceased; it is offered when crops have failed and when sickness occurs; wherever, in fact, the ancestor spirits, good or evil, might be.

Ownership of pigs therefore, is not an asset in regard to food supplies. Rather their possession and circulation are values in themselves in enhancing a man's social status and prestige. Pigs form the basis of most exchanges. In large presentations at pig ceremonies, men of each segment of a donor group pool their offerings to display the group's contribution. Members of the recipient group who are not satisfied with the return for a previous payment, feel and express both an individual and group grievance. In this way the individual contributes through a surplus of pigs, not only to his own prestige, but also to that of his group.

The pigs are owned and herded by the extended family but circulate so rapidly in some communities that it is difficult to assess individual ownership. In the Enga this has been estimated by Meggitt (1958) as a mean of 5.4 pigs per married man and 1.9 per single man. This figure would be lower in the Chimbu and Gahuku clans. The number of pigs held by a tribe at any one time, of
course, increases from the last large pig kill. These occur at 3
to 10 year intervals but small scale pork feasting for marriage
payments and the like continue all year. It has been noted that
small amounts of pork are eaten at monthly intervals (Murrell,
1961) and the observations of Roderigue (1964) in the southern
highlands confirm this. A delayed food shortage in the months
following the larger festivals which resulted in calorie and protein
deficiency was shown by Bailey (1963b). It is the large pig kills
which affect the incidence of enteritis necroticans for reasons
which become apparent later in the text.

The pig cycle varies in form from place to place. The "Te"
festival or gift exchange (Bus, 1951) of the Enga involves an
exchange of live pigs. The pig killing part of the "Te" also
serves the purpose of credit repayments and occurs in other highland
areas on a large scale. These exchanges occur in one direction
along well defined trade routes which may take up to six months to
conclude. The pig kill at Tampul, which marked the commencement of
a succession of pig killing for a period of two months, had trade
remissions with the Huri and Mendi peoples of the Southern
Highlands, and the Hagen tribes near Mount Hagen and Biyer River.
In the Lai, Tecak and Wabag valleys the "Te" affects some 40,000
people. Mesgitt (1958) estimates that the total number of pigs
involved in a given cycle to be about 30,000 of which at least half
are slaughtered at the end of the cycle and their porks sent back
along the valleys.

These big pig kills are controlled essentially by the size of the pig populations. Over a four year period they were known to take place at Upper Asaro, Baiyer River, Mendi, Tari, Wabag, Bumi and Chimbu (see Figures 2 to 5). Within one large linguistic group alternating cycles do take place. For instance, in central Chimbu, the Naregu, Siambuga, Wanga, Kabuku, Gena, Sim and Kamanegu, two clans held ceremonies during 1955 and 1956 whilst the Siku, Nogar, Kamanegu 1, and Kup 1 and 2 held theirs in 1959 and 1960 (Brown and Brookfield, 1963). The tribes near Bumi commenced their ceremonies in January 1964. The cycle in the upper Chimbu commenced in May and was concluded near Gembogl in June 1964, the feast being called the "bogla ganea" (Nilles, 1950). Extension of this activity to the Sisimi, Dang and Kup census divisions of the Chimbu took place in September and October of this same year (Figures 3 and 4). The last large festival at Gembogl had taken place three years earlier (Bailay, 1965a).

This alternation evens out the distribution of pork consumption to some extent and makes herd rebuilding easier, but it does result in an imbalance in diet and an increase in the time interval between the cooking and consumption of the pork. The possibilities of food poisoning are thus increased because (a) the volume of pork distributed is so great that it encourages storage in unsanitary
Figure 2: The geographical location of reported and observed cases of pig-bel from January 1961 to November 1964.
Figure 3: A map of the Eastern Highlands District of New Guinea. Places mentioned in the text appear as follows:

Upper Asaro lat. 6°0’ long. 145°15’
Bundi lat. 5°45’ long. 145°15’
Upper Chambu lat. 6°0’ long. 154°0’
Figure 23 A map of the Central and Upper Chimbu. Investigations were conducted at Gogina, Gorowa, and Koromogl. Directional movements of pork followed pig killing in May. The direction was reversed in September-October when the Southern Chimbu tribes had their celebrations. Each round dot represents a case of pig-bel.
Figure 5: A map of the Western Highlands. The directional movement of the pig exchange is shown by the arrows. The pig killing commenced at Tambul. Lake Kopingo appears in the extreme western tip of the district.
surroundings, and (b) the incubation of food contaminants is
prolonged by the distances over which the meat has to be carried.
This may be up to four or five days' walk in the Southern or Western
Highlands. Another factor of some importance in this practice is
the number of times one piece of meat may change hands in a series
of gift exchanges. Surprisingly, the final recipient is still
quite happy with his payment but may be forced to carve his portion
under water because it is so putrid. The possessor then shares it
with his immediate relatives. If it is really too foul, they save
the fat for decorations and throw away the meat.

The rotten pork is never blamed for deaths which are always
attributed to ghostly malevolence. Latterly, with migrations away
from home to areas for work and in the freer inter-tribal communi-
cations and relations, pork is travelling longer distances and
magnifying the problem. Motor vehicles are now being hired for
this purpose for far distant creditors.

A further potential source of infection comes from eating sick
and dying pigs. Illnesses associated with such practices are again
attributed to magic. Similar beliefs are held about illness in
pigs, although there are taboos and rules of pig husbandry which must
be avoided or adhered to. For example, the eating of human fceses
or menstrual blood is thought to cause harm to the pig. Healthy
pigs are kept away from a sick pig or a pig being prepared for
cooking (Meggitt, 1958).
From the foregoing statements it is apparent that pig husbandry plays an important part in the social life of the people. Most of the male pigs are castrated at a few months of age and herded with the sows. Boars are now imported and a fee of one or two piglets is charged for their service. This breeding technique has considerably improved the quality of the native pig, *Sus rupicola* (Egerton and Murrell, 1964). The main responsibility for the pigs belongs to a man's wife, a bachelor's married sister or a widowed mother. She feeds them daily and cleans their stalls which usually are included in the women's houses. During the day the pigs are turned loose to graze in unsupervised areas. Here they eat grass, herbage and shrubs, and root for worms. At night they are fed sweet potato. The prices a woman pays for neglecting her pigs is a beating by her husband. It is interesting to record that the commonest fracture treated at a Highland hospital is a mid-shaft radius and ulnar fracture of the forearm in a young woman. In so many instances this was sustained bywarding off blows from an irate husband in a dispute over the pigs.
CHAPTER III

MATERIALS AND METHODS
Case records were kept of patients with known and suspected enteritis necroticans, during the period January 1961 to November 1964. During most of 1962 colleagues provided much of the clinical information, for which acknowledgement appears elsewhere. Three circulars, one of the 5th of August 1961, another on the 6th April 1963 and the third on the 29th April 1963 (Appendix 1 to III) were circulated to medical personnel working in Highland hospitals requesting that material and patients be sent to Goroka. A list of the names, where available, of all cases reviewed in the study appears in Appendix VII.

Biological material was collected and forwarded initially to the School of Public Health and Tropical Medicine Sydney, and the Department of Pathology, University of Western Australia. Subsequently it was sent to the Veterinary Laboratory and Laboratory of the General Hospital, Port Moresby, to the Wellcome Research Laboratories, Beckenham, Kent, United Kingdom, and to the Institute of Medical and Veterinary Science and the Department of Pathology, University of Adelaide, Adelaide.

The investigations included epidemiological, clinical, bacteriological and pathological studies. Additional information pertaining to the prevalence of diarrhoeal diseases generally were obtained from hospital records at three hospitals in which the author worked, from other Administration hospitals at Mount Hagen and Tari, and from the Baptist Mission hospital at Baiyer River.
1. Case histories were obtained by direct questioning using the dictum Pidgin English, medical orderlies acting as interpreters. These findings, together with those on clinical examination, were recorded on a case card (Appendix IV). These data were then transferred to "Inwista" Sorter cards Pat. No. 4150 and analysed from these. A total of 210 cases were reviewed in this study (Appendix VII). This included specific clinical data on 160 persons.

2. Special investigations.

(a) Blood and serum examinations were performed on only a proportion of the above cases because laboratory and communication difficulties prevented a complete appraisal of each individual case. Examinations included haemoglobin and white cell estimations, erythrocyte sedimentation rate, blood urea, total serum protein and serum electrolyte estimations. Antitoxin levels to the beta toxin of Cl. perfringens were estimated in 58 cases, serial samples being taken from 21 persons with the disease. The serum was separated, kept under refrigeration and one drop of 50% o-cresol-ether added to each ml. of serum as a preservative. These were despatched in batches to the Wellcome Research Laboratories for estimation of Cl. perfringens beta antitoxin by the method outlined in Appendix VI (Glenny, Llewellyn-Jones and Mason, 1931; Glenny, Bar, Llewellyn-Jones, Dalling and Ross, 1933).

(b) Bacteriological investigations, except for cases in 1961, were conducted in the Veterinary Laboratory, Port Harresky.
Thirty-eight resected specimens of bowel were forwarded packed in ice in a thermos, after tying off a segment proximally and distally. The minimal delay for such specimens in reaching the laboratory was three days. Fecal specimens from 107 suspected and contact cases were placed in wide-necked bottles containing Stuart's transport media. During 1961, fecal specimens were placed in selenite F broth and Robertson's cooked meat media and despatched to the School of Public Health and Tropical Medicine for examination.

In the laboratory material from the lumen and necrotic wall of affected bowel was examined for the presence of known aerobic and anaerobic bacterial pathogens. A specimen was inoculated directly on to sheep blood agar, desoxycholate citrate agar plates and into enrichment media of selenite F and tetraethionate broth. The blood agar inoculations were cultured aerobically and anaerobically. Colonies morphologically and biochemically resembling Cl. perfringens were forwarded to the Wellcome Research Laboratories for identification by methods described by Egerton and Walker (1964).

(c) Radiological investigations were limited by hospital facilities. Representative X-Ray findings in the acute and subacute disease were selected from hospital records. Barium follow through studies were conducted on two patients with the subacute disease and two normal subjects. In other isolated subacute cases, barium X-Rays were taken at 30, 60, 120 and 200 minute intervals.
3. From the recorded clinical data, various treatment schedules were assessed. Two clinical trials conducted on subjects with the surgical disease were made in 1961 and 1964. In the first, polyvalent gas gangrene antiserum was given to seven subjects and withheld from twelve. In the second, 42,000–85,000 units of specific Cl. perfringens type C antiserum (batch N9595, Wellcome Laboratories) were given by slow intravenous injection to 32 consecutive cases. The results were compared with 66 earlier cases in which treatment was standardised except for the specific antiserum. Twenty-six persons with suspected mild disease were also given the serum by intramuscular injection to test for adverse serum reactions.

4. Pathological material was examined from twenty-four subjects at autopsy and seventy-three persons at laparotomy. Histopathological reports were obtained on forty-three occasions in this series. Sections were preserved in 10 per cent formol-saline. These findings were recorded on a card designed for the follow-up studies (Appendix V). It was subsequently discovered that the information in this record was too detailed so that only the relevant details were recorded.

5. Follow-up studies for malabsorption syndrome were conducted on twenty post-operative subjects and on twenty-nine patients surviving on conservative management. The period after infection of this examination ranged from four weeks to three years.
Twenty-three normal subjects were used as controls. The following investigations were undertaken.

(a) Haemoglobin estimations were measured on all but two patients in this series. Blood counts and bone marrow biopsies were performed according to the methods described by Desio (1956).

(b) Serum folie acid activity was measured microbiologically in 56 subjects with Lactobacillus casei as the test organism, using a modification of the method described by Herbert (1961). Acid cleaned glassware and containers rinsed in glass-distilled water were used for the collection of blood. There was a delay of at least five days before the specimens reached the laboratory in Adelaide. Serum levels less than 2.0 mg. per ml. were considered to be abnormal. (Deller, Ibbotson and Crompton, 1964).

(c) Vitamin B₁₂ in the same serum where possible (36 subjects) was assayed by the method of Hutner, Burch and Hess (1956) using Pseudomonas aeruginosa 2 strain.

(d) Serum protein electrophoretic patterns were obtained from 16 sera remaining after the above tests had been performed.

(e) Examination for 

Cl. perfringens of jejunal aspirations and faeces were carried out on 17 individuals, the collection and transporting methods being as previously described.
(f) Barium meal and follow-through studies were performed
on nine cases (five post-operative, two post-conservative,
two control).

(g) Faecal fat excretion on a 3-day stool collection was
undertaken in only seventeen patients where steatorrhoea
was suspected. There were four control subjects.
Greater than 30 per cent of fat in dry faeces was
considered to indicate steatorrhoea (King and Wooton,
1956).

(h) "Blind" per-oral intestinal biopsy using the Crosby
capsule (Crosby and Kugler, 1957) was attempted in
twenty-nine subjects.

6. Epidemiological information was obtained by examining 220
case records of patients with pig-bel (Appendix VII). Further
data were collected in the field on six different occasions - in
June and August 1961 in Upper Asaro; September 1963 in Tembul and
Wabag; May and October 1964 in Upper Chimbu; and from June to July
1964 in Chuave, Tari, Lake Kopiaga, Baiyer River (Figures 2 to 5).
The first four periods coincided with pig feasting activities of the
local people. The following procedures were undertaken:

(a) Comparative observations of pig slaughtering methods,
preparation of meat for cooking, cooking methods and
pork distribution. Sources of contamination were
looked for.
(b) Pig carcasses were examined for any noteworthy pathological abnormalities.

(c) Random samples of faeces and intestinal contents were taken from 322 pigs for bacteriological examination. These were collected aseptically, placed in sterile bottles containing Stuart’s transport media and sent airfreight to Port Moresby, packed in ice. At the Veterinary Laboratory Port Moresby anaerobic culture was undertaken as described by Egerton and Walker (1964).

(d) Samples of pig meat in the fresh, cooked and stored state were collected as in (c) above and also forwarded under refrigeration to Port Moresby. Here, similar bacteriological examinations were made on the samples using the same methods.

(e) Fly and soil specimens were collected at Coglane (Upper Chis要素) and subjected to the same bacteriological analysis.

(f) A systematic survey to determine the frequency of Cl. penfringsen type C in the normal population was undertaken during June to July 1964. Clean unsterile waxed paper cups were issued by random sampling to individuals on a basis of a 1 in 5 sample. About 5 grams of faeces were transferred to wide necked sterile bottles containing Stuart’s transport media and forwarded to Port Moresby and from there to London as in (c) above for Cl. penfringsen
isolation. Due to laboratory limitations this was later halved to a 1 in 10 population sample. Four hundred and fifty-five examinations were thus completed.

(g) A record of the foods eaten and estimations of the weight and volume of individual meals were made. Temperatures of the cooking process and cooked meat were taken with a mercury glass thermometer and a thermocoupled pyrometer.

(h) Blood samples were taken from 216 volunteer subjects with 30 ml. vacuum venules. These volunteers were representative of seven different Highland groups. These were from Upper Asaro, Chuave and Upper Chibbu (clans in the Eastern Highlands); Baiyer River, Wabag, and Lake Kopiago (clans in the Western Highlands), and one clan near Tari in the Southern Highlands.

(i) A population census was conducted in an endemic area at Goromau in the Upper Chibbu in October of 1964 (Biscoe, 1964). An approximate estimate of the disease incidence was obtained by equating the population figures to known persons with the disease treated at Kundiswa hospital following earlier pig feasting.

(j) Information on diarrhoeal disease patterns and causes of death were obtained from records of the Goroka, Tari, Baiyer River and Kundiswa hospitals for periods between
1st January 1962 and 30th November 1964. From the Gorsea hospital records a survey of surgical operations undertaken for acute and subacute intra-abdominal conditions for the four-year period 1st January 1961 to 30th November 1964 was also made.

7. Statistical methods.

Results were analysed according to the statistical methods outlined by Bailey (1959). List of symbols used in statistical formulae:

\[ a, b, c, d = \text{used for observed frequencies in contingency tables.} \]
\[ x^2 = \text{chi-squared.} \]
\[ n, n_1, n_2 = \text{numbers of observations in samples.} \]
\[ p = \text{significance level actually achieved by data.} \]
\[ s = \text{estimated standard deviation.} \]
\[ \sum = \text{summation symbol.} \]
\[ x, x_1 = \text{observed measurements.} \]
\[ \bar{x} = \text{mean of sample measurements.} \]

Statistical formulae:

(1) Calculation of mean and standard deviation

\[
\text{mean} (\bar{x}) \quad \bar{x} = \frac{1}{n} \sum x
\]

\[
\text{standard deviation} (s) = \sqrt{\frac{1}{n} \sum (x - \bar{x})^2}
\]
(2) Comparing the percentages of two samples in $2 \times 2$ contingency tables - $x^2$ test

$$x^2 = \frac{n \left\{ (a \times d) - (e \times b) \right\}^2}{(a + b)(c + d)(a + c)(b + d)}$$

(3) Significance tests.

The significance levels for the various tests were obtained from published tables (Bailey, 1959).

$p > 0.05$ not significant.

$p < 0.05$ significant at $p < 0.05$ or almost significant.

$p < 0.01$ significant at $p < 0.01$ or significant.

$p < 0.001$ significant at $p < 0.001$ or highly significant.
CHAPTER IV

RESULTS
1. Definition

For the purposes of this thesis pig-bell is defined as an acute patchy necrotising and inflammatory disease of the small bowel occurring in Highland people of New Guinea. Pig feasting and infection with *Cl. perfringens* type C appear to be important predisposing influences. If untreated, the fulminant form of the disease progresses to complete segmental gangrene of parts of the small intestine with the development of ileus, toxemia and oligemic shock.

Clinically, the disease is characterised by severe upper abdominal pain, bloody diarrhoea and vomiting. There are three different types: fulminant; obstructive; and enteritic. A small proportion of patients survives the initial stages of the illness without treatment, but perforation and peritonitis or the development of an acute malabsorption syndrome usually result in death.

Pig-bell was established as a specific syndrome in New Guinea on the following evidence:

1. The frequency with which the syndrome presented in hospital and surgical practice.
2. The macroscopic appearance of the small intestine, especially the jejunum, was typical and constant.
3. The absence of other mechanical and vascular causes to account for the patchy gangrene.
4. X-Ray appearances of jejunitis.
5. The isolation of Cl. perfringens type C from a significant proportion of resected bowel segments.
6. A rising Cl. perfringens beta antitoxin in the more protracted illness.
7. The association of the clinical features of the disease with the pathological and bacteriological findings described above.
8. The dramatic improvement in prognosis when Cl. perfringens type C antiserum was introduced in therapeutic trials.

2. Classification

Attempts to classify different clinical forms of enteritis necroticans became necessary in order to evaluate the etiology, pathogenesis and prognosis of the disease. Because the acute form of the disease presented as an acute abdominal emergency requiring surgical intervention, it was considered appropriate to use the following classification.

I. Acute toxic form: fulminant toxemia and shock
II. Acute surgical form: mechanical and paralytic ileus acute strangulation perforation and peritonitis
III. Sub-acute surgical form:

Acute and chronic malabsorption form:

- cicatrization and stenosis
- "lead pipe" jejunum
- sub-acute strangulation
- short circuiting via internal fistulae
- sub-acute and chronic enteritis

IV. Mild or trivial form:

- complete cure
- mild sub-acute form with chronic enteritis.

The toxic form of the disease has the appearance of fulminant food intoxication with circulatory collapse and death in 80 to 100 per cent of cases, usually within three days. A small percentage enter the second group. The acute surgical form begins with severe upper abdominal pain and diarrhoea followed in some instances by vomiting and the onset of a mechanical and paralytic ileus. Thirty per cent of patients in this group recover without surgery but as the indications for withholding surgical intervention are critical, it is preferable to classify this group as an acute surgical type of the disease. Spontaneous recovery in this group is not possible without bowel decompression and intravenous therapy, and this automatically places it in a surgical category. The third group
represents the sequelae which follow fibrotic scarring associated with a more protracted onset of the illness and which sometimes resulted from cases initially treated conservatively. Therefore cases in group III represent complications of healing or the actual healed stages of the acute surgical disease. These made up 25 per cent of the series in the clinical review.

The mild form consists of an acute gastro-enteritis following puer in which upper abdominal colic is a feature and may continue for varying periods of up to two weeks. Pain is present between the episodes of colic and stools are always occult. Persons in this group do not require fluid replacement or intestinal decompression. Full recovery can be expected, but the diagnosis is retrospective because the clinical picture is indistinguishable from other gastro-enteric infections. It is probable that large sections of the community are affected in this way, but only a small proportion of the population suffers the more severe fulminating syndrome of enteritis necroticans. It was shown that a few mild cases do progress to the sub-acute surgical form, but for purposes of assessment these have been included in group III. Certain immunological and environmental factors in the small intestine probably influence this transition from a mild to a severe infection.

In time intervals, persons with acute toxic disease had operation or died within four days, those with the acute surgical form had
Table II: Age distribution of the four clinical types of pig-bel (*enteritis necroticans*)

<table>
<thead>
<tr>
<th>Age Group (Years)</th>
<th>Acute Toxic No.</th>
<th>Acute Surgical No.</th>
<th>Sub-acute Surgical No.</th>
<th>Mild No.</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 - 5</td>
<td>17 (65.4%)</td>
<td>23 (28.4%)</td>
<td>5 (10.9%)</td>
<td>7 (12.3%)</td>
<td>52</td>
</tr>
<tr>
<td>6 - 10</td>
<td>4 (15.4%)</td>
<td>28 (34.6%)</td>
<td>12 (26.1%)</td>
<td>17 (29.8%)</td>
<td>61</td>
</tr>
<tr>
<td>11 - 15</td>
<td>0</td>
<td>10 (12.4%)</td>
<td>4 (8.7%)</td>
<td>8 (14.0%)</td>
<td>22</td>
</tr>
<tr>
<td>16 - 20</td>
<td>1 (3.8%)</td>
<td>5 (6.2%)</td>
<td>6 (13.0%)</td>
<td>10 (17.5%)</td>
<td>22</td>
</tr>
<tr>
<td>21 - 30</td>
<td>2 (7.7%)</td>
<td>5 (6.2%)</td>
<td>7 (15.2%)</td>
<td>7 (12.3%)</td>
<td>21</td>
</tr>
<tr>
<td>31 - 40</td>
<td>1 (3.8%)</td>
<td>7 (8.6%)</td>
<td>6 (13.0%)</td>
<td>5 (8.8%)</td>
<td>19</td>
</tr>
<tr>
<td>41+</td>
<td>1 (3.8%)</td>
<td>3 (3.7%)</td>
<td>6 (13.0%)</td>
<td>3 (5.3%)</td>
<td>13</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>26 (99.9%)</strong></td>
<td><strong>81 (100.1%)</strong></td>
<td><strong>46 (99.9%)</strong></td>
<td><strong>57 (100.0%)</strong></td>
<td><strong>210</strong></td>
</tr>
</tbody>
</table>
Figure 6: The clinical forms of 210 cases of pig-bell arranged by percentage frequency in age groups. The acute disease was confined mainly to infants and children, whereas relatively more adults presented with the sub-acute form of the disease.
Table III: Case fatality rates for the four clinical types of pig-bel

<table>
<thead>
<tr>
<th>Clinical Type</th>
<th>No. Cases</th>
<th>% Total</th>
<th>Deaths</th>
<th>Recovery</th>
<th>Mortality %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute toxic</td>
<td>26</td>
<td>12.4</td>
<td>22</td>
<td>4</td>
<td>84.6</td>
</tr>
<tr>
<td>Acute surgical</td>
<td>81</td>
<td>38.6</td>
<td>34</td>
<td>47</td>
<td>42.0</td>
</tr>
<tr>
<td>Sub-acute surgical</td>
<td>46</td>
<td>21.9</td>
<td>20</td>
<td>26</td>
<td>43.5</td>
</tr>
<tr>
<td>Mild</td>
<td>57</td>
<td>27.1</td>
<td>0</td>
<td>57</td>
<td>0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>210</strong></td>
<td><strong>100.0</strong></td>
<td><strong>76</strong></td>
<td><strong>134</strong></td>
<td><strong>36.2</strong></td>
</tr>
</tbody>
</table>
jejuntotomy or died between the fifth and fourteenth day and patients in the sub-acute surgical group came to operation or died between fourteen days and nine months of onset.

3. Clinical Features

(a) General

Two hundred and ten cases, reviewed in the epidemiology, are grouped according to the classification in Table II and Figure 6. Sixty-five per cent of persons with acute toxic enteritis necroticans fell in the 0 to 5 year age group. The highest percentage of patients in the other groups was 6 to 10 years of age. Relatively more adults presented with the sub-acute type of illness. The fatality rates (Table III), reviewed again in the epidemiology of the disease in section 10 of this chapter, were acute toxic 84.6 per cent, acute surgical 42.0 per cent, sub-acute surgical 43.5 per cent and mild nil.

In this series, clinical records were examined of 160 persons with pig-bel, 112 of whom were examined by the author. This total consisted of 10 acute toxic cases, 59 acute surgical cases, 40 sub-acute surgical cases and 51 mild cases. The overall mortality rate in this group was 27.5 per cent. For the severe forms it was 40.9 per cent. The distribution of the disease according to sex was 2:1 in favour of males.

A dietary history was obtained in all but 13 cases. Prior pork consumption was denied by only seven patients. The meat had been
Table IV: Dietary history (a) and incubation period (b) for 160 persons with pig-bel

(a) Diet  Not Recorded  Yes  No  Total
Pork eaten  13  140  7  160

(b) Incubation (Hours)  Acute Toxic  Acute Surgical  Sub-acute Surgical  Mild  Total
Symptom onset not recorded  0  11  6  3  20
0 - 9  6  2  2  3  13
10 - 19  4  25  9  14  52
20 - 29  0  8  8  13  29
30 - 39  0  10  10  13  33
40 - 49  0  3  3  4  10
over 50  0  0  3*  0  3
TOTAL  10  59  41  50  160
Mean onset  8 hours  18 hours  20 hours  23 hours

*3 cases had an ill-defined onset of symptoms one week after eating bad pork.
cooked or re-cooked for more than 24 hours in 72 per cent of these cases. There was a dietary history of tinned fish (four cases), green peanuts (two cases) and green pineapple (one case) in those not exposed to pork.

Analysis of figures shown on Table IV for the different clinical types indicated that the incubation in the acute toxic disease was short (mean 8 hours) and longer (28, 20 and 23 hours) for the other three types. The range was 5 hours to 7 days.

Because most patients had little concept of time it was difficult to obtain exact information on the time of onset of symptoms. For this reason time was recorded in 10 hour intervals, and the means calculated from the frequency of medians in each group excluding the three with a seven day incubation. In these patients the illness was protracted and the onset after pork rather ill-defined.

(b) Acute Surgical Pig-bel

The initial symptoms of abdominal pain and diarrhoea were not specific. Before a correct diagnosis of pig-bel could be made, the symptoms had persisted for three to five days. Most cases presented to hospital after such a delay and the differential diagnosis then became one of an "acute abdomen".

The onset was heralded by severe umbilical or left sided upper abdominal pain and this was a constant initial symptom in all types of the illness. The pain was cramp-like and extremely severe. Pain was usually preceded by anorexia and nausea. Vomiting followed
in 40 per cent of affected persons. The patient often indicated that he was struck down by the severity of onset of the disease. In a small group this pain was preceded by a prodromal period of a few days of mild and indefinite symptoms. It frequently radiated to the right of the umbilicus and upwards to the left costal margin. Some patients had difficulty in indicating the site of the pain. Occasionally it was that of a severe dyspepsia, described as a type of severe burning indigestion "hot long bel" in pidgin. A helpful feature of differentiating this onset from ordinary gastro-enteritis and dysentery was the constancy of the pain with intermittent periods of severe colic, rather than colic with intervening pain free periods. This pain continued for two or three days. Constant pain indicated strangulation. Rarely there was associated lower abdominal pain. In some instances the pain radiated to the back, shoulders and groin.

In 76.5 per cent of patients a brisk watery diarrhoea followed two to five hours after the onset of pain and lasted 24 hours. The motions became less frequent but bloodstained towards the end of this time. The blood was dark rather than bright, sometimes containing red blood despite the disease localisation to the oral portions of the intestine. Persons in this group then complained of constipation and the pain was less intense, but remained constant and localised to the right or left of the umbilicus, depending upon the locality of small intestine affected. When the upper jejunum
was involved pain was localised above and to the left of the
umbilicus. When the mid-jejunum and distal jejunum or ileum were
involved the pain centred upon the right para-umbilical area. Persons
in a second group (23.5 per cent) had no initial diarrhoea but
complained of diarrhoea on the fourth or fifth day after the
commencement of their abdominal pain. This was similar in intensity
and duration to those that suffered initial diarrhoea. The stools
in such cases were frequently "tarry" or greyish-green in colour,
frank melaena being not uncommon. Most children suffered an initial
spurious diarrhoea and adults made up the majority of this second
group. The first group, however, after two or three days'
constipation, once again developed diarrhoea which was blood-stained
and infrequent. By this time also the patient complained of
weakness, abdominal distension and vomiting.

Thus weakness, flatulence, abdominal distension and pain on
moving were late developments, and a second vomiting episode
indicated mechanical or paralytic ileus of serious consequences.
When this vomiting occurred dead or living Ascaris lumbricoides worms
came up in the vomitus in most instances, particularly in children of
the 6 to 10 age group. The symptoms were invariably attributed to
worms, particularly to round worms, by patient and parent alike and
more pork was occasionally given medicinally to a victim after the
commencement of symptoms. This was noted in 12 histories. In a
patient in New Guinea who had eaten a pork meal the diagnostic triad
of pig-bel was a refusal of food, insufferable abdominal pain, and bloody diarrhoea.

A history of persons eating the same pork meal was recorded on 42 occasions; 17 patients said that mild abdominal symptoms were experienced by others exposed to the same meal. However, the disease was firmly established on only three occasions in members of one family (Cases 89 and 90; 91, 92 and 93, and 132 and 134).

The physical findings depended on the time interval between the onset of symptoms and the presentation of the patient to hospital. No patients were seen within twenty-four hours of the onset of symptoms and most were examined between the third and seventh days. On the third day, the clinician was presented with an anxious patient, lying still in bed. In a third of the patients there was an appearance of "facies abdominalis". The skin was sometimes pale with irregularity spreading dark and light spotty areas (cutis marmorata). Loss of tissue turgor was apparent when the diarrhoea and vomiting had been severe. Delay in coming to hospital made the assessment of the pre-existing nutritional status difficult. However, in the severe case presenting to hospital early in the course of the illness, malnutrition was not a feature.

The nutritional state of patients with the disease was measured in general terms of appearance, skin fold thickness, muscle strength and tone, and skeletal development. These were scored as "good", "fair" and "poor", adequate provision being made for neglected
Table V: Analysis of 32 randomly selected blood pressure readings taken from acute surgical cases of pig-bell on admission

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>Operation</th>
<th>Died</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Hypotension* on admission</td>
<td>16</td>
<td>12</td>
<td>9</td>
</tr>
<tr>
<td>Course</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 days</td>
<td>5</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>5 days</td>
<td>11</td>
<td>9</td>
<td>8</td>
</tr>
<tr>
<td>B. Normotensive on admission</td>
<td>16</td>
<td>12</td>
<td>1</td>
</tr>
<tr>
<td>Course</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 days</td>
<td>13</td>
<td>9</td>
<td>1</td>
</tr>
<tr>
<td>5 days</td>
<td>3</td>
<td>3</td>
<td>0</td>
</tr>
</tbody>
</table>

*Less than 90 mm. of mercury systolic pressure.
complicated subjects with severe insaniion or dehydration. The results were as follows: good 56 (35.0 per cent), fair 61 (37.4 per cent), poor 34 (21.8 per cent), not recorded 9 (5.8 per cent). The height and weight curve of 55 subjects from 3 to 11 years of age were slightly lower than the standard established by Venkatachalam. This sample was however too small and scattered to be meaningful. In the acute disease, it was rare to find a marasmic child with pig-bel and kwashiorkor was not recorded in any case. Parotid gland enlargement, present in 70 per cent of the normal population (Venkatachalam, 1962) was frequently present. Jaundice was not recorded in any case. Conjunctival pallor was usual in patients seen after the fourth day of onset.

Early in the course of the illness the pulse was strong, regular and rapid. Tachycardia was a particularly constant feature the rate being of the order of 120 to 160 per minute. The temperature was rarely elevated on admission and seldom rose above 101°F. In 14 cases it was subnormal.

In the more advanced and neglected patient, profound shock was present. The pulse was rapid and feeble, and the blood pressure low. In a randomly selected series of 32 cases, hypotension was more commonly observed in patients presenting on the fifth or subsequent day (Table V). The majority of patients seen before the fifth day had a normal blood pressure and treatment then considerably improved their chances of recovery. A rapid fall in blood pressure
often heralded a melena. In such cases the prognosis was grave.

Early examination of the abdomen revealed slight distension and visible peristalsis. If ileus had developed, the distension masked the peristaltic movements. Distension was less apparent when only the upper jejunum was affected. There was usually hyperesthesia over thoracic segments T6 to T11 extending laterally to the nipple line. In cases with necrosis of the upper jejunum hyperesthesia was present over the left shoulder (L3 to 4).

Palpation of the abdomen early in the illness showed little resistance, although tenderness was usual in the left hypochondrium and hypogastrum. Signs changed over 2 to 3 days to a further resistance, and release tenderness. Commonly a sausage-shaped tender mass became palpable above and to the left of the umbilicus between the fourth and seventh day of onset. When lower jejunum or ileum was involved the mass was more diffuse and predominantly right-sided. The liver was often enlarged and tender in "toxic" cases, and occasionally the spleen was felt.

Auscultation at an early stage yielded excessive borborygmi indicating increased bowel activity. Ileus, mechanical or paralytic, developed in two to five days. The bowel sounds were then classically either of the "tinkling" metallic character or absent. Complete cessation of sound was inevitable if the ileus remained unrelieved.

The stools in the second diarrhoeal phase (first in some
instances) were thin, yellow-grey in colour and reacted strongly to
the benzidine reagent. A muddy deposit was sometimes present and
occasionally the faeces resembled the "pea soup" appearance of
typhoid fever. Mucus was not present. In many patients seen
after the fifth day the stools were "tarry" in character. In the
faeces mucosal shreds were present only in the more protracted
cases. Large mucosal casts however were not usual.

Frank haematemesis also occurred in two patients. The
gastric secretion was usually hypo-acid (pH 4.5 to 7.5), blood
stained, and foul smelling. Microscopic examination of Gram stain
smears showed a mixed bacterial flora. Gram positive rods
predominated the fields. Duodenal and jejunal smears yielded an
even higher concentration of Gram positive rods. Cellular exudate
was a constant finding on microscopy.

The nervous system only became involved in neglected advanced
instances, the patients being comatose when brought to hospital.
Opisthotonous was sometimes present. Air hunger and complete collapse
were present at this terminal stage. Nervous irritability occurred
in such patients, but otherwise the physician was confronted with a
quiet anxious patient with full mental faculties. These peculiar
mental changes: intellectual clarity, an appreciation of the gravity
of the circumstances and a pronounced terror of impending death,
were not unlike a person with wound gas gangrene.
Albuminuria was noted occasionally, being more frequent in the advanced states with associated oliguria and raised blood urea levels (Figure 7). Urobilinogen was similarly found in the urine of these patients. Transient glycosuria was present in five individuals with the disease.

A leucocytosis occurred on or about the fourth day and rose progressively without treatment. Polymorphs predominated in this increase and a relative eosinophilia was sometimes present. This may be a normal finding in populations with high helminthic infestation. The polymorphs inclined markedly to the left and showed toxic granulation. A total white cell count of over 20,000 was rare before the fifth day of illness. A secondary thrombocytopenia was sometimes noted. This apparently caused the bruising which occurred on handling the bowel and mesenterium at operation. Cutaneous purpura was not observed.

Haemoglobin estimations were generally low in cases presenting after five days. The haemoglobin value was sometimes high due to haemococoncentration when seen in early toxic or shocked states. The erythrocyte sedimentation rate rose marked as the illness progressed, figures to 80 to 100 mm. fall in the first hour (Wintrobe method) being frequently observed.

Malarial parasites were detected in the peripheral blood film in 8 out of 35 examinations: four were recognised as *Plasmodium falciparum*, three as *P. vivax* and one as *P. malariae*. 
Figure 7: Blood urea levels in 20 cases with severe disease compared with 12 controls and 20 with suspected mild disease. The dotted line represents the mean.
Blood urea examinations in seven persons ill for more than five
days were performed and these gave a mean blood urea of 96 mg. per
100 ml. and indicated a rising blood urea as the disease progressed.
This was probably due to tissue destruction combined with impairment
of renal function. In serum taken from 13 patients less than five
days after onset, the mean figure was 74 mg. per 100 ml. In the
mild disease and in normal controls this rise was not evident (Figure
7). Oliguria and amuria, present in ten of the acute cases,
indicated acute renal failure.

Loss of fluid in vomiting and later outflowing of fluid into
distended bowel resulted in a fall in blood chloride, alkalosis and
dehydration. However, the cold clammy “doughy” skin without much
loss of tissue turgor was rarely evident. Where diarrhoea had
continued for three or more days hypokalaemia and acidosis became
clinically evident and probably enhanced the development of paralytic
ileus.

Examination of faeces for parasites was done using the brine
flotation method, and where apparently heavy infestations were present
a quantitative estimate was obtained using the Gordon and Whitlock
(1939) technique.

Ascaris lumbricoides eggs were detected in 87 per cent of cases
degree
with all types of pig-bel, and hookworm eggs in 62.5 per cent of
cases. In 21.5 per cent, egg output was over 2,000 per gram of
faeces for Ascaris lumbricoides.
(e) **Acute Toxic Disease**

Early circulatory collapse is the predominate clinical picture in this group. Otherwise, the features are similar to other acute cases in severe abdominal cramp, diarrhoea and invariably vomiting. In two cases (Nos. 79 and 99) melena and haematemesis occurred. Pallor, air hunger, restlessness, cold clammy skin with thready pulse all add up to a fulminating toxic collapse and death took place within three days. The symptoms and signs therefore, resembled those of other states of acute poisoning.

In summing up the clinical features of the acute disease, one was impressed by the quiet, anxious male patient with "facies abdominalis", a rapid pulse rate, normal temperature and blood pressure and tender upper abdomen. A constant and reliable sign was the palpation of a firm, tender, elongated jejunal mass in the upper abdomen to the left of the umbilicus. In those cases in which perforation had not occurred marked peristaltic movements preceded signs of mechanical or paralytic ileus. The general toxic state and intestinal obstruction following a bloody diarrhoeal episode after prior pork consumption, led to a diagnosis of acute surgical enteritis necroticans.

As the disease progressed, circulatory collapse and toxemia advanced, and if untreated death occurred between the fifth and fourteenth days. In such cases the average length of illness
before death took place was six to seven days. Operation was performed most frequently on the fifth day once the therapeutic schedules become more standardized by experience.

To illustrate the clinical course of the acute forms of enteritis necroticans, the following representative case histories are presented:

Case 3: A native boy of Chimbu, aged 12 years, was admitted to the Kumasi hospital on April 5, 1961 after eating pork which had been cooked two days previously. The patient suffered abdominal pain, nausea and vomiting. No fever or headache was recorded. The symptoms continued for two days and the faeces became black on the third day. Constipation, abdominal distension and pain were complained of on the fifth day. On the tenth day signs of peritonitis developed, and the boy was transferred to the hospital at Goreka. At Goreka, examination showed a well-nourished male child with a rapid pulse rate, a temperature of 99°F, a blood pressure of 85/60 mm. of mercury and a distended, tender abdomen with generalized guarding. Bowel sounds were absent. An enema was given and a semi-fluid malaena stool was returned. The white cells numbered 17,200 per cubic millimetre; polymorphs predominated. An X-Ray examination of the abdomen showed fluid levels in the small bowel and gas under the diaphragm. After the patient had received a blood transfusion, laparotomy was performed. This revealed generalized peritonitis due to a perforation in the jejunum, 5 feet of which contained gangrenous
patches. Resection of the affected portion of bowel was performed, together with a side-to-side anastomosis. Both before and after operation, penicillin and streptomycin were administered, and neomycin and pthalysulphathiazole were given post-operatively by the intragastric route. The patient was discharged from hospital on the twentieth post-operative day, having made a full recovery. The pathologist (Professor E. ten Seldam) made the following report:

"Microscopic examination of a portion of the bowel showed the whole wall to be densely infiltrated with polymorphs with fibrin on the serosal surface. The mucous membranes and submucosa were gangrenous, necrotic and densely infiltrated with polymorph and pus formation. The wall was infiltrated with blood and in the submucosa many of the blood vessels revealed fresh thrombosis."

Follow-up examination of this patient three years later revealed some degree of absorptive dysfunction.

Case 5: A native male of Kwongi Village gave a four days' history of abdominal pain following participation in a pig feast. He complained of violent abdominal cramps with a burning pain between these episodes a few hours after a large pork meal. Then followed diarrhoea, nausea and vomiting about 10 hours after he had eaten the pork. He complained of no headache or fever. No blood
or mucus was seen in the faeces until the second day; the stools were then of a greyish, fluid character. The pain became constant and was more localised to the epigastrium. Abdominal distension and constipation occurred on the fourth day. Treatment at an aid post (staffed by a native medical orderly) was given on the third day, when the patient received sulphaguanidine and a bismuth sedative mixture. On admission of the patient to hospital on May 13, 1961 physical examination showed that he had tachycardia, a blood pressure of 100/60 mm. of mercury, a temperature of 97.8°F, a dry, furred tongue and slightly sunken eyes. The abdomen was distended, and generalised tenderness was present, especially in the epigastric and left umbilical regions. There was only slight rigidity and rebound tenderness over a discernible distended loop of bowel to the left of the umbilicus. Bowel sounds were absent. An enema was given and gas was returned, together with watery, greyish fluid. Microscopically this fluid contained an abundance of both Gram-positive and Gram-negative bacilli and the ova of Ascaris lumbricoides. The white cell count was 19,200 per cubic millimetre, with a "shift to the left". The haemoglobin value was 14.4 grammes per 100 ml. An X-Ray film of the abdomen showed distended loops of small bowel with the typical fluid levels "stepping" along the mesenteric axis. Laparotomy was performed on the day of the patient's admission to hospital, after he had been treated with gastric suction and an intravenous infusion of glucose and salines. Two-thirds of the small
bowel, beginning at a point 2 feet from the duodeno-jejunal angle, 
were affected by multiple transverse gangrenous areas, with several 
small perforations covered by some fibrinous exudate on the serosal 
surface. The mesenteric glands were swollen and the mesentery was 
ocedematous. The lesions decreased in severity towards the distal 
end of the small bowel. Resection of about 8 feet (200 to 250 cm.) 
of jejunum and ileum were undertaken and a side-to-side anastomosis 
was performed. Most of the gangrenous part of the bowel was 
removed, but several small "early-stage" skip lesions were left at 
the distal end, in the hope that further conservative measures would 
suffice for them. This procedure was forced on me by the already 
too extensive resection. Neomycin and phthalysulphathiazole were 
given through a gastric tube, and penicillin and streptomycin were 
systemically. The patient made an initial recovery until the fifth 
post-operative day, when a further attack of abdominal colic was 
followed by a brisk diarrhoea. His condition further deteriorated, 
and generalised abdominal tenderness, with distension and cessation 
of bowel movements, indicated the presence of peritonitis. On May 
24, 1961, the abdomen was re-opened and a further extension of the 
bowel necrosis was discovered, extending beyond and involving the 
region of the anastomosis. Several more small perforations were 
present in the distended part of the bowel, and collections of pus 
with early abscess formation surrounded the affected areas. The 
patient died on the following day and permission for an autopsy was
refused. Histopathological examination of a segment of the affected bowel "showed complete necrosis which was packed with red cells in the early stages of autolysis and degeneration. There was a peripheral inflammatory reaction, polymorphs predominating".

Case 15: A native female, aged eight years, of Asaro was admitted to hospital on July 24, 1961 for observation after food poisoning. The agent again was stale pork cooked three days prior to its ingestion. Eight hours after her meal of pork, the child had acute abdominal colic with some vomiting. Mild diarrhoea occurred, which lasted two days and the pain became constant with occasional severe exacerbations. On admission to hospital on the third day of her illness she lay quietly, with an occasional bout of colic. Her weight was 21.3 kg., her height 127 cm. and her nutritional state was assessed as only fair. The tongue was dry and dirty, the pulse rate was 100 per minute and the blood pressure was 105/70 mm. of mercury. The abdomen was slightly distended, bowel sounds were "tinkling" in character and some upper abdominal tenderness could be detected. An enema was given and a semi-fluid, greyish stool was returned, together with flatus. Twenty-four hours later the absence of pain, increasing distension and the cessation of bowel sounds indicated that ileus had developed. The white cell count was 12,000 per cubic millimetre. The stomach was aspirated and copious amounts of dark green bile containing digested blood were returned. Laparotomy
was performed after resuscitation of the patient, which was delayed for some 24 hours after admission. Gross distension of the small bowel was present at operation with the familiar haemorrhagic, circular, gangrenous patches extending from the upper part of the jejunum to the middle part (Figure 8). Small areas of early necrosis with echymosis, oedema and distension extended throughout the ileum. An almost total jejunectomy had to be performed but after the resection, cardiac arrest occurred and in spite of cardiac massage death resulted. Antibiotic treatment was the same as in previous cases. Polyvalent gas gangrene antitoxin was also given before operation. *Clostridium perfringens* type C was isolated in pure culture from the intestinal contents, and serum agglutination tests for salmonella serotypes gave negative results. Serum was not examined for beta antitoxin.

**Case 16:** A native male, aged 12 years, of Lunepi Village, Asaro, was admitted to hospital on September 7, 1961 with a few days' history of abdominal pain and vomiting, the symptoms having commenced twelve hours after he had eaten stale re-heated pork. His stools were bloodstained on the third day. He had severe upper abdominal pain, which was confined to the umbilical region. Examination showed an anxious child with a rapid, thready pulse. The extremities were cold, the temperature was 97.5°F, the blood pressure was 95/60 mm. of mercury and the patient was dehydrated. Abdominal distension
Figure 6: Upper jejunal loops at laparotomy in a female aged 8 years (Case 15), late on the fourth day of her illness. Extending from one gangrenous proximal loop, the lesions were more patchily distributed in the distal jejunum.
was generalized, and marked tenderness was present to the right of
the umbilicus, where an ill-defined, "doughy" mass could be palpated.
Guarding and rigidity were minimal and no bowel sounds could be
heard. Rectal examination yielded a few flecks of black faeces.
The white cells numbered 18,100 per cubic millimetre and the haemo-
globin value was 15.4 grammes per 100 ml. The blood urea level was
46 mg. per 100 ml. and the erythrocyte sedimentation rate was 42 mm.
in one hour. After resuscitation laparotomy was performed and this
showed a region of necrotizing jejunitis, commencing six inches from
the duodeno-jejunal angle and extending distally for six feet.
Several loops of distended bowel were matted together on the right
side, and in one patch perforation was imminent. "Skip" lesions
were present in the distal jejunum (Figure 9). The mesentery showed
the usual oedema and swollen glands containing sub-capsular gas
bubbles. Gas bubbles were also present along the mesenteric
attachment (Figure 10). Resection of the more grossly affected
segment of bowel (100 to 150 cm.) and side-to-side anastomosis was
performed. A good post-operative recovery was made, and the patient
was discharged from hospital on the fifteenth post-operative day.
Penicillin, streptomycin, neomycin and phthalysulphathiazole were
all administered, as in the previous cases, and polyvalent gas
gangrene antitoxin (3000 international units of Cl. welchii
antitoxin, 1500 international units of Cl. oedematis antitoxin)
Figure 2: Portion of resected jejunum from a boy aged 13 years on the fourth day of illness (Case 16). Rhip lesions are apparent on the serosal surface. A proximal segment, not shown, had the appearance of the bowel shown in Figure 6.
Figure 10: Erythematous bullae along the mesenteric attachment of the jejunum at operation in Case 16. *C.l. perfringens* type C was recovered from the contents of the resected jejunum.
were given before operation. **Clostridium perfringens** type C was isolated from the contents of the resected bowel. There was also a rise in **Clostridium perfringens** beta antitoxin in paired sera taken at a two week interval.

This patient was well and normal when subsequently seen two and a half years later, and showed a substantial gain in weight.

**Case 46:** A male whose estimated age was 30 years was transferred from Novi Anglican hospital following a short history of abdominal pain and vomiting six hours after eating four-day old pork. Tinned fish and a spinach-like vegetable were eaten with the same meal, shared by his family except for the pork. On the second day of his illness the abdominal pain became constant, "coffee grounds" material was in his vomitus, and the stools became a murky grey-brown colour. He had a temperature of 101°F, "facies abdominalis", peripheral pallor and cold, clammy skin, the pulse was thready (rate 140 per minute), blood pressure 70/45 mm. of mercury and there were generalised guarding and tenderness maximal in the left upper quadrant of a distended tympanitic abdomen. The patient failed to pass urine. A diagnosis of "acute toxic enteritis necroticans" was made; gastric suction and resuscitation with intravenous blood followed by isotonic dextrose (4.5%) and saline (0.18%)
commenced. At laparotomy on November 1, 1962 the small bowel from the fourth part of the duodenum to the mid-ileum was involved in a process with the appearance of segmental ecchymotic purplish areas consistent with patchy necrotizing enteritis. Subserosal gas bubbles were seen along the mesenteric attachment and the mesenteric glands were filled with small gaseous spaces. A heroic resection of over 300 cm. of small intestine with the usual antibiotic cover brought temporary relief, but the patient became uremic and died on the fifth post-operative day from an acute nephro-toxic tubular necrosis. His blood urea rose to 250 mg. and culture of bowel content and blood yielded Cl. welchii, which was not forwarded for typing. A pathology section (H5085, Dr. R.A. Cooke, Port Moresby) showed that the whole circumference of bowel wall contained only two islands of normal mucosa. Mucosal layers were the only portion of intact bowel wall. There was cellular infiltration with few polymorphs and numerous eosinophils and plasma cells. The subserosal layer was heavily infiltrated with polymorphs and the innermost lining of intestine consisted of necrotic, hemorrhagic material through which scattered clumps of Gram-positive bacteria could be seen.

A graphic record of the clinical course of three further patients with pig-bel, two acute surgical (Nos. 4 and 89, Figures 11 and 12) and the other acute toxic (No. 79, Figure 13) forms of the disease are drawn in Figures 11, 12 and 13. The former two patients illustrate the value of early bowel decompression and
resuscitation, which were delayed in the first instance. A relapse of the second case (No. 89) occurred on the 12th day and this was again managed with some success conservatively. A barium meal performed two months after the initial illness revealed some mucosal abnormalities with two narrowed areas in the mid-jejunum. Resection will quite likely be necessary at a later date. The periodic course of the illness, as illustrated by Case 89, was the exception rather than the rule in pig-bal, but indicates that reinfection may occur. A rise and fall in Cl. perfringens beta antitoxin confirmed the diagnosis in this case.

(a) Sub-acute Surgical Pig-bal

This type represented either a progression of a mild initial infection or late complications of healing of cured acute cases. Of the 40 cases reviewed in this group, 19 initially had only mild symptoms of abdominal pain or epigastric discomfort. Subsequently diarrhoea, abdominal colic and constipation developed over periods from two weeks to two months. The other 21 cases had a more acute onset, the clinical picture being similar to acute surgical forms at laparotomy or autopsy. The differing features are summarized thus:

1. There was a more prolonged incubation period.
2. Initially abdominal pain was less severe but it continued intermittently in gradually worsening spasms.
3. Initial vomiting was rare, but occurred in over half the
**Figure 11**: The clinical features of one of the earlier cases of pig-bell in which operation was delayed due to the uncertainty of diagnosis.
**EXAMPLE OF ACUTE SURGICAL DISEASE - CONSERVATIVE MANAGEMENT**

*CONSERVATIVE TREATMENT CONTINUED WITH FULL RECOVERY.*

**Figure 12:** The clinical course in a young boy with severe pig-bel. Effective early treatment obviated the need for operation although a relapse occurred on the tenth day of his illness.
M.B. F/22 Case No. 79  
A&D2700  
Admitted Kundiawa  
14/12/63

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**CLINICAL COURSE**

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<tr>
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<tr>
<td>Vomiting</td>
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**Blood**

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**Blood**

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<td>Not examined</td>
<td>110</td>
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**Treatment**

- IV fluids/blood
- Penicillin
- Chloromycetin
- IV Chloroquine

---

**Figure 13:** A short fulminant illness in a young woman aged 22 years.
cases as complications developed. Diarrhoea was a feature between periods of constipation.

4. Severe gastro-intestinal haemorrhage was more commonly met with in this group.

5. The continual refusal or fear of food resulted in weight loss and a gradual development of the "facies abdominalis".

6. Slow or rapid development of an acute malabsorption state: this was characterised by weight loss, iron deficiency, anaemia, and steatorrhoea with or without upper abdominal colic.

7. The isolation of Cl. perfringens type C from resected bowel was less likely when these complications had developed.

8. A rising beta antitoxin titre was more pronounced and significant in five such cases of pig-bel investigated.

The following case histories demonstrate the various complications that occurred.

Case 17: A native female, aged 40 years, came from Kama village, Goroka. Thirty-six hours after eating putrefied pork fat, she developed upper abdominal colic and mild vomiting. Abdominal distension and pain became continuous, with absolute constipation. Examination revealed a distended, tympanitic abdomen with localised tenderness to the right of the umbilicus. Bowel sounds were
present, and the general condition of the patient was satisfactory. Conservative treatment with phthalylsulphathiazole, given orally, and intramuscular injections of penicillin, streptomycin and gas gangrene antitoxin, was given. Bowel decompression was achieved by means of continuous gastric suction and an enema. The patient's condition settled down, but 20 days after her admission to hospital she developed symptoms and signs of sub-acute small bowel obstruction. A barium meal X-Ray examination confirmed the clinical diagnosis. Laparotomy revealed a stricture of the upper part of the ileum. A segment, two feet in length, of the lower part of the jejunum and the upper part of the ileum, was resected. The whole mucosa had been replaced by fibrous scar tissue and the intestinal wall was thickened and rigid in part of the resected segment. Perforation appeared to have been imminent in one area. The patient's post-operative course was uneventful. She was discharged from hospital after two weeks, and was well and gaining weight when examined two months later.

Case 20: This patient was a girl aged 7 years from a village near Gumine in the Eastern Highlands. She was referred from Kundisawa on November 12, 1961 with a history of severe upper abdominal pain for three weeks. Her illness commenced twenty-four hours following consumption of stale pork and was accompanied by vomiting and a melaena diarrhoea. The diagnosis on referral was peritonitis
and acute small bowel obstruction. Examination revealed a wasted anaemic girl with "facies abdominalis" weighing 24.6 kg. with distended abdomen and rebound tenderness to the right of the umbilicus. Bowel sounds were "tinkling" in character. On rectal examination a tender resistance was felt on the right side. Her haemoglobin was 9.6 grammes, white cell count 15,200, polymorphs increased, blood sedimentation rate 85 mm. and 120 mm. in the first and second hours respectively, blood urea 64 mg. per cent and albuminuria was present. Fecal culture for anaerobes yielded Cl. perfringens (untyped) and paired sera taken on admission after a two week illness and again two weeks later both yielded levels of greater than 70 units of the beta antitoxin of Cl. perfringens.

Treatment was along the lines of decompression, resuscitation, blood transfusion, sulphathalidine orally and chloromycetin succinate systemically. The patient made good progress on conservative treatment but absconded from hospital on the 42nd day. Barium X-Ray studies were not carried out. Subsequent follow-up indicated that the child died in her village some six months later from a wasting illness with symptoms of sub-acute intestinal obstruction.

Case 35: A boy aged 12 from Kangamori was admitted in January 1962 with recurrent bouts of fever, upper abdominal pain, sudden weight loss and constipation over a period of three weeks. He had eaten pig two days before the onset of pain and had one vomiting
episode. On admission he had a tachycardia, slightly distended abdomen with loud borborygmi. His fever was high (103°F) and "saddle-back" in type. Pain developed in the left hip on the third day and a diagnosis of typhoid fever was considered. Despite antibiotics and intravenous fluids, the fever continued and penicillin and streptomycin therapy was changed to erythromycin. The haemoglobin fell to 8.6 grammes and white cell count to 6,400 on the 10th day of admission. Fecal and blood cultures were negative for Salmonella typhi and Salmonella paratyphi, and serum agglutination tests were similarly negative. Anaerobic culture was not carried out. On the 14th day, following episodes of intermittent colic and post-prandial distension, the patient suffered a severe attack of abdominal pain and developed signs of peritonitis. Laparotomy was performed in Loe and multiple adhesions with one fresh ileal perforation were found. Between 100 and 150 cm. of affected ileum was resected. A stormy convalescence ensued and further indications of sub-acute small bowel obstruction by adhesive band strangulation necessitated another laparotomy one month later. The patient was discharged on April 30 1962 and subsequent follow-up a year later showed some residual evidence of non-sclerosing ileitis on X-Ray, although the patient was otherwise normal. On August 9 1964 a further examination indicated steatorrhea, hypochromic normocytic anaemia and a weight gain of only 0.5 kg. over the year.
Case 62: A girl from Laiagam in the Western Highlands whose estimated age was seven years, had eaten re-heated pork (bowl and mid-quarter) cooked four hours previously. She suffered severe upper abdominal cramps which continued for three days, but no diarrhoea or vomiting. No other members of the family were affected. She was treated at an aid post for the next two days with some relief. Intermittent diarrhoea and constipation with upper abdominal discomfort and pains, accompanied by anorexia and weight loss continued, and minimal treatment was given at the Laiagam hospital for a period of ten days. The abdominal colic became more severe and sub-acute bowel obstruction was suspected and the patient transferred to Wabag. Examination there revealed a wasted anaemic child, oedema of ankles and lumbar region, puffy but indrawn face and severe cachexia (weight 19 kg.). There were visible peristaltic small bowel movements of distended upper small intestine and excess borborygms. Tenderness was noted round the umbilicus and hyper-aesthesia present both sides over T6 to T11 distribution. The tip of the spleen was felt and the liver edge was palpable. A few basal lung crepitations were noted at the right base. Her temperature was 99°F, pulse rate 100 and blood pressure 95/60. Stools were frequent, bulky and offensive and yielded *Clostridium perfringens* type C on culture, being negative for other enteric pathogens. This organism was also cultured from her jejunal aspirant. Heavy hookworm and Ascaris infestation were present. The anaemia was hypochromic and normocytic.
(haemoglobin 10.5 grammes). The white cell count was 8,300; polymorphs 82%, lymphocytes 10%, eosinophils 6% and monocytes 2%. Bone marrow was highly cellular with a myeloid/erythroid ratio of 3:1. The erythroid series showed normoblastic development. Erythrocyte sedimentation rate was 46 mm. in the first hour and 66 mm. in the second. Widal and Weil Felix tests showed slight agglutination with protein OXX at a titre of 1 in 30. All other titres were less than 1 in 30. **Cl. perfringens** type C beta antitoxin antibody estimation was greater than 20 units per millilitre.

X-Ray of the intestine (taken two weeks after therapy commenced) showed dilated jejunal loops with no visible peristalsis, the "moulage sign" due to loss of mucosal folds with segmentation and clumping of barium and delay in transit through the small bowel. Wast fibres were in abundance in the stools which were bulky and offensive. Thus signs of intestinal dysfunction were evident on admission and this persisted with severe liniteric vomiting and distension. A diagnosis of a protracted form of necrotising enteritis was established with sprue-like manifestations.

A second faecal culture three months after admission was negative for **Cl. perfringens** type C, and her antibody level for beta welchii toxin had fallen to normal (less than 1/2 unit/ml.) at this examination. Her haemoglobin rose to 14.2 grammes and a weight gain of 7.3 kg. was achieved.

A similar patient with severe wasting (Case 49) was admitted
from the same village three months earlier, this time with an abdominal mass palpable to the right of the umbilicus. Three feet of lower jejunum were resected and the patient made a good recovery. Beta antitoxin level in the convalescent period was 1 to 2 units/ml. although *Clostridium welchii* type A and no type C was recovered from the faeces.

Figure 14 shows graphically the course of a further protracted case (No. 96) where surgical treatment was delayed for five weeks. At operation an entero-enteric fistula was discovered between two jejunal loops (Figure 15). The resected specimen is shown in Figure 16.

(e) **Mild or Suspected Pig-bell**

Mild infections occurred as was evident by the detection of beta antitoxin *Clostridium perfringens* type C two weeks after the onset of symptoms. This was present in 27 out of 34 examinations. Recovery of the organism from faeces and jejunal aspirate was, however, less successful from patients in this group.

The symptoms initially simulated those of the more acute cases although vomiting was infrequent. Left sided upper abdominal pain was the main presenting symptom which continued for five or more days. Nausea and anorexia were constant accompaniments. The abdominal pain was associated with a hyperaesthetic zone from T8 to T12 on the left side, but rarely was there any reflex rigidity or
**Figure 14:** A protracted form of pig-bell in a man aged about 27. At operation on the 35th day of illness, an enterocutaneous fistula was visualized. The patient died post-operatively. *Clostridium perfringens* type C and *Sh. flexneri* type 2 were isolated from faeces collected on admission from this patient.
Figure 15: Adherent upper jejunai loops at operation with a demonstrable entero-enteric fistula (Case 36), male aged 27 years.
Figure 16: Scarred adherent U-loop of upper jejunum with an elongated chronically ulcerated area. There is some mucosal regeneration (Case 96, male aged 27 years). Jejunectomy specimen fixed in 10% formal saline.
appreciable distension. Constant pain was again experienced by the patient between the bouts of colic. Diarrhoea with blood usually followed but this commenced later in the illness than in the severe forms. Constipation for two or more days usually preceded this diarrhoea.

Visible peristalsis of thickened small bowel loops was a constant though unreliable sign. Children with heavy helminthic infestation often displayed this sign together with a "doughy" feel on abdominal compression. A rapid but full pulse and dusky appearance indicated some degree of toxæmia.

Spontaneous recovery was usual, although in one-quarter of the patients in this group the main complaint was a persistent upper abdominal pain more than a week after their gastro-enteric episode. This was considered to be an important diagnostic criterion in the mild form of the disease. It was distinguished clinically from other forms of gastro-enteritis where continuous upper abdominal pain was not a feature, and from other forms of food poisoning where prolongation of the pain for longer than two days is unusual. The stool also was not dysenteric in appearance, although melaæma sometimes occurred.

Of twenty-two mild cases studied from the Asaro outbreaks in 1961, serum from nineteen cases was examined for beta antitoxin of which thirteen had significant detectable antibody levels, eight having levels of 5 or more units per ml. Of 17 cases studied in
the Chimbu in 1964 similar antibody levels were found in 14. In addition, six cases underwent per-oral biopsy soon after admission. These confirmed a mild form of necrotizing enteritis involving mucosa only. Barium meal examination showed loss of mucosal folds, clumping of barium and a fair degree of intestinal atony and spasm in serial pictures taken where a screen was not available. Faecal fat estimations two to six weeks after admission on 15 of these subjects had a range of 25 to 80 per cent of dried faeces indicating varying degrees of steatorrhoea. From these investigations it was deduced that a small percentage of patients (between 2 and 5 per cent) may enter a malabsorptive state resulting from a chronic necrotizing enteritis. However, it was difficult to prove that this was not due to a heavy helminthic infestation.

The diagnostic criteria indicating a mild infection may be summarised as follows:

1. Continuing upper abdominal pain following a gastro-enteric episode consequent to pork consumption.
2. Detectable antibody levels (≥ ½) to the beta toxin of Cl. perfringens one week after onset.
3. Isolation of Cl. perfringens from the faeces.
5. Steatorrhoea.
The following case histories illustrate this mild type of the disease.

**Case 175:** A male, estimated age of 7 years, was admitted with a three-day history of severe upper abdominal pain with two episodes of initial vomiting and constipation twenty hours after eating three day old pork. Two other members of the family also complained of pain following the same pork meal. On examination there was some rebound tenderness round the umbilicus and a tachycardia, but otherwise the patient was well although slightly anaemic (haemoglobin 11.4 grammes). Fecal culture yielded *Clostridium perfringens* type C and serum showed a beta antitoxin titre of 20 to 30 units ten days after the initial symptoms. *Salmonella* isolation and agglutination tests were negative. Recovery was complete with sulphathalidine, chloromycetin and copious oral fluids. Sera or faeces were not collected from contacts of this case.

**Case 91:** A woman aged about 22 years from Goromango was admitted in May 1964 following consumption of three day old pork killed in the Upper Chinsu pig-kill six days earlier. She complained of nausea, anorexia and a severe epigastric colic 12 hours later. She was constipated for two days. Diarrhoea occurred two days later, altered blood being passed on the fourth stool. The pain continued and the patient presented to hospital seven days later with constipation and continuous upper abdominal
Table VI: Isolation of Cl. perfringens in the bacteriological investigations

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<th>Type C</th>
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<tr>
<td>Follow-up (faecal smears)</td>
<td>17</td>
<td>10</td>
<td>7</td>
<td>6</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Controls</td>
<td>4</td>
<td>1</td>
<td>3</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Bowel resections from other causes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal population</td>
<td>468</td>
<td>351</td>
<td>117</td>
<td></td>
<td>0</td>
<td>*</td>
</tr>
</tbody>
</table>

*Preliminary communication (Walker, 1965) exact sample taken from 114 isolations unknown. It is known that no type C isolations were obtained from those sampled.
pain. She had the "facies abdominalis", furred dry tongue with
typical foetor and a slight degree of dehydration. The patient was
well nourished (height 151 cm. weight 41.2 kg.), had a haemoglobin of
10.2 grammes, a white cell count of 18,000 and heavy ova concentration
of _A. lumbricoides_ and hookworm in a faecal specimen obtained on
enema. Small bowel movements were visible. She was tender to the
right of the umbilicus and bowel sounds were excessive. Faecal
culture yielded _Cl. perfringens_ type C and no other pathogens, and
fourteen days after her initial symptoms her serum contained between
45 and 70 units of welchii beta antitoxin. Decompression was
achieved by gastric suction and intravenous fluids restored fluid and
electrolyte balance. Sulphathalidine and chloromyostin were given
and recovery achieved without operation. The patient was discharged
after a stay in hospital of ten days. _Cl. perfringens_ type C was
isolated from two relatives of this woman who both had the clinical
features of a mild pig-bell syndrome.

4. **Special Investigations**

(a) **Bacteriological Examinations**

Table VI shows the results of bacteriological examinations of
resected bowel and faeces from persons suspected of having pig-bell.
Thirty-eight specimens were examined for the presence of aerobic and
anaerobic pathogens.
Table VII: Biochemical reactions of the histotoxic clostridia

(after McLennan, 1963)

<table>
<thead>
<tr>
<th>Clostridium Species</th>
<th>Sugar Fermentation</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th>Litmus Milk</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Glucose</td>
<td>Lactose</td>
<td>Sucrose</td>
<td>Salicin</td>
<td>Gelatin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cl. perfringens</td>
<td>*AG</td>
<td>AG</td>
<td>AG</td>
<td>AG</td>
<td>L</td>
<td>Stormy clot +++</td>
<td></td>
</tr>
<tr>
<td>Cl. novyi</td>
<td>AG</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>L</td>
<td>No change</td>
<td></td>
</tr>
<tr>
<td>Cl. septicum</td>
<td>AG</td>
<td>AG</td>
<td>-</td>
<td>AG</td>
<td>L</td>
<td>Stormy clot + or +</td>
<td></td>
</tr>
<tr>
<td>Cl. bifermentans</td>
<td>AG</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>L</td>
<td>Digested</td>
<td></td>
</tr>
<tr>
<td>Cl. histolyticum</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>L</td>
<td>Digested</td>
<td></td>
</tr>
<tr>
<td>Cl. fallax</td>
<td>AG</td>
<td>-</td>
<td>AG</td>
<td>AG</td>
<td>No change</td>
<td>No change</td>
<td></td>
</tr>
</tbody>
</table>

*AG = acid and gas production.
Table VIII: Cited from Egerton and Walker (1964)

Typing of β-producing strains of Cl. perfringens isolated during outbreaks of necrotic enteritis of man in Papua-New Guinea

<table>
<thead>
<tr>
<th>Strains</th>
<th>Patient and age</th>
<th>Origin</th>
<th>α</th>
<th>θ</th>
<th>δ</th>
<th>β</th>
<th>ε</th>
<th>ι</th>
<th>κ</th>
<th>λ</th>
<th>μ</th>
<th>H.R.†</th>
</tr>
</thead>
<tbody>
<tr>
<td>F 2113</td>
<td>F 47</td>
<td>Resected jejunum</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>F 4853</td>
<td>M 8</td>
<td>Resected ileum</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>F 5701</td>
<td>M 42</td>
<td>Resected ileum</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>F 5702</td>
<td>F 8</td>
<td>Resected jejunum</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>263</td>
<td>M 22</td>
<td>Resected jejunum</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>331</td>
<td>M 4</td>
<td>Resected ileum</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>924</td>
<td>F 7</td>
<td>Duodenal aspirate</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>926</td>
<td>F 32</td>
<td>Faeces</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>934 A</td>
<td>M 7</td>
<td>Bowel contents</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>934 B</td>
<td>M 7</td>
<td>Jejunum</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>986</td>
<td>F 7</td>
<td>Jejunum</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
</tbody>
</table>

* tr = Trace  † H.R. = Heat resistance
The morphological, cultural and biochemical characters of organisms corresponded closely with those described for *C. perfringens* in standard tests. Colonies from 24-hour blood agar plates cultured anaerobically contained plump Gram-positive rods similar to those predominant in Gram stain smears of duodenal and jejunal contents from cases of pig-bel. Spores were not apparent and the organisms were not motile. These colonies showed a distinct double zone of haemolysis - an inner clear zone and an outer incomplete zone. Their size was 2 to 4 cm. in diameter, they were slightly convex and had a crenated edge.

Biochemically there was gas and digestion in Robertson's cooked meat medium, gelatin was liquified in 48 hours and a stormy clot formed in litmus milk giving an acid reaction. Fermentation of glucose, maltose, sucrose and lactose produced both acid and gas but fermentation of salicin was invariable. These biochemical reactions distinguished this organism from other histotoxic clostridial organisms (Table VII).

*C. perfringens* was isolated from the lumen and necrotic wall of 36 bowel specimens. Seventeen type C strains and nine type A strains were subsequently identified from the isolates. Ten were not submitted for complete identification. The toxicological analysis of 11 beta-producing strains are shown in Table VIII.

From a group of 107 in which no surgical intervention was undertaken and which included contacts of known cases, *C. perfringens*
strains were isolated from 54 faecal specimens (Table VI). Only
nine of these proved to be type C isolates with the same toxin
production as the strains in Table VIII. *Shigella flexneri* type 2
was isolated from three persons in this group, once in association
with *Clostridium perfringens* type C and twice was the sole demonstrable
pathogen. This organism was recovered from one case in follow-up
examination. Blood cultures were not taken. Pathogenic *Escherichia
coli* or viral causes were also not investigated.

(b) Animal Pathogenicity Tests in Guinea Pigs

Death occurred in two guinea pigs inoculated intramuscularly 24
to 48 hours after injection of a 24-hour cooked meat broth culture of
*Clostridium perfringens* type C. One control animal injected with a saline
placebo survived. At the site of inoculation a haemorrhagic
gelatinous oedema was present and gaseous distension of the sub-
cutaneous tissues had taken place. Muscles surrounding these lesions
were necrosed and a smear revealed Gram-positive encapsulated
organisms.

The same strains in broth culture were introduced into the
normal pellet ration of two more guinea pigs. Both these animals
and two other controls on normal feed remained unaffected after
their respective meals.

Two animals were given intra-duodenal suspensions of a 0.1 ml.
broth culture through a 20 gauge needle and a further strain in
another animal was injected into the peritoneal cavity. The first
Table IX: Randomly selected persons with pig-bal in whom paired serum sampling was undertaken for beta antitoxin

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex</th>
<th>Age</th>
<th>Week of illness</th>
<th>Beta antitoxin (units/ml.)</th>
<th>Isolation</th>
<th>Cl. perfringens</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>16</td>
<td>M</td>
<td>12</td>
<td>1</td>
<td>&lt; 1/2</td>
<td>Yes</td>
<td>Type C</td>
<td>Resection performed on 4th day. Full recovery.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3</td>
<td>1/2 - 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>F</td>
<td>14+</td>
<td>2</td>
<td>1/2 - 1</td>
<td>Yes</td>
<td>Type A</td>
<td>Conservative management initially. Ileal stenosis 6 weeks later.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3</td>
<td>1/2 - 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>M</td>
<td>5</td>
<td>1</td>
<td>1/2 - 1</td>
<td>Not examined</td>
<td></td>
<td>Conservative management. Malabsorption 3 years later.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2</td>
<td>10 - 15</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>M</td>
<td>37</td>
<td>1</td>
<td>1/2 - 1</td>
<td>Negative</td>
<td></td>
<td>Conservative management. Full recovery.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2</td>
<td>2 - 5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>F</td>
<td>6</td>
<td>2</td>
<td>&gt; 70</td>
<td>Not examined</td>
<td></td>
<td>First serum taken at 2 weeks. Conservative management. Died 6 months later from intestinal obstruction.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3</td>
<td>&gt; 70</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>M</td>
<td>7</td>
<td>1</td>
<td>1/2 - 1</td>
<td>Yes</td>
<td>Not identified</td>
<td>Resection on 4th day. Complete recovery.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2</td>
<td>2 - 5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>62</td>
<td>F</td>
<td>8</td>
<td>2</td>
<td>1 - 2</td>
<td>Yes</td>
<td>Type C</td>
<td>Conservative management. Residual malabsorption 1 year later.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3</td>
<td>&gt; 20</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>6</td>
<td>&lt; 1/2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>89</td>
<td>M</td>
<td>5</td>
<td>1</td>
<td>&lt; 1/2</td>
<td>Yes</td>
<td>Not identified</td>
<td>Conservative management. Normal examination 6 months later.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2</td>
<td>&gt; 10</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>4</td>
<td>2 - 5</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

continued
<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex</th>
<th>Age</th>
<th>Week of Illness</th>
<th>Beta antitoxin (units/ml.)</th>
<th>Isolation</th>
<th>Cl. perfringens</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>90</td>
<td>M</td>
<td>12</td>
<td>1</td>
<td>&lt; 1/2</td>
<td>Yes</td>
<td>Type A</td>
<td>Suspected case. Brother of Case 69.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2</td>
<td>&lt; 1/2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3</td>
<td>&lt; 1/2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>94</td>
<td>F</td>
<td>22</td>
<td>1</td>
<td>1/2 - 1</td>
<td>Not examined</td>
<td></td>
<td>Mild case. Complete recovery.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2</td>
<td>1/2 - 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3</td>
<td>1/2 - 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>96</td>
<td>M</td>
<td>27</td>
<td>1</td>
<td>1/2 - 1</td>
<td>Yes</td>
<td>Type C</td>
<td>Conservative management initially. Chronic strangulation and stenosis developed. Died post-operatively 3 weeks later.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2</td>
<td>1/2 - 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>4</td>
<td>1/2 - 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>100</td>
<td>M</td>
<td>27</td>
<td>2</td>
<td>10 - 15</td>
<td>Negative</td>
<td></td>
<td>Conservative management early. Resection &quot;lead pipe&quot; jejunum 4 weeks later with recovery.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3</td>
<td>10 - 15</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>5</td>
<td>5 - 10</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>7</td>
<td>2 - 5</td>
<td>1 - 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>101</td>
<td>M</td>
<td>7</td>
<td>3</td>
<td>&gt; 10</td>
<td>Yes</td>
<td>Type A</td>
<td>Treated conservatively. Not followed up.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>4</td>
<td>&gt; 10</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>151</td>
<td>F</td>
<td>27</td>
<td>1</td>
<td>&lt; 1/2</td>
<td>Yes</td>
<td>Type A</td>
<td>Mild case. No complications.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2</td>
<td>2 - 5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>154</td>
<td>M</td>
<td>22</td>
<td>1</td>
<td>&lt; 1/2</td>
<td>Yes</td>
<td>Type C</td>
<td>Resection on 12th day with recovery.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3</td>
<td>1 - 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>157</td>
<td>M</td>
<td>8</td>
<td>1</td>
<td>&lt; 1/2</td>
<td>Yes</td>
<td>Type C</td>
<td>Conservative management. Complete recovery.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2</td>
<td>&gt; 20</td>
<td>1 - 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>159</td>
<td>M</td>
<td>44+</td>
<td>2</td>
<td>1 - 2</td>
<td>Yes</td>
<td>Type A</td>
<td>Treated conservatively. Long convalescence.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3</td>
<td>10 - 15</td>
<td>1 - 2</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table IX - continued
<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex</th>
<th>Age</th>
<th>Week of illness</th>
<th>Beta antitoxin (units/ml)</th>
<th>Isolation Cl. perfringens</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>161</td>
<td>M</td>
<td>6</td>
<td>2</td>
<td>$\frac{1}{2}$ - 1</td>
<td>Not examined</td>
<td>Resection on 5th day with full recovery.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3</td>
<td>$\frac{3}{4}$ - 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>167</td>
<td>M</td>
<td>41</td>
<td>2</td>
<td>$\frac{3}{2}$ - 1</td>
<td>Yes</td>
<td>Conservative management.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3</td>
<td>$\frac{1}{2}$ - 5</td>
<td>Type A</td>
<td></td>
</tr>
<tr>
<td>168</td>
<td>F</td>
<td>27</td>
<td>1</td>
<td>$\frac{3}{2}$ - 1</td>
<td>Yes</td>
<td>Conservative management.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3</td>
<td>$\frac{1}{2}$ - 5</td>
<td>Type A</td>
<td></td>
</tr>
<tr>
<td>191</td>
<td>F</td>
<td>27</td>
<td>2</td>
<td>$\frac{1}{2}$ - 2</td>
<td>Not examined</td>
<td>Given Cl. perfringens type C antiserum intramuscularly 5 days before second examination</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3</td>
<td>200 - 300</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Figure 17: Paired Cl. perfringens beta antitoxin levels in 12 patients with pig-bel. A rising titre was evident in the second and third weeks of illness.
two animals died within 24 hours from ileus and toxemia but the third died in 48 hours from peritonitis. Autopsy of the first two animals revealed irregular patchy haemorrhagic areas diffusely spread along both small and large intestine which macroscopically resembled the human form of the disease. Histological studies confirmed the appearances of acute necrotising enteritis which when stained with Gram-Weigert stain showed numerous Gram-positive rods on the gut epithelium with some invasion of the submucosal layer. Culture yielded *Clostridium perfringens*. The third animal had a haemorrhagic sero-sanguinous peritonitis with some small haemorrhagic areas over the serosal surfaces of the bowel. The mucosa and submucosa were normal on microscopic examination. These investigations were carried no further due to a lack of guinea pigs.

(c) **Serological Investigations**

Paired serological examinations for *Clostridium perfringens* beta antitoxin were made in 21 persons with pig-bel, and are shown in Table IX (Figure 17). Significant rising titres occurred in 19 persons who were either treated conservatively or in whom operation was delayed. The maximum rise was 45 to 70 units. The rise was not so apparent in cases where early operation was undertaken. In four instances more than two sera were collected during the course of an illness. A rise and fall in antitoxin level resulted in each case. The maximum period over which the serum was collected was seven weeks (Case 100). This man had 50 cm. of "lead pipe" jejunum removed
four weeks after the onset of symptoms. From Figure 17 it may be
deduced the maximal immunity developed between the 12th and 20th
day of illness. It is doubtful whether this immunity is permanent.

(d) X-Ray Investigations

The radiological findings depended on the type and stage of the
disease. In early cases there were distended loops of intestine in
round arcs one on top of the other. Twenty-four to 48 hours later
fluid levels appeared. A selection from a few cases was made in
order to trace the development of the disease.

Figure 18 shows the appearance of the intestine on the fourth
day of illness in an eight year old male patient (Case No. 21).
Abdominal distension, tenderness to the left of the umbilicus with
"tinkling" bowel sounds were noted. At operation 100 cm. of upper
jejunum showed patchy segmental necrosis of mucosa and submucosal
areas. The serosa and subserosa were thickened by fibrinous exudate,
edema and empty cystic spaces.

Figure 19 was taken standing 10 days after symptoms appeared in
a 42 year old woman (Case No. 24). The "step-ladder" pattern was
more apparent once ileus was established, particularly if the lower
jejunal or upper ileal segments were involved. Gas was invariably
present in the colon. Reflex distension of the colon from the
toxaemia distinguishes this condition from small bowel obstruction
of other aetiology. At this stage perforation had usually taken
place which was indicated by gas under the right diaphragm. In
Figure 18: Plain X-ray of the abdomen taken in the standing position of an 8-year-old male with pig-bel. Early distension and "step-ladder" fluid levels are shown. There is an associated peritonitis (Case 21).
Figure 12: Advanced ileus and peritonitis in a female aged approximately 42 years with pig-bel (Case 24). 150 to 200 cm of affected jejunum and ileum were resected.
supine position the fluid levels disappeared and bowel folding due
to the oedema became apparent.

Figure 20 demonstrates the more pronounced effect of matted
distended small bowel loops with the appearance of coiled springs
(Case 65). These films were taken four weeks following initial
symptoms and at operation there was stenosis of the lower jejunum
and multiple areas of mucosal ulceration. The bowel loops were
adherent to each other and enclosed by omental adhesions. Barium
study after 15 days' illness in Case 165 showed a grossly distended
proximal jejunum and irregular mucosal areas deficient of contrast
medium. This appearance indicated ulceration and loss of normal
mu cosa. Considerable delay in passage of barium through these
dilated segments occurred (Figure 21). At this stage the roentgen-
ological findings were classical. Even without barium there was a
typical mucosal pattern with coarse transverse folds in a "herring-
bone" pattern. The distended jejunal coils lay in round area with
no visible fluid levels. The walls appeared rigid with a tendency
to be stretched out. The valvulae conniventes were broad, irregular
and ulcerated or eroded when barium was given (Figure 21). These
appearances differ from other forms of ileus where the distended
jejunum exhibits a clear regular and well defined arrangement of the
transverse mucous folds. Caseous distension is also greater.

In pig-bel, distension with gas became more apparent when
peritonitis had occurred. The colon, too, was frequently distended
Figure 20: Supine film showing distended, adherent upper small intestinal loops in a protracted case of pig-belt (Case 65, male aged 6 years).
Figure 21: Barium filled duodenum and proximal jejunum after 3 hours in a man with plg-bol of 15 days' duration. 50 to 100 cm. dilated thickened upper jejunum were removed and the patient made a complete recovery (Case 154). *C. perfringens* type C was recovered from the resected specimen.
with gas, this being more pronounced in the caecum and ascending colon. When gastritis and duodenitis was associated, these organs showed gaseous distension in the plain films.

A localized area of rigid constriction, 20 cm. long, is seen in Figure 22 which was taken five weeks after the onset of symptoms in a 27 year old male (Case 96). In this case the pathology commenced in the fourth part of the duodenum and a bowel short circuit was suspected in the proximal jejunum. Screening was not available to confirm this impression, but an entero-enteric fistula was confirmed at laparotomy. In some places the intestinal wall was completely destroyed leading to free communication between the edges. X-Ray also showed complete loss of the valvulae conniventes. Irregularities with constipation and "lead pipe" upper jejunal lumen was apparent in the 15 minute film of Case 100 (Figure 23) where successful resection of 50 cm. of multiple rigid bowel segments including the terminal part of the duodenum was achieved (Figure 24). In such instances the cicatrizing process was multiple producing multiple stenotic and rigid areas denuded of normal mucosa.

Where single constrictions developed following conservative treatment, massive gaseous dilatation simulating volvulus was apparent in the plain supine film. The barium meal in Case 29 showed massive dilatation of a proximal loop of jejunum (Figure 25). This patient, a male aged 37, developed stenosis following a gastro-enteric episode nine months prior to operation. During this time,
Figure 22: X-ray taken 3 hours after ingestion of barium. There is gross abnormality with stricture, loss of normal mucosal pattern and a short circuiting in the upper jejunum (Case 96, male aged 27 years). The bowel of this patient at operation and after resection is pictured in Figures 15 and 16.
Figure 23: A "lead pipe" segment of jejunum representing a progression of the disease from the 15 day stage shown in Figure 22. 50 cm. of rigid bowel denuded of normal mucosa (Figure 24) were removed during the 5th week of illness (Case 100, male aged 27 years).
The commencement of a rigid fibrous segment of jejunum in a complicated case of pig-bel. Mucosal regeneration amidst ulcerous granulating areas is apparent. The villous pattern of the mucosa was flatterened, resembling that in tropical sprue.
Figure 25: Massive proximal dilatation and hypertrophy in the jejunum proximal to a single stricture. This resulted from pig-bowel disease sustained 9 months previously (Case 29, male aged 57 years).
Figure 26: Film at 150 minutes in a girl aged 8 years with pig-bell (Case 62). Film was taken six weeks following the jejunal initial infection. The mucosa showed villous atrophy when intestinal biopsy was performed 12 months later.
Severely affected upper segments of jejunum in Case 65. (A plain film of the abdomen taken early in the illness is shown in Figure 16.) Resected bowel showed multiple areas of ulcerative jejunitis with strictures and adherent bowel loops. The "string sign" and demonstrable entero-enteric fistula are similar to the radiological findings of Crohn's disease.
Figure 26: Control barium contrast film in a normal subject (female aged 16) showing normal mucosal pattern in the jejunum after 40 minutes. Elongated tubular filling defects are due to Ascaris lumbricoides.
post-prandial upper abdominal colic, weight and appetite loss and
signs of severe malabsorption became apparent.

Segmentation, scattering and disturbed motility were common
findings in milder cases of pig-bel examined by contrast radiographic
methods. In three cases screening was possible, and in Case 62,
taken six weeks after initial infection, dilated jejunal loops with
no visible peristalsis were seen.

Figure 26 shows the typical "moulage sign" due to loss of
mucosal folds with segmentation and clumping of barium and delay in
transit through the small bowel. Segmentation occurred in dilated
portions which had completely lost their valvular commimentes.
Similar findings are shown in Figure 27 (Case 65) taken six weeks
after initial infection.

The above findings in mild and sub-acute cases of pig-bel must
be interpreted in the light of clinical findings. Only two patients
were submitted as controls to the radiologist in this series, and
although one showed ascaris infestation, both had normal motility.
However, both showed slightly dilated jejunal loops with thickened
mucosal folds (Figure 28).

The following X-Ray features of the disease may be summarized
as follows:

(1) In acute cases, diffuse gaseous distension of the small
intestine may indicate paralytic ileus resulting from a
severe enteric infection with Cl. perfringens type C.
Segmental necrotizing areas of upper small intestine leading to mechanical ileus is indicated by marked distension, fluid levels or coiled-spring appearance of loops of bowel. A "step-ladder" appearance in the erect abdominal X-Rays is seen when the lower jejunum and ileum are involved. If perforation with general peritonitis is present, gas may be seen under the diaphragm.

(2) In persons examined after seven days, there is distension of the duodenum and jejunum with coarse irregular rugae or valvulae conniventes. With contrast medium, there is retention of barium between these enlarged folds giving a "herring-bone" appearance. The contrast is retained for several hours in the oedematous jejunal coils as a thin coating along the walls. These findings are diagnostic of acute jejunitis.

(3) Sub-acute surgical forms may show signs of irregularities indicating a "lead pipe" bowel or constriction of the lumen. The "string sign" typical of regional enteritis may occur. Bowel loop inter-communications or fistulations, massive loop distension, and mucosal "skip" areas with filling defects in ulcerated jejunal areas have all been demonstrated. All these complications are associated with delayed transit of barium through affected
segments with an associated slowing of gastric emptying.

(4) A residual chronic enteritis may reveal itself by clumping and scattering indicating disturbed motility patterns.

5. **Diagnosis**

Difficulties in the diagnosis of enteritis necroticans may be deduced from the following table, showing the provisional diagnosis in acute and sub-acute cases admitted to the Goroka or Kumiai hospitals.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Enteritis necroticans</td>
<td>33</td>
</tr>
<tr>
<td>Intestinal obstruction</td>
<td></td>
</tr>
<tr>
<td>Small bowel</td>
<td>15</td>
</tr>
<tr>
<td>Due to Ascaris</td>
<td>5</td>
</tr>
<tr>
<td>Post-dysenteric ileus</td>
<td>6</td>
</tr>
<tr>
<td>Large bowel</td>
<td>2</td>
</tr>
<tr>
<td>Intussusception</td>
<td>1</td>
</tr>
<tr>
<td>Peritonitis</td>
<td>13</td>
</tr>
<tr>
<td>Malnutrition</td>
<td>8</td>
</tr>
<tr>
<td>Gastro-enteritis/dysentery</td>
<td>7</td>
</tr>
<tr>
<td>Bleeding peptic ulcer</td>
<td>4</td>
</tr>
<tr>
<td>Pyloric stenosis</td>
<td>2</td>
</tr>
<tr>
<td>Ruptured spleen</td>
<td>1</td>
</tr>
<tr>
<td>Appendicitis</td>
<td>1</td>
</tr>
</tbody>
</table>

The correct diagnosis was made therefore in only 29.4% of this series.
Table X: Acute and sub-acute abdominal conditions for which laparotomy was undertaken at Goroka Hospital for period 1.2.61 to 30.11.64

<table>
<thead>
<tr>
<th>Condition</th>
<th>No. of Cases</th>
<th>Condition</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pig-bell:</td>
<td></td>
<td>Abdominal trauma:</td>
<td></td>
</tr>
<tr>
<td>Acute</td>
<td>40</td>
<td>Ruptured spleen</td>
<td>9</td>
</tr>
<tr>
<td>Sub-acute</td>
<td>29</td>
<td>Bowel rupture or penetration</td>
<td>21</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ruptured kidney</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ruptured bladder (intraperitoneal)</td>
<td>1</td>
</tr>
<tr>
<td>Strangulation:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A. By adhesions</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post-inflammatory</td>
<td>18</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post-operative</td>
<td>8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Congenital</td>
<td>7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>B. By internal hernia</td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C. Volvulus</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Appendicitis:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Indigenous</td>
<td>16</td>
<td></td>
<td></td>
</tr>
<tr>
<td>European</td>
<td>26</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peritonitis:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ruptured pyosalpinx</td>
<td>9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Perforated colon</td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ascarsis perforation</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Puerperal (excluding ruptured uterus)</td>
<td>6</td>
<td></td>
<td>39</td>
</tr>
<tr>
<td>Systemic</td>
<td>8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sub-phrenic</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post-dysenteric</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tuberculous</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>301</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table I lists various conditions which required laparotomy for
the period February 1, 1964 to November 30, 1964. Pig-bel was the
commonest acute surgical abdominal condition encountered in Highland
practice. Early diagnosis improved the prognosis as it did in all
other cases, where acute abdominal pain called for prompt diagnosis
and treatment.

6. Treatment

The primary pathological lesion was destruction of the surface
mucosa of the small bowel, followed by a massive exudation and loss
of fluid and blood. Fluid loss was much greater than that suggested
by the volume of vomit or fluid stools when mechanical or paralytic
ileus was established. Therapy varied according to the site, degree
and duration of obstruction.

(a) Fluid and Electrolyte Replacement

The disease mainly affected children under 10 years of age and
the importance of fluid replacement was paramount. Water loss
through the lungs and skin was an important consideration because
of the hot dry day time conditions. Patients were carried to
hospital after a full day’s walk. Occasionally the pre-existing
nutritional state of the patient was poor. Where dehydration was
pronounced treatment with one-fifth normal saline was given prior to
blood transfusion. Whether potassium replacement should be
instituted was difficult because of the lack of facilities for
routine electrolyte estimations and the strong possibility of the presence of impaired renal function. Where it was established that diarrhoea had occurred for a period of three or more days prior to the onset of ileus and where renal function was established, small amounts of potassium chloride (3 mEq./kg. or in adults 2.0 gm./litre or 52 mEq.) were given.

Intravenous blood was considered the best fluid replacement to re-expand the extracellular volume: 10 to 20 ml./kg./body weight in children and 1 to 2 litres in adults. When blood collection and cross matching facilities were not available and severe acidosis was clinically apparent, prompt intravenous infusion of either a mixture of two parts normal saline and one part sixth molar sodium lactate or Ringer lactate solution in 20 to 30 ml./kg./body weight in two or three hours were substituted.

For these cases transfusion with blood gave the best results. The mortality rate in 65 acute and sub-acute cases not given blood transfusion initially was 48.4 per cent and in 44 cases in which blood was given the mortality was only 22.0 per cent. In a further group of nine cases in which no intravenous therapy was given at all, eight deaths resulted.

Clinical response following transfusion was assessed by a slower and fuller pulse, increased blood pressure, slower respirations and improvement in the skin colour and temperature. Improvement was usual within 6 to 12 hours, and if not the outlook was considered grave.
An intravenous maintenance phase was sustained, along with adjustments for physiological losses using standard 0.45 per cent sodium chloride with 2.5 or 5 per cent dextrose. In this situation potassium was given via gastric tube or orally in a mixture having the following composition: sodium chloride 1.5 gm., potassium chloride 2.0 gm., raspberry syrup 60 gm. and water to 1000 ml. The dose was 300 to 500 ml. daily in divided doses in the absence of ileus or vomiting. This solution was again used in the transition to oral feedings after 24 to 48 hours of alimentary rest or 48 to 72 hours after resection pending the return of normal bowel function.

(b) **Bowel Decompression**

The second part of the initial treatment was decompression of the upper small bowel by gastric or duodenal suction. Whether distension was clinically evident or not, the presence of infrequent "metallic" bowel sounds indicated the development of ileus and decompression was deemed necessary. This was achieved in most cases by the use of a Ryles tube with a weighted tip and attached to continuous suction apparatus or intermittent half hourly suction if this was not possible. Suction was maintained until physical signs indicated the return of normal intestinal function. This occurred usually between 24 and 72 hours. A small percentage of these patients (16.4 per cent) experienced a complete return to normal bowel function. The remainder developed later complications of perforation and peritonitis. The conservative and operative management of 141 persons with severe
Table XI: Case mortality rates related to management in severe pig-bell disease. The prognosis appeared better for patients treated by operation.

<table>
<thead>
<tr>
<th>Clinical Type</th>
<th>Total Cases</th>
<th>Operative Management</th>
<th>Mortality %</th>
<th>Conservative Management</th>
<th>Mortality %</th>
<th>No Treatment</th>
<th>Mortality %</th>
</tr>
</thead>
<tbody>
<tr>
<td>I Acute Toxic</td>
<td>26</td>
<td>4</td>
<td>50.0</td>
<td>17</td>
<td>88.2</td>
<td>5</td>
<td>100.0</td>
</tr>
<tr>
<td>II Acute Surgical</td>
<td>81</td>
<td>49</td>
<td>34.7</td>
<td>27</td>
<td>44.4</td>
<td>5</td>
<td>100.0</td>
</tr>
<tr>
<td>III Sub-acute Surgical</td>
<td>46</td>
<td>32</td>
<td>37.5</td>
<td>12</td>
<td>50.0</td>
<td>2</td>
<td>100.0</td>
</tr>
<tr>
<td>TOTAL</td>
<td>153</td>
<td>85</td>
<td>36.5</td>
<td>56</td>
<td>59.9</td>
<td>12</td>
<td>100.0</td>
</tr>
</tbody>
</table>

\[\text{Significance:} \text{Operative vs. Conservative} \text{ Not Significant}\]
disease is assessed in Table XI. There was an improvement in mortality in those surgically treated (36.5 per cent as against 59.9 per cent). This trend in the prognosis was evident in all three groups treated by surgical resection of affected bowel, being most pronounced in the very acute disease (groups I and II of Table XI).

(e) Antibiotics

When the disease was initially recognised various antibiotic combinations were tried empirically (Murrell and Roth, 1963).

Sensitivity tests using Difco bacto-sensitivity discs conducted subsequently on seven **Clostridium perfringens** type C cultures gave the following results:

- **Penicillin**
  - Sensitive
  - +++
- **Streptomycin**
  - Resistant
  - -
- **Chlormephenicol**
  - Sensitive
  - +
- **Chlortetracycline**
  - Sensitive
  - ++
- **Oxytetracycline**
  - Sensitive
  - ++
- **Bacitracin**
  - Sensitive
  - +++

Large doses of sulphathalidime (phthalylsulphathiazole) or sulfasaltpur (each tablet containing streptomycin sulphate BP 65 mg., sulphamerazin BP 65 mg., sulphadiazin BP 100 mg. and sulphathiazole BPC 100 mg.) were given via intra-duodenal tube at four hourly intervals to eradicate the Gram-negative flora of the bowel. The dose of the former in the adult was 2 g., four hourly and of the
latter four tablets every four hours. Both (personal communication) also added neomycin sulphate to assist this initial local bowel sterilisation although no effect could be anticipated on clostridial organisms. In fact, this antibiotic was used in differential media for the isolation of *Clostridium perfringens* from jejunal content (Egerton and Walker, 1964).

Of the other antibiotic combination, penicillin and chloramphenicol seemed to give the best results when intravenous fluid and intestinal decompressive measures were standardised. There was little difference in mortality rates with the more widely used penicillin-streptomycin combination despite the in vitro resistance of *Clostridium perfringens* type C to streptomycin. Chloramphenicol succinate was given intravenously in the initial fluid replacement in the severely toxic case which provided an initial loading dose of broad spectrum antibiotic to counter the possibility of a welchii septicemia. On the sensitivity results, the tetracycline group may prove to be preferable.

(d) **Additional Supportive Therapy**

In the severer forms, intravenous vitamin B group therapy 2 ml. daily (thiamine 10 mg., riboflavin 5 mg., niacinamide 50 mg., calcium pantothenate 5 mg., pyridoxine 2.5 mg.) and vitamin C 100 mg. were given, transferring later to the intramuscular and oral routes of administration. Folic acid was also given before and after operation to persons with extensive jejunal necrosis. Morphine was preferred to other pain suppressive drugs. Antispasmodics were used, but their effects could
Table XIII: Comparison of groups of patients with severe pig-bel disease treated with poly-valent gas gangrene antiserum and specific Cl. perfringens type C antiserum. There is a significant reduction in mortality in the latter group.

<table>
<thead>
<tr>
<th></th>
<th>Polyvalent gas gangrene antiserum</th>
<th>Cl. perfringens type C antiserum</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total No Yes</td>
<td>Total No Yes</td>
</tr>
<tr>
<td>Treated*</td>
<td>19 12 7</td>
<td>98 66 32</td>
</tr>
<tr>
<td>Bowel resection</td>
<td>4 8 12 ?</td>
<td>70 55 15</td>
</tr>
<tr>
<td>Died</td>
<td>9 6 3</td>
<td>35 29 6</td>
</tr>
<tr>
<td>Mortality (%)</td>
<td>47.4 50.0 42.8</td>
<td>35.7 43.9 18.7</td>
</tr>
</tbody>
</table>

*Persons with acute and sub-acute surgical disease are included in table. Twenty-six persons with suspected mild disease were also given Cl. perfringens type C and are not included in this series.
Figure 29: A comparison of the mortality rates in severe cases of pig-bell treated with polyvalent gas gangrene serum (difference not significant) and Cl. perfringens type C antiserum (difference most significant).
not be accurately assessed. Hyoscine N-butylbromide ("Buscopan") 0.02 gm. was given at four or eight hourly intervals depending on the severity of the bowel calic. Relief seemed more apparent in children. In mild cases in children a bismuth and opium mixture (Mixture bismuthi sed. BPC) was of doubtful help.

(e) Treatment with Specific Antitoxin

It was not until mid 1961 that an organism of the Cl. welchii group was considered to be a major cause of pig-bel. Confirmation of the type and strain of the organism was not available until 1962. Therefore, attempts to introduce antitoxin therapy involved initial trials with polyvalent gas gangrene serum in 1961 (19 cases) and the use of the more specific Cl. perfringens type C antiserum from June 1964 onwards. For this latter study, treatment trials of antiserum were offered to 32 severe and 26 mild cases of enteritis necroticans.

In the first series seven randomly chosen subjects received intramuscularly 15,750 units of polyvalent gas gangrene antitoxin consisting of 7,500 units of Cl. welchii antitoxin, 3750 units of Cl. septicae and 2,500 units of Cl. oedemaens antitoxin. This was given when other treatment procedures had been standardised. The results of this trial appear in Table XIII which although small, indicated no appreciable improvement in the mortality when compared with 12 cases not given the polyvalent gas gangrene serum (Figure 29).

A batch of Cl. welchii type C antiserum (Serial No. E9595) was
received from the Wellcome Research Laboratories, Beckenham, Kent, U.K.
and trials commenced in July 1964. The serum contained 8,500 units of
antitoxin to the beta toxin of Cl. welchii type C in each cubic
centimetre. Cresol (0.5%) had been added as a preservative because
the serum could not be refrigerated in transit. Following the
criteria and classification of disease types described earlier, 42 to
85,000 units of serum were included in the intravenous fluid therapy of
32 persons with severe pig-bel disease during the five month period,
July to December 1964. Fifteen of the 32 required resection and six
patients died. Three died in the post-operative period and two were
so moribund when therapy was commenced that death occurred before
laparotomy could be undertaken. The necessity to intervene surgically
therefore, fell by one-third from 66.7 per cent to 46.9 per cent
(Table XIII). This was clinically demonstrable by a rapid improvement
in the patient's condition in the 24 hour period following antitoxin.
The pulse rate fell, the patient appeared less toxic and subjectively
felt much better. The mortality fell dramatically from 43.9 per
cent to 18.7 per cent which was statistically significant (p < 0.01)
(Figure 29).

A group of subjects with mild forms of the disease was also given
5 cc. (42,500 units) of the antiserum intramuscularly as it was not
known what effect the serum would have on humans. It had previously
been prepared for use in lamb dysentery in which the recommended dose
is 7,000 units for a 7 to 12 lb. lamb (Samels, 1964). No adverse
Table XIII: The relationship between early and delayed treatment in 141 cases of pig-bel. Table demonstrates that early resuscitation and operation reduced the case mortality.

<table>
<thead>
<tr>
<th>Group</th>
<th>Total No. Treated</th>
<th>Duration of Symptoms</th>
<th>Delay in Treatment</th>
<th>Bowel Resection</th>
<th>Operative Mortality %</th>
<th>Total Mortality %</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>21</td>
<td>3 days</td>
<td>Minimal (≤ 24 hours)</td>
<td>4</td>
<td>50.0</td>
<td>76.2</td>
</tr>
<tr>
<td>II</td>
<td>38</td>
<td>4 - 7 days</td>
<td>Short (≤ 5 days)</td>
<td>28</td>
<td>16.7</td>
<td>13.2</td>
</tr>
<tr>
<td>III</td>
<td>38</td>
<td>4 - 7 days</td>
<td>Long (&gt; 5 days)</td>
<td>25</td>
<td>48.0</td>
<td>60.5</td>
</tr>
<tr>
<td>IV</td>
<td>13</td>
<td>8 - 13 days</td>
<td>Long</td>
<td>7</td>
<td>42.6</td>
<td>38.5</td>
</tr>
<tr>
<td>V</td>
<td>31</td>
<td>14 days</td>
<td>Long</td>
<td>21</td>
<td>38.9</td>
<td>45.6</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>141</strong></td>
<td></td>
<td></td>
<td><strong>85</strong></td>
<td><strong>36.5</strong></td>
<td><strong>45.4</strong></td>
</tr>
</tbody>
</table>
effects were noted.

The reduction in mortality in the severe group (18.7 per cent) compared to earlier cases provided proof of the efficacy of the antitoxin and support for the etiology of the disease. Supplies were distributed to other medical officers for trial and dramatic improvement in recovery rates have been reported to the author (Commer, Mohan and Smith, personal communications, 1965). In Commer's experience the necessity to intervene surgically following antitoxin administration was reduced by some 60 to 70 per cent.

(f) Indications for Laparotomy

In a disease of such variable severity, each patient had to be assessed individually. In most cases the victims arrived late to hospital and surgical intervention was inevitable because of the development of complications.

In an attempt to guide future medical personnel in the Highlands on the decision to transfer or operate upon a patient suspected as having the disease, pragmatic data have been assessed (Table XIII). This indicated that no didactic and absolute rules could be laid down. However, with most patients when intestinal obstruction was established and remained so despite adequate decompression, surgical exploration at the earliest time gave the best results. This conclusion is supported by the figures previously shown in Table XII.

In Table XIII, group I represents, as in other tables, patients
with acute toxic pig-bel. Operation was undertaken in only four of these patients within three days of the onset of symptoms. This was the most lethal form of the disease and the mortality in treated cases was 76.2 per cent.

In group II, 28 of 38 patients had immediate operation following institution of intravenous and decompressive measures, and the death rate was 13.2 per cent. Resuscitation and operation were undertaken within five days of the onset of symptoms.

The contrast between groups II and III when initial treatment and operation was delayed more than five days is significant. The mortality rose with this group to 60.5 per cent. The patient was more shocked, toxemic and presented as a grave anaesthetic risk, especially from the open ethyl chloride ether induction anaesthetic common in practice in New Guinea.

Groups IV and V consisted of persons with sub-acute surgical disease in whom operative delay was long because the onset was less acute. Again, a longer delay in diagnosis and operation resulted in a higher mortality. From these figures it would seem that, except in severe toxic moribund cases, early operation was favoured over that of a conservative "wait and see" management.

The indications for operative intervention were determined thus:

1. Failure to relieve mechanical or paralytic ileus after 24 hours despite intestinal decompression and intravenous therapy.
(2) Increasing signs of toxemia in spite of therapy.

(3) Signs of localised or diffuse peritonitis.

(4) Continuing abdominal pain and the palpation of a
tender upper abdominal mass. A change in pain
character from a severe colic to a more continuous
nature usually indicated strangulation.

(5) Loss of weight, continuing refusal of food, symptoms
and signs of sub-acute small bowel obstruction and
anaemia. This situation arises from one to six months
after the initial illness and indicates the development
of stricture or fistula formation.

(6) **Operative Method**

The choice lay between enterostomy, resection and anastomosis or,
where a single perforation had occurred in otherwise normal bowel,
oversewing. Enterostomy was performed on two occasions and both
patients died. Limited resection of the more severely affected parts
of the jejunum was at first tried with little success, four out of six
patients dying. In these cases the anastomosis was end to end and
leaks developed in three of them, presumably round a pre-existing
ulcerated area in the anastomosis. More success was achieved by
undertaking a radical resection of the jejunum with a side to side or
end to end anastomosis. Where the process commenced in the fourth part
of the duodenum, as much as possible of the diseased upper small
intestine was removed. In two patients a fatal haemorrhage occurred
from ulcers higher in the duodenum on the fourth and fourteenth postoperative days. An end to end anastomosis was necessary when the
disease was located near the duodeno-jejunal angle. Roth (1964)
generally preferred a side to side anastomosis whereas his successor
(Smith, 1964) achieved comparable success with an end to end join
(personal communications). Roderigue (1964) had some success with
oversewing perforations in two patients, one later dying from severe
wasting and a presumed sub-acute intestinal obstruction. The range
of resected lengths was from 20 cm. to 350 cm. In three operative
cases the whole small bowel and ascending colon were involved. The
length most usually resected was 50 to 150 cm. (three to five feet).

Post-operative management concerned the maintenance of fluid and
electrolyte balance, the return of normal bowel and renal function
and the gradual resumption of a normal diet. The following scheme
was adopted.

Four days fasting; oral fluids gradually commencing with glucose
and electrolyte fluids and grading to milk mixtures over a further
five days; during the latter part of the second week protein enriched
soft foods (for example, casimal) and mashed vegetables; three weeks
gradual resumption of normal diet.

7. Follow-up Studies

Forty-nine subjects who had had pig-bel were examined at varying
periods after their acute illness. Similar observations were made on
Table XVII: Summary of intestinal biopsy examinations in cases with known pig-bel disease

<table>
<thead>
<tr>
<th>No. of Subjects</th>
<th>Case No.</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stomach</td>
<td>2</td>
<td>3, 47</td>
</tr>
<tr>
<td>Duodenum</td>
<td>1</td>
<td>50</td>
</tr>
<tr>
<td>Jejunum</td>
<td>3</td>
<td>33, 41, 84</td>
</tr>
</tbody>
</table>

Group I: Post-operative subjects
- Stomach: Normal, Haemorrhagic gastritis
- Duodenum: Villous clubbing
- Jejunum: Flat atropho mucosa

Group II: Medically treated subjects
- Oesophagus: Normal
- Stomach: Normal, Haemorrhagic gastritis
- Duodenum: Villous clubbing, Flat mucosa, Active duodenitis
- Jejunum: Villous clubbing, Flat mucosa, Acute jejunitis

Group III: Normal controls
- Stomach: Normal
- Jejunum: Normal, Villous clubbing

TOTAL 29
23 normal subjects at the same time. The time interval between the initial infection and the follow-up examination ranged from four weeks to three and one-half years. The mean interval was ten months.

The subjects were divided into two groups. Group I, comprising 20 persons, had been surgically treated by resection. Twenty-nine persons in Group II had been medically treated. Enquiries regarding two further patients treated medically in Goroka (Nos. 2 and 20) in 1961 indicated that they had died from wasting and intestinal obstruction six months after their initial illness. These cases were excluded from the study.

Age and sex distribution of subjects in this study are listed in Table XIV. Weight records were available in 19 subjects from groups I and II. Twelve had weight gains and seven showed a weight loss. The decrease in weight ranged from 1.6 to 10.4 kg. Physical examination revealed finger clubbing in only one subject. Clinical syndromes of nutritional deficiency such as beri beri, pellagra, purpura and tetany were not observed. There was, however, liver enlargement in 15 of the 49 examined in the follow-up cases. Four persons had enlarged livers in the 23 control subjects.

A summary of the blood and serum investigations is given in Table IV. All subjects were assessed collectively irrespective of age and sex. Anemia in both sexes was diagnosed if the haemoglobin was less than 11.5 gm. per 100 ml.

In the control group, three persons (13.1 per cent) were anemic.
Table XV: Haemoglobin and serological findings in follow-up studies on persons with pig-bel and normal individuals

<table>
<thead>
<tr>
<th>Examination</th>
<th>Haemoglobin (g./100 ml.)</th>
<th>Serum Folic Acid Activity (µg./ml.)</th>
<th>Serum Vitamin B₁₂ (µg./ml.)</th>
<th>Serum Iron (µg./100 ml.)</th>
<th>Total Serum Proteins (g./100 ml.)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Group I: Post-operative subjects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(No. examined)</td>
<td>(18)</td>
<td>(12)</td>
<td>(9)</td>
<td>(2)</td>
<td>(4)</td>
</tr>
<tr>
<td>Mean</td>
<td>12.5</td>
<td>15.8</td>
<td>718</td>
<td>135</td>
<td>6.7</td>
</tr>
<tr>
<td>Range</td>
<td>7.1 - 17.6</td>
<td>1.4 - 44.0</td>
<td>420-1850</td>
<td>89-182</td>
<td>6.1 - 8.1</td>
</tr>
<tr>
<td><strong>Group II: Medically treated subjects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(No. examined)</td>
<td>(28)</td>
<td>(24)</td>
<td>(19)</td>
<td>(5)</td>
<td>(14)</td>
</tr>
<tr>
<td>Mean</td>
<td>11.1</td>
<td>13.9</td>
<td>746</td>
<td>90</td>
<td>7.0</td>
</tr>
<tr>
<td>Range</td>
<td>5.0 - 18.0</td>
<td>1.0 - 44.0</td>
<td>150-1850</td>
<td>66-128</td>
<td>5.5 - 8.5</td>
</tr>
<tr>
<td><strong>Group III: Controls</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(No. examined)</td>
<td>(23)</td>
<td>(20)</td>
<td>(10)</td>
<td>(-)</td>
<td>(4)</td>
</tr>
<tr>
<td>Mean</td>
<td>13.6</td>
<td>18.1</td>
<td>839</td>
<td>-</td>
<td>7.4</td>
</tr>
<tr>
<td>Range</td>
<td>8.3 - 17.2</td>
<td>2.6 - 46.2</td>
<td>400-3000</td>
<td></td>
<td>6.0 - 8.6</td>
</tr>
</tbody>
</table>
Figure 10: Haemoglobin values in follow-up studies on 22 normal subjects, 29 subjects with sub-acute pig-bel disease treated medically, and 16 treated operatively. The dotted lines represent the mean for each column.
Figure 31: Serum folic acid activity levels in follow-up studies of 20 normal subjects, 24 persons with pig-bel treated conservatively, and 12 surgically treated subjects with the disease. The dotted line represents the lower limit of normal (2 μg/ml).
**Figure 32:** Serum B₁₂ estimations in 9 control subjects, 19 medically treated subjects and 9 post-operative patients examined in follow-up studies. The dotted line represents the lower limit of normal.
Sixteen (57.1 per cent) had anaemia in the medically treated group and eight (44.6 per cent) had anaemia in the post-operative group. Four of the anaemias in group II patients were macrocytic and ten were normocytic or microcytic. In one patient from group I the anaemia was macrocytic. Sternal marrow in this case (No. 4) confirmed the presence of transitional megaloblastic changes. The haemoglobin values for the three groups are shown in Figure 30.

Low serum levels of folate activity were observed in persons with sub-acute disease who had been treated conservatively (Figure 31). Four patients had levels lower than a chosen arbitrary lower limit of 2 μg. per ml. (Deller, Ibbotson and Crompton, 1964). Two of these (Cases 93 and 100) had steatorrhoea and X-Ray and histological evidence of jejunal abnormality. A similar, but less marked trend towards low folate activity was present in 12 patients who had had small bowel resections. The length of jejunum resected ranged from 50 cm. to 200 cm. One patient (Case 47) had a serum folate level of 1.5 μg. per ml., macrocytic anaemia and steatorrhoea. Gastric biopsy in this lad showed hypertrophy and haemorrhage of the mucosa. Serum levels of folate activity were normal in each subject in the control group.

Serum vitamin B₁₂ levels were normal in the subjects studied except for one patient (Case 93) who had the sub-acute disease (Figure 32). (Deller and Witts, 1962).

Insufficient sera remained for a full evaluation of serum iron, calcium and alkaline phosphatase in these studies. The serum iron
level was estimated in seven subjects in groups I and II and was normal in each case. Serum calcium levels were also within normal limits for Europeans in five persons (Deller, Edwards and Addison, 1963). In group II patients examined shortly after their acute enteric illness, four of seven had raised alkaline phosphatase levels (range 13.1 to 30 King Armstrong units). Six such examinations in subjects with the more protracted illness were all normal.

Serum protein levels and electrophoretic patterns in 18 patients in groups I and II followed the normal New Guinea values (Karika, 1960). The gamma globulin levels were constantly raised and there was a reversal of the albumin/globulin ratio. However the beta globulin fraction was lower than previously found in New Guinea. In eight of the 18 patients there was an associated low albumin level. The alpha 2 fraction was raised in three of ten other examinations.

Fecal fat estimations were undertaken in four anemic subjects in group I, 13 subjects with anemia in group II and four normal persons from group III. Steatorrhoea, defined as a fat content of over 30 per cent dried faeces, was present in three persons treated surgically, in five treated medically and in one of the control subjects.

Only a small number of patients had follow-up small bowel X-Rays with screening. Four of six post-operative subjects and two patients examined in the medically treated group had abnormal small bowel radiographs. Two controls were essentially normal. The results are shown in Table XVI. Condensed reports from the radiologist (Dr. D.J.
Table XVI: Results of small bowel series X-ray examinations (10 subjects)

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Time after operation (years)</th>
<th>Approximate Amount Resected (cm.)</th>
<th>Pylorospasm</th>
<th>Jejunal Dilatation</th>
<th>Clumping Segmentation</th>
<th>Stenosis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Group I:</strong> Post-operative subjects</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>25</td>
<td>1.5</td>
<td>200</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>29</td>
<td>1.5</td>
<td>75</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+ (+ Ascaris)</td>
</tr>
<tr>
<td>33</td>
<td>1.5</td>
<td>150</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>- (+ non-sclerosing ileitis)</td>
</tr>
<tr>
<td>44</td>
<td>0.8</td>
<td>200</td>
<td>+</td>
<td>+</td>
<td>++</td>
<td>+ (residual enteritis)</td>
</tr>
<tr>
<td>45</td>
<td>0.9</td>
<td>100</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>48</td>
<td>1.0</td>
<td>125</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>- (+ Ascaris)</td>
</tr>
<tr>
<td><strong>Group II:</strong> Medically treated subjects</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>53</td>
<td>1.6</td>
<td>Nil</td>
<td>-</td>
<td>+</td>
<td>++</td>
<td>- (+ Ascaris)</td>
</tr>
<tr>
<td>62</td>
<td>0.6</td>
<td>Nil</td>
<td>+</td>
<td>+</td>
<td>++</td>
<td>-</td>
</tr>
<tr>
<td><strong>Group III:</strong> Controls</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C 79</td>
<td></td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>- (+ Ascaris)</td>
</tr>
<tr>
<td>C 80</td>
<td></td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>
Bassett) in this series are as follows:

No. 44, M/27, Post-resection:

The stomach and duodenum were hypotonic. The proximal jejunal loop was dilated and the mucosal folds were prominent in this segment. Vigorous churning movements were observed on screening. The serial films showed clumping and segmentation in the lower small bowel with narrowed and dilated segments and obliteration of the normal mucosal pattern. The rate of transit was not abnormal. Conclusion: The X-ray appearances suggest an extensive involvement of the small bowel with partial obstruction in the upper jejunum, possibly at the site of anastomosis.

No. 48, M/12, Post-resection:

The stomach, duodenal loop and proximal portion of the jejunum were dilated apparently due to partial obstruction at the site of the anastomosis. The normal active and vigorous peristalsis in this segment was not observed.

Transit of the media through the small bowel was delayed due to diminished peristaltic activity and after 2½ hours the head of the barium had only reached the proximal portion of the ileum.

Conclusion: There is segmentation and flocculation of the barium consistent with jejunitis.

No. 62, P/9, Post-conservative:

There was considerable delay in gastric emptying. Dilated
### Table XVII: Summary of intestinal biopsy examinations in cases with known pig-bel disease

<table>
<thead>
<tr>
<th>Group</th>
<th>Subjects</th>
<th>Case No.</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Subjects</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Stomach</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Group I:</td>
<td>Post-operative subjects</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Stomach</td>
<td>2</td>
<td>3, 47</td>
</tr>
<tr>
<td></td>
<td>Duodenum</td>
<td>1</td>
<td>50</td>
</tr>
<tr>
<td></td>
<td>Jejunum</td>
<td>3</td>
<td>33, 41, 84</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Haemorrhagic gastritis</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Villous clubbing</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Flat atrophic mucoza</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Group II:</td>
<td>Medically treated subjects</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Oesophagus</td>
<td>1</td>
<td>97</td>
</tr>
<tr>
<td></td>
<td>Stomach</td>
<td>6</td>
<td>18, 94, 102, 153, 163, 188</td>
</tr>
<tr>
<td></td>
<td>Duodenum</td>
<td>6</td>
<td>93, 96, 98, 100, 103, 164</td>
</tr>
<tr>
<td></td>
<td>Jejunum</td>
<td>5</td>
<td>62, 89, 90, 92, 159</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Haemorrhagic gastritis</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Villous clubbing</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Flat mucosa</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Active duodenitis</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Villous clubbing</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Flat mucosa</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Acute jejunitis</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Group III:</td>
<td>Normal controls</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Stomach</td>
<td>2</td>
<td>C85, C89</td>
</tr>
<tr>
<td></td>
<td>Jejunum</td>
<td>2</td>
<td>C86, C90</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Villous clubbing</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td></td>
<td><strong>29</strong></td>
<td></td>
</tr>
</tbody>
</table>
jejunal loops showed no visible peristalsis on screening. The films showed the typical "moulage sign" best seen in the 150 and 200 minute films, and due to advanced disease with atony and atrophy of segments of the small bowel. The barium was segmented in dilated portions of the bowel which showed complete loss of the valvulae oesintentes, a disordered X-ray pattern involving almost the whole of the small bowel. No normal mucosal folds were apparent. Conclusion: Extensive jejunitis with destruction of mucosal folds and impaired motor function.

Twenty-eight gastric, duodenal and jejunal biopsies were obtained with the Crosby capsule using a "blind" technique. The results of the histological examination of this material are given in Table XVII. A haemorrhagic gastritis was commonly found in both groups in material recovered from the stomach (Figure 33). Two degrees of mucosal abnormality were classified in the small bowel material obtained. In the first instance minor changes consisted of thickening, shortening and clubbing of the villous pattern with varying degrees of mono-cellular infiltration of the lamina propria. The surface epithelium appeared normal. These findings occurred in three biopsies from the duodenum or jejunum in the surgically treated group. In the second class, gross villous atrophy with flattened mucosa and heavy infiltration of the lamina propria with plasma cells, lymphocytes and eosinophils were present in six of the intestinal biopsies. The
Figure 33: Gastric biopsy from a patient (Case No. 18, male, 5 years) conservatively treated for pig-bel disease two and one-half years earlier. He was wasted, stunted in growth and had a megaloblastic anemia when examined. Section shows a hypertrophic haemorrhagic gastritis. H & E x 170

Figure 34: Duodenal biopsy taken from a person with pig-bel who had a distal stenosis and entero-enteric fistulae (Case 26, male aged 27 years). The villi were clubbed and fused with extensive plasma lymph cell infiltration of the lamina propria. H & E x 170
Figure 35: Jejunal biopsy taken from a person in whom resection had been undertaken six months earlier. The mucosa was flattened and the patient (Case 84, male aged 10 years) had some of the features of the malabsorption syndrome. H & E x 170

Figure 36: Jejunal biopsy from a control subject (male, aged 47 years). Villous pattern shows some slight abnormality probably due to upper intestinal helminthic infestation. H & E x 170
surface columnar cells were also distorted in these sections. In one case (No. 62) there was follicle formation adjacent to the muscularis. Figures 34 and 35 show these mucosal changes which may be compared with a normal villous pattern obtained from one of the control subjects (Figure 36). In two of the patients from group II, subsequent X-ray investigations indicated cicatrizing areas distal to the biopsy sites. At laparotomy a "lead pipe" segment of jejunum was removed from one man (Case 100) and a fistulous segment of upper intestine from another patient (Case 96).

The degree of cellular infiltration was most variable in the sections. Homogeneous patches of eosinophilic material were present in the lamina propria of the mucosa which had appeared like that in sprue or coeliac disease. There were relatively more instances of this type of abnormality in the medically treated group.

From these collective investigations, a diagnosis of malabsorption syndrome was a strong possibility in 11 of the 49 persons examined in this study. Details of these cases appear in Table XVIII.

6. Prognosis

Age had a considerable bearing on the prognosis. Mortality rates in 210 cases were considerably higher in the younger and older age groups (Figure 37). In the absence of treatment for severe forms of enteritis necroticans, the mortality is probably close to 100 per cent. Death occurred within four days in acute toxic states and within ten
Table XVIII : Patients with suspected acute and chronic malabsorption following enteritis necroticans infection

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Resection (cm.)</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Time Interval (years)</th>
<th>Weight (kg.)</th>
<th>Hb. (g./100 ml.)</th>
<th>MCV (μL)</th>
<th>Serum Folic Acid (μg./ml.)</th>
<th>Serum B12 (μg./ml.)</th>
<th>Faecal Fat (% dry faeces)</th>
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<td>3.3</td>
<td>350</td>
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<td>97</td>
<td>9.0</td>
<td>450</td>
<td>55</td>
<td>+</td>
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*Degree of villous blunting: + mild; ++ moderate; +++ severe.
Figure 37: Age and sex mortality rates in the 210 case series reviewed in the epidemiology. The prognosis was worse in the very young and elderly persons with pig-beal.
days in untreated surgical forms of pig-bel. An overall mortality of
36.2 per cent (Table III) was estimated: 42.0 per cent in acute
surgical forms; 43.5 per cent in sub-acute surgical forms. Mortality
following operative management in the acute surgical group was 34.7
per cent and following conservative management it was 44.4 per cent
(Table XII). A small percentage of this latter group (probably of
the order of 5 to 10 per cent) enter a sub-acute phase in which the
operative mortality was 37.5 per cent. Following conservative
management the death rate also rose in this group to 50 per cent.
More extensive and prolonged follow-up studies will be necessary to
determine the long term effects of mild infections, but evidence
presented in section 7 of this chapter suggests malabsorptive disorders
may result in a few instances. The long-term effects of more massive
intestinal resection can only be assessed over many years of follow-up
examinations.

It can be concluded that early decompression and resuscitation
greatly improved the chances of survival by conservative management and
reduced post-operative mortality. When positive indications for bowel
resection existed, the longer the delay the worse the prognosis (Table
XIII). Following the introduction of Cl. welchii type C antisera in
small series of cases, both the necessity to operate and the
overall mortality were significantly reduced (Table XII, Figure 29).
Table XIX: The distribution of lesions in the gastro-intestinal tract in 97 cases of pig-bel.

<table>
<thead>
<tr>
<th>Origin</th>
<th>Total</th>
<th>Oesophagus</th>
<th>Stomach</th>
<th>Duodenum</th>
<th>Jejunum</th>
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<td>5</td>
<td>8</td>
<td>24</td>
<td>20</td>
<td>9</td>
</tr>
<tr>
<td>Laparotomy or resection</td>
<td>73</td>
<td>-</td>
<td>3</td>
<td>15</td>
<td>68</td>
<td>58</td>
<td>25</td>
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<tr>
<td>TOTAL</td>
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<td>0</td>
<td>8</td>
<td>23</td>
<td>92</td>
<td>78</td>
<td>34</td>
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</tbody>
</table>
9. Pathology

An attempt has been made to correlate the appearance of the bowel at necropsy or operation with the clinical features of the illness. The bowel was examined at necropsy on 24 cases and at laparotomy on 73 occasions. In each case the length of bowel involved was measured. In 43 cases histopathological studies of the intestines were also made.

The most frequent length of intestine affected was between 50 and 150 cm. (Figure 58). Segments were as short as 20 cm., and in four extensive cases the entire small intestine and colon were affected. The anatomical distribution of the lesions is shown in Table XII. The upper limit of the process was usually the proximal jejunum commencing a few centimetres from the duodeno-jejunal junction. However at operation it was sometimes difficult to determine whether the duodenum was affected, particularly in less severe instances.

In establishing the direct cause of death, difficulty was found in separating shock, toxemia, post-operative collapse and haemorrhage. Perforation and peritonitis probably accounted for most deaths. Intestinal obstruction and dehydration followed next. Shock from haemorrhage and toxemia were frequent associations. Acute renal failure due to tubular necrosis was histologically confirmed on four occasions. Post-operative mortality resulted from anastomotic break-down, perforations beyond the anastomosis and cardiac arrest at
Figure 56: Distribution of intestine affected in 97 cases of pig-bel - 24 measured at autopsy and 73 at laparotomy.
operation or in the early post-operative period. Other deaths were
the result of pneumonia, gastro-intestinal haemorrhage and cerebral
malaria.

The shortest illness in a subject on which autopsy was performed
was on a 4 year old female child who died within 48 hours of the
onset of symptoms (Case 14). There was some free fluid in the
peritoneal cavity, the liver showed some pale areas on both the
capsular and cut surface, but other organs apart from the bowel
appeared normal. Commencing abruptly 10 cm. distal to the ligament
of Treits, there were small areas of bright and dark red discoloration
of the serosal surface of the intestine. The lesions were 2 to 6 cm.
in diameter and extended for 100 cm. The intervening bowel was
swollen and thickened with oedema and small emphysematous bullae were
seen along the mesenteric border. The mesentery and lymph nodes were
swollen but did not show any oedema. The lower jejunum, ileum
and colon all appeared normal, as were the stomach and duodenum.

The mucosal surface of the small intestine was dotted with dark
spots but otherwise the mucosal folds maintained their shape and form.
The mucosa was stained yellow and the darker areas contained small
pinpoint ulcers in the centre. The contents were blood stained and
muddy in the upper jejunum but in the lower small bowel the liquid
faeces were black and offensive. Gram stains revealed an abundance
of long thick Gram-positive rods and a few cocci and Gram-negative
bacilli. Microscopic examination showed a coagulative necrosis of
the mucosa and submucosa with clumps of bacilli coating some of the villi. There was a slight polymorph invasion of the submucosa and the necrosis was confined to the mucosa. Early fibrinous exudation with marked distension of the peripheral vessels was present. Thickening of the intimal layer of submucosal arteries with eosinophilic fibrinoid degeneration and early thrombus formation in the lumen of these vessels had occurred. In a few arterioles there was apparent disruption of the continuity of the wall and surrounding haemorrhage could be seen. There was also a considerable amount of haemosiderin pigment scattered throughout all layers (Figure 39).

The mesenteric lymph glands showed a few cystic spaces and polymorph infiltration but no focal necrotic areas (Figure 40). Some of these spaces contained lakes of bluish mucinous material. The histological sections of liver, kidney, spleen and adrenals showed no abnormalities although some autolysis had taken place. The autopsy had been performed 12 hours after death.

Small intestine examined from cases on the fourth and fifth days of illness showed that the distension had increased, the reddish haemorrhagic areas on the serosal surface were darker and more purplish, and the intervening areas were of a pale grey colour (Figure 41). The wall was stiffer, thicker and became alternately striped with dark violet red and pale grey or white stripes 1 to 9 cm. wide. Changes again were confined to the mid and upper jejunum. At this stage the peritoneal exudate was usually quite blood stained. Fine white
Figure 39: Section of lower jejunum from autopsy of a male aged 12 years. Shows coagulative necrosis of mucosa and submucosa with a mild acute inflammatory infiltration (4th day).
H & E x 60

Figure 40: Section of lymph gland showing gaseous cysts near surface from same case as above.
H & E x 60
Figure 41: Serosal surface appearances of jejunum and ileum in an acute toxic case of pig-bel (Case 79, female aged 22). Autopsy specimen removed two hours post-mortem.
fibrinous strings formed on the serosal surface, the bowel loops
starting to adhere together causing kinking. The mesentary was
cedematous and the lymph glands took on a deeper shade of red, gas
cysts being much more noticeable under the gland capsules. Larger
ecchymotic areas were also present over the mesentery and along the
bowel mesenteric border where surgical emphysema was most marked.
The mucosal surface of the jejunum, and sometimes duodenum, contained
hemorrhagic areas surrounding blackened pinpoint ulcers. Intervening
mucosa was thrown into rigid folds, stained yellow by bile and was
quite adherent to the thickened submucosal tissues (Figure 42). From
their pinpoint beginning, the ulcers enlarged into punched out
hemorrhagic craters 2 to 10 mm. in diameter. Figures 43, 44 and 45
show the progressive appearances of the jejunum during the development
of the necrosis. The bowel when palpated was crepitant, as were the
lymph glands. There were no specific features in the macroscopic
appearance of other organs examined during the early stages of the
disease.

An acute non-specific inflammatory reaction dominated the micro-
scopical appearances of the lamina propria and submucosal layers of the
bowel wall after the third day. Polymorphs and large mononuclear
macrophages with some eosinophils featured in this response. In Gram
stained sections, thick Gram-positive bacilli were seen as deep as the
outer muscle coat.

Vessels in the submucosa showed necrosis of the arteries and
Segment of opened upper jejunum from Case 82 (male aged 4 years). The intact wall at the left shows white "infarct" areas. Autopsy specimen, removed six hours after death. Collection of gas can be seen along the mesenteric attachment.
Dark punched out ulcers in the mucosal rugae of the jejunum. The patient died on the fourth day of illness and an autopsy was performed (Case 103, female 42 years).

Figure 44: Opened jejunum with sloughing mucosa and prominent lymphoid areas. The appearances at autopsy are shown in Figure 45.

Figure 45: Gross appearances of jejunum and ileum at autopsy in a female aged 8 years. The child had been ill for 6 days, and the autopsy performed 4 hours after death. The peritoneal fluid is sero-sanguinous and slightly turbid. There are patches of haemorrhagic and avascular necrosis with distension and adherence of bowel loops throughout the jejunum and upper ileum. Gas bubbles can be seen between the loops. Cause of death was an overwhelming clostridial toxæmia.
distension of the veins. The walls of the small and middle sized arteries had hyaline thickening. Where avascular necrosis had taken place from a more proximal thrombosis, these and surrounding tissues were necrotic. Hyaline thrombi were present in arteries with some preservation of nuclear outline and enlarged veins in the deeper sub-mucosal layer contained fresh thrombi formed by platelet agglutination (Figures 46 and 47). Until the fourth day the inflammatory reaction was not severe. The histopathological findings suggested that the initial necrosis was due to absorbed toxin, then ischaemia from multiple arterial and venous thromboses.

Figures 1 and 45 show in colour the appearance of the small intestine on the fifth to seventh days in an acute case. Classically transversely arranged yellow patches appeared which were surrounded by a purple and red rim of congestion. These were most apparent along the anti-mesenteric border. Emphysematous bullae were again prominent along the mesenteric border and in the lymph glands, which were crepitant on palpation. Gas bubbles were sometimes seen in the mesenteric veins (Figure 46).

The now extensive patchy and haemorrhagic areas became confluent and complete loop necrosis occurred. The appearance was the same as in bowel strangulation. Pulsation of the terminal vessels at laparotomy and dissection at autopsy excluded mesenteric artery occlusion as a cause for this. Fibrinous exudate was present over the whole serosa especially where perforations were imminent, loops
Figure 46: Severe necrosis, acute inflammatory infiltration and emphysema of submucosa. Dilated veins and arteries containing fresh thrombi. There is a lacework of cystic spaces, some containing hyaline material. (Case 15, female aged 6 years — resected jejunum.) H. & E. x 60.

Figure 47: Two submucosal vessels showing advanced and organized thrombus with surrounding polymorphs and haemorrhages (Case 196, female aged 10 years — resected jejunum.) H. & E. x 700.
Figure 48:
Gas bubbles seen in the distal arches of the mesenteric veins in Case 134.

Figure 49:
"Washboard" mucosal appearance in less acute disease seen on the 9th day. Hookworms still clinging to mucosa (Case 198 - resected jejunum).

Figure 50:
A more protracted case of healing stage with chronic ulcerative jejunitis and associated round worm (A. lumbricoides) infestation (Case 25).
adhering together. The peritoneum contained 1000 to 2000 ml. of 
sero-sanguinous fluid which on Gram stain showed a mixed flora of 
Gram-positive and negative rods. Other organs showed no specific 
abnormality.

The microscopic appearances showed a progression of the necrosis 
of the mucosa, surface and submucosal colonies of Gram-positive and 
negative bacilli and a dense infiltration with polymorphs. The 
submucosa was distended with empty cystic spaces, some filled with 
blood. The necrosis had extended through to the serosa where 
perforation was imminent. Polymorph infiltration had extended to the 
muscle coat on other areas. Hyperaemia, acute ulceration with 
separation of the mucosa and supplicative peritonitis became manifest. 
The submucosa was enormously thickened by the inflammatory and 
fibrinous exudate. In one-third of the histological examinations 
there was an accompanying eosinophilic infiltration in the sections. 
This may have been due to associated helminthic infestation (Figures 
49 and 50).

"Skip" lesions were a feature of the less severe forms of the 
disease. Perforation was rare before the seventh day of illness and 
by this time extensive fibrin deposits on the serosal surface between 
loops had formed causing adherence and further mechanical obstruction.

During the next seven days, in persons who survived, shreds of 
necrotic mucosa separated, the luminal surface took on a velvety 
appearance (Figure 51) and the process of healing became evident.
Figure 51: Mucosal appearances of bowel removed between the tenth and fourteenth days. Necrotic membranes is separating with chronic granulation and mucosal cell regenerative tissue apparent (Case 154, male aged 22 years).
Where strangulation was incomplete, regeneration began from the residual elements restoring the continuity of the wall. Between the ulcerated areas the intervening mucosa was blunted and flattened with clubbed villi. The ulcers were deep and indurated and consisted of very vascular granulation tissue densely infiltrated with plasma cells and lymphocytes. The oedema subsided, but the bowel wall became more rigid and stenosed. Bowel loops were stuck firmly together by peritoneal fibrin threads. In persons undergoing laparotomy at this stage the affected loops were usually shut off from the rest of the peritoneal cavity and covered by omentum. Multiple perforations were covered with omentum, fibrin or an adjacent bowel surface and sudden perforation with faecal peritoneal spilling often followed.

Spread of the necrotizing process reached a limit by about the seventh day. The process of healing was further established in the second and third weeks after the initial acute inflammatory reaction (Figure 52). The results and sequelae of this healing were directly proportional to the severity and depth of the initial necrosis. Collections of histiocytes, although apparent after the fifth day, became prolific in the second week. Necrotic material and blood pigment were removed by large numbers of pigment filled macrophages. New capillaries grew into the necrosed areas and thrombosed vessels recanalised in this reparative process. This commenced in some instances on the longitudinal muscle surfaces and in others on the
<table>
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<th>No. Cases</th>
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<th>Died</th>
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<td>3</td>
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<td>3</td>
<td>2</td>
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<td>4</td>
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<tr>
<td>Sub-acute strangulation by adhesions</td>
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<td>6</td>
<td>3</td>
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<td>Single stenotic area</td>
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<td>5</td>
<td>0</td>
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<td>Entero-enteric fistula</td>
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<td>4</td>
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<td><strong>31</strong></td>
<td><strong>18</strong></td>
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</table>
submucosa deep to the muscularis mucosae. Upon the scaffold of
collapsed blood, fibrin and inflammatory exudate, the fibroblasts
and vascular endothelium built up granulation tissue. Where
necrosis was confined to the mucosa only, islands of newly formed
villi, usually flattened and clubbed, appeared after the second
week. The early polymorphonuclear leucocytes gave place in the
deeper layers to macrophages, lymphocytes, plasma cells and
eosinophils. After the third week the fibroblastic proliferation
at the base of this granulation tissue had left segments of bowel
which were rigid, sometimes twisted and kinked and adherent to other
segments.

In Table IX are listed the complicated cases observed by and
reported to the author in 39 sub-acute and chronic forms of the
disease. The "lead pipe" and ulcerative jejunitis state was most
frequently observed, then followed examples of stenosis, strangulation
and perforation.

Three cases are particularly noteworthy in that they represent
some of the more unusual complications of the protracted disease.
Case 47, a male aged about 12 years, was transferred from Chinese
hospital following a protracted illness after pig feasting five weeks
earlier. His initial symptoms were bloody diarrhoea, intermittent
upper abdominal cramps and the vomiting of dead roundworms. This
settled down but for the next three weeks he suffered post-prandial
upper abdominal colic, anorexia, weakness, weight loss and intermittent
Figure 52:
The microscopic appearances in the second week showing (1) layers of necrotic mucosa and debris; (2) thick layer of pus, polymorphs and eosinophils; (3) vascular granulation tissue with lymphocytic and macrophage infiltration, particularly around vessels; (4) necrotic inner longitudinal muscle, and (5) intact outer circular muscle layer. (Case 84 - resected jejunum.) H & E x 60

Figure 53:
Histological section of cyst and luminal portion of bowel from case shown in Figure 54. H & E x 170.
bouts of diarrhoea and constipation. A small bowel X-ray showed a sub-acute bowel obstruction and at laparotomy 150 cm. of thickened upper jejunum were removed. The mucosal surface was narrowed by fibrous strictures in several areas, the wall containing numerous cystic spaces surrounded by fibrous strands. The wall was considerably thickened and normal mucosa in some sections separated large necrotic ulcers with sloughing floors and hard indurated bases. The "honeycomb" appearance of the bowel shown in Figure 54 resembled the secondary form of "pneumatosis cystoides intestinalis". This intestinal emphysema was commonly seen in the primary form in pigs in the Highlands (Egerton and Murrell, 1965). Koss (1952) reviewed this condition and described both primary and secondary types of the disease. A microscopic section of the wall of one of these cysts and the necrotic granulating luminal surface of the bowel is shown in Figure 53. The patient made a good recovery but when seen 18 months later had steatorrhaea, a macrocytic anaemia, and a gastric biopsy showed a haemorrhagic atrophic gastritis.

Case 54, a male aged 8 years, was admitted in September 1963 with a three week history of recurrent abdominal pain, diarrhoea, severe anorexia and cachexia. Conservative treatment failed to alleviate his symptoms and at laparotomy a further three weeks later the whole small bowel except for the distal 50 cm. was a conglomerate adherent mass with multiple internal fistulae. Resection was attempted but the patient died during the operation. Figure 55 shows a sagitally
Figure 54: The jejunal appearances after a 5 to 6 week illness in a male aged 12 years (Case 47). There were multiple subserosal and submucosal gas cysts between ulcerated and stenosed areas of bowel wall.
Figure 55: Adherent conglomerate small bowel mass in chronic case of pig-bel (Case 54, male aged 8 years). Magnification x 1.5.
Specimen shows macroscopic appearance of Crohn's disease. The histology failed to show this.
Figure 56: Cisatrised bowel with a developing entero-enteric fistula indicated by the arrows (Case 96).
cut segment of the resected small bowel mass after fixation. The microscopic appearances showed large areas of bowel wall partly replaced by old granulation tissue, particularly in the subserosal layer. The granulation tissue was oedematous and infiltrated with plasma cells, and there were extensive formations of connective tissue with intercommunicating spaces between previously covered perforations of bowel.

Case 96, a male aged between 25 and 30 years, was admitted with a protracted illness of five days' duration initially commencing with a dysenteric episode. Intermittent severe upper abdominal colic and melena brought him to hospital. Initially it was thought he had bacillary dysentery which was confirmed by the recovery of *Sh. flexneri* type 2 from his stool. After a further two weeks' treatment, pain continued and an upper abdominal tender mass and barium meal confirmed a more protracted form of pig-bel. At laparotomy on the 36th day of his illness upper jejunal loops were adherent to the duodenum and there was a fistulous communication between mid and upper jejunum (Figure 56). The histopathology showed partly intact mucous membranes, infiltrated with a few polymorphs amongst which were numerous eosinophils and plasma cells. There were an unusual number of goblet cell and villous blunting in non-ulcerated areas. The inflammatory infiltration extended throughout the muscle coat with fibroelastic proliferation and new capillary formation in the subserosal layers.

One finding also seen in sections from other cases was an
Table XXI: An approximate estimate of the disease incidence derived from hospital and reported cases from January 1961 to August 1964.

Population census figures those of 1960.

<table>
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<tr>
<th>Year</th>
<th>Place</th>
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<th>Cases</th>
<th>Incidence (per 10,000)</th>
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<td>Upper Asaro</td>
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<td>22</td>
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<td>1962</td>
<td>Baiyer River</td>
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<tr>
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<td>Bundi</td>
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<td></td>
<td>Upper Chimbu</td>
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<td>1964</td>
<td>Central Chimbu</td>
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<td>TOTAL</td>
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Table XIII: Census figures taken in October 1964 at Goromango. Incidence of pig-bel was estimated at 4.8,1 per 10,000 and mortality at 13,1 per 10,000.

<table>
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<td>123 108</td>
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<td>Kumbuglkane</td>
<td>27 44</td>
<td>71 43</td>
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<tr>
<td><strong>TOTAL</strong></td>
<td><strong>265 285</strong></td>
<td><strong>489 399</strong></td>
</tr>
</tbody>
</table>
associated villous flattening between ulcerated areas in histopathological sections from protracted cases. Whether this was antecedent to or resultant from the necrosis could not be determined. It was also noted that the regenerating epithelium was flat, similar to that seen in sprue and celiac disease.

10. Epidemiological Studies

(a) Incidence of Pig-bel in General Population

An approximate estimate of the incidence of enteritis necroticans was made by equating hospital admissions of known cases with population census figures of 1960 in areas where the disease was known to occur. In Table XXI five such cases are listed. The overall incidence was found to be 15 per 10,000. The incidence ranged from 10 per 10,000 in the Chikwanda to 23 per 10,000 at Bandi.

A more accurate assessment of the incidence of the disease was made from census figures taken of five clans at the Government Rest House at Goromugo (Upper Chikwanda - Figure 4) in October of 1964 (Biscoe, 1964). The total population, including absentees, was 1,448 (Table XIII). Following pork feasting in May and August of the same year, seven persons were known to have contracted pig-bel. The diagnosis was established bacteriologically in three cases, serologically in two and by autopsy in a further two. The incidence in this area was therefore assessed at 48.3 per 10,000. This figure could be regarded as unusually high, and Goromugo was selected with
Table XXIII: Reported cases of pig-bel in Highland districts 1961-1964 inclusive.

<table>
<thead>
<tr>
<th>Year</th>
<th>Cases</th>
<th>Deaths</th>
<th>Per cent</th>
<th>District Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>1961</td>
<td>63</td>
<td>24</td>
<td>38.1</td>
<td>46</td>
</tr>
<tr>
<td>1962</td>
<td>39</td>
<td>16</td>
<td>41.0</td>
<td>20</td>
</tr>
<tr>
<td>1963</td>
<td>25</td>
<td>13</td>
<td>52.0</td>
<td>10</td>
</tr>
<tr>
<td>1964</td>
<td>90</td>
<td>27</td>
<td>30.0</td>
<td>67</td>
</tr>
<tr>
<td>TOTAL</td>
<td>220</td>
<td>80</td>
<td>36.4</td>
<td>146</td>
</tr>
</tbody>
</table>
bias because of evidence produced later in the text, supporting a
spread of the disease by pork prepared there. A high prevalence of
Clostridium perfringens beta antitoxin in population groups sampled in the
Upper Chiru supported the impression that Carumsungo represented a
high incidence area.

(b) Outbreaks and Geographical Distribution

An outbreak of pig-bel was arbitrarily defined as the appearance
of three or more cases in any area over a period of a month. Such
an event does not infer a food poisoning outbreak in the normally
accepted sense. The geographical distribution of cases over the four
years January 1st 1961 to November 31st 1964 is shown in Figure 2 and
Table XXIII. Epidemics occurred in the Upper Asaro during the months
June to September of 1961; in the Tari Basin just prior to this;
mess of Beiyer River in the Western Highlands in March to June 1962
and in Bundi and the Chiru throughout 1964. The largest outbreak
occurred around Bundi on the northern slopes of the Central Divide in
the Madang District early in 1964. Three children died of the
disease at the Mission School, and baptismal records indicated that
at least 10 other children died in the surrounding villages (Morrison,
1964). Autopsies on the children, performed by Dr. L. Malcolm,
confirmed the diagnosis. It is possible that the disease reached
Bundi via trade ramifications with tribes in the Upper Asaro. A
dysentery outbreak at Gembogl in the Upper Chiru, in which Sh.
flexneri was recovered, coincided with the Bundi outbreaks of pig-bel.
Subsequently the dysentery spread to the Lower Chimbu but only two cases of pig-bel were encountered in association with this epidemic.

When pig feasting activities commenced in May in the Upper Chimbu, the prevalence of enteritis macroicama rose (Figure 57). There was a further increase following the pig cycles in the Central Chimbu (Dem, Kup and Kainigl Census Divisions, Figure 4). Following the "To" festival of the Enga people of the Western Highlands late in 1963, only six cases of the disease were reported. Sero logical sampling earlier in the year confirmed the impression that the Wabag-Wapenamanda area was a low incidence area.

*Clostridium perfringens* type C was isolated from cases at the Upper Asaro, Chimbu, Baiyer River, Wabag, Wapenamanda and Mendi. Bacteriological examinations from cases elsewhere at Bumdi, Kaimantu, Henganofi, Tari and Minj were not undertaken, the diagnosis being established only on the morbid anatomy of the small intestine at operation or autopsy. In retrospect, several medical officers with longer experience than the author have indicated their familiarity with the autopsy appearances of cases. Ivinskas and Malcolm saw cases as early as 1954 in the Chimbu and 1957 in Mendi.

A specific focus or geographical location of the disease cannot be defined and it seems likely that the affection has always been endemic in New Guinea and that recognition and detection of the cause was not made until 1961 - 1962. Whether *Clostridium perfringens* type C has been introduced with the advent of civilization remains open to
speculation.

(c) **Evidence of Local Spread**

During 1964, seven cases of pig-bel were detected from clans near Goromango in the Upper Chibvu (Nos. 91 to 93, 97, 99, 101 and 155). *Cl. perfringens* type C was recovered from three of these, significant beta antitoxin was found in two others and the diagnosis established at autopsy in two more.

Following the pig killing at Goromango there was a bridal exchange of pork between members of a Pagakama line and one man, Ambane Umba (Case 96), of a Kurumogi line. Ambane subsequently developed a more protracted form of the disease which was confirmed by a rising beta antitoxin and recovery of *Cl. perfringens* type C from his faeces. Sera taken from eight relatives exposed to the same meal and from the man who had prepared the meal all contained significant amounts of beta antitoxin.

A further case (No. 100) occurred in a man at Gere (Upper Chibvu) who had eaten pork originating from a line near Goromango. Contacts again had immunological evidence of exposure to beta toxin. A "lead pipe" piece of jejunum, denuded of normal mucosa, was removed at laparotomy on the 24th day. The patient also had a rise and fall in beta antitoxin.

Further evidence of local spread became manifest in August when pork exchanges between clans at Mai and Goromango resulted in two further acute cases (Nos. 150 and 155). Autopsy appearances in the
Table XXIV: Admissions to the Kundisawa hospital of persons with diarrhoeal diseases classified under categories 045-049, 571 and 785-6 and persons with pig-bel for period 1.12.63 to 30.11.64.

<table>
<thead>
<tr>
<th>Month</th>
<th>Total Admissions</th>
<th>Diarrhoeal Diseases</th>
<th>% Total</th>
<th>Cases of pig-bel</th>
</tr>
</thead>
<tbody>
<tr>
<td>December 1963</td>
<td>333</td>
<td>34</td>
<td>10.2</td>
<td>5</td>
</tr>
<tr>
<td>January 1964</td>
<td>457</td>
<td>89</td>
<td>19.5</td>
<td>1</td>
</tr>
<tr>
<td>February 1964</td>
<td>374</td>
<td>61</td>
<td>16.3</td>
<td>2</td>
</tr>
<tr>
<td>March 1964</td>
<td>459</td>
<td>59</td>
<td>12.9</td>
<td>2</td>
</tr>
<tr>
<td>April 1964</td>
<td>757</td>
<td>73</td>
<td>9.6</td>
<td>0</td>
</tr>
<tr>
<td>May 1964</td>
<td>321</td>
<td>93</td>
<td>30.6</td>
<td>12</td>
</tr>
<tr>
<td>June 1964</td>
<td>455</td>
<td>94</td>
<td>20.7</td>
<td>7</td>
</tr>
<tr>
<td>July 1964</td>
<td>407</td>
<td>36</td>
<td>8.8</td>
<td>4</td>
</tr>
<tr>
<td>August 1964</td>
<td>379</td>
<td>40</td>
<td>10.6</td>
<td>9</td>
</tr>
<tr>
<td>September 1964</td>
<td>310</td>
<td>45</td>
<td>14.5</td>
<td>10</td>
</tr>
<tr>
<td>October 1964</td>
<td>374</td>
<td>58</td>
<td>15.5</td>
<td>12</td>
</tr>
<tr>
<td>November 1964</td>
<td>303</td>
<td>42</td>
<td>13.9</td>
<td>7</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>4929</strong></td>
<td><strong>724</strong></td>
<td><strong>14.7</strong></td>
<td><strong>71</strong></td>
</tr>
</tbody>
</table>
Figure 57: Pig-bul admission numbers at the Bundiasa Hospital related to admissions for diarrhoeal diseases. The pigs represent pig-feasting episodes.
former case are shown in Figure 1 which prefaces the introduction.
The disease was firmly diagnosed three times in members of one family
(Nos. 89 and 90, 92 and 93, 132 and 134). A dietary history of
persons consuming the same pork meal was recorded on 42 occasions
and in only 17 instances (38 per cent) were mild symptoms of food
poisoning recorded in persons at risk.

(d) Hospital Admissions

At the Kundima hospital for a 12 month period 1st December 1963
to 30th November 1964, admissions for diarrhoea conditions under
categories 045 - 049, 571 and 795.6 of the W.H.O. International
Disease classification, were reviewed. These results are shown in
Table XXIV. There were also 71 (1.4 per cent) admissions for mild
and severe forms of pig-bel. The two sets of figures are shown
together in Figure 57. Peaks in admissions for the disease
corresponded to the maximal rates for the diarrhoeal disease admissions
except in December 1963. These periods were preceded by periods of
heavy pork consumption in areas within two hours walk of the hospital.
The relative rise in diarrhoeal diseases treated as compared with
other disease in May and September-October was statistically
significant. This significance was not as great from August to
September when the pig-killing moved first to Cembeig and then to
the Central Chimbu.

Operative records analysed for almost a four year period (1st
February 1961 to 30th November 1964) at the Gereka hospital
**Table XXV:** Acute and sub-acute abdominal conditions for which laparotomy was undertaken at Goroka Hospital for period 1.2.61 to 30.11.64

<table>
<thead>
<tr>
<th>Condition</th>
<th>No. of Cases</th>
<th>Condition</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sig-bel:</strong></td>
<td></td>
<td>Abdominal traumas:</td>
<td></td>
</tr>
<tr>
<td>Acute</td>
<td>40</td>
<td>Ruptured spleen</td>
<td>9</td>
</tr>
<tr>
<td>Sub-acute</td>
<td>29</td>
<td>Bowel rupture or penetration</td>
<td>21</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ruptured kidney</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ruptured bladder (intraperitoneal)</td>
<td>1</td>
</tr>
<tr>
<td><strong>Strangulation:</strong></td>
<td></td>
<td>Peptic ulcer:</td>
<td></td>
</tr>
<tr>
<td>A. By adhesions -</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post-inflammatory</td>
<td>18</td>
<td>Stenosis</td>
<td>11</td>
</tr>
<tr>
<td>Post-operative</td>
<td>8</td>
<td>Rupture</td>
<td>9</td>
</tr>
<tr>
<td>Congenital</td>
<td>7</td>
<td>Bleeding</td>
<td>6</td>
</tr>
<tr>
<td>B. By internal hernia</td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C. Volvulus</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Appendicitis:</strong></td>
<td></td>
<td>Large bowel obstruction:</td>
<td></td>
</tr>
<tr>
<td>Indigenous</td>
<td>16</td>
<td>Carcinoma</td>
<td>14</td>
</tr>
<tr>
<td>European</td>
<td>26</td>
<td>Adhesions or stenosis</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>18</td>
</tr>
<tr>
<td><strong>Peritonitis:</strong></td>
<td></td>
<td>Ectopic pregnancy:</td>
<td></td>
</tr>
<tr>
<td>Ruptured pyosalpinx</td>
<td>9</td>
<td>Paralytic ileus:</td>
<td></td>
</tr>
<tr>
<td>Perforated colon</td>
<td>5</td>
<td>Idiopathic</td>
<td>5</td>
</tr>
<tr>
<td>Ascaris perforation</td>
<td>4</td>
<td>Neonatal</td>
<td>4</td>
</tr>
<tr>
<td>Puerperal (excluding ruptured uterus)</td>
<td>6</td>
<td>Twisted ovarian cyst:</td>
<td>8</td>
</tr>
<tr>
<td>Systemic</td>
<td>8</td>
<td>Idiopathic gastrointestinal bleeding:</td>
<td>3</td>
</tr>
<tr>
<td>Sub-phrenic</td>
<td>4</td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>Post-dysenteric</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tuberculous</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TOTAL</td>
<td>301</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
emphasised the importance of enteritis necroticans as a surgical
disease in the Highlands. The conditions are listed in Table XXV.

Laparotomy for acute and subacute intra-abdominal conditions
was undertaken on 301 occasions during this period. Sixty-nine
patients (22.6 per cent) had acute and complicated forms of pig-bel
and this was the largest group requiring surgical exploration of
the abdomen. When acute and subacute surgical conditions of the
gastro-intestinal tract only were analysed, excluding trauma, the
frequency rose to 31.2 per cent. As a surgical disease, therefore,
pig-bel was the most common cause of an "acute abdomen". It is
possible also that some of the causes of strangulation by post-
inflammatory bands, listed in the second group of Table XXV, were
also sequelae of the disease. The experience of other medical
officers in Highland hospitals supports the experience of the
surgeons at Goroka (Burchett and Roderigue, 1964 - personal
communication).

(e) Seasonal Distribution

In Figure 58 admissions to hospitals in the Highlands and Madang
from enteritis necroticans are grouped at three-monthly intervals.
Peaks occurred mostly in the "dry" season between the months of
April and September. Major epidemics coincided with the larger pig-
killing ceremonies held at this time of the year because of the
prevailing climate and good harvest.

The relationship of pig-bel and epidemics due to other diseases
Figure 58: Seasonal distribution of reported cases of pig-bel from January 1961 to December 1964. Pig feasting episodes are shown along the top of the histogram.
was not investigated in detail. A widespread influenza epidemic preceded the commencement of pig-killing in the Upper Chimbu in 1964 and a measles epidemic was known to occur from October to December of 1962 in the Western Highlands. Bocraft (1962) in a personal communication noticed that a gastro-enteritis epidemic was prevalent prior to the pig-killing near Baiyer River in April 1962.

The disease was most prevalent during the months of June and August 1961 in the Upper Asaro, in April and May of 1962 (Baiyer River), the Upper Chimbu in May and June 1964 and the Central Chimbu later that same year. The seasonal prevalence of all the infective diarrhoeal diseases shows similar trends in the drier months of the year.

(f) Race, Age and Sex Incidence

Of the 210 persons reviewed in this series, only one was a European. All other persons, with one exception, were inhabitants from areas where they contracted the disease. The exception was a Mount Hagen native working as a plantation labourer on Kar Kar Island, in the Madang District. The European, a 28 year old Polish linesman, contracted his disease at Tari following a meal of native pork.

Ages and sexes are listed in Table XXVI, trends being shown diagrammatically in Figures 59 and 60. The greatest number of patients were 2 to 10 years old (52.3 per cent). Children in the 6 to 10 year group made up the largest segment of overall cases
<table>
<thead>
<tr>
<th>Age Group</th>
<th>Males</th>
<th>%</th>
<th>Females</th>
<th>%</th>
<th>Total</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 - 1</td>
<td>1</td>
<td>0.4</td>
<td>2</td>
<td>1.1</td>
<td>3</td>
<td>1.4</td>
</tr>
<tr>
<td>2 - 5</td>
<td>36</td>
<td>17.1</td>
<td>13</td>
<td>6.2</td>
<td>49</td>
<td>23.3</td>
</tr>
<tr>
<td>6 - 10</td>
<td>46</td>
<td>21.9</td>
<td>15</td>
<td>7.1</td>
<td>61</td>
<td>29.0</td>
</tr>
<tr>
<td>11 - 15</td>
<td>17</td>
<td>8.1</td>
<td>5</td>
<td>2.4</td>
<td>22</td>
<td>10.5</td>
</tr>
<tr>
<td>16 - 20</td>
<td>10</td>
<td>4.8</td>
<td>12</td>
<td>5.7</td>
<td>22</td>
<td>10.5</td>
</tr>
<tr>
<td>21 - 30</td>
<td>13</td>
<td>6.2</td>
<td>8</td>
<td>3.8</td>
<td>21</td>
<td>10.0</td>
</tr>
<tr>
<td>31 - 40</td>
<td>12</td>
<td>5.7</td>
<td>7</td>
<td>3.3</td>
<td>19</td>
<td>9.0</td>
</tr>
<tr>
<td>41+</td>
<td>10</td>
<td>4.8</td>
<td>3</td>
<td>1.4</td>
<td>13</td>
<td>6.2</td>
</tr>
<tr>
<td>TOTAL</td>
<td>145</td>
<td>69.0</td>
<td>65</td>
<td>31.0</td>
<td>210</td>
<td>99.9</td>
</tr>
</tbody>
</table>
Figure 59: Age distribution of 210 cases of pig-bel. The relative age mortality is also shown.
### Table XXVII: Age and sex mortality rates in 210 cases

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Males</th>
<th>% Total</th>
<th>Age Mortality</th>
<th>Females</th>
<th>% Total</th>
<th>Age Mortality</th>
<th>Total</th>
<th>% Total</th>
<th>Age Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 - 1</td>
<td>1</td>
<td>0.5</td>
<td>100.0</td>
<td>1</td>
<td>0.5</td>
<td>50.0</td>
<td>2</td>
<td>1.0</td>
<td>66.7</td>
</tr>
<tr>
<td>2 - 5</td>
<td>16</td>
<td>7.6</td>
<td>44.4</td>
<td>12</td>
<td>5.7</td>
<td>92.3</td>
<td>28</td>
<td>13.3</td>
<td>57.1</td>
</tr>
<tr>
<td>6 - 10</td>
<td>16</td>
<td>7.6</td>
<td>34.8</td>
<td>3</td>
<td>1.4</td>
<td>20.0</td>
<td>19</td>
<td>9.0</td>
<td>31.1</td>
</tr>
<tr>
<td>11 - 15</td>
<td>4</td>
<td>1.9</td>
<td>23.5</td>
<td>2</td>
<td>1.0</td>
<td>40.0</td>
<td>6</td>
<td>2.8</td>
<td>9.1</td>
</tr>
<tr>
<td>16 - 20</td>
<td>1</td>
<td>0.5</td>
<td>10.0</td>
<td>2</td>
<td>1.0</td>
<td>16.6</td>
<td>3</td>
<td>1.4</td>
<td>13.6</td>
</tr>
<tr>
<td>21 - 30</td>
<td>4</td>
<td>1.9</td>
<td>30.8</td>
<td>2</td>
<td>1.0</td>
<td>25.0</td>
<td>6</td>
<td>2.8</td>
<td>28.6</td>
</tr>
<tr>
<td>31 - 40</td>
<td>4</td>
<td>1.9</td>
<td>33.3</td>
<td>2</td>
<td>1.0</td>
<td>28.5</td>
<td>6</td>
<td>2.8</td>
<td>31.6</td>
</tr>
<tr>
<td>41+</td>
<td>5</td>
<td>2.4</td>
<td>50.0</td>
<td>1</td>
<td>0.5</td>
<td>33.3</td>
<td>6</td>
<td>2.8</td>
<td>46.2</td>
</tr>
<tr>
<td>TOTAL</td>
<td>51</td>
<td>24.3</td>
<td>35.2</td>
<td>25</td>
<td>11.9</td>
<td>38.5</td>
<td>76</td>
<td>36.0</td>
<td></td>
</tr>
</tbody>
</table>
Figure 62: Sex distribution of 210 cases of pig-kid.
(29.0 per cent). This distribution of persons with the disease under 10 years of age was most significant, indicating that the affliction primarily affected pre-adolescent children. Males were affected more than females in the ratio of 2.2:1. This ratio was generally maintained in all age groups except in the 16 to 20 year group when more females were recorded with the disease. The numbers recorded in this group, however, were too small to be significant.

(g) Mortality

Two of the seven persons known to have contracted pig-bel during 1964 at Gerumago died. The overall mortality was estimated therefore at 13.1 per 10,000 population.

Of the persons known to have suffered from mild and severe types of the disease, the overall death rate was 36.0 per cent. In the severe forms of the disease the case mortality was 49.8 per cent. This was highest in the acute toxic group (84.6 per cent). The mortality rose in the younger and older age groups (Table XXVII and Figure 60). The rate was 57.7 per cent in children under five years of age and 46.2 per cent in persons over 40 years of age. The lowest death rate occurred in the 11 to 15 year group. The overall rate for females was slightly higher (38.5 per cent) than that for males (35.2 per cent).

For all ages, enteritis necroticans accounted for 2.1 per cent of deaths at the Goroka and Kundina hospitals and 3.5 per cent at the Baiyer River hospital during the period April 1st 1962 and
### Table XXVIII (a, b, c): Causes of death for diseases listed at three Highland hospitals

#### DEATHS IN VOKOMA HOSPITAL DURING THE PERIOD 1.4.66 - 30.9.66

<table>
<thead>
<tr>
<th>INTERNATIONAL CLASSIFICATION</th>
<th>ALL AGES</th>
<th>UNDER 1 YEAR</th>
<th>1 YEAR BUT UNDER 10 YEARS</th>
<th>12 YEARS AND OVER</th>
</tr>
</thead>
<tbody>
<tr>
<td>DISEASE GROUP CODE NUMBERS</td>
<td>no. of deaths</td>
<td>% of all deaths</td>
<td>% of age group</td>
<td>no. of deaths</td>
</tr>
<tr>
<td>All causes</td>
<td>436-0999</td>
<td>436</td>
<td>100.0</td>
<td>100.0</td>
</tr>
<tr>
<td>Pneumonia and respiratory disease</td>
<td>470-577,763</td>
<td>470</td>
<td>21.9</td>
<td>100.0</td>
</tr>
<tr>
<td>Gastro-intestinal, etc</td>
<td>440-440,571</td>
<td>440</td>
<td>5.0</td>
<td>100.0</td>
</tr>
<tr>
<td>Enteritis necrotica</td>
<td>550-583</td>
<td>550</td>
<td>4.2</td>
<td>100.0</td>
</tr>
<tr>
<td>Cirrhosis of liver, hepatitis and related diseases of the liver</td>
<td>580-582</td>
<td>580</td>
<td>3.7</td>
<td>100.0</td>
</tr>
<tr>
<td>Malnutrition</td>
<td>266,2206,6,772</td>
<td>266</td>
<td>3.3</td>
<td>100.0</td>
</tr>
<tr>
<td>Other causes</td>
<td>110-117</td>
<td>110</td>
<td>22.3</td>
<td>100.0</td>
</tr>
</tbody>
</table>

#### DEATHS IN KUNYOMA HOSPITAL DURING THE PERIOD 1.4.66 - 30.9.66

<table>
<thead>
<tr>
<th>INTERNATIONAL CLASSIFICATION</th>
<th>ALL AGES</th>
<th>UNDER 1 YEAR</th>
<th>1 YEAR BUT UNDER 10 YEARS</th>
<th>12 YEARS AND OVER</th>
</tr>
</thead>
<tbody>
<tr>
<td>DISEASE GROUP CODE NUMBERS</td>
<td>no. of deaths</td>
<td>% of all deaths</td>
<td>% of age group</td>
<td>no. of deaths</td>
</tr>
<tr>
<td>All causes</td>
<td>436-0999</td>
<td>436</td>
<td>100.0</td>
<td>100.0</td>
</tr>
<tr>
<td>Pneumonia and respiratory disease</td>
<td>470-577,763</td>
<td>470</td>
<td>21.9</td>
<td>100.0</td>
</tr>
<tr>
<td>Gastro-intestinal, etc</td>
<td>440-440,571</td>
<td>440</td>
<td>5.0</td>
<td>100.0</td>
</tr>
<tr>
<td>Enteritis necrotica</td>
<td>550-583</td>
<td>550</td>
<td>4.2</td>
<td>100.0</td>
</tr>
<tr>
<td>Cirrhosis of liver, hepatitis and related diseases of the liver</td>
<td>580-582</td>
<td>580</td>
<td>3.7</td>
<td>100.0</td>
</tr>
<tr>
<td>Malnutrition</td>
<td>266,2206,6,772</td>
<td>266</td>
<td>3.3</td>
<td>100.0</td>
</tr>
<tr>
<td>Other causes</td>
<td>110-117</td>
<td>110</td>
<td>22.3</td>
<td>100.0</td>
</tr>
</tbody>
</table>

#### DEATHS IN SAWAYA HOSPITAL DURING THE PERIOD 1.4.66 - 30.9.66

<table>
<thead>
<tr>
<th>INTERNATIONAL CLASSIFICATION</th>
<th>ALL AGES</th>
<th>UNDER 1 YEAR</th>
<th>1 YEAR BUT UNDER 10 YEARS</th>
<th>12 YEARS AND OVER</th>
</tr>
</thead>
<tbody>
<tr>
<td>DISEASE GROUP CODE NUMBERS</td>
<td>no. of deaths</td>
<td>% of all deaths</td>
<td>% of age group</td>
<td>no. of deaths</td>
</tr>
<tr>
<td>All causes</td>
<td>436-0999</td>
<td>436</td>
<td>100.0</td>
<td>100.0</td>
</tr>
<tr>
<td>Pneumonia and respiratory disease</td>
<td>470-577,763</td>
<td>470</td>
<td>21.9</td>
<td>100.0</td>
</tr>
<tr>
<td>Gastro-intestinal, etc</td>
<td>440-440,571</td>
<td>440</td>
<td>5.0</td>
<td>100.0</td>
</tr>
<tr>
<td>Enteritis necrotica</td>
<td>550-583</td>
<td>550</td>
<td>4.2</td>
<td>100.0</td>
</tr>
<tr>
<td>Cirrhosis of liver, hepatitis and related diseases of the liver</td>
<td>580-582</td>
<td>580</td>
<td>3.7</td>
<td>100.0</td>
</tr>
<tr>
<td>Malnutrition</td>
<td>266,2206,6,772</td>
<td>266</td>
<td>3.3</td>
<td>100.0</td>
</tr>
<tr>
<td>Other causes</td>
<td>110-117</td>
<td>110</td>
<td>22.3</td>
<td>100.0</td>
</tr>
</tbody>
</table>
August 31st 1964. The Kumiana period reviewed was slightly longer (June 1st 1962 to November 30 1964). These rates were significantly higher in the 1 to 12 year age group. At Baiyer River 16.1 per cent of deaths occurring in this age group were due to pig-bell. Deaths occurring from this disease and other diseases at these hospitals are listed in Table XVIIIb, b and c) for comparison. The enteritis necroticans group forms a considerable proportion of deaths from overall diarrhoeal disease in all age groups being 15.3 per cent at Goroka, 17.3 per cent at Kumiana and 26.7 per cent at Baiyer River.

(b) The Relationship of Pig-Feasting with Enteric Diseases

The number of persons treated for diarrhoeal diseases (Categories 045-048, 571 and 785.6 W.H.O.) for the period 1st April 1962 to 31st March 1964 at four Highland hospitals is shown in Table XXIX. These data were collated on the basis of a 1 in 3 sample of admission and discharge registers for hospitals at Mount Hagen, Wabag, Baiyer River and Tari. Figures for the Kumiana hospital for all admissions over a 12 month period (1st December 1963 to 30th November 1964) were previously shown in Table XXIV. These figures have been related to local pig-killing activities in Figures 57 and 61. The diagnosis of the category of diarrhoeal disease was made by qualified medical practitioners at these hospitals.

In all five hospitals there were elevations in the percentage of patients treated for acute diarrhoeal disease. Preceding each peak, large scale pig-killing had taken place in the vicinities of the
Table XXIX: Patients treated for conditions classified under categories 045-048, 571 and 785.6 in Mount Hagen, Wabag, Baiyer River and Tari hospitals between 1st April 1962 and 31st March 1964.

<table>
<thead>
<tr>
<th></th>
<th>Persons Treated</th>
<th>Percentage of All Persons Treated</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mount Hagen</td>
<td>Wabag</td>
</tr>
<tr>
<td>1.4.62 - 31.3.64</td>
<td>208</td>
<td>1076</td>
</tr>
<tr>
<td>Quarter 1 1962-63</td>
<td>44</td>
<td>352</td>
</tr>
<tr>
<td>Quarter 2 1962-63</td>
<td>132</td>
<td>100</td>
</tr>
<tr>
<td>Quarter 3 1962-63</td>
<td>60</td>
<td>196</td>
</tr>
<tr>
<td>Quarter 4 1962-63</td>
<td>44</td>
<td>44</td>
</tr>
<tr>
<td>Quarter 1 1963-64</td>
<td>188</td>
<td>65</td>
</tr>
<tr>
<td>Quarter 2 1963-64</td>
<td>168</td>
<td>52</td>
</tr>
<tr>
<td>Quarter 3 1963-64</td>
<td>124</td>
<td>184</td>
</tr>
<tr>
<td>Quarter 4 1963-64</td>
<td>72</td>
<td>84</td>
</tr>
</tbody>
</table>

(a) No figures available.
Figure 61: Diarrhoeal disease admissions expressed as a percentage of total admissions in four Highland hospitals related to pig feasting activities from April 1962 to March 1964.
Figure 62: Pigs tethered to rows of stakes at a "Te" exchange near Babag. During this phase of the festival the pigs were not slaughtered.

Figure 63: Pigs being cruelly clubbed to death in a ceremonial area of the Upper Chimbu.
hospitals under review.

(1) Pig-Feasting Investigations

(1) Description of a pig-kill

Preparations months in advance take place when long houses are built to accommodate residents and visitors round central courtyard clearings. Tables are also built in readiness to receive the pork slabs for distribution. In the Eastern Highlands two to four weeks before a large pig-kill, a smaller celebration takes place, the smaller pigs being sacrificed on such an occasion.

All large pig-killing ceremonies take place at the time of a full moon. Whenever possible they are held during favourable weather which is usually during the dry season. Dancing and singing festivities are held before the killing commences. In the Chimbu the pork preparation and distribution takes place over a period of three or four days, whereas only a little over a day is needed in the Huli and Enga areas. The description which follows is basically applicable to all groups except for the important difference just mentioned. Pig-kills were witnessed at Karfena (July 1964), Tontol and Wayemamia (September 1963) and the Upper Chimbu (May 1964). The reader is referred to Figures 3, 4 and 5 in Chapter II for the geographical location of these places.

The tethered pigs are clubbed to death by a near relative of the owner, four to five sharp blows to the head with a 2 to 3 inch diameter stick usually being sufficient to kill the animal (Figure 63).
There is no bleeding of the carcass although some blood from the head wounds spills out over the ground. Cassowaries, chickens and dogs are sometimes killed along with the pigs (Figure 65) but are cooked separately. The Chimbu people place the dead pigs in radiating lines from a central spirit or "Belim" house, rather like the spokes of a cartwheel, each spoke representing pigs to be distributed from one man’s house. The clan leaders then welcome visitors and incantate upon the significance of the occasion. After about two hours, the butchering starts. This was well under way by 10 a.m. in the Enga situation. The haste of Enga in preparing and cooking the pork is probably due to the longer distances over which exchanges must take place and the greater number of pigs slaughtered.

The hair is singed over an open fire (Figure 64) and the carcasses then lifted onto leaf mats of banana, breadfruit and tree fern leaves. An intricate dissection in the dorsal position is commenced with double lateral incisions in the anterior axillary line (Figure 66). The outermost continues down behind the anus and the inner two meet anterior to it. Here, spillage may occur and the butcher’s hands can become contaminated directly by pig faeces (Figures 68 and 70). The abdominal skin flaps are dissected up towards the head and the thoracic cage opened laterally by axe cuts. A careful butcher then removes the diaphragm, peritoneal sac and contents intact. However, sometimes the peritoneal cavity is penetrated by the less adept and very occasionally bowel may be
Figure 64:
The hair of the slain pig is singed off over an open fire, later used to heat stones for the earth cooking ovens.

Figure 65:
Cassowaries are a delicacy offered at the larger pig kills.
Figure 66:
Butchering proceeding on
leaf mats. The carcass is
not bled and small boys mop
up the blood spillage and
squeeze it into a bowl under
the head.

Figure 67:
A "baggis" being prepared
whilst a boy displays a
"bladder balloon" in the
background.
**Figure 68:** Abdominal viscera being removed with spillage.

**Figure 69:** Shows example of contamination by dogs and human feet.
perforated at this stage (Figure 68). This evisceration is preferably performed with bamboo knives, which being new, are more hygienic than unwashed steel knives. Women take no part in this process and sit aside watching the proceedings. Stray dogs, piglets, fowls and children wander at will about the dissectors, all contributing to a cumulative spoiling of the fresh meat. The boys also help mop up pooled blood with a paralay type of plant *Oenanthe javandica* squeezing it into a bowl under the head (Figure 66). Thoracic viscera, tongue and oesophagus are then removed and the solid organs separated. Not infrequently, butchers had infected and dressed scours on their hands from which meat became contaminated. *Pseudomonas proteus* was isolated from the sore shown in Figure 70. Figure 71 shows a man with a dirty finger bandage.

The women take the bowels in their "biluma" (string carrying bags along over the head) to the nearest stream for washing. The Chimu and Gahuma (Asare) women evert the small intestines by intussuscepting a stick into the bowel lumen, then siphon and blow water through the large bowel, the anal skin and anus acting as a funnel (Figures 72 and 73). Enya fashion is a little less crude as the whole bowel is slit open and cleaned more thoroughly. The bowels are plaited and wrapped in leaves ready for cooking. Stomach is packed with chopped fat, greens and herbs and cooked in the form of a haggis (Figure 67). It is eaten after "maturing" for two or three weeks. While the bowel washing is in progress, the men
Further examples of contamination by festering and dressed skin lesions. In the top picture, contamination from the arms also occurs.
**Figure 72:** Bowel cleaning performed by mucosal intussusception.

**Figure 73:** A woman blowing bowel contents through the colon using the anal skin as a mouthpiece.
continue the final filleting of the carcase: skull, rib cage and backbone being removed in one section. Here, contamination by feet was most apparent as assistants were required to pull the head and spine forward and ventrally.

Children eat raw morsels such as ear tips and snouts. Parents wrap entrails and sex organs about their wrists to promote future fertility. Bladders on inflation are sometimes used as balloons or footballs (Figure 67). The extraneous blood is used as a garnish, but the bulk is mixed with chopped fat and leaves, rolled in a banana leaf and cooked over an open fire as a blood sausage. Most of the viscera is consumed soon after cooking. Organs such as liver, spleen and kidney are regarded as having special magico-religious significance. Cooked bowel is eaten mainly by the women and children.

After the butchering is completed, which is in about 2 to 4 hours, the night is given to more singing and dancing. Earth pits are dug, long and shallow for the half sides of meat and deep and wide for the offal and prime cuts (Figure 74). The ovens are lined with banana leaves, tree fern fronds (Cycads contimana) and leaves of a variety of breadfruit (Ficus sammuremba). The meat, corn, coha, taro, sweet potato and chopped greens are mixed with pre-heated stones. Men handling them use long wooden tongs with great dexterity (Figure 75). Layer by layer the oven fills up until the large leaves roof over a final insulating layer formed by the pig’s quarters and flanks.
**Figure 74:** Men preparing oven and cooking stones.

**Figure 75:** Stones and pork flanks being placed in a long shallow oven at Tombul (Western Highlands).
Table XXX: Cooking time and procedure. The temperature readings were taken from the centre of oven using a thermocoupled pyrometer.

<table>
<thead>
<tr>
<th>Cooking Process</th>
<th>2/10/63</th>
<th></th>
<th>3/10/63</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Time No. 1</td>
<td>Temp. °C.</td>
<td>Time No. 2</td>
<td>Temp. °C.</td>
</tr>
<tr>
<td>Commenced putting meat in ovens. Water introduced.</td>
<td>11.30</td>
<td>38</td>
<td>11.45</td>
<td>35</td>
</tr>
<tr>
<td>Sealed with earth and cooking commenced.</td>
<td>11.45</td>
<td>82</td>
<td>12.00</td>
<td>79</td>
</tr>
<tr>
<td>Temperature rise.</td>
<td>11.58</td>
<td>93</td>
<td>12.11</td>
<td>92</td>
</tr>
<tr>
<td>Further gradual rise to</td>
<td>13.00</td>
<td>104</td>
<td>13.15</td>
<td>102</td>
</tr>
<tr>
<td>Started opening pit to remove partially cooked meat (dirt shovelled off).</td>
<td>13.15</td>
<td>104</td>
<td>13.22</td>
<td>104</td>
</tr>
<tr>
<td>Exposed meat.</td>
<td>13.25</td>
<td>99</td>
<td>13.34</td>
<td>99</td>
</tr>
<tr>
<td>Carcases now cut in half down spinal centre section, meat</td>
<td>13.37</td>
<td>93</td>
<td>13.40</td>
<td>93</td>
</tr>
<tr>
<td>extracted for disposal, i.e. head, thoracic cavity and everything removed.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Actual cooking time.</td>
<td>1 hr. 37 mins</td>
<td></td>
<td>1 hr. 34 mins</td>
<td></td>
</tr>
<tr>
<td>Kay-keu and bananas well cooked, soft and crumbly and eaten</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>almost immediately.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table XXXI: Temperature observations taken 12" below oven surface with 200°C mercury glass thermometer. The readings were taken 15 minutes after insertion.

<table>
<thead>
<tr>
<th>Tambul ) earth</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>Mean</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Goglme ) oven</td>
<td>84</td>
<td>92</td>
<td>92</td>
<td>88</td>
<td>88</td>
<td>88</td>
<td>88</td>
<td>85</td>
<td>86</td>
<td>86</td>
<td>87</td>
<td>1.8</td>
</tr>
<tr>
<td>Wooden oven</td>
<td>86</td>
<td>86</td>
<td>90</td>
<td>90</td>
<td>90</td>
<td>88</td>
<td>86</td>
<td>86</td>
<td>89</td>
<td>89</td>
<td>2.0</td>
<td></td>
</tr>
</tbody>
</table>
Water is poured in and the oven is again sealed by another layer of leaves and sometimes mound up with dirt, so that the food is cooked under steam pressure in its own moisture. The juices of all the contents are thus retained in the cooking process. The Chimbu women also cook beef and other morsels in wooden barrels or "stone ovens".

(2) **Temperature Observations**

Temperature readings were taken at Tambul and Gogoe with a 15 inch 400°C mercury glass thermometer. This was wrapped in leaves in similar fashion to the way a piece of meat was cooked, the thermometer bulb being placed 12 inches below the outer leaf layers. Results taken from ten ovens selected at random are present in Table XXX.

Observations in the Tseak Valley (Table XXX) indicate temperature readings taken using a pyrometer (clock face). The thermocouple was placed more deeply in the earth ovens and temperatures taken at set time intervals during the cooking. The centre of a hind quarter immediately after removal from an oven after two hours' cooking had a mean temperature of 78°C, for five pieces sampled. The stones after cooking were a little hotter than warm and could be handled with bare hands.

The results indicated that large chunks of meat were not thoroughly cooked and the lower mean temperatures at Tambul were due to the shallow ovens which also contained relatively fewer hot stones. Following cooking the meat rapidly cools, further handling
occur, and the same sources of contamination are present—feet, flies, dogs and so on. It is after cooking that contamination is most significant and the arguments and discussions concerning the distribution may continue for a whole day (Figures 76 and 77). In transit to its destination, a half side of pork may change hands two to five times and reach consumption point one to four days later. A day or more may then elapse before the pork is further cut up and a second distribution held. The meat is re-heated in a smaller earth oven so that any prior bacterial spoilage becomes a potent culture with a high infective dose. It may be assumed that the cooking temperatures will be the same. The redistribution and piece-meal consumption of this meat increases further the possibilities of food poisoning occurring and it is probably this meat which causes the prolonged upper abdominal pain of which so many people complain after eating pork (Milles, 1950).

From the foregoing, it may be concluded that the likelihood of disease spread is much greater in the large pig ceremonies than in the much smaller marriage and death distributions, where there is a limit to the number of pigs killed and a smaller number of recipients. Bridal payments do take place within the framework of the larger ceremonies and stacks of pork quarters and halves were seen at Goglue for this purpose.

(3) Sanitation

Deep pit latrines were in use in Goglue village. Of 20
**Figure 76:** Cooked pork ready for distribution.

**Figure 77:** Final distribution taking place at destination of exchange.
(By courtesy Dr. P. Brown.)
latrines, 18 were considered insanitary, due to inadequate housing (5), inadequate roofing to the pit (16), overfilling (7), and no hole covers (18). Nineteen latrines serving a population of 380 people, plus approximately 220 immigrants for the feast, were regarded inadequate and insanitary without considering their efficiency.

Water supplies for drinking purposes from the mountain springs were not examined for contamination.

Flies trapped during the pigkill at Goglme were identified as follows: *Chryosia* sp., *Orthellia* sp., *Musca domestica*, and *Musca* sp. Diptera of the *Calliphora* sp., *Phaonia* sp., and *Chrysomyia* sp. have been incriminated as vectors of *Clostridium perfringens* (Hobbs et alii, 1953). The resting fly density on a fly counting grill (Watt and Lindsay, 1954) was 30 to 36 per square foot. The fly index was far in excess of the normal figure of 10 when breeding areas were measured.

(4) Diseases in Pigs

Diseases in the alain pigs were noted at Tambul and Goglme. One thousand one hundred and forty carcasses, representing about one-half of the total killed, were examined at the Tambul and Wapenamanda pigkills. Sixty-one per cent of the pigs had intestinal emphysema (*Pneumotaxis ovitoides intestinalis*) and 4 per cent had the macroscopic appearances of enteritis. Reports of these findings have appeared elsewhere (Egerton and Murrell, 1964). At Goglme the
incidence of intestinal emphysema was only 5 per cent in a total of
530 pigs examined. Only 1 per cent had macroscopic enteritis. No
cases of anthrax or trichinosis were seen at either of these pig-
kills.

(5) Conclusions

In the process of butchering, cooking and distribution of meat
and food at pig-feasting ceremonies, contamination may be implied
from the following sources: the hands of the butcher due to a
universal lack of personal hygiene; the feet of butchers and food
handlers; direct and indirect soil contamination; leaves and
foliage used as mats and coverings; the faeces of the pig during
butchering and from inadequate washing of the bowels after evisceration;
other animals "moing about" during food preparation; multiple
handlers, especially children, during carcass preparation; flies
settling on the food before and after cooking; the bodies and
hands of individuals transporting the meat to its recipients and
its subsequent redistribution; and water containing heat resistant
spores poured over carcasses and into ovens.

Bacterial multiplication is favoured by the following factors:
delay between butchering and cooking with the meat exposed under
atmospheric conditions, sometimes in open sunlight for 6 to 10 hours;
temperature at the initial cooking inadequate to kill anaerobic
organisms and to thoroughly cook the big joints; considerable delay
in consumption and storage of meat for long periods without refrigeration; and inadequate temperatures during repeated re-coking processes, and in the initial cooking of inadequately washed bowel.

(4) Sources of Infection

(1) Pigs and Pork

_Clostridium perfringens_ was recovered from the intestinal contents of 53 of 322 pigs chosen at random from pigs killed at Korfena, Tambul and Gogins. All strains isolated proved to be type A and no type C strains of this organism were recovered. From 115 samples of cooked and uncooked pork, collected mostly from the medial aspect of the hindquarters where contamination was considered to be most likely, 18 isolations of _Clostridium perfringens_ type A were obtained. Again no type C strains were recovered. These samples included 18 cooked bowel specimens. On only five occasions, remnants of a pig (hair, bone and fat) suspected as a source of origin for a case of enteritis necroticans, were obtained and sent for anaerobic culture. _Clostridium perfringens_ type A was obtained each time but no type C strains.

The failure to enlarge the samples from this latter group was due primarily to the late arrival of cases to hospital and the difficulty in obtaining meat remnants. All traces of meat had gone by the time attempts to get such samples were made. The general reluctance of the people to blame pork as a cause also added to this difficulty.
*Cl. perfringens* was recovered from two fly samples (*Chrysomyia* sp.). These isolations were not taken to complete identification. Fecal samples from other possible vectors such as dogs, rats, fowls and cockroaches, were not investigated.

(2) Human Population

A one in ten systematic sample of feces of a normal human population failed to yield any *Cl. perfringens* type C strains. This survey was carried out independently of any pig feasting activity, although in the Upper Chimbu sampling at Goglo, Goromango and Kurumogi was undertaken one month following the pig killing there. The results were as follows:

<table>
<thead>
<tr>
<th></th>
<th>Feces examined</th>
<th>Isolation <em>Cl. perfringens</em></th>
<th>Per cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chirave</td>
<td>100</td>
<td>40</td>
<td>40</td>
</tr>
<tr>
<td>Upper Chimbu</td>
<td>100</td>
<td>36</td>
<td>36</td>
</tr>
<tr>
<td>Wabag</td>
<td>68</td>
<td>12</td>
<td>18</td>
</tr>
<tr>
<td>Tari</td>
<td>100</td>
<td>16</td>
<td>16</td>
</tr>
<tr>
<td>Lake Kapiauo</td>
<td>100</td>
<td>13</td>
<td>13</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>468</strong></td>
<td><strong>117</strong></td>
<td><strong>25</strong></td>
</tr>
</tbody>
</table>

An unknown proportion of these isolations was selected for complete identification. They all proved to be type A strains (Walker, 1964 - personal communication).

(3) Soil

Twelve soil samples taken from the ground over which carcasses
<table>
<thead>
<tr>
<th>Group</th>
<th>Place</th>
<th>Units of Beta Antitoxin per ml.</th>
<th>No.</th>
<th>&lt; $\frac{1}{2}$</th>
<th>$\frac{1}{2}$ - 2</th>
<th>2 - 5</th>
<th>5</th>
<th>$&gt;\frac{1}{2}$</th>
<th>% $&gt;\frac{1}{2}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Known recent pig feasting</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>i. Low prevalence pig-bel</td>
<td>Webag</td>
<td></td>
<td>66</td>
<td>34</td>
<td>26</td>
<td>3</td>
<td>3</td>
<td>32</td>
<td>48.5</td>
</tr>
<tr>
<td>ii. High prevalence pig-bel</td>
<td>Upper Chimu</td>
<td></td>
<td>39</td>
<td>4</td>
<td>29</td>
<td>5</td>
<td>1</td>
<td>35</td>
<td>89.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Total</td>
<td>105</td>
<td>38</td>
<td>55</td>
<td>8</td>
<td>4</td>
<td>67</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B. Doubtful recent pig feasting</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>i. Low prevalence pig-bel</td>
<td>Watabung</td>
<td></td>
<td>17</td>
<td>9</td>
<td>8</td>
<td>0</td>
<td>0</td>
<td>8</td>
<td>47.1</td>
</tr>
<tr>
<td>ii. High prevalence pig-bel</td>
<td>Upper Asaro</td>
<td></td>
<td>20</td>
<td>6</td>
<td>12</td>
<td>1</td>
<td>1</td>
<td>14</td>
<td>70.0</td>
</tr>
<tr>
<td></td>
<td>Tari</td>
<td></td>
<td>34</td>
<td>1</td>
<td>11</td>
<td>21</td>
<td>7</td>
<td>1</td>
<td>29 (85.3)</td>
</tr>
<tr>
<td></td>
<td>Baiyer River</td>
<td></td>
<td>17</td>
<td>0</td>
<td>13</td>
<td>4</td>
<td>0</td>
<td>17</td>
<td>100.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Total</td>
<td>88</td>
<td>20</td>
<td>54</td>
<td>12</td>
<td>2</td>
<td>68</td>
</tr>
<tr>
<td>iii. Unknown prevalence</td>
<td>Lake Kupiago</td>
<td></td>
<td>23</td>
<td>4</td>
<td>17</td>
<td>2</td>
<td>0</td>
<td>19</td>
<td>82.6</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Total</td>
<td>216</td>
<td>62</td>
<td>126</td>
<td>22</td>
<td>6</td>
<td>154</td>
</tr>
<tr>
<td>C. Overall N.G. population (A+B)</td>
<td>Grand total</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D. Known cases pig-bel</td>
<td></td>
<td></td>
<td>24</td>
<td>1</td>
<td>9</td>
<td>4</td>
<td>10</td>
<td>23</td>
<td>95.8</td>
</tr>
<tr>
<td>E. Suspected mild cases pig-bel</td>
<td></td>
<td></td>
<td>34</td>
<td>7</td>
<td>20</td>
<td>2</td>
<td>6</td>
<td>27</td>
<td>79.4</td>
</tr>
<tr>
<td>F. Relatives of pig-bel cases</td>
<td></td>
<td></td>
<td>38</td>
<td>6</td>
<td>23</td>
<td>6</td>
<td>3</td>
<td>32</td>
<td>84.2</td>
</tr>
<tr>
<td>G. Follow-up of cases of pig-bel ( &gt; 6/12)</td>
<td></td>
<td></td>
<td>9</td>
<td>4</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>5</td>
<td>55.6</td>
</tr>
<tr>
<td>H. European controls</td>
<td></td>
<td></td>
<td>42</td>
<td>38</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td>9.5</td>
</tr>
</tbody>
</table>
Figure 78: Statistical Significance of Differences in % Frequency of Groups of Persons with detectable Cl. perfringens β Antitoxin
had been prepared were collected from three village compounds in the
Upper Chimbu. *Clostridium perfringens* type A was isolated from four of
these.

(k) **SEROLOGICAL INVESTIGATIONS**

Sera were examined for *Clostridium perfringens* beta toxin from seven
different population groups (A - H Table XXXII). A level greater
than or equal to 0.5 units of antitoxin per ml. was regarded as
significant. This figure corresponds with that which provides
sheep with immunity against the *Clostridium perfringens* beta toxin (Bullen,
1955). The last group (group H) in Table XXXII consisted of a
control group of 42 Europeans. There had been no exposure to native
pork except in three of these persons. Four, which included the
three at risk, had levels of $\frac{1}{2}$ to 1 unit per ml. In the sera of
24 known cases of pig-bel, 23 had detectable antitoxin, 10 of which
were greater than 5 units per ml. In the overall normal population
sample of 216 persons (group C = A + B) 154 (71.3 per cent) had
significant antibody levels to the beta toxin. The statistical
significance of these results is shown diagrammatically in Figure
78. In the normal population there were significantly different
levels between groups chosen from areas where the disease was thought
to have a high and low prevalence respectively, for example, groups A
(i) + (ii) and B (i) + (ii). It was not possible to sample a large
group of individuals before and after pig feasting. In comparing
high prevalence groups represented in A (ii) and B (ii) where known pig feasting had taken place and where it was doubtful, there was no significant difference between the frequency of detection. Detectable levels in relatives of persons with known pig-boil was significantly higher than in the normal population of groups C and F.

With group A the following table may be formed.

<table>
<thead>
<tr>
<th>Suspected incidence</th>
<th>Units beta antitoxin per ml.</th>
<th>Total</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$&lt; \frac{1}{2}$</td>
<td>$\geq \frac{1}{2}$</td>
<td></td>
</tr>
<tr>
<td>Low incidence</td>
<td>34</td>
<td>32</td>
<td>66</td>
</tr>
<tr>
<td>High incidence</td>
<td>4</td>
<td>33</td>
<td>39</td>
</tr>
<tr>
<td>Total</td>
<td>38</td>
<td>67</td>
<td>105</td>
</tr>
</tbody>
</table>

$X^2$ for this table proved to be 14.114 which is highly significant ($p < 0.001$). Here and elsewhere in the analysis of the serological data, $X^2$ was corrected for discontinuity by the method of Yates.

A similar table for group B, omitting row iii, gave the following figures:

<table>
<thead>
<tr>
<th>Suspected incidence</th>
<th>Units beta antitoxin per ml.</th>
<th>Total</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$&lt; \frac{1}{2}$</td>
<td>$\geq \frac{1}{2}$</td>
<td></td>
</tr>
<tr>
<td>Low incidence</td>
<td>9</td>
<td>8</td>
<td>17</td>
</tr>
<tr>
<td>High incidence</td>
<td>11</td>
<td>60</td>
<td>71</td>
</tr>
<tr>
<td>Total</td>
<td>20</td>
<td>68</td>
<td>88</td>
</tr>
</tbody>
</table>
Again the $\chi^2$ value is significant ($0.01 > p > 0.001$). If the above two tables were considered homogeneous, the frequencies could be pooled to yield the following table combining the two groups:

<table>
<thead>
<tr>
<th>Suspected incidence</th>
<th>Units beta antitoxin per ml.</th>
<th>Total</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$&lt; \frac{1}{2}$</td>
<td>$\geq \frac{1}{2}$</td>
<td></td>
</tr>
<tr>
<td>Low incidence</td>
<td>43</td>
<td>40</td>
<td>83</td>
</tr>
<tr>
<td>High incidence</td>
<td>15</td>
<td>95</td>
<td>110</td>
</tr>
<tr>
<td>Total</td>
<td>58</td>
<td>135</td>
<td>193</td>
</tr>
</tbody>
</table>

For this combined table $\chi^2 = 30.9984$ ($p < 0.001$). For row iii of group B where it was not known whether enteritis necroticans existed, the percentage $\geq \frac{1}{2}$ was 82.60 which differed significantly from the low incidence row of the combined table above ($0.01 > p > 0.001$) but was insignificantly different from the high incidence row.

The comparisons of row iii of group B and each of the other groups, except in the total population represented in group C, with the low incidence frequencies and high incidence frequencies of the combined table, were then summarised as follows:

<table>
<thead>
<tr>
<th>Comparison with:</th>
<th>Percentage</th>
<th>High incidence sample</th>
<th>Low incidence sample</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Row iii</strong></td>
<td><strong>Group B</strong></td>
<td>$&gt; \frac{1}{2}$</td>
<td>$p &gt; 0.05$</td>
</tr>
<tr>
<td></td>
<td><strong>Group D</strong></td>
<td></td>
<td>$p &gt; 0.05$</td>
</tr>
<tr>
<td></td>
<td><strong>Group E</strong></td>
<td></td>
<td>$p &gt; 0.05$</td>
</tr>
<tr>
<td></td>
<td><strong>Group F</strong></td>
<td></td>
<td>$p &gt; 0.05$</td>
</tr>
<tr>
<td></td>
<td><strong>Group G</strong></td>
<td></td>
<td>$p = 0.05$</td>
</tr>
<tr>
<td></td>
<td><strong>Group H</strong></td>
<td></td>
<td>$p &lt; 0.001$</td>
</tr>
</tbody>
</table>
Table XXXIII: Age Distribution of Antitoxin in Normal Population

<table>
<thead>
<tr>
<th>Age Group</th>
<th>No. examined</th>
<th>Detectable beta antitoxin per ml.</th>
<th>Per cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 - 5</td>
<td>7</td>
<td>5</td>
<td>71.4</td>
</tr>
<tr>
<td>6 - 10</td>
<td>11</td>
<td>7</td>
<td>63.6</td>
</tr>
<tr>
<td>11 - 15</td>
<td>22</td>
<td>14</td>
<td>63.6</td>
</tr>
<tr>
<td>16 - 20</td>
<td>18</td>
<td>10</td>
<td>55.6</td>
</tr>
<tr>
<td>21 - 30</td>
<td>39</td>
<td>30</td>
<td>76.9</td>
</tr>
<tr>
<td>31 - 40</td>
<td>55</td>
<td>40</td>
<td>72.7</td>
</tr>
<tr>
<td>40+</td>
<td>31</td>
<td>28</td>
<td>90.3</td>
</tr>
<tr>
<td>TOTAL</td>
<td>183</td>
<td>134</td>
<td>73.2</td>
</tr>
</tbody>
</table>

*Total population surveyed differs slightly from that in Table XXXII because age and sex were not recorded on 33 occasions.

Table XXXIV: Sex distribution of antitoxin levels in the normal New Guinea

<table>
<thead>
<tr>
<th>Sex</th>
<th>No.</th>
<th>Detectable beta antitoxin</th>
<th>Per cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>M</td>
<td>74</td>
<td>56</td>
<td>67.9</td>
</tr>
<tr>
<td>F</td>
<td>109</td>
<td>78</td>
<td>75.7</td>
</tr>
<tr>
<td>TOTAL</td>
<td>183</td>
<td>134</td>
<td>73.2</td>
</tr>
</tbody>
</table>
Figure 79: Quantitative distribution of C. perfringens beta antitoxin in normal New Guinea population, in contacts and in persons with enteritis necroticans and in Europeans.
Row iii group B and groups D, E and F corresponded with high incidence frequencies. Group G corresponded most closely with low incidence frequencies but the percentage $\geq \frac{1}{2}$ will be seen to be in a rather equivocal region between the two extremes. Group H differed significantly from both high and low incidence groups with a percentage $\geq \frac{1}{2}$ of 9.52 which is highly significantly lower than the low incidence percentage.

Quantitative levels of beta antitoxins greater than 2.0 units per ml. were considerably higher in groups D, E and F. This upward trend is shown diagrammatically in Figure 79. These results suggest that the immune response in individuals in these groups is the result of a greater or more prolonged exposure to beta toxin. They also indicate that antitoxin detection may be diagnostically significant.

The frequency of detectable beta toxin in different age groups is shown in Table XXXIII. It was higher in 31 persons above an estimated age of 40 years (90.3 per cent). In the 16 to 20 year age group only 55.6 per cent of those examined had detectable levels. Between these limits the frequencies were evenly distributed between 63.6 per cent and 76.9 per cent. Males had a slightly greater immune status than females (Table XXXIV). The difference, however, was not significant.
CHAPTER V

DISCUSSION
Accurate measurement of the occurrence of enteritis necroticans proved difficult because there was no single pathognomonic criterion of clinical diagnosis. However, the minimal incidence was estimated to be between 10 per 10,000 and 20 per 10,000. At Germershausen, where the disease was prevalent, the incidence was established only in retrospect from census figures obtained well after the pig-killing had ceased. The figure of 46.1 per 10,000 at Germershausen must therefore serve only as a rough guide to assess the size of the problem.

In North Germany, with the exception of Lübeck, the incidence of Darmsbrand ranged from 0.40 per 10,000 in 1946 to 1.77 per 10,000 in 1947. The maximal incidence was 3.59 in Kiel in 1947 (Kloua and Brummond, 1950). In Lübeck the incidence was much higher, being 16 per 10,000, and the mortality was estimated to be 3.5 per 10,000 (Hansen, 1949). The mortality figure at Germershausen was 13.8 per 10,000. The discrepancies between the two German figures were influenced rather by difference in definition and classification of the less severe forms of the disease.

Of 264 Darmsbrand cases reviewed by Hansen (1949) the overall death rate was 22 per cent. Kloua and Brummond (1950) in their series of 355 cases estimated the case fatality rate at 41.4 per cent. These latter writers were concerned mainly with the severe forms of the disease and included only 38 in the mild group. Three other German authors gave high mortality rates for their case series. Ernst (1948) reported a mortality of 50 per cent in Hamburg, Nissen
(1950) a figure of 44 per cent for the Landesteil-Schleswig area, and Griessmann (1950) a mortality rate of 46 per cent for 124 cases. These figures compare with an overall New Guinea case fatality rate of 36.4 per cent. Excluding the mild infections, the rate here rose to 48.8 per cent. This case fatality rate was maximal at 52.0 per cent in 1963 but fell to 30.0 per cent in 1964 following the introduction of antiserum therapy in June of that year. The cumulative experience of managing cases pre-operatively also helped bring about this reduction.

It is interesting to record that among Australians wounded in the New Guinea campaign against the Japanese, wound gas gangrene occurred at the rate of 4.5 per cent, an exceptionally high rate compared with other theatres of World War II (Ross and Ryan, 1944). These writers also recorded that among 103 Japanese prisoners of war during the same period there were no less than 21 cases of gas gangrene.

The German outbreaks of Darabrand had well defined seasonal distributions over the years 1947 - 1948 and the disease reached pan-epidemic levels during the mid and late summer months (July - September) of those years. The maximum incidence occurred in 1948 and then, in the following two years declined and the disease disappeared as mysteriously as it had arrived. Jeckeln (1957) who had experience with a large number of patients in Lübeck, expressed the view that this intestinal disease, the most severe he knew, had
abated. A report of the possible role of _Cl. parvum_ type F in the etiology of infant dyspepsia appeared as late as 1952 (Blechova and Novotny) but no other isolations of either type F or type C strains from man have been made between then and the discovery of strains in cases of pig-bel.

The seasonal or annual appearance of the disease in New Guinea was generally confined to the middle six months of the calendar year. This is the "dry" season of the year and as such, gardens were at maximum production and the absence of rain favoured outdoor festivities.

A noteworthy difference in the New Guinea disease from its German counterpart was the age distribution. The number of persons in the 0 to 10 year age groups was significantly higher (53.8 per cent) than in any other decade. This distribution contrasts with Hamburg, where most cases occurred in the fourth, fifth and sixth decades. The mortality rose in the very young and most elderly patients, which was also the case in New Guinea. However, Joschins (1947) reported the condition in 13 infants under one year of age. The disease had an acute toxic course and 12 of these cases succumbed.

The occurrence of the disease in childhood in New Guinea is readily explained by the dietary practice associated with pork feasting. With the larger pig cycles occurring at 3 to 10 yearly intervals, it is possible that the younger age groups have an initial exposure to massive pork meals. Infants without teeth are not
offered solid foods, and toddlers, who are breast fed up to the age of 2½ to 3 years, are given only token amounts of pork. Only three patients in the series reviewed were under two years. The belief that pork imparts strength into the individual is practically demonstrated by encouraging children, especially males, to consume as much as possible. Particular organs, such as the genitalia, liver and kidneys, are also reserved for children to eat. Burchett (1964) reported to the author that in the Baiyer River outbreaks in 1962 children actively refused further meals offered to them by their parents because of their fear of becoming ill.

The reluctance of older people to seek medical care, the general regard that illness in the older generation is due to "old age" and the lower life expectancy, account for the fewer case reports in people over 40 years of age. The life expectancy of the Highlander is probably lower than that estimated in life tables by Scrugg (1954) for New Ireland natives of New Guinea.

The sex distribution in New Guinea corresponded with that in Germany (2.2 males : 1 female). This distribution may be influenced by the greater frequency with which males seek medical care as indicated by a higher bed occupancy rate both in Papua (Campbell and Arthur, 1964) and the Territory as a whole (Department of Public Health, 1964). However, the survey at Gengame indicated that twice as many males than females were affected. The women eat more of the cooked bowel of the pig than the men. If heat resistant
spores of the clostridial organisms survive the cooking, as well they might, a higher morbidity of illness should be expected in the females. The fact that the reverse is probably true rather indicates that Clostridium type C is not resident in the gut of the pig. However, the bowel is cooked in the hottest part of the oven and normally eaten soon after its removal, and the type C strains did not show the heat resistant quality that the type F strains did in Europe (Egerton and Walker, 1964).

More females contracted the disease than males in the 16 to 20 year age group. At marriage feasts the bride and prospective brides are encouraged to consume unusually large amounts of pork to encourage fertility. This possibly explains the distribution of the disease in this group.

The evidence that large scale pig-killing activities influence the prevalence and spread of enteritis necroticans in the Highlands is circumstantial. It would appear from the trends in admission figures that this cultural practice influences the prevalence of diarrheal disease in the localities of the hospitals reviewed. The possibility that coincident gastro-enteric infections due to a virus or other causes occurred has still to be excluded. In the presence of such a coincidence, the spread of these pathogens would be greatly assisted by consuming contaminated pork segments involved in exchanges. The relative increase in hospital admissions for diarrhoea at Kundiana following this activity in two separate areas
during two different times of the same year strongly suggests an etiological relationship.

The experience of the author at Wabag and Kundiana left the impression that pig-bel varied considerably in density in the two areas. This impression was borne out by the differing levels of beta antitoxin in individuals sampled in the Upper Chimbu and Wabag areas after pig-feasting. An alternative explanation of the variable prevalence may lie with the actual feasting habits of the tribes concerned. In the Chimbu it became apparent that the admission rate rose as the feasting continued. In the Goroka and Chimbu areas, there was also a small introductory pig-kill which was the overture to the larger massive feast. This took place two to four weeks earlier and was not a feature of pig-kills witnessed in the Western Highlands. This may explain the different disease prevalence on the etiological basis of a sensitisation to pork at the preliminary feast.

A more accurate index of the morbidity and mortality following pig-feasting could be obtained by measuring the diarrhoeal attack rate in a population sample. Difficulties apparent in this approach were found to be a reluctance of the people to implicate pork as a cause of diarrhoea, and the problem of accounting for people before, during and after the feasting, due to the transient migrations which naturally occurred at these times.

The proven cases at Goromango and the subsequent appearance of
documented cases at Kurumגד (Case 96) and Gere (Case 100) after consuming pork prepared by people from Goruma-go supports the argument that pork may be a vector of the disease. Against this hypothesis remains the fact that few other contacts of known cases developed symptoms suggestive of food poisoning. A significantly high level of Cl. perfringens beta antitoxin was found however in contact persons at risk. Perhaps some toxin neutralizing substance in the bowel limits the distribution of the disease in persons at risk. This has been postulated in the case of food poisoning due to Cl. perfringens type A (Goudie and Duncan, 1956).

According to the latest edition of "Hospital Disease Statistics" published by the Department of Public Health in the Territory (1964) figures for gastro-enteric infections appear third in the list of "Principle Causes of Death" for all age groups in Highland hospitals. These infections are second only to acute respiratory disease as a cause of death in children under 12 years of age. The figures in Tables XXIII A, B and C support this report. By contrast, figures are lower in coastal hospitals. The significance of this difference is reflected in the poorer standards of personal hygiene and environmental sanitation of Highland communities. The whole balance between this situation, protein malnutrition and the denser population is rather crucial. There can be little doubt that unhygienic pork consumption produces an overall increase in the prevalence and mortality of infective enteric disease.
Exposure to pork seems to play a vital role in the epidemiology of enteritis necroticans in New Guinea. The dietary history, distinct incubation period and significant antitoxin levels in proven and suspected cases and in their contacts support this hypothesis. The failure to recover the organism from pigs, pork and the normal population provides weighty evidence against this.

The immunological testing of sera provided a more reliable index of exposure than was possible by bacteriological methods. An antitoxin level of 71.3 per cent in the normal Highland population compared to the 9.5 per cent in European controls indicated a constant widespread exposure to the perfringens beta toxin. The degree and frequency of this exposure were higher in areas where pig-bel was more prevalent (for example, Upper Chimbu). No previous study of a similar nature has been carried out in humans before. It is unfortunate that a parallel study was not undertaken in pigs because this may have provided some added information on the origin of Cl. perfringens type C.

The generally high immune status of the population as a whole could explain the absence of massive epidemics of the disease. At Bundi, where the disease was most prevalent, the previous large pig feast had been held seven years earlier. Here the pig population was not as dense as in the Chimbu (Morrison - personal communication) and a significantly high non-immune population may have built up during the seven years' absence from massive pork feasting. In this
regard, the fluctuations in the disease prevalence simulate that of some other diseases such as chicken pox and measles, their endemicity being dependent on the non-immune pool. In the Chimbu, it was established that the time interval between the larger feasts was only three years. This factor could therefore influence the variance of disease from one place to another.

A future investigation of the immune status of new born infants and toddlers may provide information on the age at which exposure to \textit{Cl. perfringens} type C first takes place. Samples taken in the 0 to 5 year age group were too small to make any meaningful conclusions. Examination of faeces for the presence of beta toxin is another important avenue for further elucidation of the epidemiology and aetiology of pig-bel.

The origin of \textit{Cl. perfringens} type C remains unsolved. It was thought that the human population was adequately sampled to exclude a human "carrier" source for the organism. In order to maintain a human source of infection, the organism should have been recovered from at least 10 per cent of the normal population. Collecting and isolation techniques were fairly well standardised although the recovery of \textit{Cl. perfringens} varied from 40 per cent at Chuave to only 13 per cent at Lake Kopiago. These discrepancies were probably due to a delay factor between collection and plating of faecal samples. The greatest delay was at Wabag, Tari and Lake Kopiago, where the frequencies were lowest. Overgrowth by \textit{Proteus} sp. probably
occurred in the transport media. The clostridia, being strict anaerobes, may not have survived under such conditions. It is also possible that some perfringens strains produce substances inhibiting the growth of other strains (Sasarman and Antoki, 1963).

It will be noted, however, that the recovery rate of \textit{C.l. perfringens} type C was only 17 (44.7\%) from 38 bowel samples from cases of enteritis necroticans. The material presented in this group for bacteriological examination came from resected gut specimens, and not from faeces passed per rectum. The more protracted the illness, the lower this rate of recovery became from the resected gut lumen.

The organism was isolated from one of 12 recovered from pig-bel patients. The presence of type C in this patient 12 months after infection was the only bacteriological evidence of the natural source of the disease.

Unknown factors remain to be revealed. Is \textit{C.l. perfringens} type C resident only in the upper small intestine as the type D organism is in ruminants (Bullen, 1954)? Is this organism more sensitive to environmental changes than the type A strains? The colonial appearances of type A and type C strains are identical so this reduces by half the chance selection of a type C colony for identification.

The possibility still remains that pigs are the source of the organism. Samples taken were adequate only at Tambul from where only two known cases eventuated. Egerton (1964) in a survey of
more than 100 pigs in the Chimbu failed to isolate \textit{Cl. perfringens} type C. In the Asaro and Chimbu surveys only 30 random faecal samples from more than 300 pigs were submitted for examination. No one was known to have contracted severe pig-bel from the pigs sampled. Most of the meat sampled likewise came from the Enga "Te" pig-killing at Tambul and Wapenwambe. Bacteriological examination of meat samples showed that faecal contamination had occurred. Apart from \textit{Cl. perfringens} type A, \textit{Proteus vulgaris} and \textit{Escherichia coli} were commonly found.

It is unfortunate that diseased pigs could not be examined. The people were reluctant to bring in the carcasses of diseased animals for examination. An enterotoxemic-like disease is known to the people of the Chimbu among their piglets (Brockfield - personal communication). Several local leaders expressed a familiarity with the signs of this disease but were not so helpful in producing carcasses for autopsy. It is in this direction that future investigations could be applied in an attempt to discover the origin of the organism in man. When a more specific dietary history was taken, only twice was there a suspicion that pork from a diseased animal had been consumed. More often than not, however, the meat was more than 24 hours old, and in a few instances it was up to two weeks old. Putrefied meat, of itself, may be a sufficient stimulus to start the pathogenic sequence.

It is perhaps interesting to speculate that the disease
incidence is increasing now that more pigs are being bred and communications less restricted. Whether the disease was present before the advent of Europeans will never be known. Only recently has there been an awareness of the disease among the native population. Prior to 1962 no aetiological association with pork was held by the Highlander, otherwise the syndrome would have been known by name in the local vernacular. Diseases such as goitre, dysentery, leprosy and cirrhosis (for example, ascites and oedema) have specific names. The latter three are particularly feared and regarded as infectious. Abdominal pain after the pork engorgement is the rule rather than the exception and, generally speaking, a sign that one has had enough.

The question as to whether *Clostridium perfringens* type C has been introduced into the Highlands by immigrant pigs or Europeans remains. Has the disease been introduced by missionaries from Germany? This is a speculation which would be impossible to prove. The toxigenic differences between the German and New Guinea strains are sufficiently distinct to be geographically separate. Even if it were assumed that the disease appeared first in 1960 – 1961, it is difficult to understand so rapid a spread from Tari to Goroka. The experience of earlier workers in the Highlands seems to confirm that the disease has been endemic since at least 1954. The findings of *Clostridium perfringens* beta antitoxin levels in a high proportion of the population at Lake Kopiago, where there has been only recent limited contact with
Europeans, suggests a long standing contact with the antigen. These people are sufficiently isolated from neighbouring Huli tribes near Koreba in the Southern Highlands to exclude this possibility. In a health survey at Lake Kopiago in 1963, the health of the people there differed little from that found in other Highland groups, although the people were shorter in stature, bordering on pigmy size (Walsh, Murrell and Bradley, 1964).

It seems unusual that the disease has not been recognised outside the Highlands of Papua-New Guinea. For many years Highlanders have been recruited for work as labourers on coastal plantations. If the disease is a human-carried one, then cases should have appeared elsewhere than the Highlands. Roth has had more (including Papua and Wewak) and had seen no similar cases prior to those reported in January 1961 (Murrell and Roth, 1963). These observations throw the balance in favour of pigs as a source, as these animals are scarce in coastal areas. The author is not aware of any similar human culture where the pig is of such importance as a symbol of status and worship as in the Highland culture of New Guinea.

Imported pigs first appeared in the Highlands in 1954. Distribution and interbreeding with the native pig, *Sus carmenae*, commenced soon after and is continuing at an ever increasing rate through both private and Administrative influences. There are no available statistics on just how many imported pigs have filtered
into and survived in native herds.

The pathogenesis of pig-bel will now be considered. The
different grades of severity in the histopathology of pig-bel led to
speculation on the exact pathological sequence of events in the severe
disease. The clinical picture and macropathology were easy to
differentiate from the suspect group which, without bacteriological
confirmation, was indistinguishable from the multitude of other causes
of gastro-enteritis. Grissman (1947) believed that "Darabrand"
should be separated from the condition "darmphlegmon", necrosis in
the latter being confined to the mucosa and known now as "pseudo-
membranous enterocolitis". This writer attempted to simplify the
pathogenesis but was unable to fix any particular factor which
influenced the rapid submucosal necrosis which typifies the severe
forms. He believed that a liquefactive necrosis, inflammation with
pus formation, occurred by secondary invasion of the primary "phlegmon".
Siegmund (1948) agreed with this view but Jeckeln (1948) believed the
two stages interchangeable and synonymous.

The distribution of the bowel lesions in pig-bel was usually
confined to the jejunum, although extension to the ileum was often
seen in children. Involvement of the colon was not uncommon in
Germany (Dorman, 1948; Jeckeln, 1948; Klee, 1947; Siegmond, 1948).
Segments involved were usually less than 50 cm. in length and in
severe instances the greater part of the intestine could have been
affected (Friedman, 1953). Delayed treatment in the New Guinea
disease resulted in larger and longer gangrenous segments. The inflammation commenced in limited areas in the proximal intestine and spread distally. The lesions were also either continuous or occurred segmentally in the form of "cuffs" with interposed normal segments of intestine (Dormanns, 1948; Heins, 1947; Sieg mund, 1948). Although not as apparent in the New Guinea cases, old and recent lesions were sometimes seen in the one patient.

The first lesion is a coagulative necrosis which is almost certainly the result of a powerful toxin produced in the bowel or introduced with the diet. The fact that the upper jejunum was primarily the seat of this necrosis, sometimes the stomach and duodenum but rarely the lower ileum or colon, plus the short but variable incubation period, suggested that a food borne toxin was the "excitor" to the pathogenic sequence of events. Each dying process was inevitably connected with inflammatory infiltration and the end pathological result was unquestionably that of an anaerobic infection of the upper intestine. The appearance of the bowel was consistent with multiple segmental areas of strangulation. Histopathological findings of mucosal and submucosal thrombi were indicative that this was one step in the pathogenesis. It has been well established by Cohn (1956) that following strangulation in experimental obstruction in dogs, bacteria invade the intestinal wall, and by their growth and production of toxins, increase vascular permeability and thus destroy the bowel wall. The lecithinase of
the alpha toxin of \textit{Clostridium welchii} is thought to be responsible for many of these effects (Cohn and Hawthorne, 1951). Williams (1927) first emphasised the importance of the toxemia due to anaerobic organisms in intestinal obstruction. In the dog experiments, Cohn (1954) showed that in strangulation of long loops of bowel, death was due to blood loss, but in strangulation of short loops of bowel, death was due to perforation and peritonitis. This phenomenon was true to some extent in pig-bel.

The toxins produced by the New Guinea strains of \textit{Clostridium perfringens} type C were alpha, beta, theta, kappa and mu toxins. The importance of this organism to the clinician rests not on its behaviour in vitro but on its ability to produce soluble agents capable of poisoning and destroying animal cells under certain fortuitous circumstances. The clostridia are primarily and essentially saprophytes. If one of the pathogenic clostridia of gas gangrene is cultivated in a suitable fluid medium for some hours, the intramuscular inoculation of the bacteria-free culture filtrate will produce in an experimental animal most or all of the signs and symptoms of the disease with the exception of tissue emphysema (Moerman, 1962). This forms the basis of the test to estimate levels of circulating antitoxin in the host (Gleny et alii, 1931). Gleny, Oakley and their collaborators first clearly demonstrated the multiplicity of toxins produced by \textit{Clostridium perfringens} (Gleny et alii, 1933; Oakley, 1943).
The distribution of antigens and heat resistance amongst the different types of Clostridium perfringens

<table>
<thead>
<tr>
<th>Type</th>
<th>Occurrence (Country where first described)</th>
<th>Major antigens *</th>
<th>Minor antigens *</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>α</td>
<td>β</td>
</tr>
<tr>
<td>A</td>
<td>(1) Gas-gangrene of man and animals</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Intestinal commensal, man and animals</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Putrefactive processes soil, etc. (United States)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(2) Food poisoning (Britain)</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>B</td>
<td>(1) Lamb dysentery</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>(2) Enterotoxaemia of foals (Britain)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(2) Enterotoxaemia of sheep and goats (Iran)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>(1) Enterotoxaemia (&quot;Struck&quot;) of sheep (Britain)</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>(2) Enterotoxaemia of calves, lambs (United States)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(3) Enterotoxaemia of piglets (Britain)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(4) Necrotic enteritis of man (formerly type F)† (Germany)</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>(5) Necrotic enteritis of man (Papua-New Guinea)</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>D</td>
<td>Enterotoxaemia of sheep, lambs, goats, cattle and possibly man (Australia)</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>E</td>
<td>Sheep and cattle, pathogenicity doubtful (Britain)</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

* * Produced by most strains; + + produced by some strains; + produced by few strains; 0 not produced by any; – not tested.

* The major antigens are those defining the type and predominantly responsible for pathogenicity; the minor antigens are of a lower order of toxicity and of little or no importance in pathogenicity.

† Occasionally the presence of this antigen must be assumed from the production of the appropriate antitoxin by hyperimmunised horses.

‡ Type F has been abandoned and the strains included in it transferred here.

§ In this column + + + means heat resistant; 0, not heat resistant.
From Table XXXV it will be seen that the alpha toxin is a lecithinase C (Macfarlane and Knight, 1944) which hydrolyses lecithin to phosphorylcholine and a diglyceride. It is haemolytic, demeco-
rotizing and lethal. The biochemical nature of beta toxin is not known but it is regarded as a lethal toxin (Gleny et alii, 1935) and may very well be a collagenase similar to the beta toxin produced by Cl. histolyticum and the kappa toxin of Cl. perfringens (McLennan, 1953). The theta toxin is haemolytic, demeco-
rotizing and lethal (Todd, 1944), and considerable antigenic relationship exists between theta toxin and the oxygen-sensitive haemolysins produced by certain streptococci and possibly other clostridia. Chain and Duthie (1940) were able to identify spreading factors whose property was to hydrolyze the cementing mucopolysaccharide of the tissue hyaluronidase from gas gangrene infections. Baker, Webster, Freeman, Cary and Sanders (1956) later purified hyaluronidase from Cl. perfringens type A. Oakley and Warrack (1953) suggested the name "mu-toxin" for this enzyme.

From the foregoing it can be seen that the major and minor toxins produced by Cl. perfringens type C could all synergistically unite to produce the pathology of pig-bel. For these toxins to be absorbed through the mucosal barrier pre-existing defects by way of mucosal atrophy, ulceration and inflammation must exist. The villi of the intestinal biopsies on follow-up studies were mostly abnormal which in the acute cases may have been pre-existent. Hookworm and
roundworms both indirectly and directly could provide this pre-
disposing state of affairs. The irritant voluminous pork meal with
its subsequent stasis, alkaline pH and motility upset could also
influence or enhance this situation. Cohn (1956) showed in his
obstructive experiments in dogs that pressures higher than 10 mm. of
mercury produce vascular damage, thus assisting the necrosis. Early
gaseous distension was a clinical feature in severe pig-bell.

Once necrosis had occurred, particularly if beyond the lamina
propria, the environmental conditions just described would favour a
rapid proliferation of the electridia so that as "facultative
pathogens" they gain a footing only when tissues of the primary
lesion have been altered to suit their limited and peculiar needs.
The pathogenic sequence of events may then basically follow that of
a gas gangrene infection in wounds, of which a reduction in the
local oxidation-reduction potential is vital (McLemore, 1962).
Speculation about such a reduction suggests this could be brought
about by:

1. Failure of blood supply to the affected area; thrombosis
   of submucosal vessels and distension of bowel wall by
   atony and gas both occur in the pathogenesis.

2. Presence of necrotic tissue from the initial necrotizing
   process and hemorrhage into the bowel wall following
   capillary necrobiosis.

3. Secondary invasion by normal bowel flora as further
necrotizing agents, with putrefaction and gangrene.

iv. Maintenance of an alkaline pH in luminal contents.

Obviously all of these factors are interrelated and the process probably becomes irreversible once the smooth muscle layers of the wall become involved. When toxigenic enterobacteria gain access to muscle, further toxins are certainly elaborated and these diffuse into the peritoneum. These toxins, and specifically the necrotizing alpha, beta and theta toxins of *Cl. perfringens* type C may then diffuse out, aided by hyaluronidase production, (mu toxin) and kill surrounding tissues, which in their turn are colonized by other anaerobic bacteria. So the disease process continues gaining momentum all the time.

The patho-physiology, as suggested by Oakley (1954) for wound gas gangrene is such that in areas of reduced oxygen tension the pyruvate in the muscle is incompletely oxidized, or reduced to lactate so that the local pH falls. This lowered redox potential and acidity together favour the activity of endogenous proteolytic enzymes in the tissues with a consequent release of amino acids into the lesion, producing conditions ideal for the multiplication of anaerobic micro-organisms. In ordinary gas gangrene infection, Wood Power (1945) stressed the importance of blood supply to muscles. When this occurred to the muscle wall of the jejunum in enteritis necroticans the probability of irreversible changes in the defensive powers of the host became critical.
The essential lesion in the muscles is a disruption of the sarcolemma and fragmentation of the muscle fibres with preservation of the myofibrils. There is also nuclear karyolysis and the appearance of extracellular fatty droplets. Early there is no fibrin formation, no vascular hyperaemia and no neutrophil cellular reaction; in fact leucocytes in the vicinity of a lesion undergo karyolysis and cytolysis. The alpha toxin being a lecithinase, accounts for the death of tissue and by similar effects on capillary cell walls it doubtless alters their permeability and produces the gross oedema characteristic of the lesion. This oedema by reducing the blood supply and bowel lumen still further lowers the redox-potential of the tissues and permits multiplication of clostridia. The theta toxin, which is a leucocidin, may well play a part in the notable absence of polymorphonuclear cells characteristic in the early stage of the disease. The beta and mu toxins, probably act upon the supporting tissues, the collagen fibrils, the intercellular cementing material and any defensive fibrin that may be laid down. In particular, the beta toxin, possibly being a collagenase as is the kappa toxin, may be responsible for the disappearance of collagen and reticulum in the submucosa and muscularis mucosae. By thus removing the supporting reticulum round the capillaries and small vessels, haemorrhage and thrombosis is thereby favoured (Macfarlans and McLennan, 1945).

In pig-bel, as in gas gangrene, even in the most advanced and
moribund cases, no obvious intravascular hae-molysis was observed
clinically except in two cases (46 and 79) where a concurrent
clostridial septicemia was apparent. Blood cultures for clostridia
were negative.

The pathogenesis probably therefore starts by the action of
powerful toxins in the upper intestine and this genesis is then
stimulated by additional factors, intrinsic and extrinsic, favouring
an overwhelming toxemic infection of the host. The role that other
faecal and oral flora play in this toxemia requires further investiga-
tion. Differential bacteriological analysis of organisms of the
bowel flora such as B. coli, Pseudomonas sp., Bacteroides Sp., Strept.
faecalis and Proteus mirabilis normally present in bowel above
obstructed areas (Bishop and Alcock, 1960) needs to be worked out in
the New Guinea disease.

The large pork meal, which may or may not contain pre-formed
toxin/and a high helminthic infestation, possibly predisposes to
motility changes, spasm on one side and atony on the other. This
favours further bacterial growth and toxin production setting in
motion a chain of events which eventually leads to total bowel
necrosis.

A hypothetical mechanism for the pathogenesis of pig-bel has
been drawn in Figure 80. It shows the various clinical and
pathologic stages through which the disease develops as related to
time intervals. It is difficult to pin point precisely the exact
**Figure 60:** Some possible pathways in the pathogenesis of "pig-bel", related to time.
chain of events as there are so many changing individual and environmental variables. The constant variation and change in the symptomatology alone indicate a complex pathogenic sequence. Until further more precise bacteriological and physico-chemical measurements can be made, the complete story of the pathogenesis of the severe forms of pig-bel remains clouded. It may be postulated that predisposing influences operate prior to the consumption of the large, contaminated or otherwise, pork meal. These are shown in the following diagram:

Pre-existing villous atrophy and flattening due to folic acid deficiency, helminths, giardiasis.  

Pre-existing mucosal sensitisation to pork protein, fat or Cl. perfringens beta toxin.

Large contaminated pork meal + Cl. perfringens type C.  

Colliquative necrosis mucoza + submucoza.  

Associated inflammation caused by other enteric pathogens; virus, Shigella, Salmonella.

In further discussing the aetiology of pig-bel, evidence has been presented to suggest that Cl. perfringens type C plays some role, if not a major one, in the pathogenesis. The only previous
isolates of strains of this organism with similar toxin production were from animals. Isolates from calves and lambs were described by Griner and Brenchen (1953) and by Griner and Johnson in the following year. These strains produced delta toxin. Other type C isolations producing non-delta haemolysis were made from cases of piglet enterotoxaemia by Field and Gibson in 1955. In New Guinea, the possibility of enterotoxaemia in piglets remains unexplored and this could well be the origin of the disease, having undergone a synanthropic ecosia (cf. brucellosis, trichinellosis).

The very complex toxicology of Cl. perfringens has been exhaustively studied by Oakley (1943) following initial work by Wiledon in 1931. Today at least 12 toxins are recognized (Table XXXV).

The German cases of "Darmbrand" were believed to be caused by Cl. perfringens type F which differs mainly in its heat resistance from type C. There are also slight antigenic differences. Schüts (1947) first realized that these strains differed in their morphology from the classical type A variety, the rods being longer, thicker and occurring in chains.

The strains isolated from cases in this thesis produced alpha and beta toxins but were not heat resistant. They have therefore been assigned to the type C group. As these strains are implicated in a syndrome similar epidemiologically, pathologically and toxicologically to the German disease due to the type F organism
(Zeisal and Bassetto-Sternberg, 1949), it strengthens the view of Brooks, Stennes and Warrack (1957) and Stennes and van Heyningen (1958) that the type F classification should be abandoned and these strains included in the type C group. Stennes and Warrack (1964) have taken this step and reclassified the type F strains as a result of the New Guinea discovery (Table XXXIV).

Schmid (1944) and Krause (1949) carried out exhaustive animal pathogenicity tests with the German strains. As well as demonstrating pathogenicity by pure culture filtrate inoculation into the thigh, peritoneum and duodenum of guinea pigs, they also inoculated bowel contents from severe cases directly into the bowel lumen which reproduced the disease. This was later confirmed by Zeisal (1949). In the intramuscular lesions a bloody gelatinous oedema was formed but very little gas was noted in the subcutaneous tissues. Due to a shortage of animals, pathogenicity tests were limited in size and it is felt that further work will be necessary to clarify this aspect of the aetiology.

The beta toxin of the New Guinea strains when injected subcutaneously into guinea pigs gave a bluish gelatinous necrotic lesion. This was the basis of the method of determining antitoxin levels (Semela, 1964). Zeisal maintained that the variable pathogenicity in his type F strains was probably due to slight differences in toxin production by the organism and host susceptibility. He believed that individual variations in this isolate pathogenicity
explained the wide range in severity of the disease both clinically and pathologically. Most German writers on the subject concluded that Clostridium perfringens type F was probably the initiating infective agent introduced in the diet. The infection failed to establish itself unless other dietary indiscretions and unknown local conditions in the upper intestinal tract were concurrently present.

McLennan (1962) in his excellent monograph on the histotoxic clostridial infections of man states "Clostridia are at most "facultative pathogens", able to gain a footing and produce these effects which we speak of as disease, only when the tissues of the primary lesion have been altered to suit their limited and peculiar needs. . . . . By far the most important prerequisite for clostridial infection (as distinct from clostridial contamination) is that there must be present an area of lowered oxidation-reduction potential."

Rising antibody levels were detected in the few protracted cases of enteritis necroticans studied. The isolation of Clostridium perfringens type C from resected bowel contents or faeces in a significant proportion of those in which coupled serological investigation was undertaken, presupposes infection by this organism. Examination of faecal and bowel material for the presence of beta toxin was not possible. This is an investigation which could well be undertaken at some future date.

Because a direct food borne epidemiological sequence was rarely demonstrated and because of the rather sporadic dispersion of the
disease, further factors in the causal mechanisms of the disease have to be considered. Attention was therefore directed to answer such questions as to why some patients failed to develop ileus, whether other exciting stimuli were operative before or after the pig meal, and why all participants of a contaminated meal were not equally affected. Some of these answers lie partly in the pathogenesis which has been discussed earlier in this chapter.

Dietary histories taken from 140 patients showed that only 7 categorically denied a pork meal. Of these 7, 4 had an earlier meal of tinned meat or fish, and 2 had eaten a meal of green peanuts the previous day. In most instances legumes and other green leafed vegetables, collectively known as "kumu" together with the staple food, sweet potato ("kau kau") and other starch foods listed earlier were eaten with the meat. The meat was freshly prepared in only 18 per cent of cases, the meat having been from 2 days to 3 weeks old. Meat, even when putrid, is eaten after re-cooking or more correctly, re-heating.

The Highlander eats his pork in huge quantities over a period of weeks or months during the longer pig festival seasons. Then follows a relative fast until the next celebration. Heavy and continuous pork indulgence therefore occurs only once or at the most, twice every 3 to 7 years or may be longer. There are occasions however, when smaller festivities as death, marriage or illness call for a small sacrifice of one or more pigs.
The mean weights of pork eaten at such a ceremony were estimated by Venkatachalam (1962) at 29 and 26 grams per day in children in the 10 to 15 and 5 to 10 year age groups respectively. At a large pig kill at Gogina the mean weight of five pork meals consumed by adults was 652 grams. Volumetric measurements were not made but the total engorgement of food at one sitting would approximately amount to between 2 and 3 litres, more than half of which was made up of pork. This unaccustomed diet is both qualitatively and quantitatively in excess of the average daily diet.

The diet of the Chiru people has been studied by several workers (Gemen and Malcolm, 1958; Venkatachalam, 1962; Bailey and Whitesman, 1963). This consisted of a daily protein intake of between 25 and 30 grams. The calorie intake was between 1850 and 2883, very little of which was made up by fat. The conclusion was that such a low protein intake, mostly arising from vegetable protein, amounted to chronic protein malnutrition, particularly in the very young and elderly. Therefore, an intestinal tract primarily conditioned to a vegetarian diet, the bulk of which is a carbohydrate staple of sweet potato, suddenly becomes confronted with large meals rich in animal protein and fat. Considerable gastric distention probably results from this but may not be significant because the stomach of the Highlander is well conditioned to meals of large bulk. As much as 1500 to 2000 grams of sweet potato make up a normal meal (Bailey and Whitesman, 1963). However, in order to handle the qualitative change in dietary
content, there must be a change in bacterial flora and enzyme secretion in the gastro-intestinal tract. In children it is possible that this change may induce bowel atony. The high helminthic load, so often present in children of the 4 to 10 year age groups, may adversely influence this change, the net result being stasis of intestinal content and bowel distension favouring the proliferation of existant and ingested bacteria. Yasnogorodsky (1963) drew attention to the production of paralytic ileus caused by an over-abundant, unaccustomed, indigestible or spoiled food. In South India, a condition of spontaneous paralytic ileus has also been reported. This is considered to be due to a diet of spoiled millet ragi, the staple food (Roantree, 1949).

Pietzonka and Rassfeld-Sternberg (1950) supported Schüts in promoting the association of an antecedent diet rich in protein, usually of improperly cooked or canned meat preparations, in persons with enteritis necroticans in Germany. Most of their patients also confessed to engorgement. Thus in Germany, soon after World War II, a similar situation existed in the population to that in New Guinea whereby a population subsisting on meagre rations, was suddenly tempted by an influx of richer foods when hostilities ceased. This unbalanced situation, in which the diet consisted chiefly of carbohydrates, is believed to have been a factor underlying the epidemics in both Norway and Germany.

Nordmann (1947) observed the disease on the Russian front in 1941
and concluded that the nutritional status of affected persons
influenced changes in their bowel environment and bacterial flora.
Dietary indiscretions by such individuals predisposed to infection by
the anaerobic clostridial organisms sometimes resident in the lower
small intestine. Siegmund (1948) and Dormanns (1948b) supported
this view and the latter writer stressed the fact that changes in
bowel motility resultant from stasis possibly led to circulatory
disturbances in the terminal collateral arterioles. This caused the
"multiple infarct" appearance of bowel in advanced cases. On the
other hand, Siegmund believed that dietary changes assisted the
fermentative bacteria to become pathogenic under ill-defined changes
in the "internal milieu" of the duodenum and jejunum. Toxins
produced by these faulty mechanisms then caused arteriolar spasm.

The experience of the veterinarians is perhaps most relevant to
the study of the ecology of enteritis necroticans in man in New
Guinea. Without being in any way derogatory, the average New
Guinese lives his life, and this particularly applies to the women and
children, in an unhygienic environment akin to that of domestic or
herd animals.

The enterotoxaemias caused by Cl. perfringens types B, C and D
in principally ruminant animals are infectious diseases that result
from the absorption of bacterial toxins formed in the intestine of
the host. The disease in lambs called "lamb dysentery" or "bloodpens"
is largely restricted to young lambs less than 2 weeks old (Bleszchner,
1951). This disease causes a loss of a few lambs in the first year and increasingly larger losses in successive years until the mortality reaches 20 to 30 per cent of the lambs. The haemorrhagic enteritis with mucosal ulceration is believed to be the result of the beta toxin. The disease in foals caused by Cl. perfringens type B (Montgomery and Rowlands, 1937) occurs in the first two days of life; the signs of abdominal pain and bloody diarrhoea being similar to those in lambs.

Calves are also susceptible to Type B enterotoxaemia. Hepple (1952) demonstrated the beta toxin in the liquid and bloodstained intestinal contents of affected animals, the pathology being the same type of haemorrhagic enteritis as in the other animals. Calves, however, took longer to die - 1 to 4 days - and some recovered very slowly over periods of up to two weeks.

Enterotoxaemia caused by Cl. perfringens type C, known as "struck" in sheep has been well known in the Romney Marsh area in England and in North Wales (McKwen, 1929). The characteristic lesions are haemorrhagic enteritis with ulceration of the mucosa, particularly the jejunum and duodenum. Dietary changes in early spring were considered to play a role in the development of the condition. Haemorrhagic enteritis in newborn animals due to this organism was not recognised until 1952, when Griner isolated type C strains from calves. A similar disease was also found in lambs (Griner and Johnson, 1954) and in piglets (Field and Gibson, 1955). From the work of Griner (1952 -
1954) it appears that type C strains are carried as part of the normal bacterial flora of the intestine of adult animals. It also seems likely that these bacteria produce some beta toxin, although in amounts insufficient to cause death or clinical disease. Grimer and Baldwin (1954) found antitoxin in the sera of 14 per cent of apparently normal cows and 24 per cent of apparently normal calves that they studied. It seems reasonable to suppose that newborn calves of non-immune cows pick up the organism from their environment, which has been contaminated by adult animals carrying the organism. The spread of Cl. perfringens type C from one infected calf to another is a very real possibility due to fodder spoilage. The disease also occurs most frequently in large calves receiving an abundance, possibly an excess, of milk in calves of beef breeds.

The signs of this disease vary with the degree of intoxication (Grimer and Bracken, 1953). Subacute cases occur with signs of diarrhoea and lack of appetite, and these calves recover in a few weeks. They showed the effect of even this mild infection by a growth rate less than normal. In more acute cases, there is loss of appetite, evidence of abdominal pain and diarrhoea in which the faeces may or may not be bloodstained. The calves are unable to rise and often die in convulsions with no rise in body temperature.

The most marked post mortem change is a haemorrhagic enteritis of the small intestine, particularly the duodenum, jejunum and ileum, with necrosis and desquamation of the mucosa, which is bright red and
velvety. The intestinal contents are fluid and dark red from haemorrhage into the lumen. The mesenteric lymph nodes are frequently congested or haemorrhagic. *Cl. perfringens* beta toxin may be demonstrated in the intestinal contents from which the type C strain can be readily isolated. The post mortem findings in cases of type C enteritis in lambs and piglets are much like those in calves, with marked haemorrhage and necrosis of the intestinal mucosa.

Enterotoxaemia caused by *Cl. perfringens* type D ("pulpy kidney" of sheep) had been known as a clinical entity for many years. Bennetts (1952) first demonstrated the causative agent in Australia. Bullen (1952, 1953) has worked out the pathogenesis of this disease which bears considerable relevance to pig-bel in man. He examined 100 normal sheep at slaughter and was able to demonstrate this organism in some part of the alimentary tract of 46 of them. In 19 cases he found it in the rumen, in 15 in the abomasum, in 14 in the duodenum, in 17 in the jejunum, in 30 in the ileum and in 22 in the colon. Determinations of epsilon antitoxin in the sera of non-immunised sheep indicate that as high as 90 per cent of a flock of sheep are, or have been, carriers of *Cl. perfringens* type D. This organism apparently is restricted to an intestinal habitat and cannot long survive in the soil, probably being unable to compete with the type A strains that are present. The epsilon toxin that is responsible for this enterotoxaemia is not produced by the bacteria in the active, toxic form. Instead, it is produced as the relatively
non-lethal prototoxin which can be converted into the active toxin by
the action of a number of proteolytic enzymes (Bullen, Scarisbrick
and Maddock, 1953).

The disease has occurred in sheep of all ages. In almost all
cases, except for day-old lambs, the enterotoxaemia is associated with
the heavy intake of nutritious food whether it be milk, fresh grass or
grain. The mechanism by which over-eating induces an attack of enterotoxaemia was further studied by Bullen and his colleagues (Bullen and
Betty, 1957; Bullen and Scarisbrick, 1957). They showed that the
disease cannot be reproduced in sheep fed on normal diets such as hay
even if large quantities of pure Cl. perfringens type D cultures were
introduced in the intestine for long periods of time. This appeared
to be due to the absence of large quantities of fermentable substrate
in normal intestinal contents limiting the growth of the organisms
and the rapid flow of the intestinal contents soon removed any
accumulation of bacteria and toxin. The disease was reproduced
consistently when the diet was changed suddenly with a large wheat
meal eaten before the culture was introduced into the intestine.

Under these changed circumstances the ruminal flora was unable to
adapt itself rapidly to the changed environment and enterotoxaemia
resulted when Cl. perfringens type D was introduced into the diet.
They showed that low concentrations of toxin were harmless, but high
concentrations, if maintained for a few hours, rapidly increased the
permeability of the intestine. Enterotoxaemia in goats due to this
organism develops under similar conditions of a sudden change to a
more succulent feed (Omer, 1956).

In veterinary medicine, enterotoxaemia has been controlled in
several ways. The amount or quality of the food may be reduced in
young lambs. This necessitates placing the ewes on a less
succulent and less lactogenic diet. The establishment of a
permanent state of immunity in sheep, as well as other aspects of
immunisation against enterotoxaemia, is an established veterinary
practice (Thompson and Betty, 1953; Montgomery and Thompson,
1954).

The pig-bell syndrome in man is analogous to the enterotoxaemias
of animals. Further research with experimental animals and the New
Guinea strains of Clostridium perfringens type C will have to be undertaken
to prove this analogy, and clarify the pathogenesis of the disease.
It is felt that the extensive experience of the veterinarians in
their field of enteric infections caused by Clostridium perfringens cannot
be lightly put aside.

Supposing that pig-bell is in some way associated with a drastic
dietary insult, the role that resident and introduced bacterial flora
play in the aetiology may be considered briefly. In man the spectra
of normal intestinal flora are not clearly understood and depend at
least in part on age, species, diet and environment of the host as
well as upon interactions that may occur between various organisms
present (Donaldson, 1964). Gowdie and Duncan (1956) have shown
that faeces may contain a substance that behaves like a trypsin which neutralizes the alpha toxin derived from Cl. perfringens. Florey (1933) made observations on the function of mucus in the early stages of bacterial invasion of intestinal mucosa. Stark (1960) demonstrated some antibiotic activity of haemolytic streptococci in the bowel. One species may inhibit another by lowering the pH or the redox potential, by producing metabolites that are toxic, or by synthesising more complex antibiotics (Donaldson, 1964). The exact mechanisms involved remain as yet unknown.

Cregan and Hayward (1953) have shown that when the human intestinal tract is healthy the small intestine is not colonised by resident flora. In further intestinal floral studies in subjects with gastric disease, they also found that the small intestine contained only a sparse transient population, irrespective both of gastric acidity and of the degree of contamination of the stomach (Cregan, Dunlop and Hayward, 1953). Bowel distension and stasis which possibly follow large meals are favourable to normal flora multiplication. Dixon (1960) has shown that micro-organisms are removed from the small bowel primarily as a result of mechanical cleansing of the lumen by intestinal peristalsis, and confirmed Florey’s work that mucus assists in this process. The development of the syndrome of enteritis necroticans clinically manifests itself early by increased peristaltic activity, seen as peristaltic waves visible in the upper abdomen. Later there is decreased activity assessed by "meteorism" and metallic
bowel sounds indicating ileus. This situation favours a rapid and
glominant growth of the anaerobic clostridial organisms initiating a
vicious cycle which, if unrelieved, results in further toxemia and
death of the patient. The organisms responsible for such must
surely be introduced per oris.

Diet has long been recognised as one of the determinants of the
bacterial types present in the intestinal flora (Dudgeon, 1923).
Although both host and bacteria appear to thrive together, the
relation between the two is not strictly commensal or saprophytic.
Some bacteria proliferate only with altered environment brought about
by diet. In the malabsorption states, Dellipiani and Girdwood (1964)
proposed the concept of "transient" and "permanent" flora. They
concluded that it is possible that faecal type flora are constantly
ingested in low concentrations, and these remain undetected until
some change in the pattern of bowel function allows their multi-
plication. This situation probably exists in the high helminthic
infestation with associated mucosal abnormalities seen in patients
followed up in the present series.

Ova of *Ascaris lumbricoides* were found in 87 per cent of severe
cases of enteritis necroticans and the rate fell to 69.1 per cent in
mild cases. These rates are higher than those quoted independently
for the Chimu by Venkataschalam (1962), and Bearup and Lawrence (1950).
Rates for the recovery of hookworm ova were 62.3 per cent and 60.2
per cent, similar to the survey results of these writers. Vines
(1964) in a recent morbidity survey covering all Highlands gave an overall ascaris incidence figure of 54.8 per cent for 241 faecal samples. Variations between different Highland areas were however considerable. Increased activity and migration of adult ascarids following pig feasts is evident by the complaints of abdominal dyspepsia, pain and passage of worms per rectum made following pork meals. The people believe that most of their ill-defined abdominal pains are due to round worms.

It is conceivable that heavy ascaris infestation influences the development of partial mechanical obstruction in the small intestine favouring strangulation. The heavier the worm load the more likely would this be so. The quantitative data failed to verify this point. One writer (Dormann, 1948a) noted the association of this helminth in cases of Darmbrand.

Hookworms by virtue of their attachment to the jejunal mucosa probably create defects in the surface continuity. This could allow access to the submucosa of toxins and bacteria, thus initiating the necrosis.

High helminthic infestation probably acts as an irritable influence in the upper small intestine, clinically evident by visible bowel loops in the epigastrum sometimes seen when ascaris infestation is high. This in itself may predispose to a mild inflammation of the mucosa, to an increase in bowel flora and to the development of mucosal atrophy.
Symptoms of distress, nausea, eructation, vomiting, indigestion, flatulence and epigastric fulness have been attributed to the ingestion of pork (Burch, Phillips and Wood, 1957). These writers in a survey of pork eating negroes of the southern United States claimed that 35 per cent of the population suffer from one or more of these symptoms. The factor in pork causing this remained unidentified. Symptoms akin to these are described by the people in New Guinea, but are regarded as essential to their enjoyment of the feast.

Just over one-third of the histopathological sections at all stages of pig-bel in New Guinea showed some eosinophilic infiltration of the bowel wall, particularly the lamina propria. This finding was more frequently seen in bowel removed from a more protracted type of disease. In a few cases where serial differential leucocyte counts were undertaken, an eosinophilia was present. These changes may well be explained by the high incidence of helminthic infestation. However, the apparent rise in prevalence as the pig feasting continued in the Chimbu, and the different cultural pattern between eastern and western Highland tribes, resulting in a higher prevalence in the former district, supports a theory that the disease may be initiated by an allergic process. This implies that a Schwartzman phenomenon may be involved (Schwartzman, 1928). Koslowaki (1950, 1951) proposed this theory as a cause of Darmbrand and supported the hypothesis by sensitising guinea pigs to beef proteins. The high antitoxin levels in the population possibly indicated repeated exposure to the antigenic
influence of *Clostridium perfringens* beta toxin. This analogy may be extended further by postulating that sensitivity to *Clostridium perfringens* type C in the allergic individual may be responsible for the jejunitis. There was no other systemic evidence of sensitivity reactions, apart from the eosinophilic infiltration in sectioned material. This, however, was not massive.

The most violent symptoms of gastro-intestinal allergy following ingestion of offending food are sudden onset of nausea, vomiting, diarrhoeas which may be bloody, and violent abdominal pain, the picture in acute toxic forms of pig-bel. A more delayed type of reaction may result in a form of chronic jejunitis, typical of the sub-acute form of pig-bel.

Host nutrition may affect the types rather than the absolute numbers of organisms in the small intestine (Dubos and Schaedler, 1960). Under conditions of poor sanitation, hygiene and marginally adequate diets, certain micro-organisms which, although moderately virulent, ordinarily might tend to overwhelm the host and establish disease when the diet is drastically changed.

A state of sub-clinical infection in the pig-bel syndrome has been neither supported or refuted by the current investigations, although the normal New Guinea population shows a high immune status. Studies by Dubos et alii (1960) indicate that the relation between a state of chronic enteritis and the nutrition of the non-ruminant host is poorly understood and at present seems complex. Experiments using
antibiotics and germ free techniques in mice raised in extremely
sanitary environments suggest that the character of the enteric flora
may influence host nutrition.

In most New Guinea Highland communities the relation of the human
host to his insanitary environment is crucial to his nutritional
state, particularly in infancy. This problem is synergistic to the
protein deficiency patterns described by Bailey (1962, 1963). If the
recurrent infant diarrhoeal attack rate could be lowered by improved
hygiene and sanitation, the problem of protein malnutrition may well
be eliminated. Undernutrition as such in the tropics may in fact be
regarded as diseases of under-developed or poor countries.

The nutritional status of patients with enteritis necroticans
was measured in general terms of appearance, skin thickness, muscle
strength, tons, skeletal development assessed where possible by height
and weight measurements. Fluid loss associated with diarrhoea tended
to give lower weights than the established weight survey for Chimbu
children (Venkatachalam, 1960). The general impression was that the
children with pig-bel were not marasmic or malnourished.

With Darmbrand, opinions on this point differed. The disease in
Germany rarely affected people from rural areas and was more prevalent
in the urban areas where post-war malnutrition was significant (Ernst,
1948). Griessman (1950) found no association between malnutrition
and Darmbrand but Kloes (1946) and Molennan (1956) suggested that
severe malnutrition showed a predisposition to necrotizing enteritis.
Fick and Wolken (1949) and Pietzonka and Rasafeld-Sternberg (1950) believed that the nutritional deficiency manifested itself in thin atomic small bowel segments which predisposed to the disease.

It is felt that pig-bel and pig-feasting in New Guinea antedates rather than follows the prevalence of severe protein deficiency states. In countries where there is the problem of malnutrition, other unhygienic conditions exist. In these human societies the multiplicity of such factors greatly complicates the problem of assessing their respective contributions to morbidity and mortality. For the present this seems to be a philosophical question. The need for maintaining a proper critical attitude in approaching the problem of nutrition must not be disregarded, especially since their solution is bound up with so many other social factors. In spite of this, consideration must be given to the nutritional status with respect to resistance and antibody formation. There is substantial evidence that serious nutritional deficiencies, particularly of animal proteins, lessen the ability of the infected host to form antibodies (Cannon, 1945). In order to cope with new infections, what is less apparent but probably equally real, is that during periods of serious malnutrition those antibodies which had been produced in response to earlier infections become utilized for other and overriding metabolic needs. The individual then becomes vulnerable to infections against which he was formerly protected. In young children, malnutrition has even graver repercussions, for not only are special amino-acids
required for current catabolic activities, but the pressure of growth makes added demands upon an already inadequate supply, so that the normal acquisition of immunity to chance infections suffers accordingly. This may assist in explaining why pig-bel predominantly affects children in New Guinea, in age groups affected most by protein undernutrition.

It has been shown that 71.3 per cent of the normal New Guinea Highland population have significant amounts of circulating beta antitoxin to *C. perfringens* type C. The age distribution of this antibody does not favour any one particular age group except possibly in the elderly. This implies that the population is constantly exposed, at all ages, to infection with *C. perfringens* type C. As with other enteric bacteria, this exposure must also occur very early in life.

Since the initial lesion in enteritis necroticans is probably of toxigenic etiology rather than infective, individuals already possessing antitoxin titre will promptly neutralise any further toxins produced by bacteria so that little tissue damage results. The protective action of specific alpha antitoxin in gas gangrene for exposed defensive cells of man was shown experimentally by Stewart in 1943, although the toxin itself is a poor antigen (McLeman, 1962). Robertson and Keppie in the same year demonstrated that protection in animals depended on the presence of a level of circulating antitoxin of at least 0.1 unit per ml. of serum; if this level were attained then massive infections could be withstood. Similar levels of anti-
toxin in sheep protected them from enterotoxaemia due to \textit{Cl. perfringens} types B, C and D (Montgomery and Thompson, 1954; Bullen, 1957).

Levels of 0.2 units per ml. were thought to be significant in assessing exposure to beta toxin by Oakley (1949) when he investigated convalescent sera from six persons with Darmbrand in Germany. It can be assumed therefore that levels of beta antitoxin of 0.5 units per ml. or more must provide some protective action to a large section of the community. The considerable fall in mortality of cases treated with high doses of \textit{Cl. welchii} type C antiserum lends support to the idea that the immune status of the community may control the prevalence of pig-bel. Probably the most significant predisposing factor in the development of severe pig-bel disease in a New Guinean is his pre-existent immune status before exposure to the toxins of \textit{Cl. perfringens} type C.

Difficulty in producing a specific gas gangrene toxoid has been experienced because of the poor antigenic properties of the alpha toxin of \textit{Cl. perfringens} type A, the main organism responsible for the disease. It has not been found possible to produce an alpha toxoid which is constantly antigenic and protective (McEwan, 1962). The beta toxin, by contrast, has excellent antibody stimulating properties and specific toxoid production for prophylactic use in New Guinea is a practical possibility. Such preparations are widely used in veterinary practice. The antiserum provided for therapeutic trials in New Guinea was originally prepared for sheep, being first tested and
and adapted for use in man by the Wellcome Research Laboratories.

The early diagnosis of pig-bel was important when therapeutic measures were considered. The development of threatening complications worsened the outlook for the patient. Because the clinical features were variable, diagnosis was sometimes difficult.

Staphylococcal and anthrax enteritis were initially excluded on bacteriological grounds (Murrell and Roth, 1963). Attention was directed to intestinal anthrax, which has the pathological features of a haemorrhagic enteritis, because pulmonary anthrax was an established disease in pigs in New Guinea (Symes, Egerton and Clatworthy, 1958). Dysenteric malaria was a possible cause but only 11 per cent of patients in the series had parasites in the thin peripheral blood films. Histopathological section did not show either parasites or malarial pigment. Bacillary dysentery and salmonella causes were looked for but not found.

Bowel gangrene complicating dysenteric, typhoid or paratyphoid infections are rarely recorded in the literature. Morrow (1955) had experience with one case in New Guinea during the last war in which poor nutrition and hypotension played important roles in the pathogenesis. Gangrenous infection of the gastro-intestinal tract has been reported with other coincident bowel pathology such as carcinoma (Wyman, 1949), peptic ulceration (Fethers, 1959), bowel perforation, (Silver, 1961), appendicitis (Capiello, Liccose and Crecca, 1950) and in association with leukaemia (Boggs, Frei and
Thomas, 1958). Fine (1959) has shown that in dogs, shock becomes irreversibe because of damage to the intestine. Marston (1962) has postulated that in the older age group, pre-existing ischaemia of the bowel is an important cause of the numerous syndromes of enteric necrosis described in the elderly, debilitated patient with a fatal illness. Berger and Byrne (1961) also reviewed 23 cases of non-corlusive massive bowel infarcton associated with decompensated heart disease. The pathological findings of intracapillary and arteriolar fibrin thrombi in the mucosa and submucosa leading to infarct necrosis in pig-bel, is similar to that in the syndromes reviewed by Marston. However, pig-bel was not a disease of the elderly nor of the debilitated patient with other coexistent chronic disease. Shock, however, probably adds to the overall clinical and pathological picture in cases of neglected pig-bel. It is possible also that the endotoxins of E. coli may be sympathomimetic adding to the overwhelming toxemia produced by the clostridial toxins (Carter and Ashley, 1960).

The diagnosis of pig-bel rested upon an integration of the dietary history, clinical features, X-ray appearances, isolation of Cl. perfringens type C from resected bowel or aspirated jejunal contents, and the detection of beta antitoxin in the sera of convalescent patients.

Patients with the severe disease presented as surgical emergencies. Experience in their management indicated that early vigorous
conservative measures were life-saving. Evidence against the
continuation of the initial conservative approach was the higher
mortality figure in patients in which surgery was delayed. Opinion
has been divided as to whether laparotomy should be undertaken in the
acute disease.

Conservative treatment was recommended initially by many authors.
(Beckermann and Laas, 1946; Brynjulfsen, 1948; Dammermann, 1947;
Dormannes, 1948; Fick and Wolkers, 1949; Lesius, 1949; Williams and
Pullan, 1953). When gangrene, perforation, stenosing cicatrization
that threaten perforation, intestinal obstruction, or bleeding was
present, resection was essential, despite the poor condition of the
patient. Lesius (1949) expressed the view that late complications
and recurrences of Barmbrand required more active surgery. Wiklander
(1964) considered that laparotomy and resection were governed primarily
by the severity of the pathologic changes and the presence of
intoxication. In the view of the author, the degree of toxaemia was
proportional to the extent and severity of the necrosis in the acute
surgical disease. Surgery, until the introduction of specific Cl.
perfringens type C antiserum, was considered to be life-saving in
cases of New Guinea. A conservative "wait and see" attitude was
certainly not in the best interests of the patient once complications
became manifest. In the early toxic stage, however, the decision
became a difficult one. Lesius (1949) considered that the great
danger in the acute stage was of a toxic nature and was combated
better by conservative measures than by early operation. He
maintained that the best results were obtained if operation could be
performed in the non-toxic stage. He based this conservative
approach on the fact that intestinal perforation did not occur in
the acute stage, but only when 6 to 10 days had elapsed. One of his
conditions for conservative treatment was that the patient should be
kept under careful surveillance to detect complications, if any.
These optimal conditions were difficult to reach with hospital
facilities in New Guinea. For this reason, a more radical approach
to the management of pig-bel was adopted. Early experience, when
limited resections were undertaken, often resulted in an extension
of the process beyond the anastomosis which frequently broke down.

It has been shown that most of the small intestine can be
removed (Meyer, 1962; Trafford, 1956). The most extensive resection
undertaken with survival in a case of pig-bel was about 250 cm. The
long term effects of resections over 200 cm. will need to be followed
up at a later date, but certainly the immediate prognosis improved
when more extensive resection was undertaken in severe cases.

The observations recorded in the follow-up studies demonstrated
some residual malfunction of the upper intestinal tract. The
intestinal biopsies indicated two abnormal groups on the basis of
whether the mucosa appeared "flat" or had a clubbed villous pattern.
The former, which is morphologically similar to that found in sprue
and idiopathic steatorrhoea (Fons, Cooke, Maynell, Brewer, Harris and Cox, 1960) was a constant finding in the more chronic forms of pig-bel. Histological pictures similar to that seen in adult coeliac disease and tropical sprue have been observed in neomycin-induced malabsorption (Jacobson, Prior and Falston, 1960), in hookworm disease (Smeby, Meroney, Cox and Soler, 1962), in giardiasis (Townley, Cass, Anderson, 1964), bacterial dysentery (Sprins, 1962), in some cases of post-gastrectomy steatorrhoea (Baird and Dodge, 1957) and in post-irradiation enteritis (McGovern, 1963). Layrisse, Blumenfeld, Carbonell, Desenne and Roche (1964) in studies of hookworm disease and intestinal absorption concluded that hookworm infection per se, without the association of other factors such as malnutrition and other parasitic infections, does not as a rule lead to histological changes in the mucosa which might cause malabsorption of nutrients. King and Joake (1960) also reported two cases of malabsorption with similar mucosal appearances following enteric infections.

In sections from resected sub-acute cases of pig-bel, the intervening mucosa between areas of ulceration showed the flat atrophic pattern of sprue. The abnormal motility resulting in such instances will influence the existence of an abnormal bowel flora in the upper small intestine. The low serum folate levels could be due to this or the associated helminthiasis (Layrisse, Blumenfeld, Dagarte and Roche, 1959). The comparatively normal vitamin B₁₂ findings were possibly related to the coprophagic contamination of food occurring
with the insanitary environment.

Hartsberg (1954) believed that a transition from an acute to chronic necrotising jejunitis does not occur. Sub-acute and chronic forms of Darmbrand, regarded as complications, were not uncommon in Germany (Ruppert, 1947; Hansen, 1949; Jeckeln, 1949). The transition from an acute to a chronic ulceration with fibrosis was dependent upon the degree of initial strangulation. The exact proportion of cases in which this took place, resulting in chronic ulcerative jejunitis, steatorrhea and malabsorption was not accurately determined. Spontaneous cure must have occurred in most of the less severe instances. It is felt that a longer time interval in the follow-up examinations for an assessment of the long-term effects of the disease will be necessary before any meaningful conclusions can be drawn.

The very serious nature of pig-bel and the mortality that results from it provokes the response that "prevention is better than cure", both for the exposed population and the health budget.

In considering the prevention of pig-bel in New Guinea, the physician is faced with the problem of unhygienic pork consumption and its cultural ramifications. Another problem in planning a control programme is the present lack of an adequate supporting economic and educational system.

Data obtained in the epidemiological studies are essentially qualitative and, therefore, an inadequate measure for evaluating
control. The prevalence of specific enteric pathogens is usually too low, even in under-developed countries having areas of high intensity, to provide a statistically reliable index and this is certainly the case with enteritis necroticans and the distribution of *C. perfringens* type C.

The increase in hospital admission rates for diarrhoeal diseases following large pig feasting activities, indicates that control of the physical factors involved in the spread of pig-bel and other food-borne diseases must focus on pork-eating habits, although the public health significance of this disease and other diarrhoeal infections is related to combinations of other sanitary and environmental factors. Rates are high in all countries where one or a combination of the following conditions exists: (1) An inadequate knowledge of personal hygiene to prevent the transmission of enteric organisms by personal contact; (2) Sanitation facilities are inadequate to prevent contamination of the environment with human excrement; (3) Economic resources are insufficient to provide enough educational and nutritional requirements; (4) Maternal care is inadequate to protect children from infection; (5) Medical resources are inadequate or not used.

Experience indicates that diarrhoeal diseases and the accompanying mortality diminish in the face of economic development with the improvement of living standards. But, the difficult question is where to begin a control programme against such
formidable problems whose causes are deeply rooted in the society
and whose elimination depends upon extensive economic and social
changes.

Rural health centres are now developing in the Highlands of
New Guinea following the establishment of Local Government
Councils. The aim of these centres is to promote and improve the
health of the Highlander. The following plan for the control of
pig-bel should result in a general reduction in morbidity and
mortality from all diarrhoeal infections of food-borne origin.
These recommendations were put to the Papua and New Guinea Medical
Research Council (Murrell, 1964):

A. Education

1. An increased awareness of the disease and its proper
management will reduce the immediate mortality. Most
medical workers in the Highlands are still unaware of
the problem, particularly of the mild and sub-acute
forms of the disease which, if unrecognised, remain
ineffectively treated.

2. Inclusion of the condition in the syllabus of teaching
establishments run by Missions and the Department of
Public Health is suggested which would help in the
recognition of the disease. Specific instruction to
health inspectors in pig slaughtering and meat
preservation would also be necessary.

3. General education of the public may be promoted through agencies of hospitals, aid posts, rural centres, child and maternal welfare clinics, local government councils, women's welfare clubs, schools and so on. This could be achieved by visual aids developed round the story of pigs which should be most acceptable and topical for adaptation in the Highlands. In larger Highland centres press and radio are becoming an effective education media.

4. A programme for training indigenous agricultural field workers in hygienic slaughtering and carcass preparation will become necessary as well as one concerned with simple pork curing and meat preserving methods.

B. Pig Husbandry Improvements

A complete appraisal of this aspect of control relevant to gastro-enteric infections is outside the medical officers' sphere of influence. However, it is apparent that little has been achieved thus far in improving and developing pig rearing as an industry.

Small distributions of European type pigs have been made to improve pig herds in the Highlands, but these pigs are previously reared in pens at a piggery. There are no figures available to refute a statement that in its own environment a native pig does better than a European or crossbred pig. The few available figures on the
growth rate of Berkshires introduced to villages are in fact alarming (Egerton, 1964). Therefore, the development of small scale piggeries in association with Local Government Councils or co-operative societies would do much to improve standards of husbandry and stimulate an industry.

C. **Improving Slaughtering Techniques**

Environmental sanitary projects are now being introduced through Local Government Councils in the Highlands, and it seems feasible to include in the plans for these projects, simple slaughtering floors or sheds, designed and built of local materials according to specifications laid down by the Agricultural Department. Facilities for hygienic killing would have to be made available within walking distance of the majority of pig owners. Pigs being killed for feasting, bridal payments and credit exchange could be brought to the floor and bled and prepared under hygienic conditions. Conceivably, the gastro-intestinal tract could be taken as a slaughter fee and the dressed carcase and solid viscera returned to the owners.

With reference to the Slaughtering Ordinance 1951 and its specific provisions (Egerton, 1964), it is essential that its application in the Highlands should not be allowed to interfere with the enormous increase in the standard of hygiene of pig-killing which these simple facilities described would provide.
D. Pork Curing

This could be developed along the lines of:

1. "Do-it-yourself" smoking of ham in small central
   smokehouses set up a month or so before the pig-kills.
   The idea of smoking pandanus mats for storage is not
   foreign to Enga and Duman cultures. As a pilot project
   one man (preferably an influential co-operative leader)
   could be persuaded to cure some sides in readiness for a
   distribution and the selling point could be that such
   pork would travel further and last longer, so that bigger
   and better debt relationships would be possible.

2. Alternatively, pork salting alone might be investigated
   by placing salting bins strategically near slaughter-
   houses. Salting of beef has been adopted by some
   Northern Territory Aborigines following experience on
   cattle stations (Meggitt, 1963 - personal communication).

3. A meat industry would be a sounder means of introducing
   meat preservation techniques due to its economic potential.
   Pigs could be sold to Local Government Councils or to a
   co-operative buying organisation for sale and distribution
   in larger centres such as Kainantu, Goroka, Kundiesa,
   Minj, Mount Hagen, Wabag, Mendi and Tari. The floors at
   these places would have facilities for meat preservation,
   salting, smoking, canning or sausage making. Many
Missions now operate small scale plants for local staff requirements, e.g. Wingende R.C. Mission, Chirumu.

E. Immunisation

Direct immediate control of enteritis necroticans could be achieved by the use of a toxoid anti-welchii C serum as with tetanus prophylaxis. The antigenic stimulus of the beta toxin of Cl. perfringens type C is good in sheep (Thompson and Betty, 1963; McLerran, 1963) and this study indicates that it is in humans. Alternatively, a mass cover of the population before pig feasts with antitoxin inoculations may be feasible although this may not be practicable or acceptable with the people. Immunisation in infancy along with tetanus, diphtheria and whooping cough antigen as is now conducted at infant welfare clinics may provide the easiest and most immediate results of controlling pig-bel. Further research is needed to discover whether this is likely and feasible.

The long term effects, if the above changes took place, are particularly applicable to the Chirumu and Webag area, and may be summarised as follows:

1. A change in pig husbandry methods from a nomadic type of foraging to one of fenced pigeries would be labour-saving in that gardens would not have to be fenced, and timber needs would be considerably reduced. More land for the cultivation of subsistence and cash crops would also become available,
particularly in the two areas mentioned above.

2. Pigs confined to piggeries would do much to raise sanitary norms in villages or hamlets where, at present, they roam and defecate at will.

3. At pig feasting activities, meat killed and preserved correctly would provide a more balanced distribution of available animal protein, thus assisting present attempts to improve the low protein diet of the Highlander (Bailey, 1963a, 1963b). It would also help eliminate one of the possible predisposing causes of pig-bel, pork engorgement and reduce the probability of food contamination and poisoning.

4. Through the media of improved slaughtering floors, adult education in improved personal hygiene would provide a valuable adjunct to general health education in food preparation.

5. The basic cultural pattern of pork exchange and barter would be preserved and uplifted as bigger and better debt relationships would be possible, because the meat would last longer. This is an important consideration now that migrations away from clan boundaries for work and other reasons are accepted cultural changes, necessitating longer distances for pork exchange.
6. A new industry would be established in the Highlands of benefit to the general economic development of the country as a whole. Further by-product industries would also evolve in support of this industry.

7. The plan is particularly adaptable to areas of high population density and could be moulded to a developing cattle industry in areas where there is more available land.
CHAPTER VI

SUMMARY AND CONCLUSIONS
This thesis concerns the investigation of a gangrenous enteritis associated with the practice of pork feasting in the New Guinea Highlands. The disease was first noticed in subjects during laparotomy at Goroka in 1961. Pig-bel (enteritis necroticans) was defined as an acute necrotising inflammatory disease of patchy distribution of the small bowel commencing in the upper jejunum. It was characterised clinically by severe upper abdominal pain, bloody diarrhoea and nausea with occasional vomiting. The disease progressed, if untreated, to complete segmental gangrene of parts of the small intestine with the development of mechanical ileus, oligaeemic shock and severe toxemia. A small proportion of persons with the disease survived the initial stages without treatment, but perforation and peritonitis or the development of an acute malabsorption syndrome resulted in death. The malabsorption was due to chronic small bowel obstruction by adhesive bands, stenosis by cicatrization or the development of short circuits, blind loops and rigid scarred bowel segments denuded of normal mucosa. The histopathological findings were non-specific except for the presence of angioneurosis and thromboses in the mucosal and submucosal vessels which resulted in ischaemic and haemorrhagic necrosis. There was no apparent mechanical or vascular cause for this. Confusion in terminology with this disease and a large group of allied disorders has necessitated the designation of the specific name "Pig-bel" to the condition.
A similar disease, "Darmbrand", appeared in epidemic form in Northern Europe in the latter and post-war years, and was believed to be due to Clostridium perfringens type F. The pig-bell syndrome has been classified clinically into four groups: acute toxic, acute surgical, sub-acute surgical, and mild forms. The diagnosis was established on the history of pork consumption, the clinical features, X-ray findings, bowel appearances at operation or autopsy, the isolation of Cl. perfringens type C from a significant proportion of resected intestinal segments, and the detection of rising Cl. perfringens beta antitoxin levels in the sera of recovered patients.

In the treatment of pig-bell, it has been shown that the prognosis improved with early resuscitation with blood and bowel decompression. In patients treated surgically by bowel resection the case mortality was slightly lower. The indications for laparotomy were signs of peritonitis, failure to relieve an ileus after 24 hours, the development of "metallic" bowel sounds despite decompression and intravenous fluids, or the continuation of abdominal pain and the palpation of a tender upper abdominal bowel mass. Loss of weight, a continuing refusal of food, anaemia and symptoms of sub-acute small bowel obstruction some four weeks to six months later were also indications for operative treatment. Following the introduction of specific Cl. perfringens type C antiserum in June
of 1964, the fatality rate in severe forms of the disease fell from 43.9 per cent to 18.7 per cent. Many antibiotic combinations were tried empirically until it was found that *Clostridium perfringens* type C was most sensitive to penicillin and tetracycline.

The following conclusions from epidemiological studies were made:

1. Enteritis necroticans has not been reported in epidemic members elsewhere than in North Germany and New Guinea.

2. The disease appeared in both epidemic and sporadic members in New Guinea and was basically confined to the Highlands region. Only two sporadic cases were reported outside the Highlands region, one of these being a Highland labourer working on the coast. Of the 210 cases reviewed only one was a European, the others all being Highland Melanesians.

3. The disease was the commonest acute abdominal condition requiring surgical treatment in Highland hospital practice during the years 1961-1964 under review.

4. The incidence of pig-bel varied from an approximate lower limit of 16 per 10,000 to a crudely estimated upper limit of 48 per 10,000 of the population.

5. *Clostridium perfringens* beta antitoxin levels in the normal population in high and low prevalence areas differed significantly.
6. Levels of beta antitoxin in the overall native population were significantly higher than in European controls. This indicated a constant exposure to *Cl. perfringens* type C by the Highland populations sampled.

7. Pig-bel predominantly affected children under 10 years of age and had a sex distribution of 2:2:1 in favour of males.

8. The mortality rate in 210 cases was 36.2 per cent. This was 84.6 per cent for acute toxic forms and 42.0 per cent and 43.5 per cent for the two surgical forms of the disease respectively.

9. Some strains of *Cl. perfringens* isolated from cases of pig-bel have been placed, on toxicological and morphological grounds, in the type C group. As such, strains of this organism have not previously been recovered from man. As a result of this discovery, the strains of *Cl. perfringens* type F, believed to cause "Darmbrand", have been reclassified on toxicological grounds as a type C variety.

10. There was an increased prevalence of the disease together with other enteric infections following large scale pig-killing and feasting ceremonies. Qualitative evidence indicated that these cultural activities were involved in the epidemiology of the disease.
11. The disease was established in more than one member of a family on only three occasions. The failure of contacts to develop the disease does not favour a simple food poisoning epidemiology.

12. PORK may act as a vector in the transmission of the disease, but there was no bacteriological proof of this.

13. The origin of the suspected cause, _Clostridium perfringens_ type C, remains unknown, although it seems likely that the pigs harbour the organism. It is possible that a somatic transfer of the disease from pigs to man may have taken place.

It must be emphasised that some aetiological features of pig-bel remain obscure. It was established that infection with _Clostridium perfringens_ type C occurred, but the sequence of events leading up to the establishment of this infection may involve a number of mechanisms, superadded or otherwise.

The most logical cause for the gangrene seems to be invasion by clostridial organisms, in particular _Clostridium perfringens_ type C. The environmental changes induced by a dietary change and engorgement of pork and the individual's immunity probably play the most significant roles in the severe pig-bel. This situation is analogous to the enterotoxaemia of animals caused by type B, C and
D organisms of the \textit{Cl. perfringens} group.

A review and study of the cultural aspects of pig-feasting have been undertaken and a suggested programme put forward to control the disease. These public health measures, if adopted, may help to reduce the prevalence of diarrhoeal diseases associated with the practice of unhygienic pork consumption. An exploratory trial of a toxoided preparation of specific \textit{perfringens} beta antitoxin could also be tried in the immediate control of the disease.

The discovery of pig-bel and its likely cause, opens the way for a reappraisal of the significance of \textit{Cl. perfringens} as an enteric pathogen in man. The importance of typing such strains in the diagnostic and epidemiological study of necrotising enteritis, pseudomembranous enterocolitis and similar synonymous conditions is brought into relief by this work. Attention is also directed to the role that dietary influences may play in the etiology of such diseases. Finally, this study is a demonstration in ecology of a disease or syndrome in man in New Guinea. In determining the pattern of disease and its control in a particular location, factors to do with man himself: his race, society, customs, attitudes, education and movements are necessary foundations upon which to build a healthier community.
Appendix I

Department of Public Health
Goroka
Eastern Highlands
5th August, 1961

CIRCULAR MEMO

All Stations Highlands Region

Subject: Enteritis Necroticans in the Highlands

Since January of this year several deaths have occurred at the Goroka Hospital from this disease.

Symptoms of an acute gastro-enteritis, with vomiting and diarrhoea, precede signs of ileus and diffuse peritonitis which develop over a period of from three days to one week.

At operation or post-mortem there is seen several transverse haemorrhagic necrotic areas in the upper and mid-jejunum, with or without perforation.

Investigations have revealed several common factors:

1. Usually follows the ingestion of putrefied pork;
2. Highest proportion of cases in children due to the delayed consumption of pork after pig feasts;
3. Concurrently several cases of gastro-enteritis occur which recover.
4. Clinical details accompanying the patient or specimen should include details of previous diet, onset and duration of symptoms (an accurate time relationship is essential), course of the illness and the date and time of obtaining specimens. Name, age, sex, village, nutritional state, height and weight must be included.

5. If possible every attempt should be made to obtain specimens of suspect food previously eaten by the patient and/or relatives.

The disease poses a considerable threat to the lives of many Highlanders and your co-operation in the gathering of basic epidemiological data is an absolute necessity before an attempt can be made to introduce effective control measures.

F.L.C. Tuza
Regional Medical Officer
On behalf of Dr. T.G.C. Murrell
Appendix II

Department of Public Health
Goroka
Eastern Highlands
8th April, 1963

CIRCULAR MEMO

All Stations Highlands Region

Subject: Enteritis Necroticans in the Highlands

A circular concerning this problem was sent out in August, 1961. In the period between then and now little progress has been made in elucidating the epidemiology of this disease which is a serious health problem in the Highlands and probably preventable.

Initially the symptoms are those of an acute or subacute gastro-enteritis which lasts for periods from two to twelve hours. The patient may recover or die (sudden fulminating type) during this stage. In some cases severe upper abdominal pains (colic at first) then precedes the development of distension and signs of ileus and/or diffuse or localized peritonitis. A small proportion of cases do recover during the initial phase of this stage but only after a stormy and protracted convalescence with complications of small bowel stenosis, short circuits and/or malabsorption syndrome developing some weeks later.
Once paralytic ileus becomes established it has been shown that resection of the affected small bowel segments provides the best chance of survival. Necrotic areas commencing in the upper jejunum are seen as purplish congested patches initially which become confluent and eventually gangrenous.

One common factor is that the disease follows the ingestion of putrefied pork and the disease initially therefore is one of food poisoning. A specific organism has yet to be determined, should this be the cause.

It is requested therefore that the following procedures be undertaken:

1. Any infected case must be reported by radio to Garoka immediately and steps taken to transfer the patient urgently without waiting prior approval.

2. If the patient cannot or refuses to be transferred, specimens of vomit, faeces and bowel (if resected) should be placed in sterile bottles, deep frozen and forwarded packed in ice in an ordinary thermos.

3. All patients dying from this disease must have a post-mortem performed by the nearest Medical Officer who, if necessary, must fly to an E.M.A. hospital to do it. Specimens can then be forwarded by the method described above.
A *Clostridium welchii* organism has been isolated from the gangrenous cases but as yet has not been fully identified.

It has been ordered by Headquarters that this disease is notifiable in the Highlands Region and any suspected cases must be reported to Goroka immediately - by radio. A letter should follow in which the name, age, sex, place, history and examination findings are given, also if operation or post-mortem was performed to make a diagnosis. (Operation and post-mortem to be performed by Medical Officers.)

Specimens of faeces and any vomited material should be forwarded to Goroka for culture.

As a prophylactic measure propaganda should be spread to emphasise the necessity of cooking all slaughtered pork immediately and consuming same within six hours.

Patients who have gastro-enteritis and develop ileus or signs of peritonitis should be forwarded urgently to Goroka - it will not be necessary to obtain the prior approval of Goroka for any such transfer. However, please be sure to forward an explanatory letter with the patient.

F.L.C. Tuma, A/R.M.O.
On behalf of Dr. T.G.C. Murrell.
Appendix III

Department of Public Health
Goroka
Eastern Highlands
29th April, 1963

Circular to: All Medical Officers, Highlands Region

Subject: Clostridium welshii Enteritis

Mr. J.R. Egerton, Bacteriologist, Department of Agriculture, Port Moresby, is willing to do the bacteriological part of this research.

He has requested that samples should be forwarded to him in the following manner:

(a) Pack all samples with ice in a thermos.

Thermos is available from R.M.O. Goroka.

(b) Consign samples, in thermos, to

Veterinary Laboratory,

Port Moresby.

(c) Advise this laboratory by radio of consignment.

Material from cases:

(a) Medical cases

1. Fecal samples - at least 5 grams.

ii. Vomitus if present.

iii. Acute and convalescent serum.
(b) **Surgical cases**

1. Pre-operative bowel content (enema).
2. Vomitus if present.
3. Resected gut with gut content tied to either end placed with aseptic technique in plastic bags then into thermos container with ice.
4. Acute and convalescent sera.

   All samples should be taken into sterile containers. In association with clinical and surgical cases a serious attempt should be made to obtain samples of pig meat.

**Meat samples**

A sample of at least 60 grams (2 oz) is preferable. To prevent contamination from one sample to the next, samples should be packed in individual plastic bags and between sampling from different carcases knives should be sterilized or scalpel blades changes.

F.L.C. Tusa,
A/Regional Medical Officer
on behalf of Dr. T.G.C. Harrell
and Mr. J.R. Egerton.
### Appendix IV

#### AGE

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#### HISTORY:

1. **Dist:** Not recorded

   - **Pork eaten:** Yes (X)
   - **No** (Y)

   **Age of Pork:**
   - Incubation: 0-10 hrs (K)
   - 10-20 hrs (L)
   - 20-30 hrs (M)
   - 30-40 hrs (N)
   - 40-50 hrs (O)

2. **To Hospital/Operation/Death:**
   - 1-3 days (P)
   - 4-7 days (Q)
   - 1-13 days (R)
   - 14-21 days (S)
   - 22-28 days (T)
   - 29-35 days (U)
   - 36-42 days (V)
   - 42+ days (W)

#### EXAMINATION:

1. **General Hippocratic facies**
   - **Nutrition:** Good (36)
   - **Fair** (37)
   - **Poor** (38)
   - **Shocked/Dehydrated** (39)
   - **Enlarged parotids** (40)
   - **Furred tongue** (41)
   - **Abdominal distension** (42)
   - **Visible peristalsis/ Meteorism** (43)
   - **Tenderness** (44)
   - **Rebound** (45)
   - **Bowel sounds:** present (46)
   - **absent** (47)

2. **Symptoms:**
   - **Nausea/Anorexia** (1)
   - **Flatulence/Eructation** (2)
   - **Vomiting** (3)
   - **Ascaris passed** (4)

   **Offset** | **Severity** | **Duration**
   ---|---|---

   **Liver palpable** (48)
   **Spleen palpable** (49)
   **Pulse rate**
   **Temperature**
   **Blood pressure**
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<td>Diarrhoeas</td>
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</table>

**Weight**

- **Height**

2. Other systems:

3. Investigations:

- **Urine**: Albumin (50), Sugar (51), Bile (52), Urobilinogen (53)
- **Micro urine**
- **Faeces**: Faecal fat
- **Occult positive** (54)

4. Treatment:

- **Sulphaguanidine** (16)
- **Sulphathalidine** (17)
- **Penicillin** (18)
- **Streptomycin** (19)
- **Chloromycetin** (20)
- **Tetracycline** (21)
- **Neomycin** (22)
- **Agga/type C anti- serum** (23)

3. Family history: Others ill: Details

- **Yes** (14)
- **No** (15)

4. Treatment:

- **Ascarisiasis neg.** (55)
- **Moderate** (56)
- **Heavy** (57)
- **Hookworm** (58)
- **WBC** (59)
- **Muscle fibres** (60)

**Culture**:

- **Cl. welchii neg.** (67)
- **Type A** (65)
- **Type C** (66)
- **Other pasts.**
- **Not examined** (67)
- **Vomit stain**
- **G. pos. rods** (68)
- **Not examined** (69)
Resuscitation:

**Fluids** (24)

**Blood** (25)

**Suction and Decomp.** (26)

**Operation:** Yes (27)

**No** (28)

Resected length: 0-50 cm (29)

50-100 " (30)

100-150 " (31)

150-200 " (32)

250-300 " (33)

-300 " (34)

5. Result: Recovery (35)

**Death** (U)

6. Classification:

**Acute Toxic A.**

**Acute Surgical B.**

**Subacute Surgical C.**

**Mild O.**

7. Provisional diagnosis

8. Date admitted:

9. Date operated:

10. Date Discharged/Died:

11. Days in hospital:

**Blood:**

**BSR**

**Hb**

**WBC**

**Differential**

**Malaria smear**

**Electrolytes:** K

**Na**

**Cl**

**Total serum**

Cl. welchii beta toxin antibody

**Salmonella agglutination**

**MUN**

**Hauttest**

**Jejunal biopsy**

**X-Ray:**
## Appendix V

### Follow-up

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| Classification: | Acute toxic |
|                | Acute surgical |
|                | Subacute surgical |
|                | Mild |
| Autopsy: | Date: |
| Gross pathology: | |
| Microscopic pathology: | No. |
| Stool/culture: | bowel |
| Urine: | S.G. |
|         | Albumin |
|         | Sugar |
|         | Urobilinogen |
|         | Bile |
Investigations:

Blood:
- B.S.R.
- Hb.
- R.B.C.
- M.C.V.
- M.C.H.C.
- M.C.H.
- C.I.

Smear:
- Serum folic acid
- $B_12$

Proteins - total serum protein
- albumin
- alpha 1
- alpha 2
- beta
- gamma

- total serum globulin

Serum electrolytes:
- potassium
- sodium
- chloride

Glucose tolerance
- Cl. perfringens beta antitoxin

Feces:
- Ascaris
- Hookworm
- Meat fibres
- Fecal fat
- Cl. perfringens type A
- Other pathogens type C

Examination (cont'd)
- Glossitis
- Nail clubbing
- Skin changes
- Petechiae
- Sensation
- Abdominal distension
- Abdominal tenderness
- Peristaltic movements
- Liver
- Spleen

Bone marrow:
- Jejunum biopsy:
- X-ray:
- Barium meal
- Long bones
Appendix VI

The main antigen in *Cl. perfringens* type C is the beta toxin which is also the main antigen in *Cl. perfringens* type B. This is tested *in vivo* by intracutaneous injection into the flanks of depilated guinea pigs. The toxin gives a purple necrotic lesion which can vary from 5 mm. to 20 mm. in diameter and which may be spreading in character owing to the presence of hyaluronidase in toxic filtrates from the organisms. The effect of the toxin can be blocked by serum containing beta antibody.

A titration is performed with a known amount of toxin and the unknown serum. The same amount of toxin is put into each of several tubes and varying amounts of the serum added, the amounts varying in geometric progression. 0.2 ml. of the mixture is injected intracutaneously into the prepared guinea pig and the lesion examined after 48 hours. An over-neutralised mixture (too much antibody) results in a negative reaction and an under-neutralised mixture (too much toxin) results in a positive necrotic reaction. The neutral point lies in between a negative and a positive reaction and an exact point of neutralisation is indicated by a tiny (2 to 3 mm. diameter) reaction. The number of units of antibody per ml. in the serum can then be worked out as the number of unit equivalents of toxin in the mixture is known.
# Appendix VII

## Patient Histories

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