Copyright is owned by the Author of the thesis. Permission is given for a copy to be downloaded by an individual for the purpose of research and private study only. The thesis may not be reproduced elsewhere without the permission of the Author.

# THE SIGNIFICANCE OF "NAVEL ILL" AND OTHER LESIONS AT POST-MORTEM INSPECTION OF BOBBY CALVES

## MONIQUE ELISABETH BISS

A Thesis presented in partial fulfilment (70%) of the requirements for the degree of Master of Veterinary Science.

Massey University, New Zealand

1990

#### **ABSTRACT**

Calves slaughtered for bobby veal in New Zealand are considered a public health risk if "acute inflammation of the umbilicus" is present at slaughter, because it is believed that the consumption of veal derived from these calves may subsequently "cause food-poisoning" as a result of bacterial dissemination throughout the carcass. This belief, however, has not been fully validated.

During the 1989 season, 54 calves were condemned for "navel ill" (0.465% of the total slaughtered) at Waitaki International's Feilding export slaughterhouse. Carcasses from these calves, along with 31 normal carcasses, were examined for the presence of septicaemia (Part I). The study involved the detailed gross examination of the carcass and viscera, together with microbiological examination of umbilical vein, liver and muscle, and histopathological examination of the umbilical vein, liver, and kidney. The working definition of septicaemia for the purposes of this study was the presence of large numbers of bacteria in the general circulation, involving slight or absent clinical signs and with or without gross evidence of early systemic infection in the carcass. Bacteraemia was defined as the presence of smaller numbers of bacteria in the general circulation, with or without gross evidence of localisation in the carcass. Although differing from the several definitions, particularly of septicaemia, in the literature, these were proposed as appropriate definitions in the context of the slaughterhouse. The presence or absence of septicaemia was determined on the basis of the combined gross lesions in the carcass, histopathological lesions in the liver and kidney, and microbial isolates from the liver and carcass musculature. The presence of infection extending from the umbilicus was determined by histopathological and microbiological examination of the umbilical vein near the liver.

"Navel ill" could be sub-classified into three categories:

- (1) Umbilical vessel infection and carcass lesions indicative of systemic spread, with or without umbilical infection.
- (2) Umbilical vessel infection, with or without umbilical infection.
- (3) Umbilical infection alone.

Three carcasses condemned for "navel ill" had no abnormalities of the umbilicus or umbilical vessels. Of the remaining 51 carcasses, one (2%) had gross, histological and microbiological evidence of septicaemia, and seventeen (33%) had lesions indicative of bacteraemia or septicaemia. The proportion of condemned carcasses which were bacteraemic decreased as the condemnation criteria expanded to include all three categories of "navel ill". There was a significant risk of bacteraemia in carcasses from all sub-categories of "navel ill", except category (3), when compared with normal carcasses. Insufficient data were generated to allow assessment of the risk of septicaemia being present in carcasses from calves with "navel ill".

A second study was undertaken later in the 1989 season, and involved the detailed gross examination and description of 371 calves condemned for any reason at six export slaughterhouses; five in the North Island and one in the South Island. The major disease entities found in calves of this age and resulting in carcass condemnations were "navel ill" (197 (0.50%) carcasses), pneumonia (75 (0.19%) carcasses), arthritis (31 (0.08%) carcasses, and "white spotted kidneys" (30 (0.08%) carcasses). Peritonitis, jaundice, hepatic abscesses and "fever" occurred at very low rates (< 0.03%), while other lesions occurred sporadically.

The current inspection system in New Zealand requires calves with "acute inflammatory lesions" to be condemned. In this study, this requirement resulted in the condemnation of virtually all diseased calves, whether the disease was acute, chronic, generalised, localised or non-infectious in nature. There was, however, considerable variation found in the severity, age and likely pathogenesis of the lesions in each disease category. Because this "blanket" approach to disease in the bobby calf leads to unnecessary condemnation and wastage, it is suggested that more appropriate judgement criteria such as the trimming of localised infectious and traumatic lesions should be considered.

#### **ACKNOWLEDGEMENTS**

This thesis has been the result of a great deal of effort from a number of people from within the bobby calf industry, the meat industry, meat services, and Massey University.

I would like to thank first and foremost all the meat inspectors, seniors, supervisors, veterinarians, and company personnel who took this project on board and made it happen. Without their co-operation then, and now, the project would not have ach eved as much as it did. I especially thank Dr. McKenzie for supporting me through this very steep learning curve.

The meat services and the Department of Veterinary Pathology and Public Health of Massy University have been  $\infty$ -financiers of this project. That spirit of co-operation was invaluable in allowing this work to be  $\infty$ -mpleted.

The support of my supervisors is particularly acknowledged, especially those who spent a great deal of time trying to teach me the methods and principles of meat hygiene, public health, and epidemiology. Dr. Madie, Emeritus Professor Blackmore, Associate Professor Alley, Professor Wilks and Professor Manktelow have all been very patient as we discussed issues central to the theme of the thesis.

Dr. Hathaway has been a tremendous support and teacher, particularly in the area of risk analysis.

There are a great many people at Massey who have helped me learn the basics of a number of specialist disciplines in a very short time. I would particularly like to thank Ms. Cullinane and Ms. McMillan for their patience in the microbiology lab, and Associate Professor Alley for his assistance for many hours at the microscope.

There are people too numerous to name who have helped me master the computing and photography equipment, who have obtained for me the equipment I used during the season, and performed the technical tasks required for the preparation of slides and media: all these I thank.

# TABLE OF CONTENTS

	PAGE
Abstract.	i
Acknowledgements.	iii
Acknowledgements.	""
Table of Contents.	iv
List of tables.	viii
List of figures.	xi
List of figures.	Al
The Bobby Calf Industry in New Zealand.	1
The Development of Specialty Dairy Breeds.	1
The Beginnings of the Bobby Calf Industry.	1
New Zealand's Bobby Calf Industry in Perspective.	1
Legislation Concerning the Bobby Calf.	3
Calves Used in the Bobby Calf Industry.	4
Calf Managament.	4
The Bobby Calf Pool System.	5
The Slaughter and Processing of Bobby Calves.	10
Calf Products, Economics and Markets.	20
The Post-Mortem Inspection of Bobby Calves.	22
Judgements in Other Countries in Relation to those	25
used in New Zealand.	
The Economic Impact of Disease in the Bobby Calf at	26
Slaughter.	
Diseases of the Bobby Calf.	28
Omphalophlebitis, -arteritis, and -urachitis	28
"Navel III".	
Pleurisy/Pneumonia/Pericarditis.	30
Arthritis.	35

	Enteritis.	36
	Miscellaneous Bacterial Infections.	40
	Septicaemia/Bacteraemia.	41
	Working Definition of Septicaemia for this Study.	
		45
	Objectives of the Present Study.	46
Materia	als and Methods.	
Part I:	Comparison of the Gross Lesions, Histological	48
	Lesions, and Microbial Flora of Bobby Calf Cohorts	
	With and Without Omphalophlebitis ("Navel ill").	
	Selection of Cohorts.	48
	Examination of Samples.	49
	Estimation of the Prevalence of Haematomas	54
	Associated with the Umbilical Arteries in Bobby	
	Calves Submitted for Slaughter.	
Part II:	Observations of the Lesions Present in Calves	55
	Condemned by Meat Inspectors.	
Results	5.	
Part I:	Comparison of the Gross Lesions, Histological	57
	Lesions, and Microbial Flora in the Carcasses	
	from Bobby Calf Cohorts With and Without Omphalo-	
	phlebitis ("Navel III").	
	Normal carcasses and viscera.	57
	Condemned carcasses and viscera.	65
	The Relative Risk of Bacteraemia in Carcasses	79
	from Calves with "Navel III".	. 0
	Changes in the Rate of Detection of Bacteraemia Between	81
	Categories of "Navel III".	31
		83
	The Accuracy of the Meat Inspection Judgements.  Estimation of the Prevalence of Haematomas Associated	
		85
	with Umbilical Arteries in Bobby Calves Submitted for	

.

# Slaughter.

Condemned by Meat Inspectors.  The Rate of Condemnation for Disease, Contamination and Wounds and Bruising.  "Navel III".  Focal interstitial nephritis.  Pleurisy/Pneumonia/Pericarditis.  Arthritis.  Wounds and Bruising  Miscellaneous Conditions  Discussion.  Part I: Comparison of the Gross Lesions, Histological Lesions, and Microbial Flora in the Carcasses from Bobby Calf Cohorts With and Without Omphalo- phlebitis ("Navel III").  Study Design.  The Lesions of "Navel III" in Bobby Calves at Slaughter. The Baseline Test for Septicaemia. The Relationship Between "Navel III", Bacteraemia and Septicaemia. The Risk of Bacteraemia in Calves with "Navel III".	2 3 7 9 9
Wounds and Bruising.  "Navel III".  Focal interstitial nephritis.  Pleurisy/Pneumonia/Pericarditis.  Arthritis.  Mounds and Bruising  Miscellaneous Conditions  Discussion.  Part I: Comparison of the Gross Lesions, Histological  Lesions, and Microbial Flora in the Carcasses  from Bobby Calf Cohorts With and Without Omphalo- phlebitis ("Navel III").  Study Design.  The Lesions of "Navel III" in Bobby Calves at Slaughter.  The Baseline Test for Septicaemia.  The Relationship Between "Navel III", Bacteraemia  and Septicaemia.	0 2 3 7 9
Wounds and Bruising.  "Navel III".  Focal interstitial nephritis.  Pleurisy/Pneumonia/Pericarditis.  Arthritis.  Mounds and Bruising  Miscellaneous Conditions  Discussion.  Part I: Comparison of the Gross Lesions, Histological  Lesions, and Microbial Flora in the Carcasses  from Bobby Calf Cohorts With and Without Omphalo- phlebitis ("Navel III").  Study Design.  The Lesions of "Navel III" in Bobby Calves at Slaughter.  The Baseline Test for Septicaemia.  The Relationship Between "Navel III", Bacteraemia  and Septicaemia.	2 3 7 9 9
"Navel III". 90 Focal interstitial nephritis. 92 Pleurisy/Pneumonia/Pericarditis. 98 Arthritis. 107 Wounds and Bruising 108 Miscellaneous Conditions 108  Discussion.  Part I: Comparison of the Gross Lesions, Histological Lesions, and Microbial Flora in the Carcasses from Bobby Calf Cohorts With and Without Omphalophlebitis ("Navel III").  Study Design. 113 The Lesions of "Navel III" in Bobby Calves at Slaughter. 113 The Baseline Test for Septicaemia. 114 The Relationship Between "Navel III", Bacteraemia 114 and Septicaemia. 115	2 3 7 9 9
Focal interstitial nephritis.  Pleurisy/Pneumonia/Pericarditis.  Arthritis.  Mounds and Bruising  Miscellaneous Conditions  Discussion.  Part I: Comparison of the Gross Lesions, Histological Lesions, and Microbial Flora in the Carcasses from Bobby Calf Cohorts With and Without Omphalo- phlebitis ("Navel III").  Study Design.  The Lesions of "Navel III" in Bobby Calves at Slaughter.  The Baseline Test for Septicaemia.  The Relationship Between "Navel III", Bacteraemia and Septicaemia.	2 3 7 9 9
Pleurisy/Pneumonia/Pericarditis.  Arthritis.  Mounds and Bruising  Miscellaneous Conditions  Discussion.  Part I: Comparison of the Gross Lesions, Histological Lesions, and Microbial Flora in the Carcasses from Bobby Calf Cohorts With and Without Omphalo- phlebitis ("Navel III").  Study Design.  The Lesions of "Navel III" in Bobby Calves at Slaughter.  The Baseline Test for Septicaemia.  The Relationship Between "Navel III", Bacteraemia and Septicaemia.	3 7 9 9
Arthritis. 107 Wounds and Bruising 108 Miscellaneous Conditions 108  Discussion.  Part I: Comparison of the Gross Lesions, Histological 113 Lesions, and Microbial Flora in the Carcasses from Bobby Calf Cohorts With and Without Omphalophlebitis ("Navel III").  Study Design. 113 The Lesions of "Navel III" in Bobby Calves at Slaughter. 113 The Baseline Test for Septicaemia. 115 The Relationship Between "Navel III", Bacteraemia 116 and Septicaemia.	7
Wounds and Bruising Miscellaneous Conditions  Discussion.  Part I: Comparison of the Gross Lesions, Histological Lesions, and Microbial Flora in the Carcasses from Bobby Calf Cohorts With and Without Omphalo- phlebitis ("Navel III").  Study Design. The Lesions of "Navel III" in Bobby Calves at Slaughter. The Baseline Test for Septicaemia. The Relationship Between "Navel III", Bacteraemia and Septicaemia.	9
Miscellaneous Conditions  Discussion.  Part I: Comparison of the Gross Lesions, Histological Lesions, and Microbial Flora in the Carcasses from Bobby Calf Cohorts With and Without Omphalo- phlebitis ("Navel III").  Study Design. The Lesions of "Navel III" in Bobby Calves at Slaughter. The Baseline Test for Septicaemia. The Relationship Between "Navel III", Bacteraemia and Septicaemia.	9
Discussion.  Part I: Comparison of the Gross Lesions, Histological Lesions, and Microbial Flora in the Carcasses from Bobby Calf Cohorts With and Without Omphalo- phlebitis ("Navel III").  Study Design. The Lesions of "Navel III" in Bobby Calves at Slaughter. The Baseline Test for Septicaemia. The Relationship Between "Navel III", Bacteraemia and Septicaemia.	
Part I: Comparison of the Gross Lesions, Histological Lesions, and Microbial Flora in the Carcasses from Bobby Calf Cohorts With and Without Omphalo- phlebitis ("Navel III").  Study Design. The Lesions of "Navel III" in Bobby Calves at Slaughter. The Baseline Test for Septicaemia. The Relationship Between "Navel III", Bacteraemia and Septicaemia.	3
Lesions, and Microbial Flora in the Carcasses from Bobby Calf Cohorts With and Without Omphalo- phlebitis ("Navel III").  Study Design.  The Lesions of "Navel III" in Bobby Calves at Slaughter.  The Baseline Test for Septicaemia.  The Relationship Between "Navel III", Bacteraemia  and Septicaemia.	3
Lesions, and Microbial Flora in the Carcasses from Bobby Calf Cohorts With and Without Omphalo- phlebitis ("Navel III").  Study Design.  The Lesions of "Navel III" in Bobby Calves at Slaughter.  The Baseline Test for Septicaemia.  The Relationship Between "Navel III", Bacteraemia  and Septicaemia.	3
from Bobby Calf Cohorts With and Without Omphalophlebitis ("Navel III").  Study Design.  The Lesions of "Navel III" in Bobby Calves at Slaughter.  The Baseline Test for Septicaemia.  The Relationship Between "Navel III", Bacteraemia  and Septicaemia.	
phlebitis ("Navel III").  Study Design.  The Lesions of "Navel III" in Bobby Calves at Slaughter.  The Baseline Test for Septicaemia.  The Relationship Between "Navel III", Bacteraemia  and Septicaemia.	
Study Design.  The Lesions of "Navel III" in Bobby Calves at Slaughter.  The Baseline Test for Septicaemia.  The Relationship Between "Navel III", Bacteraemia  and Septicaemia.	
The Lesions of "Navel III" in Bobby Calves at Slaughter.  The Baseline Test for Septicaemia.  The Relationship Between "Navel III", Bacteraemia  and Septicaemia.	
The Baseline Test for Septicaemia.  The Relationship Between "Navel III", Bacteraemia  and Septicaemia.	3
The Relationship Between "Navel III", Bacteraemia 11 and Septicaemia.	3
and Septicaemia.	5
	6
The Risk of Bacteraemia in Calves with "Navel III".	
	8
Hazard Identification. 119	9
The Cost of Condemned Carcasses to the Producer. 12	1
Conclusions. 123	2
Don't H. A Otania of the Legione December Online	4
Part II: A Study of the Lesions Present in Calves	4
Condemned by Meat Inspectors.	
Focal Interstitial Nephritis.	4
Pleurisy. 124	4
Arthritis. 125	5

	Jaundice.	126
	Enteritis.	126
	Wounds and Bruising.	127
	Conclusions.	127
Append	dix I: Development of Bacteriological Materials	129
	and Methods.	
	Pilot Trial A.	129
	Pilot Trial B.	104
	The Effect of the Sticking Procedure on the	105
	Sterility of Heart Blood in Bobby Calves.	
	Investigation of the Effect of Heparin on the Via-	108
	bility of a Broth Containing Escherichia coli.	
Append	dix II: Details of Microbiological and Histological	138
	Methods and Materials.	
Append	dix III: Recording Form.	145
		454
Append	dix IV: Random Times Table.	151
Refere	neas	153
HEIEIG	HUGS.	100

## LIST OF TABLES.

TABLE		PAGE
1.1	List of products derived from the bobby calf and their market destination.	21
1.2	Tickets used in the inspection of bobby calves, with the defects covered by each ticket used.	23
1.3	Numbers of defective carcasses identified (prevalence) and condemned under standard ticketing categories.	24
1.4	Causes of carcass loss to the producer after collection from the farm gate.*	27
3.1	Location and dates of visits to slaughterhouses during the 1989 bobby calf season.	55
4.1	The number of carcasses with gross, microbiological and histological abnormalities from 31 calves passed as normal by meat inspectors.	62
4.2	Comparison of gross findings of detailed examination with meat inspection dispositions on carcasses with and without "navel ill".	65
4.3	Number and identification of bacterial isolates from the sample sites in condemned carcasses. (In some cases, more than one isolate was recovered from one sample site.)	70
4.4	Histological lesions in 42 grossly normal livers from	74

4.5.	Histological findings in 45 grossly normal kidneys from calves condemned for "navel ill".	78
4.6	Number of carcasses in each category of "navel ill" which were truly bacteraemic (D+ve), false test positives (D-ve), and the positive predictive value (P(T/D+)) of the test.	79
4.7	Relative risk, 95% confidence intervals and $\chi^2$ statistics calculated for each category of "navel ill" in Table 4.6.	80
4.8	Detection rates (DR), wastage rates (WR), and their 95% confidence limits for the sub-categories of "navel ill" detected by meat inspectors.	82
4.9	Comparison of the meat inspection judgement (T+/-) with the "true" presence of bacteraemia (D+/-) determined using the combined microbiological, histological and gross findings.	83
4.10	Numbers of carcasses with substantial haemorrhage in the serosa supporting the bladder and umbilical arteries.	85
5.1	Calves slaughtered, and condemned for disease by meat inspectors, at six slaughterhouses in New Zealand during the 1989 season.	89
5.2	Calves slaughtered, and condemned by meat inspectors for "navel ill", at six slaughterhouses in New Zealand.	90
5.3	The numbers of carcasses condemned for "navel ill" with phlebitis, arteritis and/or urachitis in six slaughter-houses.	92
5.4	Gross lesions in kidneys and carcasses condemned for "white spotted kidneys" (Focal Interstitial Nephritis).	93

5.5	Histopathological findings in 27 calves condemned by meat inspectors at six slaughterhouses on the basis of lesions in the kidneys.	97
5.6	Gross findings and severity of lung and pleural lesions in 75 calves which were condemned by meat inspectors for pleurisy at six slaughterhouses.	99
5.7	Lesion categories ticketed for pleurisy which were associated with gross signs of a concurrent bacteraemia.	100
5.8	Gross features of the joints of 32 carcasses condemned for arthritis by meat inspectors in six slaughterhouses.	108
5.9	Diseases detected by meat inspectors which resulted in the condemnation of carcasses using the tickets OC, PYO, and SAL.	111
1.1	Comparison of two methods of surface sterilization: hot sear with a spatula vs. sear with an alcohol flame.	131
1.2	Comparison of contamination rates of duplicate blood samples from 10 calves taken in the yards and at the inspection stand. (Expected distribution of values for $\chi^2$ are subscripted.)	135
1.3	Total viable count (TVC) of an E. coli broth cultured with and without heparin.	137

## **LIST OF FIGURES**

FIGUR	E	PAGE
1.1	Numbers of lambs (), sheep (), cattle () and bobby calves () slaughtered per month in New Zealand during the 1989 season.	2
1.2	Numbers of calves slaughtered from each pool during the 1989 season.	6
1.3	Percentage of each pool supplying slaughterhouses processing bobby calves during the 1989 season.	7
1.4	Number of calves slaughtered in each slaughterhouse during 1989.	8
1.5	The approximate geographic boundaries of each pool in New Zealand.	9
1.6	A calf with severed external carotids after the Halal cut.	15
1.7	The Halal cut is followed with a thoracic stick to ensure that the calves do not recover sensibility during exsanguination.	15
1.8	The calf is suspended from "spreaders" to facilitate freeing of the pelt in the forequarter area prior to removal.	16
1.9	The pelt is freed from the cheeks to facilitate pelt removal at the hide-puller.	16
1.10	The pelt is removed mechanically, much like a sleeve.	17
1.11	The removal of the abdominal contents (evisceration).	17

1.12	The carcass (foreground) and viscera move together past the inspection area.	18
1.13	MAF inspectors assess the carcass and viscera for their suitability for human consumption.	18
1.14	Carcasses are directed in two directions if not con- demned outright: those to the left are detained for trimming, while those on the right are regarded as suit- able for human consumption and are moving directly to the grader's stand.	19
4.1	Normal umbilici, illustrating the size range possible in the tissue. Normal umbilical veins are attached to each example.	59
4.2	An example of normal umbilical arteries and their relationship with the bladder.	59
4.3	Longitudinal section of a normal umbilical vein in a bobby calf. H&E, x10.	63
4.4	Normal bobby calf kidney. H&E, x100.	63
4.5	Bobby calf liver with vacuolated hepatocytes. H&E, x 200	64
4.6	Infection of the apical remnants of the umbilical arteries.	64
4.7	A large localised area of necrosis involving the urachus and the umbilical arteries. The ileum is adherent to the lesion.	68
4.8	Extensive necrosis of the urachus in case N <sup>0</sup> 11.	68

4.9	Longitudinal section of an umbilical vein showing severe inflammation, leucocytic infiltration, and fibrinous effusion of the mural tissues. H&E, x10.	72
4.10	Longitudinal section of a numbilical vein with severe leucocytic infiltration and fibrinous effusion. H&E, x10.	73
4.11	High power of the same tissue showing granulation tissue developing beneath the zone of infiltrating leucocytes.  H&E x 40.	73
4.12	Bobby calf liver, showing moderate periportal cuffing with mononuclear cells. H&E, x100.	75
4.13	Bobby calf liver with an inflammatory focus dominated by monocytes in the parenchyma. H&E, x200.	75
4.14	Bobby calf kidney with chronic focal interstitial nephritis in the cortex, and severe tubular destruction.  H&E, x100.	76
4.15	Bobby calf kidney showing an acute suppurative inflammatory reaction in the cortex. H&E, x100.	76
4.16	Focal interstitial nephritis involving both acute suppurative foci and early fibrosis of the cortical tissue. H&E, x100.	77
4.17	Variation in severity of haematomas associated with the umbilical arteries. These are presumed to arise from inadequate contraction of the umbilical arteries at birth.	87
4.18	Haemorrhage associated with the umbilical arteries and urachus.	87

5.1	A representative range of the lesions of "white spotted kidneys". "Acute", hyperaemic lesions in the kidney on the right, and two examples of "chronic" lesions to the left.	94
5.2	Severe infection of the kidney, with fibrinous exudate in the perirenal tissues and renal swelling.	94
5.3	Kidney with acute pyelonephritis. Note the radial distribution of the inflammatory lesions. H&E, x40.	95
5.4	Acute enzootic pneumonia with areas of hyperaemia and consolidation with an anteroventral distribution.	102
5.5	An old, well-encapsulated pulmonary abscess with fibrous adhesions to the surrounding tissues.	102
5.6	Bobby calf lung with acute suppurative bronchopneumonia. H&E, x10.	103
5.7	High power of the above showing intense neutrophilic accumulation in a bronchiole. H&E x 100.	103
5.8	Bobby calf lung with fibrinous bronchopneumonia containing an area of coagulation necrosis. There are bacterial colonies in the centre. H&E x 40.	104
5.9	Bobby calf lung showing severe distension of the interlobular septa with fibrin and necrotic material.  H&E, x10.	104
5.10	Bobby calf lung containing streaming macrophages ("oat cells") typical of infection with <i>Pasteurella haemolytica</i> . H&E, x 200.	105

5.11	The edge of an old abscess in a bobby calf lung. There is encapsulation of the necrotic material and extensive	105
	destruction of the normal lung tissue. H&E, x10.	
5.12	Bobby calf liver with one encapsulated abscess in the left lobe.	112
5.13	Generalised fibrinous peritonitis in a bobby calf.	112
IV 1	Random times table	152

#### THE BOBBY CALF INDUSTRY IN NEW ZEALAND

#### The Development of Specialty Dairy Breeds

During the 16th and 17th centuries in Europe and Britain, changes in attitude to the products available from cattle occurred. Increases in prices realised for milk, milk products and meat prompted better feed management and a geographical distribution of cows according to the quality of grass occurred. Distinct dairy and beef breeds were then developed at the expense of the dual-purpose breeds (Urquhart 1983).

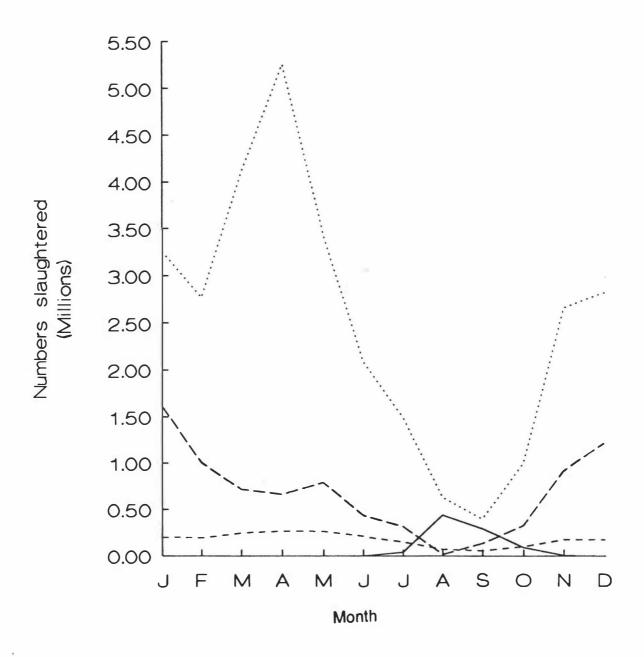
#### The Beginnings of the Bobby Calf Industry

In today's intensive dairying areas, the necessary result of high levels of milk production is also the production of calves far in excess of that which occurred in early times, when the calf was fattened for slaughter on special occasions using milk surplus to household requirements (Urquhart 1983, Harrison 1964). In modern times, as only a minor proportion of calves produced are needed as replacements in the herd, the disposal of the other, unwanted calves presents very real problems. To address the problem in part, a bobby calf industry has developed in many countries, including New Zealand. The meat from these calves is largely used in specialty processed meats, where its blandness and tenderness are desirable properties, and also in the production of primal cuts such as veal cutlets and veal striploins.

#### New Zealand's Bobby Calf Industry In Perspective

There are over forty licensed Meat Export slaughterhouses distributed throughout New Zealand which process lambs, sheep and adult cattle for export to overseas markets. The slaughter of these species for export follows a markedly seasonal pattern (Figure 1.1). The bobby calf season occurs at a time when there are low numbers of other species being processed, beginning slowly from late May, peaking through August and September, then tailing away until cessation in late October or early November.

Figure 1.1: Numbers of lambs (...), sheep (---), cattle (- - -), and bobby calves (\_\_\_\_) slaughtered per month in New Zealand during the 1989 season.



Calves differ from the other slaughtered species from the meat industry's point of view, in that they do not become the property (and responsibility) of the processing company. The company is contracted by Dairy Meats New Zealand Ltd (DMNZ) to slaughter and process the calves on behalf of the dairy farmers, who maintain ownership of the calves and the subsequent products.

During the 1989 season, eleven slaughterhouses were contracted by DMNZ for the slaughter and processing of the 873,361 calves collected from dairy farmers during the spring calving season.

In terms of volume of throughput, the bobby calf industry is a minor, but lucrative part of the processing companies' incomes. Since they are under contract, the companies do not bear the economic effects of calf losses due to disease, contamination or bruising. Ironically, they receive additional income from the by-products generated from such losses.

## Legislation Concerning the Bobby Calf

New Zealand is one of a few countries which practise the slaughter and processing of calves less than one month of age for the commercial production of bobby veal. The legal description of a bobby calf in New Zealand is "a calf which is intended to be slaughtered for the production of bobby veal; and includes any other calf that has a liveweight of less than 100 lb (metric conversion 45 kg) (Anon. (a) 1969)

Until 1989, when the Bobby Calf Marketing Regulations, 1955, were rescinded, there was a further legal requirement from these regulations for bobby calves to have acquired "a minimum liveweight ... so as to ensure that such calves upon slaughter will yield a dressed weight of not less than 22 lb (metric conversion 10 kg) cold weight" (Anon. 1955). Since the removal of these regulations from the law, the minimum liveweights and ages of calves submitted for slaughter are now negotiated annually between the calf marketing arm of the New Zealand Dairy Board, Dairy Meats New Zealand Ltd (DMNZ), and the New Zealand Ministry of Agriculture and Fisheries (MAF). For the 1990 season, the minimum liveweight will be 25 kg, and the minimum age four days.

#### Calves Used In the Bobby Calf Industry

The dairy industry is the major source of bobby calves in New Zealand. Calves which are not being reared as replacement heifers or for slaughter at a later date as bull beef are submitted as soon as the required weights and ages are attained, and killing space is available. The age of calves submitted for slaughter is therefore expected to range from the minimum of four days to approximately fourteen days. During the peak of the season, in August and September, the calves will tend towards the minimum liveweight, since there are daily pick-ups by the trucks. At the beginning and end of each season, when the trucks are picking up calves only once or twice weekly, the calves will tend to be older. The predominant breed types presented for slaughter for the production of bobby ver! were subjectively observed by the author during 1989 to be Jersey and Jersey crosses. Data from 1966 reported by O'Connor in 1979 support this observation, citing Jersey calves as contributing 72.6% of the total available surplus calves produced, Friesian-Jersey crosses 7.4%, Friesians 12.1%, and "others" 7.9% (O'Connor 1979). Despite the increased proportion of Friesians currently in the national dairy herd as compared to 1979, the use of dairy bull calves from factory supply herds for the bull-beef market has also increased. A total of 161,963 calves were reared for beef in 1982/3, increasing to 412,573 in 1987/8 (Anon. 1988/89). Friesian calves predominate in those kept for bull beef and the contribution of this breed to the bobby calf pool is unlikely to have increased substantially.

#### Calf Management

Calves are reared under a variety of systems. Generally, part of the milking shed, or a utility building already present, is converted in part or *in toto* into calf pens. Some farmers have purpose-built calf sheds, especially if rearing calves for bull-beef is also part of their production system. The sheds are generally wooden with a variety of flooring materials including concrete, wooden slats or beds of sawdust or straw. Ventilation and hygiene of the shed vary greatly. Calves may be kept in the shed until they are sent for slaughter, or they may be given access to pasture at a few days of age.

The bobby calf is usually removed from its dam after 24 hours. Thereafter, it may be fed by recently calved nurse cows at milking time twice a day, or the colostrum may be collected at milking time, pooled, and fed to the calves using either individual buckets, communal troughs, or alternatively a communal ("calfeteria") or individual ("udder mother") teat system. Only calves kept for rearing as replacement heifers or for the bull beef market are likely to be fed a commercial milk replacer.

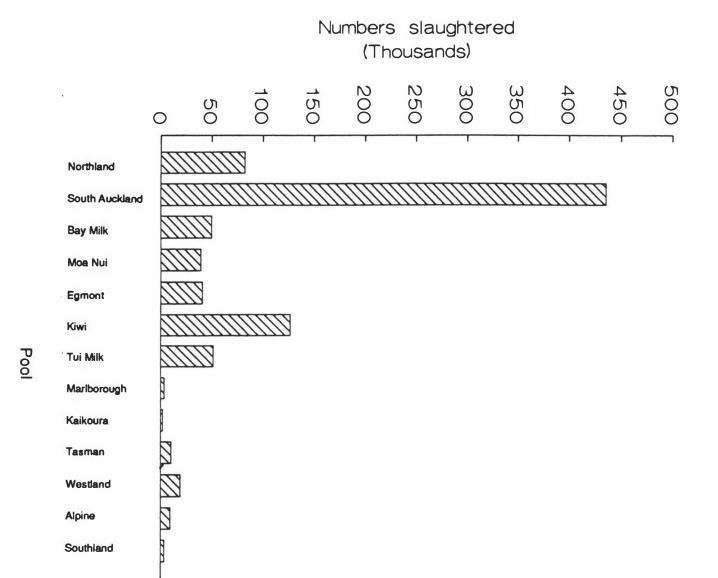
In response to concern by importing countries, particularly the United States of America, about antimicrobial residues in carcass meat derived from calves, much effort has been invested by regional personnel from MAF educating farmers directly or through their practising veterinarian in appropriate rearing management practices which maximise productivity and minimise disease. As research into calf diseases has elucidated the epidemiology of major disease entities, particularly neonatal diarrhoea, septicaemia and calf pneumonia (Roy 1989, (a),(b),(c)), so preventative management strategies have developed to minimise overt disease and the subsequent need for the use of antimicrobial drugs. Of particular importance has been research into colostrum supply to the new-born calf and the vital role colostrum plays in the subsequent health of the animal (Amstutz 1970, Jones et al 1977, Woolcock (b), 1979, Radostits & Acres 1980, Roy 1989 (d)). The result has been a supply of improved calves to the bobby calf industry, as well as a reduction of antibiotic and sulphonamide residues present in their carcasses. Calculations from the number of sulphonamide residue violations detected in 1986 indicate that a probable mean of 84,000 carcasses with sulphonamide residues could have reached the USA, whereas in 1989, the mean number of carcasses calculated to have residues dropped to about 600 (Pers. comm. B. Marshall, MAF, 1989).

Statistics from 1963 give the average bobby calf carcass weight as 30 lb (13.6 kg), and the dressing out percentage as 57 to 58%. No figure on meat yield was available (O'Connor 1979). Figures for the 1988 season give the average carcass weight as 16.5 kg, with a meat yield of 8.95 kg (Dairy Meats NZ statistics 1989). These improvements in carcass weights are probably due to improvements in animal health, management and herd quality, coupled with the market demand for calves of greater liveweight.

### The Bobby Calf Pool System

It was apparently not until two dairy farmers from Taranaki, visiting Europe in 1922, were dining with a number of directors of a Dutch Dairy Company, that any inkling of the possible export potential of the meat from very young calves was learned. Until then, surplus calves in New Zealand were destroyed on the farm, with only their pelts saved. Inquiries in New Zealand led to the first practical shipment of veal from Taranaki in 1926. Others soon became interested since the venture proved to be profitable, and the bobby calf industry came into being in New Zealand (O'Connor 1979).

The collection of calves was organised through a co-operative pool system, rather than an individual farmer system, with all profits returned to the producer. Each pool



Numbers of calves slaughtered from each pool during the 1989

season.

FIGURE 1:3: PERCENTAGE OF EACH POOL SUPPLYING SLAUGHTERHOUSES PROCESSING BOBBY CALVES DURING THE 1989 SEASON. (Data courtesy of DMNZ.)

Meat Export Slaughterhouse (ME) Number and Name.

Moe- rewa	Aote- oroa	Horo- tiu	Kaiti	10 Tomo- ana	32 Feild- ing	39 Imlay	2 Wai- tara	40 Stoke	41 Kai- apoi	80 Blue Sky	Total
100.00											82,509
65.60	13.40	21.01									34,033
		97.75									51,992
4.67											44,314
				6.35							111,952
							0.52				106,187
											86,744
			48.65	0.20			0.00				49,914
						6.45	92.23				39,014
											40,535
	0.72				0.46						126,579
							<b>J</b>				19,744
	1.09			21.75							31,702
						_,.00		100.00			3,295
									78 77		2,007
									70.77		10,085
									47 87		19,022
											9,230
								17.77		88 11	3,549
		46.66		53.33					11.00	30.11	60
n (ME 16)		.0.00		30.00							864
	100.00 65.60 4.67 1.92 0.54	100.00 65.60 13.40 2.25 4.67 2.62 1.92 7.81 0.54 55.91 43.25 28.90 1.29 0.72 1.09	100.00 65.60 13.40 21.01 2.25 97.75 4.67 2.62 92.70 1.92 7.81 83.92 0.54 55.91 5.34 43.25 42.63 28.90 22.44 1.29 0.02 0.72 1.09	100.00 65.60 13.40 21.01 2.25 97.75 4.67 2.62 92.70 1.92 7.81 83.92 0.54 55.91 5.34 43.25 42.63 28.90 22.44 48.65 1.29 0.02 0.72 1.09	100.00 65.60	100.00 65.60	100.00 65.60	100.00 65.60	100.00 65.60 13.40 2.25 97.75 4.67 2.62 92.70 1.92 7.81 83.92 6.35 0.54 55.91 5.34 37.69 0.52 43.25 42.63 8.26 28.90 22.44 48.65 1.29 0.02 6.45 92.23 5.03 94.97 0.72 0.46 46.08 52.74 10.66 89.33 1.09 21.75 49.35 27.80  100.00 21.23 100.00 52.13 14.47	100.00 65.60	100.00 65.60

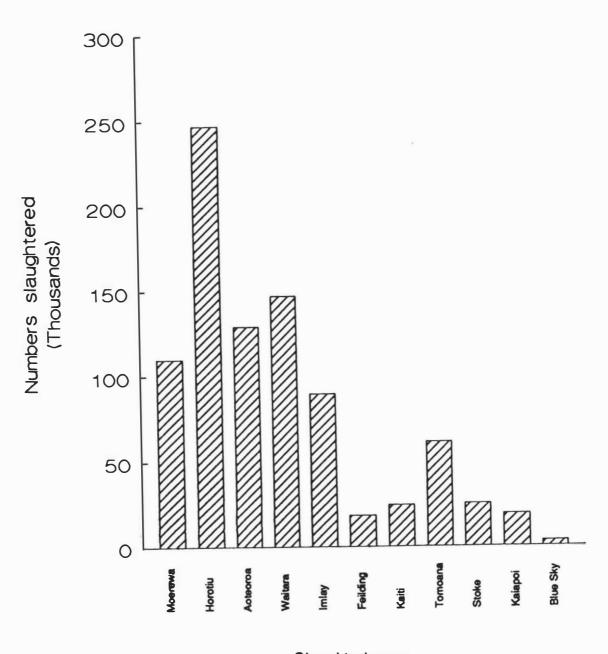
Total Calves Slaughtered 873,361

Haur/Coro = Hauraki/Coromandel
Wair/HB = Wairarapa/Hawkes Bay

PPCS = Primary Producers Co-operative Society

CFM = Canterbury Frozen Meats

Figure 1.4: Number of calves slaughtered in each slaughterhouse during 1989.



Slaughterhouse

Figure 1.5: The approximate geographic boundaries of each pool in New Zealand.



constitutes a defined geographic area (Figure 1.5): calves are collected from the farm gate, and once on the truck lose their individual farm identity. Farmers are thus paid an average price for each calf based on the performance of the pool as a whole.

The entire system is managed by the Dairy Board through its subsidiary, DMNZ, which arranges all aspects of the bobby calf industry, on behalf of the dairy farmers, including organising transport of the calves to the slaughterhouse, negotiating slaughter contracts and marketing the product to importing countries. The relative contribution of each pool to the total kill is illustrated in Figures 1.2 and 1.3. Figure 1.4 illustrates the number of calves handled at each of the slaughterhouses contracted by DMNZ for the 1989 season.

#### The Slaughter and Processing of Bobby Calves

The calves are processed on a mechanised chain system designed and used largely for the processing of sheep and lambs. Processing details vary from slaughterhouse to slaughterhouse, but the essentials of the process follow a sequence as shown below:

- (1) Calves are collected at the farm gate by trucks contracted by DMNZ, and transported to the nearest slaughterhouse.
  - The truck driver weighs the calves before loading them onto the truck to ensure that the minimum liveweight is reached, and at the end of his "run", delivers the calves to the appropriate slaughterhouse.
- (2) Stockmen at the slaughterhouse put the the delivered calves into pens and coordinate their transfer to the race leading to the slaughter area. The calves must be presented for slaughter within 24 hours of arrival at the slaughterhouse and have been submitted to an ante-mortem inspection by a veterinarian during that period. Any calves already dead, or considered unfit for slaughter by the veterinarian, are condemned and removed from the pen. The yards and stockmen are strictly separated from the processing area and personnel, which begins at the stunner where the calves are stunned and exsanguinated.
- (3) As with all animals slaughtered in export slaughterhouses in New Zealand, calves must be stunned so that they are insensible to pain prior to exsanguination and until the onset of permanent insensibility occurs as a result of cerebral anoxia (Anon. (b) 1969).

The stun may be delivered by a number of methods, each slaughterhouse using that which satisfies animal welfare obligations and best meets its operational needs. Methods used include electrical stunning, (head-to-back, head-to-leg and head-only) and a non-penetrative percussive stun. The penetrative captive bolt method of stunning was not used on calves in any of the slaughterhouses visited by the author.

- (4) The animals are exsanguinated ("stuck") after stunning using traditional or Halal methods, depending on market requirements.
  - (i) The thoracic stick (traditional).

This involves the severance of the major blood vessels via the thoracic inlet. The method is used after a stun which produces permanent insensibility, and is suitable for products destined for any except the Moslem market.

#### (ii) The Halal stick.

This method is used following a reversible stun and involves the bilateral severance of the carotid arteries across the extended neck of the animal (Figure 1.6). The method has Islamic religious significance, and must be delivered to an animal which is "alive". The head-only electrical stun and non-penetrative percussive stun are considered by Muslims to be reversible and therefore acceptable as a method of producing insensibility in animals.

In the case of calves, the Halal stick must be followed by a thoracic stick (Figure 1.7) to ensure that the calf does not regain sensibility during exsanguination. This problem arises because the electroencephalogram of calves during exsanguination has been found to take 80 seconds or more to become consistent with permanent insensibility following bilateral severance of the carotids (Newhook & Blackmore 1982). This is believed to be due to a significant supply of cerebral blood being delivered by the vertebral arteries which are not severed by a Halal cut (Newhook & Blackmore 1982).

The duration of insensibility following a head-only stun is only of the order of 30 seconds, so in a proportion of calves there is the possibility that sensibility will be regained before cerebral anoxia results in permanent insensibility. In order to satisfactorily interrupt the blood supply via the vertebral arteries, the brachiocephalic trunk or the heart must be severed, hence the necessity for the additional use of the thoracic stick in calves slaughtered by the Halal method (Newhook & Blackmore 1982).

The knife used for the thoracic stick has been found to be more reliable in ensuring the severance of the brachiocephalic trunk if it has a blade 16 cm in length, rather than the usual 12.5 cm length (Leigh & Delaney 1986).

- (5) The process of removing the pelt generally begins with the head and forequarters. The pelt is freed from the cheeks, ventral neck, brisket, and forelegs. The oesophagus is freed in preparation for the later removal of the contents of the abdominal cavity. To facilitate access to these areas of the carcass, the calf is suspended by all four legs by means of "spreaders" (Figures 1.8 & 1.9).
- (6) The forelegs are removed at the carpal joint either by an intra-articular knife cut, or by mechanical cutters. The skin must be removed before an intra-articular cut is made. This process usually drops the forequarters, so the carcass is suspended by the hind legs. If an inverse dressing system is used, the forelegs are held above the carpal joint and removal of the forelegs does not result in dropping of the forequarters.
- (7) In standard dressing systems, the calf passes the "legging table", where the skin over the hindquarters is freed and the hind legs removed using an intraarticular cut at the hock. In some inverse dressing systems, the hind legs are removed using mechanical cutters applied just below the hock joint.
- (8) The pelt is freed from the carcass along the midline and towards the flank region, so that the pelt does not tear, or the carcass become mutilated, during subsequent pelt removal.

- (9) The hind legs are usually removed at this stage during an inverse dressing system, usually below the hocks.
- (10) More commonly, and in all slaughterhouses visited, the pelt was removed mechanically using either a pulley system or a ratchet system. The carcass was fixed by the forelegs, and in some cases also the head. The pelt was then attached to the pulling mechanism, and removed like a sleeve (Figure 1.10).
- (11) If the slaughterhouse employs an "inverse" dressing system, where the calf has previously been suspended by the forelegs, it must now be suspended by the hind legs prior to a pre-evisceration wash.
- (12) After the pre-evisceration wash, the anal region is freed from connective tissue, the abdomen opened down the midline, and in some instances the terminal colon and/or the bladder removed.
- (13) The abdominal contents, sometimes excepting the kidneys, is removed and placed on a stainless steel viscera tray (Figure 1.11). Edible organs such as the liver are placed in a separate compartment from the intestines.
- (14) The brisket is cut, and the lungs and heart are removed from the thorax and placed in the edible compartment of the viscera tray.
- (15) The kidneys must be "enucleated" from their capsule, and may be placed on the viscera tray with other edible products, or left in position in the carcass.
- (16) The carcass and viscera move as a unit on the chain (Figure 1.12), and are inspected by NZMAF Meat Services inspectors (Figure 1.13). One of three dispositions are assigned to the carcass and viscera:
  - (a) Total condemnation of both carcass and viscera.
  - (b) Partial condemnation of the viscera or parts of the carcass.
  - (c) Carcass and viscera are passed in their entirety as suitable for human consumption.

Different chains are used for the carcass, depending on whether the carcass has been passed, or has been detained for trimming of the condemned parts (Figure 1.14).

- (17) All condemned material is sent via closed chutes to the rendering department and heat treated to produce sterilised products.
- (18) Edible offals are directed to the fancy meats room for packaging prior to freezing and export.
- (19) The carcass is weighed and if it complies with the appropriate specifications, may be graded for the small market requiring frozen carcasses. All carcasses are then taken to the cooling floor and finally left in chillers overnight. The following day the vast majority of carcasses are boned out and packed in plastic-lined cardboard cartons. A limited number are used to supply a small market for chilled "bone-in" hindlegs, veal cutlets, or frozen carcasses.

Figure 1.6: A calf with severed external carotids after the Halal cut.

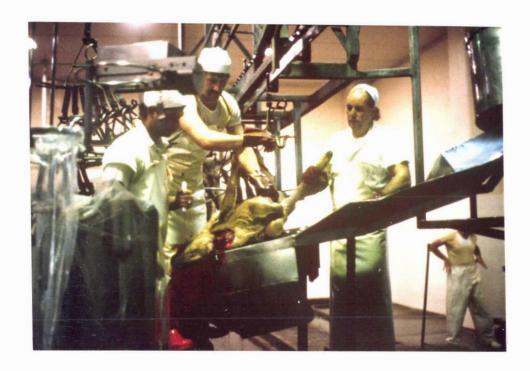


Figure 1.7: The Halal cut is followed with a thoracic stick to ensure that the calves do not recover sensibility during exsanguination.



Figure 1.8: The calf is suspended from "spreaders" to facilitate freeing of the pelt in the forequarter area prior to removal.



Figure 1.9: The pelt is freed from the cheeks to facilitate pelt removal at the hide-puller.



Figure 1.10: The pelt is removed mechanically, much like a sleeve.



Figure 1.11: The removal of the abdominal contents (evisceration).



Figure 1.12: The carcass (foreground) and viscera move together past the inspection area.



Figure 1.13: MAF inspectors assess the carcass and viscera for their suitability for human consumption.



Figure 1.14: Carcasses move in one of two directions if not condemned outright: those to the left are detained for trimming, while those on the right are regarded as suitable for human consumption and are moving directly to the grader's stand.



# Calf Products, Economics and Markets

Each calf results in export earnings to New Zealand of NZ\$98.25 (DMNZ, 1988 figures) through the following products:

PRODUCT	\$(NZ)	
skin	29.00	
4.14 kg legs	31.00	
4.37 kg trunks	20.75	
0.44 kg backstraps	3.50	
abomasum (vell)	10.00	
offals	4.00	
TOTAL	98.25	

The processing companies also generate earnings through calf by-products contributing to the production of tallow, meat and bonemeal, dried blood, and the production of long bones. The net return to the farmer after DMNZ has deducted all the costs incurred for the processing, marketing and administration of the bobby calf industry was NZ\$51.00 for the 1988 season (pers. comm. DMNZ).

Altogether the bobby calf industry produces about NZ\$100 million in export earnings, (pers. comm. DMNZ).

The bulk of products derived from calf processing are exported to a limited number of countries. Personal inquiry at a number of slaughterhouses provided information about the major products and their market destinations (Table 1.1).

TABLE 1.1: LIST OF PRODUCTS DERIVED FROM THE BOBBY CALF AND THEIR MARKET DESTINATION.

Product	Market destination
Boneless veal, including diaphragm	USA, Japan
Carcass form, 18kg plus	French Polynesia
Bone-in hindlegs	Switzerland
Liver < 750g > 750g others	Canada USA France
Kidney, heart, tongue, brain, thymus	France
Long bones (humerus, femur, tibia)	Japan
Abomasum (vell) (Rennet)	NZ Rennett Company
Skin	Italy, Japan, Mexico
Lung, trachea, tongue roots	UK, local trade (petfood)
Blood (inedible)	USA, Scotland
Small intestinal submucosa (inedible)	Japan
Meat and bonemeal	Malaysia
Other byproducts	Local trade

#### The Post-Mortem Inspection of Bobby Calves

The MAF Meat Services in New Zealand uses trained, non-veterinary personnel to perform the routine inspection of individual carcasses and viscera at the chain. The meat inspectors are supervised by senior meat inspectors, who also maintain the appropriate disease records and ensure the smooth running of the MAF-related aspects of the slaughter floor. The meat inspectors and senior meat inspectors in each slaughterhouse are managed by the supervising meat inspector. The supervising veterinarian, along with the supervising meat inspector, form the interface between the inspection staff and the company running the slaughterhouse. The meat veterinarian and the supervising meat veterinarian, often assisted by senior meat inspectors, perform an audit role over all processing departments in the slaughterhouse, and manage national disease surveillance duties and emergency response preparedness within the slaughterhouse. Their role in public health extends beyond the slaughter floor to all processing departments.

A manual ticketing system is used to identify and categorise carcasses with defects for disposition and statistical purposes. The tickets and the general defects they cover are listed in Table 1.2.

The company grader records the weight of the carcass, and if a carcass has been condemned, the appropriate tickets are transferred to the grader for recording by the company. The tickets used by MAF are serially numbered, so MAF are able to maintain their own records of disease and defect prevalence and condemnation rates. No grade is assigned to the majority of slaughtered calves since bobby veal is exported almost entirely in a boneless form to be used as manufacturing meat.

Regulations relevant to the production of meat and meat products are transformed into working guidelines for the export slaughterhouses in 18 Meat Manuals (Anon. 1977). Manual 16 deals with the actual inspection routines and appropriate judgements for all species of slaughtered stock and follows the guidelines written for third and fourth schedule diseases and defects in New Zealand (Anon. (a) 1969).

In the determination of judgements to be applied to calves with diseases and defects present at the time of slaughter, two important assumptions appear to have been made:

- (a) The calves are too young to have developed an effective immune system, therefore any disease process is likely to be ongoing and systemic in nature (personal inquiry, MAF staff).
- (b) Most diseases affecting calves at this age are likely to have been contracted antenatally, and are therefore also likely to be systemic in nature (Anon. 1977).

Severe judgements resulting in total condemnation of both carcass and viscera have thus evolved for bobby calves with virtually any pyogenic or inflammatory lesion. This is reflected in the disease and defects statistics generated by MAF from the ticketing system, where prevalence and condemnation rates are virtually identical for every defect except contamination and wounds and bruises (Table 1.3).

**TABLE 1.2:** TICKETS USED IN THE INSPECTION OF BOBBY CALVES, WITH THE DEFECTS COVERED BY EACH TICKET LISTED.

Ticket	Defect
ANY	a special purpose ticket, usually used for trial purposes
ACT	actinobacillosis or actinomycosis
ART	arthritis
CONT	contamination
EMA	emaciation
NP	neoplasia
ocs	other causes: diseases, including "navel ill", not
	covered by other tickets.
PLU	pleurisy
PYO	pyogenic lesions
SAL	septicaemic-like lesions
ТВ	tuberculosis
WB	wounds and bruises
XAN	xanthosis
CONDEN	A condemned: used in conjunction with the defect tickets as nec

TABLE 1.3: NUMBERS OF DEFECTIVE CARCASSES IDENTIFIED (PREVALENCE) AND CONDEMNED UNDER STANDARD TICKETING CATEGORIES.

Defect	Prevalence*		Condemned*	
	Number	(%)	Number	(%)
CONT	107,874	(12.35)	1,312	(0.15)
WB	45,069	(5.16)	1,154	(0.13)
EMA	27	(0.003)	27	(0.003)
PYO	1,769	(0.20)	1,720	(0.19)
ART	1,340	(0.15)	1,308	(0.15)
SAL	5,046	(0.58)	5,038	(0.57)
NP	22	(0.002)	22	(0.002)
PLU	1,898	(0.22)	1,805	(0.21)
ACT	0	(0.00)	0	(0.00)
XAN	1	(0.0001)	1	(0.0001)
ОС	2,621	(0.30)	1,963	(0.22)

Bobby Calf Q.A. Programme Summary, MAF, 1989.

The calf is unique among animals routinely slaughtered for meat in this country in that the umbilicus and umbilical vessels are still atrophying after the birth process, so this site is given specific inspection procedures in addition to those directed at the usual organs. "Acute inflammation of the umbilicus" is described in Manual 16 as a potential source of "disease which may cause food poisoning" (Anon. 1977). "Navel ill" (omphalitis or omphalophlebitis) has not, however, been assigned a ticket category of its own. Meat inspectors therefore use one or other of three existing tickets to cover "navel ill": SAL, OC, or PYO, depending on the nature and extent of pathological changes present in the carcass. It is therefore difficult to extract epidemiological information concerning this disease from national disease statistics.

# Judgements In Other Countries In Relation to those use 1 In New Zealand

The New Zealand requirements for the judgements of disease in bobby calves bear distinct similarities to requirements in the federal procedures of the United States Department of Agriculture, with the addition of some regulatory requirements relevant to this country. For instance, section 76.1 in Manual 16 is similar to judgements cited in section 311.16, of the United States Department of Agriculture Federal Regulations (Anon. 1989), and also to those in the Australian Export Meat Manual (volume 1, section 20) (Anon. 1985 (b)). Manual 16 goes further than either of these two documents, however, in requiring that calves with omphalitis alone are condemned because of the potential risk to the consumer.

It is interesting to note that many of the conditions for which the New Zealand requirement is blanket condemnation under 16.77.1 (Anon. 1977), are found under category (5) of the USDA inspection summary sheet for "bob veal" (calves weighing 150 pounds or less), where passing of the carcass for human consumption is an option (Anon. 1987).

Both the USDA (Anon. 1989) and the Canadian (Anon. 1982 (a)) disposition codes recognise that even a young calf may have disease conditions which are localised in nature, particularly arthritis, abrasions, bruises, abscesses (Anon. 1982 (a), anon. 1989), and pneumonia and pleurisy (Anon. 1982 (a)). All the above conditions are covered in general terms, with no special judgements cited when dealing with bobby calves.

The Codex Alimentarius commission has published an international code of practice for ante-mortem and post-mortem judgements of slaughter animals and meat. Recommendations relevant to the slaughter of calves include the approval of carcasses and viscera if any pathological lesions present are localised and/or chronic in nature. The umbilicus is only mentioned if phlebitis of the umbilical vessels or systemic invovement of the carcass is also present (Anon. 1985 (a)). In documents concerned with the appropriate judgements of lesions in calves, "omphalophlebitis" is listed as a condition for which total condemnation of the carcass and viscera is required. Nowhere is there recognition that infection of the other vessels arising from the omphalus, namely the umbilical artery and urachus, may also constitute a hazard to the consumer, nor is infection of the umbilicus alone considered a serious hazard.

The only apparent European Community (EC) requirement for the judgement of calves is that "fresh meat (must not be acquired) from animals which are slaughtered too young", stated in Article 20(f) of the EC Official Agricultural Journal (Anon. 1983). France is the only EC member country which imports bobby calf products from New Zealand, and the criterion found in Manual 12 (FRANCE) for determining whether or not an animal was slaughtered "too young" is a minimum weight for the organs derived from the animal.

#### The Economic Impact of Disease In the Bobby Calf at Slaughter

The value of the bobby calf to the producer has been steadily rising over a number of years with the farmer receiving \$NZ51.00 for each calf in the 1988 season (DMNZ, 1989). In 1988, 18,511 calves out of a total of 873,361 collected at the gate were rejected for processing for a variety of reasons, representing lost earnings of \$944,061 to New Zealand producers (DMNZ, 1989).

TABLE 1.4: CAUSES OF CARCASS LOSS TO THE PRODUCER AFTER COLLECTION FROM THE FARM GATE.\*

Reason for	Number	
Rejection	Condemned	
Dead on Arrival	413	
Dead in Pens	1,533	
Unfit for Slaughter	621	
Disease	11,884	
Wounds and Bruises	1,154	
<b>Excessive Contamination</b>	1,312	
Underweight (under 10kg)	1,594	
TOTAL	18,511	

<sup>\*</sup> Bobby Calf Q.A. Programme Summary, MAF, 1989.

As can be seen from Table 1.4, "disease" was the most important reason for total condemnation of calf carcasses. The loss of entire carcasses as a result of excessive bruising or contamination was not high, but the prevalences of these two defects, at 5% and 12% respectively (MAF statistics, 1989), are high in comparison to other slaughtered species. The loss of meat due to the trimming of bruises and contamination from affected carcasses is therefore presumed to be significant, but to the author's knowledge, no effort has been made to calculate such losses.

#### **DISEASES OF THE BOBBY CALF**

A percentage of the bobby calves submitted for slaughter in New Zealand are diseased; MAF statistics for 1989 show a national average for "disease", as ticketed by meat inspectors, of 1.362% of calves collected, representing 11,884 calves. Infectious diseases which can affect calves of this age in New Zealand and which may result in grossly visible lesions and/or a risk to public health are briefly discussed below under the categories commonly used by meat inspectors.

# Omphalophlebitis, -arteritis, and -urachitis ("navel III")

The elements of the umbilical cord, which comprises one vein, two arteries and the urachus, are patent at birth. Both the vein and the arteries have "special" anatomical features: the arterial intima consists only of endothelium without the usual internal elastic layer of a large calibre artery, whilst the media is comprised of two thick muscular layers which are sharply demarcated from each other. The inner layer is aligned longitudinally, the outer transversely. The media also contains a small number of elastic fibres. The umbilical vein contains longitudinal or tangential smooth muscle fibres in the subendothelial connective tissue layer of the tunica intima (Maximov & Bloom, 1957).

During the birth process, the smooth muscle cells present in the walls of the umbilical vessels contract in response to stretch stimuli and changing oxygen tensions (Keatinge, 1979). The arteries contract with such strength as to retract into the abdomen. However, due to the more intermittent nature of the contractions of the vein, this remnant remains in the umbilicus and therefore outside the peritoneum (Baxter, 1989). Vasoconstrictive agents released from platelets during blood clotting in the umbilical vessels can also cause powerful contraction of blood vessels. This mechanism is of greater importance in veins than arteries, since clotting is too slow and weak to halt bleeding from a major artery. The best established, and perhaps most important of these vasoconstrictors is serotonin. Other monoamines, including the more recently discovered thromboxane A2, are candidates for mediating the remaining vasoconstrictor activity (Keatinge 1979).

#### Aetiology

Poor sanitation of the environment the calf is born into, along with poor subsequent management of the calf and unhygienic housing conditions, may predispose the calf to infections of the umbilical stump.

Bacteria commonly involved include *Escherichia coli*, *Streptococcus spp*, *Staphylococcus spp*, *Actinomyces (Corynebacterium) pyogenes* and other soil and faecal contaminants (Blood & Radostits 1989 (b)). *Salmonella spp*, important meatborne pathogens, have also been cited as a cause of omphalophlebitis (Gracey 1986), and omphalophlebitis has been described as one of the lesions present in calves with clinical salmor ellosis (Greene and Dempsey 1986). Direct extension of any of these infections along the umbilical vein, arteries, urachus, or a combination of these may result in the lesions described below (Baxter 1989, Blood & Radostits 1989 (b)).

#### Gross lesions

The umbilicus itself is the only umbilical tissue visible externally in the live calf. Lesions will range from an enlarged umbilical stalk through to umbilical abscessation (Baxter 1989, Roy 1990 (e)).

Omphalophlebitis may be localised anywhere along the vein or extend to the liver, although the infection can be quite severe without hepatic involvement (Jubb & Kennedy 1985 (e), Baxter 1989). Possible sequelae include hepatic abscesses, thrombophlebitis of the vena cava, endocarditis, pulmonary abscesses, polyarthritis and osteomyelitis (Jubb & Kennedy 1985, Firth *et al* 1987, Blood & Radostits 1989 (b), Roy 1990 (e)). Surgical intervention is practised in some countries where the value of the calf warrants this expense (Trent & Smith 1984 (a & b), Baxter 1989).

The urachus is the most commonly infected umbilical remnant, with abscessation or necrosis occurring variably along its length (Baxter 1989).

The arteries are perhaps the least commonly infected remnant due to their contraction into the abdomen at birth. Infection may occur in one or both remnants anywhere along their course. Clinical signs of toxaemia and ill-thrift may be associated with the infection (Blood & Radostits 1989 (b)). McFarlane (1965) has cited hyperaemia and thickening of the broad ligament of the bladder with inflammatory changes of the peritoneal tissues as one manifestation of "navel ill" in lambs which have died in the post-parturient period.

Systemic infections, particularly those which are pyogenic or necrotising, gained by the foetus *in utero* via the umbilical vein have been described by Dyce *et al.* (1987) as having a greater likelihood of manifesting (as abscesses or necrotic foci) in the left lobe of the liver (umbilical moeity) since this area is vascularised almost exclusively by the umbilical vein in the foetus.

#### Histological lesions

Histopathological descriptions of omphalitis, omphalophlebitis, or other umbilical infections could not be found in the literature.

#### Clinical signs

Clinical sequelae to urachitis may include dysuria, pollakuria, pyuria and cystitis (Trent & Smith 1984 (a & b), Hylton & Trent 1987, Baxter 1989). General unthriftiness, inappetence, pyrexia and prostration may all be evident in calves with umbilical infections (Blood & Radostits 1989 (b), Roy 1989 (e)). Signs such as generalised peritonitis or abdominal pain, which could be expected as a result of infection of the umbilical remnants could not be found described in the literature.

#### Pleurisy/ Pneumonia/ Pericarditis

This disease classification covers a number of frequently interrelated lesions arising from a variety of aetiologies. Bobby calves, however, have a limited exposure time to many of the predisposing factors, pathogens and opportunists which can cause pneumonia in cattle. The three major respiratory conditions of importance in calves are aspiration pneumonia, enzootic pneumonia, and pneumonia associated with septicaemia. Pericarditis may also occur in association with pneumonia, so will also be discussed under this category. A common sequel to pneumonia important in the context of a slaughtered animal is the development of pleuritic adhesions.

#### (a) Aspiration Pneumonia

# Aetiology

In calves of this age group, the lesions will be principally caused by inhalation of milk or milk substitute during trough, bucket or hand feeding. Alley (1975) recovered

Pasteurella haemolytica, Escherichia coli and Staphylococcus aureus from lesions of acute pneumonia in very young hand-reared lambs (0-2 months), some of which had lesions consistent with aspiration of milk. No comprehensive review of the bacteria present in aspiration pneumonia in calves could be found.

#### Gross lesions

There is often hyperaemia of the lung and small amounts of exudate are able to be expressed from the airways of the cut surface. The lesions may extend to large focal areas of necrosis or suppuration in the ventral aspects of the lobes of the lung. There may be a fibrinous or fibrinopurulent pleuritis, which may result in adhesions between the lung and the pleurae (Blood & Radostits 1989). The severity of the lesions increases if bacteria are introduced to the lung (Cheville 1983 (a), Blood & Radostits, 1989 (i)). Cheville (1983 (a)) in fact deals with aspiration pneumonia under the heading of "gangrenous pneumonia".

# Histological lesions

Acute bronchiolitis with varying amounts of alveolar inflammation is seen, but lesions may progress to an acute gangrenous pneumonia with extensive pulmonary abscessation (Jubb & Kennedy 1985 (b)).

#### Sequelae

Animals surviving the acute phase may present with chronic persistent ill-health due to low grade toxaemia from a residual focus of infection in the lung tissue. If an overwhelming toxaemia develops, death may occur as soon as one day after aspiration (Blood & Radostits 1989 (i)).

#### (b) Enzootic Pneumonia

This is more common in calves which are housed rather than raised on pasture and is usually associated with high humidity and fluctuating temperatures in the immediate environment (Anderson 1983, Blood & Radostits 1989 (a & f)).

#### Aetiology

It is generally believed to be the end result of the interaction between environmental factors, including housing, temperature fluctuations, and ventilatory inadequacies, with

viruses such as Parainfluenza 3, and secondary bacterial infection, particularly with *P. haemolytica* (Jubb & Kennedy 1985 (b), Blood & Radostits 1989 (f), Roy 1990 (c)). Yates (1988) cites Bovine Respiratory Syncitial Virus (BRSV) as another important viral agent in the pathogenesis of respiratory disease, but there are no reports of this virus being isolated from cattle in New Zealand. The immune status of the calf, indicated by the level of systemic immunoglobulins, is also an important contributing factor in this disease, as with all diseases of calves (Roy 1990 (c)).

# Gross lesions

Uncomplicated cases of enzootic pneumonia are described by Yates (1988) as patchy areas of atelectasis (perhaps better termed collapse) in the anteroventral areas of the lung. Secondary infections with opportunists will result in lesions typical of the agent concerned.

*P. haemolytica*, a normal inhabitant of the upper respiratory tract of healthy cattle of all ages (Frank 1988), can superimpose itself on viral infections causing acute fibrinous pneumonia in very young calves. Lesions are usually cranioventrally distributed with extensive areas of consolidation affecting up to 60% of the lung volume. There may also be multiple necrotic foci present in the consolidated areas. The consolidation ranges in colour from red-black to red-brown or grey, and is uniformly spread throughout the cranioventral lung tissue. The interlobular septa are prominent and gelatinous. Any necrotic areas are sharply demarcated with thick white boundaries of fibrous tissue. Fibrinous pleurisy will occasionally also be present (Jubb & Kennedy 1985 (b)).

Pyogenic opportunists such as *Actinomyces (Corynebacterium) pyogenes* will result in a purulent bronchopneumonia and abscess formation (Omar 1966).

#### Histological lesions

The lesions of uncomplicated cases of enzootic pneumonia vary, but primarily involve a bronchopneumonia or interstitial pneumonia (Yates 1988). Secondary invasion by pyogenic or necrotising bacteria will further result in bronchiectasis or abscess formation (Yates 1988, Frank 1988).

If a massive growth of *P. haemolytica* similar to that which occurs in "shipping fever" in cattle is superimposed on enzootic pneumonia of calves, a severe fibrinous pneumonia

will ensue. Alveoli fill with eosinophilic, homogeneous fibrinous deposits, and there is distention of interlobular septa and lymphatics with serofibrinous or fibrinous exudate (Yates 1988, Jubb & Kennedy 1985 (b)). The necrotic areas seen grossly correspond to areas of coagulative necrosis surrounded by inflammatory cell debris and bacteria. The presence of clustered inflammatory cells with elongated, compressed nuclei known as "oat cells", is also characteristic of pneumonia due to *P. haemolytica* (Rehmutulla & Thomson 1981). Pneumonic pasteurellosis as a clinical entity is not described in adult or young cattle in New Zealand, although *P. haemolytica* and *P. multocida* are important potential pathogens of sheep and pigs, respectively, in this country (Manktelow 1984).

In a survey of lung and pleural lesions in veal calves (assumed to be about four months old) which was recently conducted in the Netherlands, 17.2% of calves at slaughter had extensive lung and/or pleural lesions involving one or more lung lobes. Of these, 93.6% had what was described as "cuffing/exudative pneumonia", 5.8% had "exudative pneumonia" with bronchiectasis, and 0.6% had abscesses or necrotic lesions (van der Mei & van den Ingh 1987). "Cuffing pneumonia", so termed due to the peribronchiolar lymphoid cuffs which develop, is also mentioned by Yates (1988), and is cited to be due to chronic infections with mycoplasmas.

## (c) Pneumonia as part of a Systemic Infection

Any infectious process which becomes systemic in nature may localise in organs where there is opportunity for penetration from the microcirculation. Organs where localisation commonly occurs are the liver, kidney, spleen and lung. This is due to two main factors:

- (a) Some organs, such as the lung, have a relatively large capillary bed, where blood perfuses at very low flow rates. This results in higher levels of exposure of the organ to organisms present in the blood. The lung has the largest capillary bed in the body, the surface area being 70m<sup>2</sup> in the adult human lung (Yates 1988).
- (b) Some organs have intrinsic mechanisms which retain organisms. Examples are the Kupffer cells lining hepatic sinusoids, similar macrophages lining the sinusoids of the spleen and bone marrow, and fenestration of the renal capillaries (Mims 1976).

#### Aetiology

Any pathogen capable of inciting a systemic infection is potentially capable of localising in the lungs and producing pneumonia (Jubb & Kennedy 1985 (b)). During a clinical septicaemia caused by *Salmonellae*, pneumonia has been occasionally reported as one of the pathological findings (Gibson 1961, Petrie *et al.* 1977, Greene and Dempsey 1986).

#### Gross lesions

Very numerous, widely distributed, small abscesses are most likely to arise from septic emboli of haematogenous origin. Lesions in the dorso-caucial areas of the lung are also most likely to be haematogenous in origin, but without an obvious source of septic emboli elsewhere in the body, the pathogenesis of such lesions is difficult to determine (Jubb & Kennedy 1985 (b)).

#### Histological lesions

The histological appearance of embolic pneumonia is not discussed by Yates (1988), while Jubb & Kennedy (1985 (b)) describe it as initially interstitial, with damage primarily to the alveolar walls and interstitial tissues. Other tissues in the lung become involved so rapidly, however, that pneumonias of this type cannot be strictly classified as "interstitial".

#### (d) Pericarditis

#### Aetiology

Fibrinous pericarditis may result from an infection by non-pyogenic bacteria such as coliforms, *Clostridium spp.* or *Mycobacterium bovis*. These bacteria are cited by Jubb & Kennedy (1985 (e)), and Blood & Radostits (1989 (c)), as being disseminated by the haematogenous route, or occuring as a direct extension of pleurisy or myocarditis. Van Fleet and Ferrans (1988) consider fibrinous pericarditis to be usually the result of haematogenous infection, and Smith & Jones (1966) dispute the concept of direct extension of infections from the pleurae to the pericardium and contend that the pericardial sac is an effective barrier against such extension. These authors also mention that viral infections may initiate pericardial infection.

#### Gross lesions

The fibrinous exudate is usually yellow and the pericardium is thickened. As the lesion progresses, adhesions between the pericardium, myocardium and the pleurae develop to varying degrees. In severe cases there may be complete obliteration of the pericardial sac (Blood & Radostits 1989 (c), Jubb & Kennedy 1985 (e)).

#### **Arthritis**

Lesions may involve from one to most of the major joints of the affected animal. Arthritis in farm animals is almost always infectious, haematogenous in origin and at least initially polyarticular (Jubb & '(ennedy 1985 (c)).

If a lesion of traumatic origin becomes infected, arthritis will develop in the joint as described below.

# Aetiology

Streptococci and coliform bacteria are common causes of polyarthritis in neonatal calves. Infections with *Salmonella spp, A. pyogenes*, or *Erysipelothrix rhusiopathiae* may occur at any age (Radostits & Acres 1980, Jubb & Kennedy 1985 (c), Blood & Radostits 1989 (k)).

#### **Gross lesions**

# (a) Fibrinous Arthritis

This occurs as bacteria invade the synovial stroma and fluid. Villi develop and hypertrophy, and the synovial fluid increases in volume. The joint fluid may become turbid and contain fibrin strands. If the infection is overcome, complete resolution may occur. Although the villi continue to enlarge as the inflammation progresses, synovial effusion lessens, and a pannus may develop between articular surfaces, causing adhesions which result in permanent stiffness (Jubb & Kennedy 1985 (c), Doige 1988).

#### (b) Purulent Arthritis

This is a more severe and destructive form of arthritis than fibrinous arthritis. It may arise from primary infection of a single joint, but is more commonly haematogenous in

origin. In the early stages of infection the synovial fluid is thin and cloudy, but as the lesion progresses, the fluid becomes thick and purulent. The articular cartilage may degenerate and become necrotic. The reaction may extend to result in periarticular abscesses, osteomyelitis, or fistulation to the skin. If spontaneously or therapeutically terminated, the resultant lesions will depend on the extent of damage incurred. The degree of healing ranges from complete resolution to permanent stiffness due to intra-articular ankylosis (Jubb & Kennedy 1985 (c)).

In both types of arthritis, there will be filling of the joint with excess synovial fluid or purulent material so that it appears puffy in the early stages. If the disease progresses far enough, it will eventually result in permanent boney and periarticular enlargement and/or distortion.

The initial inflammation may subside in many, particularly smaller, joints, with the larger joints (stifle, hock, carpus) developing a more chronic reaction, usually bilaterally (Jubb & Kennedy 1985 (c)).

With arthritis of haematogenous origin, there may also be evidence of other disease entities such as hypopyon, endocarditis, pericarditis, meningitis and septic emboli in the kidney(s) or spleen (Blood & Radostits 1989 (a)).

#### **Enteritis**

It is generally recognised that neonatal calf diarrhoea is not a simple disease, but rather a group of diseases sharing common characteristics but with different aetiologies and a variety of predisposing factors. It results from the interaction between enteropathogenic organisms, herd and individual immunity, and environmental and management factors (Amstutz 1970, Radostits & Acres 1980, Blood & Radostits 1989 (d), Roy 1990 (a)).

#### Aetiology

In the United States, the four main infectious agents associated with diarrhoea in young calves are rotavirus, *Cryptosporidium*, bovine coronavirus and enterotoxigenic strains of *E. coli* (Tzipori 1985).

The most common bacterial pathogens involved in neonatal calf diarrhoea and enteritis are enterotoxigenic strains of *E. coli* (ETEC), although *Salmonella spp* may infect young calves, often resulting in signs of systemic infection as well as enteric disease (Tzipori

1985). Enterotoxigenic *E. coli* classically produce disease in hypogammaglobulinaemic calves less than a week old (Wray & Morris 1985). *Salmonella spp*, on the other hand, generally affect calves older than one week of age (Amstutz 1976, Radostits & Acres 1980).

It is now believed that the susceptibility of very young calves to ETEC infections is not necessarily related to the adequacy of colostral intake, since resistance develops rapidly after two or three days once the normal enteric flora has established itself (Woolcock 1979 (b), Radostits & Acres 1980)). The ingestion of colostrum does, however, appear to reduce the severity and duration of the disease process (Radostits & Acres 1980, Jones et al 1977, Roy 1990 (d)). The immunoglobulin content of colostrum is predominantly IgG, with lesser amounts of IgA and IgM. All are rapidly absorbed into the circulation, thus conferring immediate humoral antibodies to the calf. The IgA absorbed by the neonatal calf is lost again via external secretions, including those to the gut, by normal transudative processes. This low level of antibody confers some protection from infection, while not interfering with the development of the intestinal microflora (Woolcock 1979 (b)). The flora initially comprises E. coli, Streptococcus spp., and Clostridium perfringens. These are followed by Lactobacillus spp., particularly in the abomasum and small intestine, and Bacteroides spp in the large intestine. The coliform population is then much reduced and restricted to the ileum and large intestine (Woolcock 1979 (a), Roy 1990 (a)).

#### Enteric coliform infection

In veterinary medicine, disease due to coliforms has been classified into three major entities: septicaemia, mastitis, and diarrhoea (Wray & Morris 1985). The coliform strains producing diarrhoea are strictly enteric in their location, and in human medicine are further subdivided on the basis of their mode of action on the gut (Matthewson & DuPont 1986):

(1) Enteropathogenic E. coli (EPEC): strains described in human medicine, where hospitalised infants and their mothers represent the major reservoirs of infection. They do not produce enterotoxin, nor are they enteroinvasive, but they adhere to an area of the intestine, causing localised destruction of the villi. Although serotyping will identify some EPEC strains, not all strains belonging to a particular serotype are necessarily enteropathogenic.

- (2) Enterotoxigenic E. coli (ETEC): These strains adhere to the gut and produce enterotoxins which cause the diarrhoea. Similar strains commonly cause diarrhoea in calves as well as humans.
- (3) Enteroinvasive E. coli (EIEC): These strains occasionally produce diarrhoea in humans, invade the intestinal epithelial cells, multiply in the cells, and result in inflammation and ulceration of the lower intestine.

E. coli serotype O157:H7 has recently been identified as a cause of haemorrhagic colitis in humans. It has been found to be associated with the ingestion of a number of retail meats, including rare, ground beef (Doyle & Schoeni, 1987).

Roy (1990 (a)) describes a similar categorisation of *E. coli* infections in calves, pointing out that calves have been inferred to harbour strains capable of producing enteritis in humans. As far as neonatal calves are concerned, the major enteropathogenic strains appear to be the ETEC strains. The disease entity may follow an acute course, with the production of two enterotoxins which result in fluid and electrolyte imbalances and death although lesions may not be detected at *post-mortem* examination (Wray & Morris 1985, Roy 1990 (a)). ETEC strains of *E. coli* have no particular propensity to produce septicaemia (Cheville 1983 (b)).

#### Clinical signs

Diarrhoea is more severe if infection is acquired in the first two weeks of life, this also being the age when *E. coli* is most likely to infect calves. The onset of diarrhoea is acute, with profuse, watery, pale yellow or green diarrhoea usually without blood. The tail and buttocks become soiled by faecal material. There is initially abdominal distension, and fluid-filled intestines may be detected. Many cases rapidly develop dehydration and anorexia. Death may occur in only a few days, with calves in the terminal stages becoming prostrate and suffering cardiac arrhythmias due to electrolyte imbalances (Radostits & Acres 1980, Roy 1990 (a)). Mildly to moderately affected calves may spontaneously recover from diarrhoea of a few days duration.

#### Gross lesions

Acutely affected calves may have the small intestine and caecum distended with yellow watery material, poorly digested milk, and gas. There are, however, no pathognomonic lesions to differentiate coliform, viral, or cryptosporidial infections.

#### **Enteric salmonellosis**

Salmonellosis usually affects calves of the two to six week age group. As a clinical entity, the disease in calves in New Zealand is apparently not a common event (only 3.9% of cases submitted to Wallaceville Animal Research Centre for confirmation of salmonellosis were confirmed by de Jong & Ekdahl in 1965). The documented rate of recovery (3.5 to 13%) of salmonellae from the faeces, liver, spleen and mesenteric lymph nodes of apparently healthy calves submitted for slaughter, however, can be very much higher (Nottingham & Urselmann 1961, Nottingham et al 1971).

# Clinical signs

The course of the disease in young calves is usually acute, with common clinical signs of moderate to severe diarrhoea consisting of liquid, malodorous intestinal contents mixed with mucus and blood. The perineal area may be scalded and matted by yellowish-brown faeces. The calf quickly becomes lethargic, dehydrated and emaciated, and death rapidly follows (Radostits & Acres 1980, Wray & Morris 1985, Blood & Radostits 1989 (e), Roy 1990 (b)).

#### Gross lesions

Submucosal and subserosal haemorrhages of the gut may be present and the mesenteric lymph nodes are oedematous and petecchiated. There may be focal necrotic lesions in the liver and spleen. Epicardial haemorrhages, splenomegaly, and icterus are often seen. Lesions may not be observed at *post-mortem* examination of some fatally affected calves. In the terminal stages, a secondary, exudative and necrotic pneumonia may develop. If the animal develops a less fulminating form of the disease and survives, it may develop polyarthritis or meningitis (Gibson 1961, Petrie *et al* 1977, Radostits & Acres 1980), while some non-fatal infections may manifest as transient or chronic diarrhoea, growth retardation and unthriftiness, osteomyelitis, pneumonia or a shifting arthritis (Gibson 1961, Robinson 1966, Greene & Dempsey 1986, Firth *et al* 1987).

The serotypes of salmonellae most commonly producing disease in cattle in New Zealand are *S. typhimurium* and *S. bovis-morbificans*, both of which show little host specificity (Anon. 1980).

# Other pathogens

#### Clostridium perfringens

Infection with *C. perfringens* has been described as a cause of enteritis in neonatal calves. The most common biotypes to produce disease in neonatal calves are believed to be A, B, and C, all of which are members of the normal gut and soil flora. Type A, however, is the major type involved in outbreaks of human food poisoning (Genigeorgis 1981). The infection occurs in calves up to ten days of age, with an acute and usually fatal course (Blood & Radostits (1989 (h)). The major lesion is a haemorrhagic enteritis, with ulceration and necrosis of the mucosa and fibrin exudation (Cheville 1983 (c)). Signs of a generalised toxaemia may also be present. The pathogenicity of *C. perfringens* to calves has been disputed by Roy (1989 (a)), who contends that there is no evidence of a relationship between the incidence of diarrhoea and the presence of *C. perfringens* in the gut or faeces of affected calves. Infection of domestic animals in New Zealand with *C. perfringens* type C is not known to occur (Anon. 1990 (a)).

#### Campylobacter spp

There is little evidence to implicate *Campylobacter* spp. as a primary aetiological agent of enteritis in young calves, although two species, *C. coli* and *C. jejuni* are found in intestinal disorders, dysentery, and diarrhoea in various animals (Roy 1990 (a)). The prevalence of this bacterial species is, however, high in normal cattle (Morgan *et al* 1986, Blackmore & Humble 1987).

# Miscellaneous Bacterial Infections

#### Leptospirosis

Leptospirosis is unlikely to occur in very young calves, unless the environment of the calf shed is contaminated by rodents or pigs carrying such *Leptospira interrogans* serovars as *copenhageni, ballum* or *pomona* (Hathaway 1981, Blackmore & Humble 1987). If leptospiral infection is present in the herd, the calves will be protected by maternal antibody until several months of age (Hathaway 1981). There has been increasing awareness of the zoonotic risk of leptospirosis to farmers handling cattle, resulting in the increased use of vaccination to control the problem. Leptospirosis in calves due to any of the above serovars would result in an acute clinical syndrome detectable at *ante-mortem* inspection and rendering the calves unsuitable for presentation for slaughter.

# <u>Tuberculosis</u>

The infection of young calves in New Zealand with *Mycobacterium bovis* is a very unlikely event and although congenital tuberculosis may occur at a rate of up to 0.5 % of newborn calves where the disease is common (Jubb & Kennedy 1985 (d)), bovine tuberculosis is currently at a low prevalence in New Zealand.

Since 1970, all dairy cattle in New Zealand have been under regular surveillance testing, resulting in reactor rates dropping from an initial 8.6% to 0.05% in 1980 (Anon. 1986). It was found that this rate of reduction of the prevalence of tuberculosis was not sustainable and a reservoir of infection was identified in the feral population of Australian brush-tailed possums (*Trichosurus vulpecula*). In 1988, the prevalence was still about 0.06% (Anon. 1988) and it was recognised that total eradication of the disease from New Zealand was likely to be very difficult. Nevertheless, by a combination of surveillance of stock presented for slaughter, surveillance testing, and the implementation of a control policy, the areas currently declared non-endemic could be maintained, and the endemic areas contained. As at 1986, 98% of New Zealand cattle herds are classified as accredited free of tuberculosis, and 2% as infected or on movement control (Anon. 1986).

The primary site of infection in congenital tuberculosis of calves is the liver. The disease progresses rapidly to become generalised, and calves will usually die within a few weeks or months (Jubb & Kennedy 1985 (d)). Other workers consider the feeding of tuberculous milk far more important than congenital infection as a source of infection for calves (Blood & Radostits 1989 (g)).

#### Septicaemia/Bacteraemia

The presence of bacteria in the bloodstream of animals is described as a common event, but one which does not necessarily imply that the animal is clinically ill (Walter & Israel 1975, Mims 1976, Gracey 1986, Franco 1988, Blood & Radostits 1989 (a)).

# <u>Bacteraemia</u>

has been defined as "the presence of bacteria in the blood" (Blakiston's Gould Medical Dictionary 1984). Franco (1988) considers a bacteraemia to involve the "temporary presence of bacteria in the blood without gross lesions or clinical signs of disease". In contrast to this, Walter & Israel (1975) define

bacteraemia as the "transient presence of organisms in the bloodstream", which "causes few symptoms". Cheville (1983 (d)) defines bacteraemia as "the presence of bacteria in the blood, occurring either silently or as the prelude to severe systemic disease."

# Septicaemia

has been defined as "a clinical syndrome characterised by a severe bacteraemic infection, generally involving the significant invasion of the bloodstream by micro-organisms from a focus or foci in the tissues, and possibly even with the micro-organisms multiplying in the blood" (Blackiston's Gould Medical Dictionary 1984). Walter & Israel (1975) add that septicaemi? differs from bacteraemia in the following ways:

- (1) It is associated with severe clinical symptoms.
- (2) There are more organisms in the blood.
- (3) It indicates a very inadequate host resistance.

Gracey (1986) and Franco (1988) both define septicaemia as including not only "the presence of large numbers of bacteria" in the blood, but also viruses, protozoa, and toxins. When bacteria constitute the infective organisms, septicaemia is described as a severe and persistent bacteraemia.

The term septicaemia is, however, used interchangeably with bacteraemia in some instances (Dow & Jones 1989), and one pathology text referred the reader to "septicaemia" upon searching "bacteraemia" (Anon. 1990 (b)). Cheville (1983 (d)) cites septicaemia as being applied "clinically to the syndrome of septic bacteraemia". Septicaemia is thus a clinical term, rather than a pathological entity.

The major factor adversely affecting the resistance of the animal to systemic infections is inadequacy of the immune system. Since the calf is born agammaglobulinaemic (Woolcock 1979 (b), Wray & Morris 1985, Blood & Radostits 1989 (a), Roy 1990 (d)), it requires the ingestion of colostrum from the cow to protect it until its own immune system has developed sufficiently in response to challenge. The immune system of the calf is not so much underdeveloped at birth as unchallenged. Antibody responses to various infectious agents can occur *in utero* as early as 120 days of gestation (Osburn

1986), however to overcome and survive the battery of challenges received after birth, high levels of serum IgG are needed. These are normally supplied via the colostrum, so septiacemias typically occur in calves which have received inadequate amounts of, or no colostrum. The feeding of colostrum beyond four to six hours after birth will also result in hypo- or agammaglobulinaemic calves, since after that time, the uptake of immunoglobulins from the gut is much less efficient (Woolcock 1979 (b), Radostits & Acres 1980, Blood & Radostits 1989 (a), Roy 1990 (d)).

#### Associated Infectious Agents

Bacteraemia: Virtually any bacterial species capable of penetrating the skin,

mucous membranes, lungs or intestines.

Septicaemia: E. coli strains which are distinct from those which produce diarrhoea,

and Salmonella spp, are the most common causative agents of septicaemia in young calves, but other pathogens could include Listeria monocytogenes, Clostridium spp and Streptococcus spp

(Radostits & Acres 1980).

# Clinical signs

Bacteraemia: By definition, none unless there are specific host and pathogen

characteristics which predispose to the localisation of bacteria in

tissues (Walter and Israel 1975, Cheville 1983 (d)).

Septicaemia: Pyrexia, followed by rapid prostration and death. There may also be

other signs related to the action of the causative organism, such as

diarrhoea with salmonellosis.

#### Gross lesions

Bacteraemia: Usually none. However, a common finding on post-mortem

inspection of calves, and which is presumed to result from an earlier bacteraemia, is multiple, white, necrotic foci 1-3 mm in diameter in the cortex of the kidneys. These lesions may involve anything from one lobule of one kidney to both kidneys in their entirety. They represent areas of focal interstitial nephritis and, because of their appearance, are colloquially called "white spotted kidney". These

lesions have been attributed to emboli derived from a variety of organisms including Salmonella, Brucella, (Gracey 1986) and coliforms (Jubb & Kennedy 1985 (a)). A confused mixture of other possible aetiologies have been suggested, including malignant lymphoma, embryonic mesonephric tissue or infectious emboli of uncertain origin (Smith & Jones 1966). However, coliforms are the only organism cited as having been recovered from these lesions, and then only occasionally. In the meat industry the lesions are generally regarded as incidental and unaesthetic in nature rather than hazardous, since there is usually no obvious primary site of infection or involvement of the carcass as a whole, and the prevalence rapidly decreases with age (Thomson 1978, Gracey 1986).

Septicaemia:

Hyperaemia and swelling of the liver, spleen and lymph nodes, subendocardial haemorrhages and muscle wasting. The extent of lung involvement (hyperaemia, haemorrhages, oedema) depends on the propensity of this organ for high bacterial clearance, and this varies between animal species (Cheville 1983 (d)).

Gracey (1986) and Franco (1988) both cite the following lesions as present in various combinations: carcass congestion, slow blood clotting, slight or absent rigor mortis, petechiae or ecchymoses in the kidneys, myocardium, liver, mucous and serous membranes, generalised lymphadenitis, blood stained serous exudates in the abdomen or thorax, parenchymatous degeneration of major organs, and icterus. It is unfortunate that many of the terms used by these authors (e.g. lymphadenitis and parenchymatous degeneration) are too vague to be of much value. Radostits & Acres (1980) also cite subserosal and submucosal haemorrhages, and fibrinous exudation on serous membranes including the pleurae and peritoneum as signs of septicaemia.

Acute infections, particularly those which become septicaemic, may lead to the generalised activation of the blood coagulation system. This phenomenon is known as disseminated intravascular coagulation (DIC) (Walter & Israel 1975, Slauson & Cooper 1982) and has been initiated by endotoxins (Thomson *et al* 1974), or the

widespread damage of vessel endothelium (Walter & Israel 1975, Slauson & Cooper 1982). The result is the formation of fibrin in the circulation, producing vascular obstruction and micro-infarction (Walter & Israel 1975), and the subsequent presence of petechiae in many organs.

#### Working Definition of Septicaemia for this Study

Bobby calves slaughtered in New Zealand are subjected to routine inspection procedures as described earlier. The judgements passed on the carcass, however, are very harsh when compared to other classes of livestock. Guidelines in Manual 16 (Anon. 1977) describe most infections in bobby calves as ante-natally acquired, and therefore requiring total condemnation of the carcass. Discussions with meat inspection staff further revealed that as part of their training, calves were described as immunologically incompetent, so most, if not all, disease detected was likely to be generalised, and a public health risk. The severity of the judgements thus appears to stem from the assumption that any disease acquired by the calf prior to slaughter will almost invariably render the calf septicaemic at the time of slaughter.

Calves presented for slaughter are examined by MAF staff at the farm gate and in the yards at the slaughterhouse to assess their suitability for slaughter. The calves which are slaughtered will have few, if any, overt clinical signs of disease, so in practical terms, the severe clinical signs associated with septicaemia cited in the definitions above will be absent in calves presented for slaughter. There must be concern, however, about the high numbers of pathogenic bacteria which could be present in an animal which has not yet been overwhelmed by an early septicaemia. Such an animal could conceivably be presented for slaughter, and its exclusion from human consumption will rely on the detection of generalised, non-specific abnormalities in the carcass.

It appears that in the case of bobby calves in New Zealand, septicaemia is further implied by the presence of virtually any lesion in the carcass or viscera. From the literature reviewed, it was considered more likely that for the animal to survive for a period of time sufficient for the development of specific lesions in the carcass, the animal will have or have had a bacteraemia involving much fewer bacteria in the circulation for a shorter period of time, rather than a septicaemia.

The definition of septicaemia for the purposes of this study is therefore the presence of large numbers of bacteria in the general circulation, involving slight or absent clinical signs and with or without gross evidence of early systemic infection in the carcass. Bacteria should be isolated from all tissues sampled, and histological evidence of early septicaemia, such as thromboses in blood vessels, organ congestion and leucocytosis, should be present in the tissues examined.

The definition of bacteraemia adopted in this study is the presence of smaller numbers of bacteria in the general circulation, with or without gross evidence of localisation in the carcass. Clinical signs will be slight or absent, and if present affect sites of bacterial localisation (e.g. the joints). Gross lesions will similarly reflect sites of bacterial localisation rather than generalised infection of the carcass. Bacteria are only likely to be isolated from tissues which present a suitable environment for survival, and predispose to localisation in that tissue (e.g. Kupffer cells in the liver), particularly since only small numbers of bacteria are expected in cases of bacteraemia. Histological lesions consistent with the presence of bacterial emboli, or a mild host response to the infection, may be present in tissues sampled.

"Navel ill" is one of the most frequent categories resulting in carcass condemnation within the group condemned for disease. New Zealand is not consistent with the requirements of other countries in its judgement of "navel ill", and may be causing significant losses to the producers for no appreciable benefit to public health. It is also possible that similar wastage is occurring in other disease categories.

#### **OBJECTIVES OF THE PRESENT STUDY**

The present study was undertaken to fulfil the following objectives:

- (1) To record the prevalence of gross pathological lesions in slaughtered bobby calves which are detained by meat inspectors in New Zealand.
- (2) To provide a practical description of the lesions associated with omphalophlebitis in gross, microbiological and histopathological terms.
- (3) To calculate the accuracy of organoleptic meat inspection techniques in detecting acute cases of omphalophlebitis with or without systemic effects.
- (4) To determine the prevalence of septicaemia associated with potential pathogens in calves presented for slaughter in New Zealand.

(5) To comment on the significance of any public health hazard associated with lesions seen in bobby calves in New Zealand which require detention by meat inspectors, with special reference to omphalophlebitis.

#### MATERIALS AND METHODS

# PART I: COMPARISON OF THE GROSS LESIONS, HISTOLOGICAL LESIONS, AND MICROBIAL FLORA OF BOBBY CALF COHORTS WITH AND WITHOUT OMPHALOPHLEBITIS ("NAVEL ILL")

A detailed characterisation of the pathological lesions associated with omphalophlebitis was conducted at Waitaki International's slaughterhouse at Feilding (ME 58) during 1989.

The bobby calf season for 1989 began at Feilding on 20-7-89, when calves were slaughtered on only one day during that week. From 31-7-89 until 15-9-89, slaughtering took place on four week days, Wednesdays being the no-kill day. The season was terminated prematurely due to commercial factors and for the final two weeks, calves which would normally have been slaughtered at Feilding were diverted to Waitaki's Imlay slaughterhouse located at Wanganui.

#### Selection of Cohorts

The prevalence of omphalophlebitis at the Feilding slaughterhouse was subjectively estimated by MAF inspectors to be 0.25-0.5% of calves slaughtered. The predicted season's tally was around 25,000 animals, therefore approximately 62 to 125 animals were likely to be condemned by meat inspectors for gross lesions of "navel ill". With this relatively small number, it was judged possible that all of the carcasses which were condemned for "navel ill" could be examined in detail and selected tissues obtained for microbiological and histological examination.

Carcasses identified by inspectors as having gross lesions of "navel ill", were identified with an AGM 74 (detain) ticket skewered to one leg. The carcass was then diverted to the veterinary inspection rail for further examination. The viscera corresponding to the carcass were placed in a bucket by a viscera inspector and placed alongside the carcass. Carcasses and viscera were kept in strict chronological order to ensure that no mismatching occurred.

This study was designed as a cohort study, with "exposed" cohorts those condemned by meat inspectors for "navel ill", while "non-exposed" cohorts were selected from carcasses passed by meat inspectors as normal. "Navel ill" is considered in the meat industry to lead to septicaemia, the disease state of interest.

Non-exposed cohorts were selected by using a series of random times tables (Appendix IV). The use of random times tables resulted in the systematic selection of a predetermined number of carcasses, with a random start time, during the periods when carcasses were passing the inspection stand. The selection was made only from carcasses which were considered fit for human consumption by the meat inspectors, so that carcasses with other disease entities such as pneumonia were excluded from the group, and would not confound the results.

Consideration was not given to sample size requirements, since the purpose of the study was to gather preliminary data on the gross, histological and microbiological aspects of carcasses from calves with "navel ill", and from this data estimate the true rate of septicaemia in both grossly normal calves, and those with "navel ill". As many carcasses were included for examination as was practicable in terms of time and financial resources.

#### **Examination of Samples**

The working definitions of septicaemia and bacteraemia were stated earlier. The preliminary trials which led to the sampling regime used in the histological and microbiological examination of the carcasses are presented in Appendix I.

#### Gross pathology

All carcasses and viscera were examined in detail by the author once they had been rerouted from the main chain to the veterinary inspction rail, and the results were entered on a recording form (Appendix III). The gross appearance of all organ and carcass surfaces, visceral lymph nodes, heart valves, anterior chamber of the eye, the character of the fluid from major joints, and the extent of any lesions involving the umbilicus or associated vessel remnants was recorded. All superficial lymph nodes were palpated and investigated further if abnormalities in size or texture were found. A pictorial record of the extent of incisor eruption was kept for retrospective ageing purposes, and the sex

of the carcass noted. The non-exposed cohorts were examined in exactly the same manner except for the characterisation of the joint fluid. In these carcasses, joint fluid was aspirated using an eighteen gauge needle and a 1ml tuberculin syringe, since meat from carcasses contaminated by joint fluid would be ineligible for export to the USA. Offals from control carcasses were condemned, since they were mutilated as a result of the examination, and had been on trays which were cleaned but not sterilised between animals.

# Histopathology

Tissue samples were fixed in 10% buffered formalin for at least 48 hours prior to processing. Blocks of tissue were processed and embedded in paraffin by routine methods and sections cut at 5  $\mu$ m. All sections were stained routinely with Haematoxylin and Eosin (HE) stain, and where confirmation of the presence or absence of bacteria was required or desirable, the Gram-Twort stain was used.

Samples taken from all carcasses examined for histology were liver (left lobe), kidney and umbilical vein two centimetres from the umbilical fissure of the liver. Umbilical samples were examined in both transverse and longitudinal section. Samples of lesions of special interest (e.g. hepatic abscesses, nephritis, urachal or umbilical arterial lesions) were occasionally taken in a similar fashion.

#### Microbiology

#### Sampling method

In all carcasses examined, samples for microbiological examination were taken from the liver, the umbilical vein at the umbilical fissure (two centimetres), and the left semimembranosus muscle after removal of the surface fascia. All samples were surface sterilised by dipping in 95% ethanol, followed by flaming. Each sample was then placed in a pre-weighed, sterile Seward laboratory "400" bag (Laboratory Services) and labelled with the calf identity, and group status.

In the laboratory, the bags containing the samples were reweighed, to enable calculation of the net weight of each sample. Sterile, normal saline at room temperature was added to each sample at the following rate:

Umbilical vein < 10 grams:

50 ml.

Umbilical vein > 10 grams:

100 ml.

Liver and muscle:

100 ml.

The samples were then homogenised in a Colworth Stomacher 400. It was found that 45 seconds of homogenisation was adequate for liver samples, while muscle samples required 3.5 minutes to achieve fraying and maceration of the tissue. The vein samples were never grossly damaged by the stomaching process, but removal of any material present in the lumen of the tissue was reliably achieved in 1.5 minutes.

#### Incubation and media

The samples were allowed to settle so that surface frothing was minimised, and sampling with a 10 microlitre, calibrated, sterile, disposable loop (Banksia Scientific Company, PTY Ltd) was not compromised. One loopful of well mixed, undiluted sample was taken and spread over each of a blood agar plate, a MacConkeys agar plate, and a Xylose-Lysine-Decarboxylase (XLD) plate, all of which were pre-incubated overnight at 37°C. The plates were labelled with the date, calf number, category (NI or NNI), and tissue sampled. The plates were then incubated for 48 to 72 hours at 37°C, and examined for evidence of growth at 24 and 48 hours. If growth was heavy or swarming, subcultures were taken at 24 hours, but otherwise they were subcultured at 48 hours. Colonies were counted and growth characteristics recorded. Pure subcultures of each colony type observed on the primary plate were made by picking off individual colonies. Identification of colonies was undertaken from broths generated from pure subcultures. This method was used to ensure that in every instance a pure culture was obtained, and small, slower growing colonies were not inadvertently picked off into broth with the primary colony of interest.

Details of many of the media, methods, and API kits used for the identification of bacteria (marked \*) are described in Appendix II. Those media obtained from DIFCO Laboratories (Detroit, Michigan 48232, USA) are marked \*\*.

# Colony identification

# (1) Gram stain.

# (a) Gram positive cocci:

One colony from a pure subculture was streaked onto a TGY\* slope and incubated for 24 hours at 37°C. The Catalase test\* for the identification of staphylococci was performed on the subsequent culture.

#### Staphylococcus species:

These were streaked onto nutrient agar, labelled with the date, calf and tissue identification, incubated at 37°C for 24 hours, and refrigerated until the end of the experimental period. They were then observed for pigment, and tested for the presence of coagulase and DNase\* to differentiate *S. aureus* from coagulase negative staphylococci.

#### Streptococcus species:

With two exceptions, these were not identified further since their growth characteristics, principally their lack of  $\beta$ -haemolysis, indicated that they were non-pathogenic.

Two isolates recovered from muscle tissue were further identified using the following method:

An entire pure subculture of each isolate was inoculated into heart infusion broth. Four drops of the broth were then inoculated into fermentative broths, aesculin agar and litmus milk at 37°C. Purity plates were prepared on blood and McConkeys agar.

#### (b) Gram positive rods:

If these morphologically resembled *A. pyogenes* on Gram staining, a series of biochemical tests was performed to differentiate them from other gram positive bacilli:

An entire pure subculture of the bacillus was inoculated into heart infusion broth. Four drops of the broth were then inoculated into or onto litmus milk, a Loefflers slope, triple

sugar iron agar slopes<sup>\*\*</sup>, and a TGY slope. Purity plates were prepared on blood and MacConkeys agar, and all preparations were incubated at 37°C.

# (c) Gram negative rods:

One colony from a pure subculture was initially subjected to the oxidase test.

### (i) Oxidase negative rods.

Those which morphologically resembled Enterobacteriaceae were identified using a biochemical test mini-kit, the API 105<sup>\*</sup>. This system is designed to identify *Enterobacteriaceae* plus other gram negative rods.

Some *E. coli* isolates were poorly differentiated from *Serratia spp.* and *Klebsiella oxytoca* on the API10S result profile. Using a chart compiled in 1973 by Dr. W.H. Ewing (Enteric Bacteriology Laboratories, Atlanta) for the differentiation of enterobacteriaceae by biochemical tests, negative adonitol and inositol tests were considered sufficient to identify an isolate as *E. coli*.

## (ii) Oxidase positive rods.

Bacteria which morphologically resembled *Pseudomonas spp.* were subjected to a more comprehensive kit test, the API 20 NE<sup>\*</sup>.

Those morphologically resembling *Pasteurella spp.* were identified by following the profile described by R.E. Weaver and D.G. Hollis (U.S. Department of Health and Human Services, Public Health Service, Centre for Disease Control, Atlanta, Georgia 30333) for the identification of gram negative organisms. Colonies were inoculated into fermentative broths containing sugars (Glucose, xylose, mannitol, lactose, sucrose, maltose, levulose, sorbitol, salicin, arabinose, dulcitol, trehalose, rhamnose, raffinose, mannose, melibiose), triple sugar iron agar slopes with lead acetate paper (Johnsons of Hendon Ltd, London NW4, England), decarboxylases (arginine- (dihydrolase), lysine-& ornithine-, nitrate infusion, aesculin agar, Christensen's urea, Simmon's citrate agar, Salmonella-Shigella agar, MacConkey's agar, all incubated at 37°C, and TGY slopes incubated at 25°C and 42°C.

# Estimation of the Prevalence of Haematomas Associated with the Umbilical Arteries in Bobby Calves Submitted for Slaughter

A smaller study was undertaken during the period spent at Feilding as a result of observations made during slaughter. It had been observed that a proportion of the calves condemned for "navel ill" had extensive haematomas involving the umbilical arteries and/or the urachus. Some of these appeared to be fresh, but others were older, and appeared to be infected. Infection of the umbilical arteries has been described in lambs which died in the post-parturient period by McFarlane (1965), but no reports could be found describing severe haemorrhages in four-to-ten day old calves. Since these cases were affecting the inspectors' judgement, it was decided to investigate the prevalence and extent of such haematomas in the slaughter population.

The examinations were carried out while carcasses were being processed and the examination of carcasses with "navel ill" was not required. The carcasses were viewed after the midline abdominal incision had been made but before the terminal colon had been removed from the carcass. Between 40 and 80 carcasses were viewed on each of six days between 17/8/89 and 27/8/89. The presence and extent of any haemorrhage involving the umbilical arteries was recorded.

# PART II: A STUDY OF THE LESIONS PRESENT IN CALVES CONDEMNED BY MEAT INSPECTORS

Detailed examinations were performed on the carcasses and viscera of all bobby calves condemned, for whatever reason, by meat inspectors in the slaughterhouses listed in Table 3.1.

TABLE 3.1: LOCAT

LOCATION OF AND DATES OF VISITS TO SLAUGHTERHOUSES DURING THE 1989 BOBBY CALF SEASON.

Company	Location	Date
Waitaki Fielding	(Fielding)	4-9 to 15-9
Weddel Crown Tomoana	(Hastings)	18-9 and 21-9
Waitaki Stoke	(Nelson)	19-9 and 20-9
Waitaki Waitara	(New Plymouth)	25-9 to 27-9
Waitaki Imlay	(Wanganui)	2-10 to 5-10
Waitaki Horotiu	(Hamilton)	6-10 to 11-10

# **Gross Pathology**

Detailed gross examinations were made in the same manner as previously outlined in Part I.

In addition to the details of the gross findings normally recorded, the ticket used by the meat inspector to classify the disease condition was also noted. A provisional diagnosis of the disease condition was made by the author.

# Histopathology

Samples relevant to the disease entity were collected in some cases and prepared for subsequent histological examination in the manner described in Part I.

# Microbiology

Microbiological examinations were not performed on samples obtained during Part II of the study.

# **RESULTS: PART I**

# COMPARISON OF THE GROSS LESIONS, HISTOLOGICAL LESIONS, AND MICROBIAL FLORA OF CARCASSES FROM BOBBY CALF COHORTS WITH AND WITHOUT OMPHALOPHLEBITIS ("NAVEL ILL")

The investigation conducted at Waitaki Feilding's slaughterhouse involved the detailed examination of 85 carcasses in two cohort groups. Group 1 consisted of 31 carcasses without "navel ill" and passed by the meat inspectors as fit for human consumption. Group 2 consisted of 54 carcasses which were condemned on the basis of the presence of "navel ill".

# (1) Normal Carcasses and Viscera

One of the 31 carcasses sampled from the population of "normal" carcasses passed by inspectors had gross lesions of "navel ill" consistent with Category 3 described later in this chapter, i.e. exhibiting inflammatory necrosis of the umbilicus alone. The rate of non-detection of "navel ill" is calculated as 1/31 (0.032). Unfortunately a very small sample of normal animals were examined, and calculation of the 95% confidence interval (Schwabe 1979) for this figure indicates that up to 9% of the 12,252 calves processed during the course of this part of the study may have been passed with similar gross lesions of the umbilicus.

#### **Gross features**

#### The carcass

The average normal calf carcass observed in this sample had the large bone structure and very prominent joints, particularly the stifles, hocks and carpi, usual in young calves. The average meat yield was 8.91 kg from a 16.5 kg carcass, or 54% of the carcass weight (pers. comm. J. Mahoney, DMNZ). The musculature was usually well-developed, but rather paler in colour than that of older cattle, or even lambs.

The carcass fat was minimal, except in the pelvic inlet and perirenal tissues. It was usually a yellowy tan colour, rather than pearly white, and was similar in texture to that

found in adult animals. The fat colour often made the detection of small amounts of faecal contamination, also yellow in colour, very difficult.

# The umbilicus

The umbilicus in the normal animal varied in appearance from very small, hard, and fibrous to large and oedematous, with varying amounts of localised bruising and cellulitis. The size of the umbilicus varied with the age and degree of cellulitis present, as shown in Figure 4.1, but usually ranged from 0.5 to 2.5 cm in diameter. The umbilical vein remnant arising from the umbilicus intra-abdominally was usually transected during the dressing operation, leaving about two centimetres attached to the peritoneum. The dry, shrivelled tag of the umbilical vein remnant visible on the exterior of the umbilicus in the live animal is usually removed with the pelt.

#### The Umbilical Vessels

The umbilical vein was a white, cord-like structure with an external diameter of about four millimetres, with five to eight centimetres of its length attached to the liver at the umbilical fissure. The remainder, one to three centimetres in length, was left at the umbilicus after the midline abdominal incision (Figure 4.1). The umbilical arteries were usually removed from the carcass with the bladder, uterus and terminal colon at Feilding. When present, they lay to either side of the bladder, their tips extending a little beyond the bladder apex. The urachal remnant was a transparent, broad sheet of tissue connecting the umbilicus and the apex of the bladder. The majority of it remained intact along the ventral aspect of the abdomen. The lumen via which foetal urine was excreted to the placenta from the foetal bladder was still present, although not patent between the bladder and the umbilicus. Figure 4.2 illustrates the position and appearance of normal umbilical arteries on each side of the bladder, and their relationship to surrounding tissues.

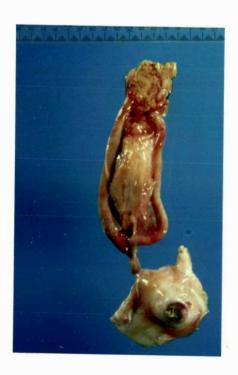
An incidental finding in normal carcasses was the presence of substantial haemorrhage apparently originating from the umbilical arteries. McFarlane (1965) mentions peritoneal haemorrhages as a result of trauma to the liver in lambs, but haematomas as seen in the calves could not be found previously described.

Figure 4.1: Normal umbilici, illustrating the size range possible in the tissue.

Normal umbilical veins are attached to each example.



Figure 4.2: An example of normal umbilical arteries and their relationship with the bladder.



# The Abdominal Contents

The abomasum is the most prominent of the four stomachs at this age. It was noted in four calves that substantial ingestion of bedding material had occurred, filling the abomasum with sawdust or wood shavings.

All other intestinal components were miniature forms of the adult organ. An occasional incidental finding was hyperaemia of the small intestine, without gaseous distension or haemorrhage into the lumen or any evidence of impaired digestion of the contents.

The liver varied in colour from yellowish tan to deep mahogany. Similarly the kidneys ranged in colour from pale tan to rich brown. One calf presented with blood speckling of the kidneys.

# The Thoracic Contents

The lungs were small replicas of the adult organ. Many of them showed small (1-3mm), recent haemorrhages over the majority of the surface, probably as a result of electrical stunning.

As with lambs, the thymus is large enough in the calf to be recovered for edible purposes. It is situated at the thoracic inlet, closely associated with the trachea.

The hearts were again small replicas of the adult organ.

#### The head

Only two features of the head tissues varied significantly from those of the adult animal: the teeth and the shape of the forehead.

In many calves all the temporary premolar teeth had erupted by the time of slaughter, indicating that they were around two weeks of age. The number of temporary incisors erupted ranged from two to six, while the temporary canines, which also appear when the calf is about two weeks old (Brown *et al* 1960), had sometimes not erupted. The incisors were either still overlapping to varying degrees, or the jaw had enlarged sufficiently to allow them to lie in a mature fan pattern.

All calves had a high, domed forehead typical of young mammals.

# Microbiological findings

The umbilical vein from one calf only yielded a pure growth of mixed lactose fermenting and non-lactose fermenting *E. coli*. This vein was normal both histologically and macroscopically.

# Histological findings

#### Umbilical vein

In most cases, the normal umbilical vein comprised a thick layer of connective tissue surrounding the lumen, which was usually bordered by a distinct endothelial lining. There was sometimes a blood clot present in the lumen, and occasionally the lumen had been obliterated by the resolution process. The connective tissue was interspersed by large bundles of smooth muscle fibres aligned tangentially across the vessel. There was a thin serosal layer continuous with the peritoneal lining surrounding the entire tissue (Figure 4.3).

#### Kidney

Calf kidney tissue had no features distinguishing it from adult kidney tissue (Figure 4.4).

Two calves considered normal and passed as fit for human consumption had microscopic lesions of focal interstitial nephritis in the renal cortex. There were small localised areas of interstitial fibrosis with infiltration by mononuclear cells. As a result of these lesions, these two calves were classified as having had a bacteraemia at some time in the past (Table 4.1).

#### Liver

The hepatic tissue had three features not normally found in adult cattle: a general appearance of decreased regularity and orderliness of the hepatic sinusoids, hepatocytic vacuolation with fat droplets (Figure 4.5), and many hepatocytes showed mitotic figures. In all other respects, the tissue was similar to adult liver.

# Summary

Table 4.1 summarises the major findings in the calves considered by the meat inspectors to be fit for human consumption.

TABLE 4.1: THE NUMBER OF CARCASSES WITH GROSS, MICRO-BIOLOGICAL AND HISTOLOGICAL ABNORMALITIES FROM 31 CALVES PASSED AS NORMAL BY MEAT INSPECTORS.

	Examination				
Summary	Microbiological	Histological	Gross		
findings					
Incidental	0	6	19		
findings*					
Localised	1	0	1		
infection**					
Bacteraemia	0	2	0		
Normal	30	23	11		
* Histology:	hepatocytic vacuolation a	•			
Gross:		al hyperaemia, renal blood sp	peckling, agonal		
	naemorrnages on lungs,	intra-pelvic haematomas.			
**Bacteriology:	bacteria cultured from	m the umbilical vein only.			
Gross:	inflammatory necros	is of the umbilicus.			

From these figures, an estimate of the rate of non-detection of bacteraemia in calves submitted for slaughter is 2/31 or 6.4% Calculation of the 95% confidence intervals for this figure (Schwabe 1979) shows that up to 15% of grossly normal calves may have lesions indicative of a past bacteraemia which has subsequently localised in the kidneys.

Figure 4.3: Longitudinal section of a normal umbilical vein in a bobby calf. H&E, x10.

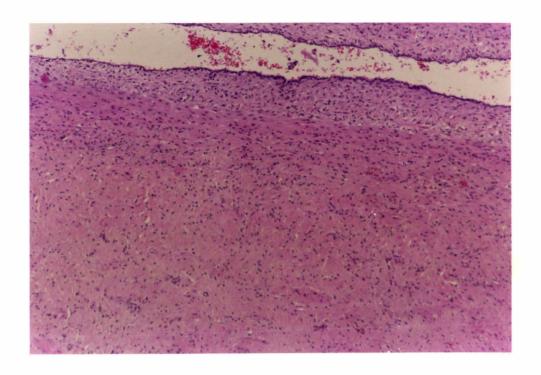


Figure 4.4: Normal bobby calf kidney. H&E, x100.

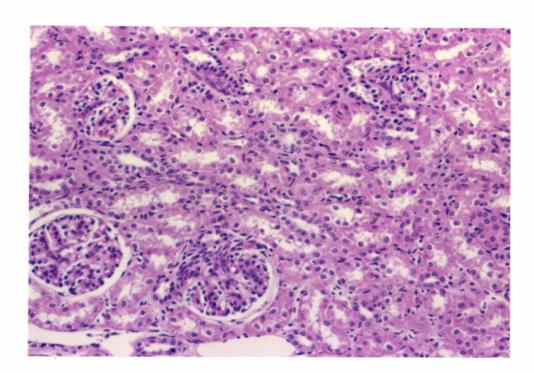


Figure 4.5: Bobby calf liver with vacuolated hepatocytes. H&E, x200.

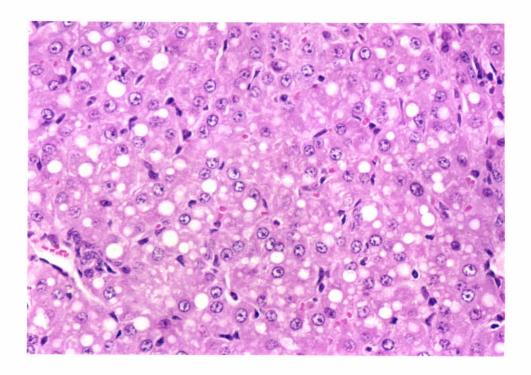
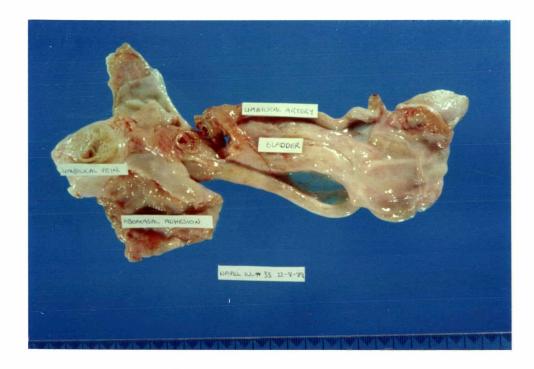


Figure 4.6: Infection of the apical remnants of the umbilical arteries.



# (2) Condemned Carcasses and Viscera

Of the 12,252 calves slaughtered during the study at Feilding, 54 were condemned for "navel ill". This represents an apparent prevalence of  $0.44 \pm 0.01\%$  A variety of lesions, which are described below, were observed in these calves.

Three carcasses condemned for "navel ill" had no gross evidence of infection in the umbilicus or the umbilical vessels, although oedema was present in two of them. The localised hepatic lesion which was present in the third of those cases, however, would probably have arisen from an infection gained via the umbilicus. Three "false positive" judgements in 54 indicated a positive predictive value for the inspection criteria of 0.952.

TABLE 4.2: COMPARISON OF GROSS FINDINGS OF DETAILED EXAMINATION WITH MEAT INSPECTION DISPOSITIONS ON CARCASSES WITH AND WITHOUT "NAVEL ILL".

# Carcass Categorisation on Gross, Detailed Examination

Meat inspection disposition	"Navel ill"	Normal	Total
"Navel ill"	51	3	54
Normal	1	30	31
Total	52	33	85

# **Gross lesions**

# Umbilicus and Umbilical Vessels

Carcasses condemned for "navel ill" typically had inflammatory, necrotic or purulent lesions of the umbilicus (Figure 4.2, in association with normal umbilical arteries), usually with concurrent involvement of one or more of the umbilical vessels. In a small

number of cases there was also gross evidence of bacteraemia. The lesions were subclassified into three categories:

(1) Lesions in the carcass or viscera indicative of past bacteraemia (six carcasses) or current septicaemia (one carcass) in association with umbilical and/or vessel infection.

The umbilicus was involved in all cases in this category. Of the six cases with gross evidence of past bacteraemia, all three umbilical vessels were involved in three cases, the umbilical vein alone in one case, the umbilical vein and the urachus in one case and no vessels in one case. Detailed descriptions of the umbilical and umbilical vessel lesions are under categories 2 and 3. In the case with gross evidence of a current septicaemia, the umbilical vein and arteries were involved along with the umbilicus.

Gross lesions in each carcass were as follows:

#### Carcass:

- 1. Hepatic abscesses.
- 2. Hepatic abscesses, focal interstitial nephritis (FIN), generalised carcass hyperaemia.
- 3. Generalised peritonitis, arthritis in one joint.
- 4. Polyarthritis.
- 5. Polyarthritis.
- 6. Polyarthritis, hypopyon.
- Emaciation, generalised carcass and visceral hyperaemia, premature rigor.
- (2) Lesions involving the umbilical vessels. There were 30 cases of omphalitis which involved one or more of these vessels.

Infected umbilical veins were enlarged, and a hyperaemic ring surrounding the contents of the lumen was apparent when the veins were examined in transverse section. The contents of the venous lumina varied from a dark red to black resolving blood clot through to dry, grey or yellowish caseous material, to purulent material. Although the infection in the venous lumen has been reported to extend only part way along the vessel, or be contained in a localised section of the vessel (Jubb & Kennedy, 1985), the lesions seen in this study

extended all the way to the liver. In these cases, the material present in the lumen of the vein terminated in a distinct line at the junction of the umbilical vein with the portal sinus in all cases except one, where the necrotic material in the core of the umbilical vein extended to the hepatic vasculature in the immediate vicinity of the portal sinus.

Similarly, infected umbilical arteries were usually enlarged, reddened, and oedematous, especially towards their tips. They usually extended from their origin at the internal iliac arteries significantly beyond the apex of the bladder, some with their tips only 2-3 cm from the umbilicus (Figure 4.6). Very rarely was there frank pus or caseous material in the lumen, but a red-black blood clot was commonly found. A ring of hyperaemia was usually seen in the arterial granulation tissue when they were sectioned. Most of the arteries were grossly affected for part of the length of the vessel, many of them also associated with urachal infections.

Ascending infections of the urachus were usually manifest by varying amounts of greyish or black, foetid necrotic material in the lumina (Figure 4.8). On occasions there was purulent material present. As indicated above, the umbilical artery was often involved in urachal infections (Figure 4.7).

Urachal lesions were commonly associated with localised peritonitis and fibrinous adhesions between the urachus and adjacent abdominal organs, particularly the abomasum and abomasal omentum (Figure 4.7).

Omphalourachitis was nearly as common as omphalophlebitis in the carcasses examined during this study. The umbilicus itself was not infected in five cases of "navel ill" which involved the umbilical vessels.

(3) Umbilical lesions alone. Fourteen cases were condemned for "navel ill" with lesions involving the umbilicus alone.

These cases had umbilical lesions ranging from enlargement (up to 4 cm) due to the excessive development of hard, white granulation tissue (two cases), through oedema and/or bruising (five cases) to necrosis (four cases) or suppuration (one case). In two cases there was both necrotic and purulent material present in the umbilicus. Necrotic material present in the centre of the lesions was usually black, and occasionally grey. Purulent material was usually thick and creamy or greenish in colour.

Figure 4.7: A large localised area of necrosis involving the urachus and the umbilical arteries. The ileum is adherent to the lesion.

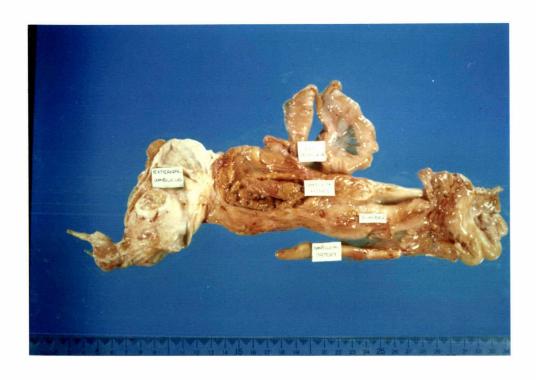


Figure 4.8: Extensive necrosis of the urachus in case N<sup>0</sup> 11.



Of the 54 cases condemned at Fielding, three were condemned with **no** lesions in the umbilicus or its associated vessels.

One of the three was considered to have an abnormal umbilicus by the meat inspector, but the author considered the degree of umbilical oedema to be within normal parameters. There were no other abnormalities in the carcass. One carcass had an abomasal adhesion to the parietal peritoneum, independent of the umbilicus or umbilical vessels, while the third had two encapsulated caseous foci in the liver parenchyma without any umbilical abnormality. In each of the three cases, there were also no abnormalities of the umbilical vessels.

# Liver

The livers from 11 condemned carcasses exhibited gross abnormalities. They were as follows:

- (1) Encapsulated, necrotic foci in the liver parenchyma, ranging in size from 0.5 cm to 2.0 cm in diameter, and numbering three to five (three cases).
- (2) Pale tan colouration (three cases).
- (3) Hyperaemia (three cases).
- (4) Enlargement (two cases).

#### Kidneys

The kidneys from five condemned carcasses exhibited gross abnormalities. They were as follows:

- (1) Small, white focal lesions on the surface of the cortex of the kidneys (two cases). The lesions were not prominent and involved only one lobule of one kidney in one of these, whereas the lesions were clearly visible and distributed over the entire surface of both kidneys in the other case.
- (2) Pale tan colouration of the cortex (three cases).

# Microbiological findings

Microbiological isolates from infected carcasses are shown in Table 4.3.

TABLE 4.3: NUMBER AND IDENTIFICATION OF BACTERIAL ISOLATES FROM SAMPLES FROM CONDEMNED CARCASSES. (In some cases, more than one isolate was recovered from one sample site.)

# Sample site

Isolate	Umbilical Vein	Liver	Muscle
Streptococcus spp.	12	5	1
(α and/or non-haemolytic)			
Escherichia coli	7	5	1
Actinomyces pyogenes	1	(2)	-
Gram +ve pleomorphs	2	-	-
Pasteurella haemolytica	2	-	-
Proteus mirabilis	1	8	
Gram -ve coccobacillus	1	-	
Pseudomonas putrefasciens	1	(4)	-

In the single case where the infection with *Streptococcus spp* extended to the musculature of the carcass, identification of the isolate was attempted. The isolate was not typable by the Lancefield method using a rapid slide latex agglutination test kit (Strepslide, Cambridge Biomedical Ltd.). A comprehensive series of biochemical tests was subsequently used and the isolates identified as *Streptococcus faecalis* and a type most resembling *Streptococcus viridans*.

Two of the more severely infected urachae and one umbilical artery were also cultured, and the following species were isolated:

Streptococcus spp. (\alpha and/or non-haemolytic)

Escherichia coli

Actinomyces pyogenes

Pasteurella haemolytica

Erysipelothrix rhusiopathiae

# Histological findings

# Umbilical vein

Thirty-one of the 54 carcasses condemned for "navel ill" had histologically normal umbilical veins.

The histological changes found in condemned carcasses with gross abnormalities of the umbilical vein were as follows:

- (a) Generalised, moderate leucocytic infiltration of granulation tissue in the wall of one "enlarged" umbilical vein. The diameter of the vein was 6 mm.
- (b) All other veins showed varying degrees of leucocytic invasion of both the mural granulation tissue and the intra-luminal clot (Figure 4.9), but the most striking feature was an intense fibrinous and leucocytic reaction at the junction of the lumen and the granulation tissue. Colonies of bacteria were often visible, bordered by a layer of necrotic leucocytes surrounded by a dense layer of viable leucocytes. The leucocytic cells were dominated by neutrophils (Figures 4.10 and 4.11). The lumen of the vein usually contained cellular components of blood often mixed with fibrinous exudate. In some cases, the peritoneum surrounding the vein was oedematous and contained fibrinous exudate.

Figure 4.9: Longitudinal section of an umbilical vein showing severe inflammation, leucocytic infiltration and fibrinous effusion of the mural tissues. H&E, x10.

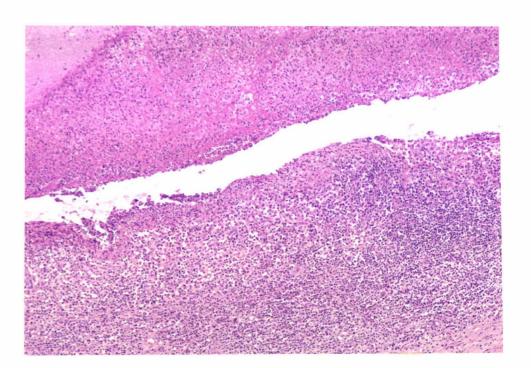


Figure 4.10: Longitudinal section of an umbilical vein, with severe leucocytic infiltration, and fibrinous effusion. H&E, x10.

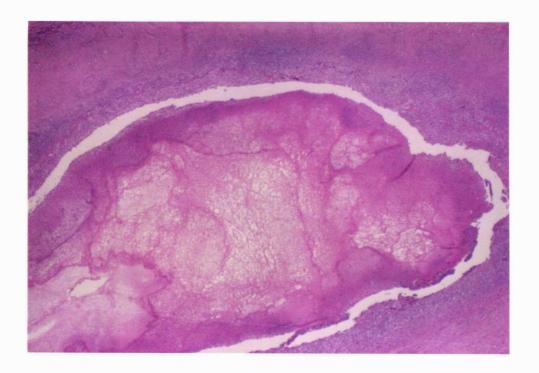
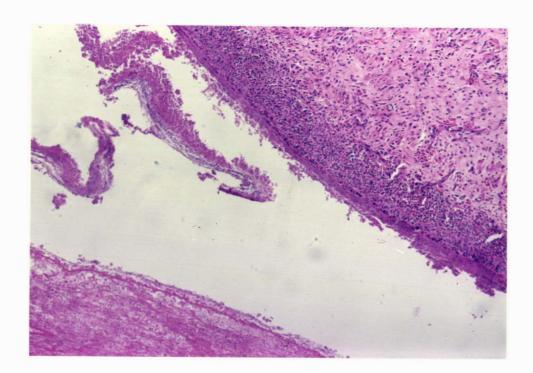


Figure 4.11: High power of the same tissue showing granulation tissue developing beneath the zone of infiltrating leucocytes. H&E x 40.



# Liver

At Feilding, 42 of the 54 condemned carcasses had grossly normal livers. The histological findings are shown in Table 4.4.

TABLE 4.4: HISTOLOGICAL LESIONS IN 42 GROSSLY NORMAL LIVERS FROM CALVES CONDEMNED FOR "NAVEL ILL".

Histology	N <sup>O</sup> cases	Illustration
Fatty infiltration of hepatocytes	2	Figure 4.5
Mononuclear periportal cuffing	13	Figure 4.12
Non-suppurative (N-S) inflammatory foci	3	Figure 4.13
Periportal cuffing & N-S inflammatory foci	2	
Periportal cuffing & single cell necrosis	2	
Normal	24	

In the livers with gross abnormalities (p. 69), the following histopathological lesions were present:

- (1) The liver parenchyma was normal except in the immediate vicinity of the lesions, where there was an intense band of leucocytes, dominated by neutrophils. In the centre, there was complete hepatocyte necrosis and amorphous debris. The lesions were well encapsulated by fibrous tissue.
- (2) Two livers had generalised fatty infiltration with vacuolation of hepatocytes, and one also showed mild periportal cuffing with lymphocytes. The remaining liver was normal.
- (3) No histological abnormalities were detected.
- (4) One had moderate periportal cuffing and fatty infiltration of the hepatocytes. The other was normal except in an area adjacent to the periumbilical region, where the necrotic core from the umbilical vein extended into the hepatic vasculature.

Figure 4.12: Bobby calf liver, showing moderate periportal cuffing with mononuclear cells. H&E, x100.

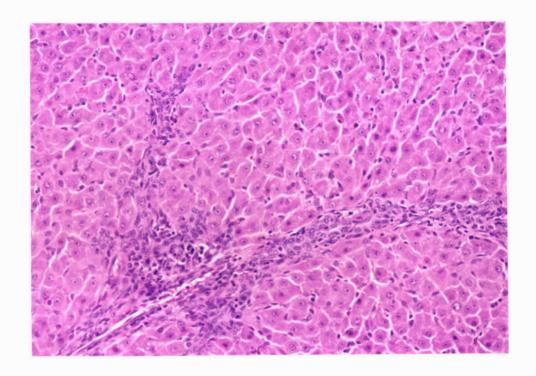


Figure 4.13: Bobby calf liver with an inflammatory focus dominated by monocytes in the parenchyma. H&E, x200.

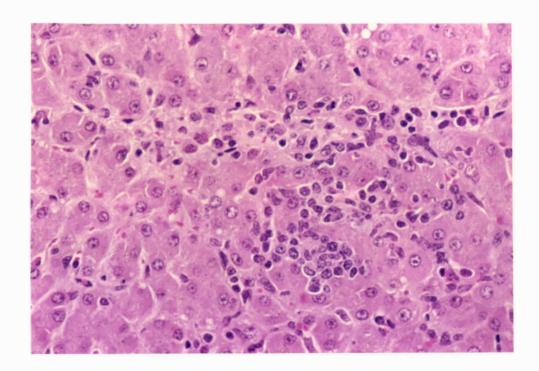


Figure 4.14: Bobby calf kidney with chronic focal interstitial nephritis in the cortex, and severe tubular destruction. H&E, x100

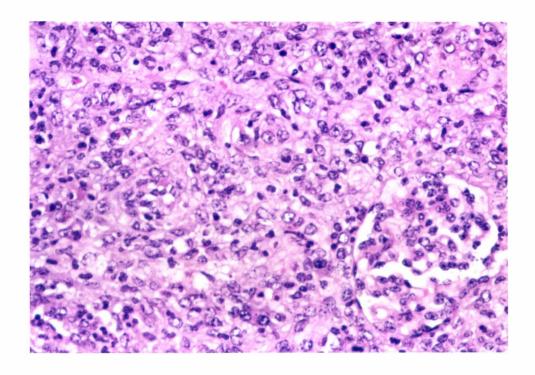


Figure 4.15: Bobby calf kidney showing an acute suppurative inflammatory reaction in the cortex. H&E, x100.

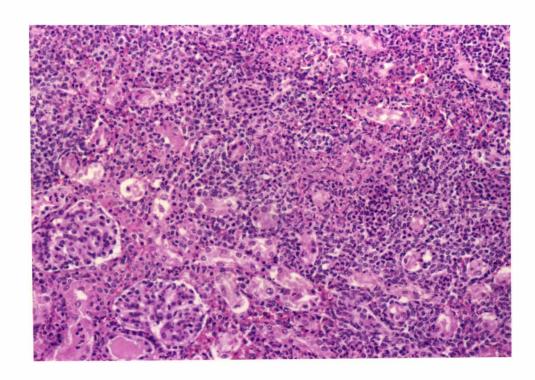
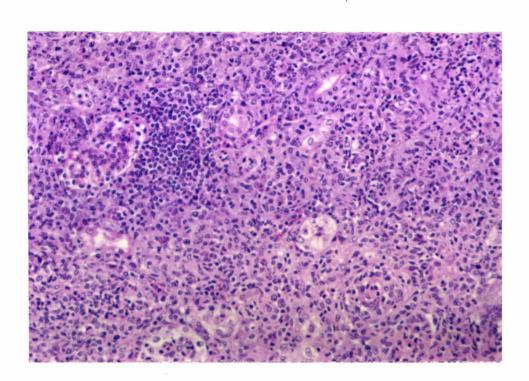


Figure 4.16: Focal interstitial nephritis involving both acute suppurative foci and early fibrosis of the cortical tissue. H&E, x100.



# Kidney

Forty-five sets of kidneys from the 54 animals sampled were grossly normal. The histological findings are shown in Table 4.5:

TABLE 4.5: HISTOLOGICAL FINDINGS IN 45 GROSSLY NORMAL KIDNEYS FROM CALVES CONDEMNED FOR "NAVEL ILL".

Histology	Number of calves	Illustration
Focal interstitial nephritis (FIN)	5	Figure 4.14
Suppurative inflammatory foci	1	Figure 4.15
FIN & suppurative inflammatory foci	1	Figure 4.16
Tubular dilatation	1	
Normal	37	

In the kidneys with gross abnormalities (pp. 69), the following histopathological lesions were found:

- (1) One of these kidneys had scattered inflammatory foci in the renal cortex. These foci were dominated by neutrophils, which obliterated the normal renal structures in the immediate area. The other showed similar inflammatory foci coupled with subacute FIN. The interstitial tissues were infiltrated by fibrous tissue.
- (2) One of the three kidneys showed histological lesions of FIN and tubular degeneration. There were no histological changes which explained the colour change in the other two cases.

The kidneys from four cases were not recovered by the meat inspectors, and were therefore not examined.

# The Relative Risk of Bacteraemia in Carcasses from Calves with "Navel III"

"Navel ill" was divided into three categories as described previously (pp.66-67):

Category (1): Carcasses which had lesions in either or both of the umbilicus and its associated vessels, plus gross lesions in the carcass indicative of a bacteraemia.

Category (2): Carcasses which had lesions in the umbilicus and one or more of its associated vessels.

Category (3): Carcasses which had lesions of the umbilicus only.

The initial results presented in Table 4.3 can therefore be expanded into each of these categories as shown in Table 4.6.

TABLE 4.6: NUMBER OF CARCASSES IN EACH CATEGORY OF "NAVEL ILL"
WHICH WERE TRULY BACTERAEMIC (D+ve), FALSE TEST
POSITIVES (D-ve), AND THE POSITIVE PREDICTIVE VALUE
(P(T/D+)) OF THE TEST.

Category T(+ve)	Total T+ve	D+ve	D-ve	P(T/D+)
(1)	7	4(a)*	3(b)	0.57
(2)	30	11(a)	19(b)	0.36
(1)&(2)	37	15(a)	22(b)	0.40
(3)	14	2(a)	12(b)	0.14
TOTAL	51	17(e)	34(f)	0.33
CONTROLS	31	2(c)	29(d)	

Subscripted letters are used in construction of formulae.

Carcasses in this study have been compared in the cohort format against systematically selected normal carcasses. The relative risk can be used to estimate the strength of association between the risk factor, in this case inflammation of the umbilicus and/or its associated vessels, and the disease process, in this case bacteraemia (Schwabe 1979, Martin *et al* 1987).

The relative risk is calculated as y = a/a + cb/b+d

Two tests were used to assess the significance of the relative risk: the calculation of 95% confidence limits in the manner described by Schwabe *et al* (1979), and calculation of a  $\chi^2$  statistic for association using the method described by Schlesselman (1982) (Table 4.7).

TABLE 4.7: RELATIVE RISK, 95% CONFIDENCE INTERVALS AND  $~\chi^2$  STATISTICS CALCULATED FOR EACH CATEGORY OF "NAVEL ILL" IN TABLE 4.6.

Category	Relative Risk	95%CI	χ2	<i>p</i> -value
(1)	8.86	[1.35,57.97]	11.03	<i>p</i> <0.005
(2)	5.68	[1.17,27.38]	8.29	<i>p</i> <0.005
(1)&(2)	6.28	[1.5, 26.04]	10.4	<i>p</i> <0.005
(3)	2.4	[0.28,17.28]	0.73	NS
(1),(2)&(3)	5.16	[1.12,23.8]	8.0	<i>p</i> <0.01

Both tests are in agreement in that all categories other than (3) have relative risk rates which differ significantly from unity, since the confidence intervals of those categories do not include one. The calculated  $\chi^2$  statistics also exceed the critical value of 3.84 at the 95% confidence level in all cases except that of category (3). This would indicate

that there is an association between all categories of "navel ill" and the presence of bacteraemia in the carcass except for that of category (3) (omphalitis only).

# Changes In the Rate of Detection of Bacteraemia between Categories of "Navel III"

The detection rates may be calculated from Table 4.6 as:

DR=a/e

and the wastage rate calculated as:

WR=b/f

for each category of "navel ill".

The 95% confidence limits can be calculated for each rate as follows:

 $p(DR) \pm z\sqrt{pq}$ 

n

where p =proportion detected (wasted)

q = (1-p)

n =total number detected (wasted)

z =1.96 at the 95% confidence level

and  $\sqrt{pq}$  = the standard error of the estimate for a binomial

n distribution (Martin et al, 1987).

The results are shown in Table 4.8.

TABLE 4.8: DETECTION RATES (DR), WASTAGE RATES (WR), AND THEIR 95% CONFIDENCE LIMITS FOR THE SUB-CATEGORIES OF "NAVEL ILL" DETECTED BY MEAT INSPECTORS.

Category	DR±SE [95%CI]	WR±SE [95%CI]
(1)	0.23±0.20	0.09±0.10
	[0.03,0.43]	[0.00,0.19]
(2)	0.65±0.23	0.56±0.16
	[0.42,0.88]	[0.40,0.72]
(1)&(2)	0.88±0.15	0.62±0.16
	[0.73,1.00]	[0.46,0.78]
(3)	0.12±0.15	0.35±0.16
	[0.00,0.27]	[0.19, 0.51]
(1),(2)&(3)	1.00	1.00

The accuracy of the current inspection system, which includes all three categories, is assumed to be 100% since umbilical lesions are readily identifiable at *post mortem* inspection.

A combination of categories (1) and (2) captures 88% of the possible "at risk" carcasses currently being condemned, while only incurring 62% of the total wastage currently experienced. It appears that little additional benefit in terms of detection rate relative to the wastage rate is gained by the inclusion of category (3).

# The Accuracy of the Meat Inspection Judgements

The gross lesion of "navel ill" as determined by the meat inspectors (T+) was compared with the total combination of gross, microbiological and histological findings which comprised the "gold standard" for the true presence of bacteraemia/septicaemia (D+/-) in the carcass. Similarly the carcasses passed by the inspectors (T-) were compared with the combined findings of the "gold standard" (Table 4.9).

TABLE 4.9: COMPARISON OF THE MEAT INSPECTION JUDGEMENT (T+/-) WITH THE "TRUE" PRESENCE OF BACTERAEMIA (D+/-) BASED ON THE COMBINED MICROBIOLOGICAL, HISTOLOGICAL AND GROSS FINDINGS.

	D+	D-	Total	Apparent Prevalence
_				
T+	17	34	51	0.62
T-	2	29	31	0.38
Total	19	63	82	
Apparent Prevalence	0.23	0.77		

the three cases which did not involve the umbilicus or its associated vessels (Table 4.2) are excluded from this table.

The sensitivity and specificity of the meat inspection test cannot be calculated from the figures generated from the study because the groups have been selected on the basis of the presence or absence of the predisposing factor, "navel ill", rather than on the presence or absence of the disease, bacteraemia. Agreement between the two tests can be assessed by calculating the Kappa value (Martin *et al.*, 1987).

Observed proportion agreement:

17+29/82

=0.56

Chance agreement positive:

0.62x0.23

=0.14

Chance agreement negative:

0.38x0.77

=0.29

Chance proportion agreement:

0.14 + 0.29

=0.43

Observed minus chance agreement:

0.56-0.43

=0.13

Maximum possible agreement

beyond chance level:

1-0.43

=0.57

Kappa:

0.13/0.57

=0.22.

# Estimation of the Prevalence of Haematomas Associated with Umbilical Arteries In Bobby Calves Submitted for Slaughter

The number of carcasses with and without substantial haemorrhage into the peritoneal ligaments supporting the bladder, urachus and umbilical arteries over a limited number of carcasses specifically examined for this lesion is summarised below:

TABLE 4.10: NUMBERS OF CARCASSES WITH SUBSTANTIAL HAEMORRHAGE IN THE SEROSA SUPPORTING THE BLADDER AND UMBILICAL ARTERIES.

Date	Haemorrhage	No haemorrhage (percent)	Total number (percent)
17/8/89	28	32	60
	(46.66)	(53.33)	
21/8/89	28	32	60
	(46.66)	(53.33)	
22/8/89	5	35	40
	(12.5)	(87.5)	
23/8/89	8	32	40
	(20.0)	(80.0)	
24/8/89	17	63	80
	(21.25)	(78.75)	
27/8/89	24	56	80
	(30.0)	(70.0)	
TOTAL	110	250	360
	(30.5)	(69.5)	(100.0)

The haematomas varied in size: the smallest were limited to within the broad ligaments of the bladder and the middle ligament containing the urachus, while the most extensive also extended under the intra-pelvic peritoneum along the ventral abdomen and encased the kidneys within a sub-peritoneal blood clot. Figure 4.17 illustrates the variability in size and age of haematomas.

The lesion was not seen by meat inspectors if the haemorrhage was fresh and involved no more than those structures removed with the terminal colon. If the lesion resulted in significant amounts of blood in the pelvis or perirenal tissues, the carcass would be detained for trimming under "wounds and bruises" (see Part II). Older lesions which had become infected or ischaemic, with the development of adhesions to and inflammation of adjacent organs (Figure 4.18), were usually condemned for "navel ill".

Figure 4.17: Variation in severity of haematomas associated with the umbilical arteries. These are presumed to arise from inadequate contraction of the umbilical arteries at birth.

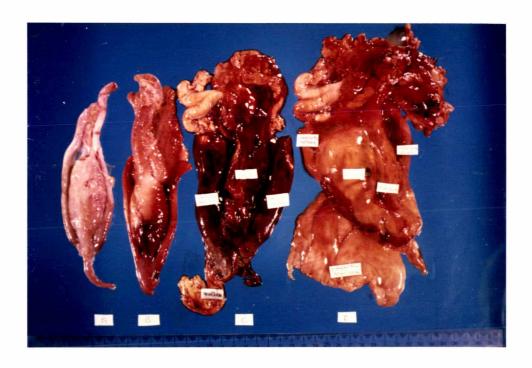


Figure 4.18: Haemorrhage associated with the umbilical arteries and urachus.



# **RESULTS: PART II**

# A STUDY OF THE LESIONS PRESENT IN CALVES CONDEMNED BY MEAT INSPECTORS

# The Rate of Condemnation for Disease, Contamination and Wounds and Bruising

The condemnation rate at the six slaughterhouses visited from 4th September 1989, to 11th October 1989 was calculated and compared with the condemnation rate over the entire season in each slaughterhouse (Table 5.1).

There appeared to be some pressure from the study on the condemnation rate within each slaughterhouse, with some condemnation rates below and others above the annual rate recorded for the season. This assessment must be interpreted with caution, since many other animal, environmental and management factors may have also been exerting an effect and confounding the effect of the study. There was marked variation between slaughterhouses during the study and on an annual basis.

TABLE 5.1: CALVES SLAUGHTERED, AND CONDEMNED FOR DISEASE BY MEAT INSPECTORS, AT SIX SLAUGHTERHOUSES IN NEW ZEALAND DURING THE 1989 SEASON.

Slaughter house & period visited	Number slaughtered	Number condemned	Condemnation Rate (percent)	Annual Condemnation Rate * (percent)
A 4-15/9/89	6,056	50	0.82	1.49
B 18&21/9/89	3,582	27	0.75	1.10
C 19&20/9/89	1,809	12	0.66	1.84
D 25-27/9/89	6,722	86	1.28	1.10
E 2-5/10/89	4,076	69	1.69	1.50
F 6-11/10/89	4,520	132	2.92	2.59
Period total	26,765	376	1.35	
National total				1.60

<sup>\*</sup> Figures obtained from statistics generated by the New Zealand Ministry of Agriculture and Fisheries.

# **DISEASES DETECTED BY MEAT INSPECTORS DURING 1989**

# (1) "Navel III"

The condemnation rate for "navel ill" was calculated for each slaughterhouse (Table 5.2).

TABLE 5.2: CALVES SLAUGHTERED, AND CONDEMNED BY MEAT INSPECTORS FOR "NAVEL ILL", AT SIX SLAUGHTERHOUSES IN NEW ZEALAND.

Slaughterhouse & period visited	Number slaughtered	Number condemned	Prevalence (percent)
A 8/9 to 15/9/89	6,056	28	0.46
B 18 and 21/9/89	3,582	12	0.33
C 19 and 20/9/89	1,809	5	0.28
D 25 to 27/9/89	6,722	25	0.37
E 2 to 5/10/8	4,076	18	0.44
F 6 to 11/10/89	4,520	55	1.22
TOTALS	39,017	197	0.50

Prior to this study, the condemnation rate for "navel ill" was estimated by meat inspectors at Feilding to be between 0.25 and 0.5%. This estimate was confirmed by the study, with the average condemnation rate 0.50±0.07%. However, the results show that Slaughterhouse F had a high prevalence of "navel ill" relative to the other slaughterhouses.

Ticket usage varied between slaughterhouses. Inspectors at slaughterhouse F using the SAL ticket virtually exclusively, whereas all others visited used a combination of PYO, OC, and SAL, depending on whether there was a purulent umbilicus (PYO), an inflammatory or necrotic reaction without pus (OC), or other lesions in addition to those centred on the umbilicus which indicated systemic involvement (SAL).

# **Gross Lesions**

The cases of "navel ill" seen in slaughterhouses other than Feilding were grossly similar to those examined in detail in Part I of this study (Ref. Results: Part I). The following lesions were observed:

- (1) Enlargement, cellulitis, necrosis, or suppuration of the umbilicus, usually with inflammatory or infectious lesions in one or more of the associated vessels.
- (2) Involvement of the urachus and/or the umbilical artery with or without umbilical involvement. There was usually localised inflammation, fibrinous exudation and adhesions of adjacent organs, particularly the abomasum and abomasal omentum.
- (3) A few carcasses exhibited excessive haemorrhage associated with the umbilical arteries and extending to the peritoneum of the pelvic cavity, ligaments of the bladder or even the renal capsule.
- (4) In some cases gross lesions indicative of systemic involvement similar to those seen at Feilding were present. These lesions included polyarthritis, focal interstitial nephritis both with and without hyperaemic haloes, hypopyon, foci of necrosis in the liver or myocardium, pericarditis, or embolic foci of necrosis in the lungs.
- (5) One carcass had an umbilical hernia. The serosa of the loop of small intestine present in the hernial pouch was reddened and adherent to the hernial sac.

As at Feilding, the umbilical vein was not necessarily the dominant tissue involved in extension of the infection from the umbilicus; the umbilical artery and the urachus were involved in a significant number of the carcasses condemned (Table 5.3).

**TABLE 5.3:** 

THE NUMBERS OF CARCASSES CONDEMNED FOR "NAVEL ILL" WITH PHLEBITIS, ARTERITIS AND/OR URACHITIS IN SIX SLAUGHTERHOUSES.

Total

Abnormality of associated vessels

Condemned

Number (percent)

	Vein	Artery	Urachus	No Abnormality
143	80	17	66	10
	(56)	(12)	(46)	(7)

In 14% of cases, more than one vessel was involved in the lesion.

# (2) Focal Interstitial Nephritis

Focal interstitial nephritis (FIN), which in the meat industry is colloquially termed "white spotted kidneys", was seen in all six slaughterhouses. No microbiological examinations were carried out on the kidneys examined.

#### Gross lesions

Twelve of the 38 calves condemned for FIN had minor lesions in other tissues which were found on detailed examination of the carcass, with the principal lesion being infection of the umbilicus.

Three calves condemned for "septicaemic-like lesions" had grossly visible FIN as part of generalised systemic changes.

The lesions seen on the renal surface ranged in size from less than 1mm to 3 mm in size, and were found to vary in density from a few lesions on one lobule of one kidney to densely scattered foci over both kidneys in their entirety. In some cases, the lesions coalesced. The lesions were either contiguous with the renal surface, or slightly raised. The presence of a hyperaemic halo around the central white area was a lesion not described in the literature, but was found regularly in cases condemned for "white spotted kidneys" (Figure 5.1). Meat inspectors assumed that such lesions are "acute" and active rather than "chronic", and a severe case is illustrated in Figure 5.2.

Table 5.4 summarises the gross lesions which resulted in condemnation of carcasses for FIN.

TABLE 5.4: GROSS LESIONS IN KIDNEYS FROM 30 CARCASSES CONDEMNED FOR "WHITE SPOTTED KIDNEYS" (FOCAL INTERSTITIAL NEPHRITIS).

"Chronic"	"Acute"	Both	Chronic or acute with other carcass lesions.		
12	12	6	12		

It is evident that despite the MAF requirement that carcasses are to be passed for human consumption (Anon 1979) if chronic "white spotted kidneys" are the only lesion present in the carcass, ten of 18 carcasses condemned (55%) with no other lesions elsewhere were in that category.

# Histopathological lesions.

Of a total of 30 calves condemned on the basis of the presence of lesions of FIN, 27 were sampled for histological examination. Lesions were largely confined to the renal cortex, and centred on the interstitial tissues surrounding the tubules and glomeruli. In long-standing cases, there was interstitial fibrosis, with infiltration predominantly by mononuclear cells, although polymorphs were often also present. There were varying degrees of tubular disorganisation, or even complete destruction of tubules and glomeruli. In more acute examples, there was general invasion of the tissues with

Figure 5.1: A representative range of the lesions of "white spotted kidneys".

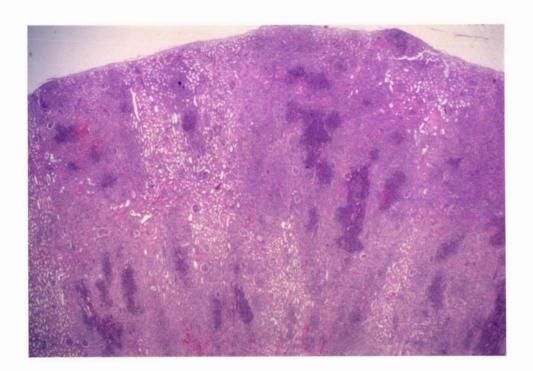
"Acute", hyperaemic lesions in the kidney on the right, and two examples of "chronic" lesions to the left.



Figure 5.2: Severe infection of the kidney, with fibrinous exudate in the perirenal tissues and renal swelling.



Figure 5.3: Kidney with acute pyelonephritis. Note the radial distribution of the inflammatory lesions. H&E, x40.



mixed inflammatory cells, and the formation of suppurative foci in the cortical tissue. In some cases there was also marked accumulation of red blood cells in the interstitium. Examples of these lesions were illustrated in Figures 4.14 to 4.16. One case examined had developed a pyelonephritis, with leucocytes present in the renal pelvis and medulla as well as the cortex, and hyperplasia of the pelvic epithelium. In two other cases, the radial distribution of the lesions and the presence of inflammatory cells within the tubules in the renal cortex and/or medulla was suggestive of pyelonephritis (Figure 5.3). Some cases exhibited only subacute lesions, some only acute inflammatory lesions, and others a mixture of the two lesions, with suppurative foci surrounded by fibrosis. Little difference was found in the age and types of histological lesions between kidneys that were grossly considered to have lesions of "acute" FIN and those considered to have lesions of "chronic" FIN (Table 5.5).

TABLE 5.5: HISTOPATHOLOGICAL FINDINGS IN 27 CALVES CONDEMNED BY MEAT INSPECTORS AT SIX SLAUGHTERHOUSES ON THE BASIS OF LESIONS IN THE KIDNEYS.

	"White spots"	"White spots" with Hyperaemia	Other Lesions
Subacute FIN	0	3	2
Suppurative Inflammatory foci	2	1	1
Both lesions	7	9	0
Normal	0	0	1
Tubular distension	0	0	1
TOTALS	9	13	5

The gross and histopathological findings in kidneys which had lesions termed "other" were as follows:

- (a) Cortical petechiations (one case). The kidney tissue was histopathologically normal, and the "petechiae" were presumed to have resulted from electrical stunning.
- (b) Cortical and medullary colour reversal (one case). On histopathological examination, there was severe subacute interstitial nephritis, with very little normal renal tissue remaining.
- (c) Cortical infarcts, with depressed centres, measuring 0.25 to 0.5 cm in diameter scattered over the surface of both kidneys (two cases). Extensive subacute

focal interstitial nephritis was present histopathologically in one case, while in the other, there was a probable congenital tubular dysplasia with disorganisation of the tubules, but no evidence of inflammation.

(d) Three necrotic foci one millimetre in diameter in the renal cortex of one lobule in one kidney, with similar small necrotic foci present in the liver, particularly on the left lobe, with hepatic lymph node enlargement (one case). Histopathologically, the renal lesions consisted of inflammatory foci of mixed inflammatory cells surrounding a necrotic centre. Some lymphocytes were present around the medullary tubules. The necrotic foci present in the liver were histopathologically similar to those in the kidney.

As well as the 30 cases condemned primarily for renal lesions, there were fifteen cases of condemned carcasses where FIN of the kidneys was a part of the total lesions present: 11 had omphalitis and associated lesions, two had other gross lesions indicative of a bacteraemia but no umbilical involvement. Two had localised lesions as well as FIN: one had compensatory hypertrophy for the congenital loss of one kidney, the other a wound to the musculature. The 11 cases with both FIN of the kidneys and "navel ill" represent only 8.0% of the total number of calves condemned for "navel ill".

# (3) Pleurisy/Pneumonia/Pericarditis

The extent of the involvement of lung tissues in all calves condemned for "pleurisy" during the study, along with the types of gross lesion present in the lungs and other tissues, are presented in Table 5.6.

TABLE 5.6: GROSS FINDINGS AND SEVERITY OF LUNG AND PLEURAL LESIONS IN 75 CALVES WHICH WERE CONDEMNED BY MEAT INSPECTORS FOR PLEURISY AT SIX SLAUGHTERHOUSES.

		Acu	Acute lesions			Chronic lesions					
			Lung area			Lung area				Total Number	
Lesion											
Category	0	1	2	3		0	1	2	3		
Trauma	6	2	0	0		0	1	0	0	9	
"Enzootic" pneumonia	0	19	2	0		0	29	3	0	53	
Large discrete abscesses	0	2	0	0		0	3	1	0	6	
Haematogenous emboli	0	1	1	0		0	0	1	0	3	
Pericarditis	0	2	0	0		0	1	1	0	4	
Total	6	26	3	0		0	34	6	0	75	

<sup>\* 0=</sup> no lung involvement

Table 5.7 shows the number and category of pneumonic lesions which had concurrent gross lesions in the carcass or viscera suggestive of bacteraemia. Such lesions

<sup>1=</sup> less than 1/3 lung involvement

<sup>2= 1/3</sup> to 2/3 lung involvement

<sup>3=</sup> more than 2/3 lung involvement

particularly include polyarthritis, but also such lesions as focal interstitial nephritis, endocarditis and swollen liver and kidneys.

TABLE 5.7: THE LESION CATEGORIES TICKETED FOR PLEURISY WHICH WERE ASSOCIATED WITH GROSS SIGNS OF A CONCURRENT BACTERAEMIA.

Lesion category	N <sup>O</sup> of cases bacteraemic	Prevalence (Percent)
Haematogenous emboli (acute)	2	100.0
Haematogenous emboli (chronic)	1	100.0
Pericarditis (acute)	1	50.0
Pericarditis (chronic)	1	50.0
Enzootic pneumonia (acute)	2	10.5
Enzootic pneumonia (chronic)	1	3.1

# **Gross lesions**

# **Traumatic Pleurisy**

These included old, callused and fibrosed fractures of the ribs, recent and unhealed rib fractures, bruised ribs, and bruised lungs. Two of the carcasses with broken ribs had developed a localised osteomyelitis within the fracture callus. There was necrotic bone tissue within the fracture calluses, which were about 2 cm in diameter.

# Enzootic Pneumonia

A pneumonic lesion was classified as enzootic if the lesions were anteroventral in distribution and involved consolidation of the lung tissue with or without necrotic foci in

the affected areas. Both acute and chronic lesions were observed. In acute cases, consolidation was red to red-black in colour, usually with a fibrinous exudate over the surface of the consolidated areas (Figure 5.4). As the pneumonia progressed, the fibrin often formed fibrous adhesions between the lung surface and the pleurae. On occasions there was severe distension and prominence of the inter-lobular septa. In many of the lungs there were greyish-white, dry, caseous nodules ranging in size from a few millimetres to about 1 cm in diameter. These were usually distributed within the consolidated tissue. In more chronic cases, the consolidated areas were greyish in colour. Occasionally the necrotic foci were present in isolation, usually with small, fibrous pleural adhesions, but no evidence of consolidation.

# Pneumonias of Haematogenous Origin

There were three cases where the distribution of the lesions did not have an anteroventral pattern. Rather, the lesions were evenly distributed throughout the lung tissue. These cases were presumed to be due to the arrest of haematogenous bacterial emboli. Two were acute in nature, while one was a more chronic lesion, with caseous nodules and fibrous adhesions present.

#### **Discrete Abscesses**

Some pneumonias exhibited very large (often seven to eight centimetres in diameter), purulent or necrotising, and malodorous abscesses in the lung (Figure 5.5). A limited number were approaching a gangrenous nature. These lesions were invariably associated with a severe pleuritic reaction, usually rendering the lung tissue unable to be removed from the thoracic cavity without massive contamination of the lower half of the carcass with purulent or necrotic material. In three of these cases there were also caseous foci present in the anteroventral parts of the lung.

#### **Pericarditis**

Two of the cases of pericarditis were associated with fibrinous adhesions of the pericardium to the pleurae, while the other two involved fibrous adhesions. In two cases, one acute and one chronic, there was not only pericardial, but also lung involvement in the pleurisy.

Figure 5.4: Acute enzootic pneumonia with areas of hyperaemia and consolidation with an anteroventral distribution.



Figure 5.5: An old, well-encapsulated pulmonary abscess with fibrous adhesions to the surrounding tissues.



Figure 5.6: Bobby calf lung with acute, suppurative bronchopneumonia. H&E, x10.

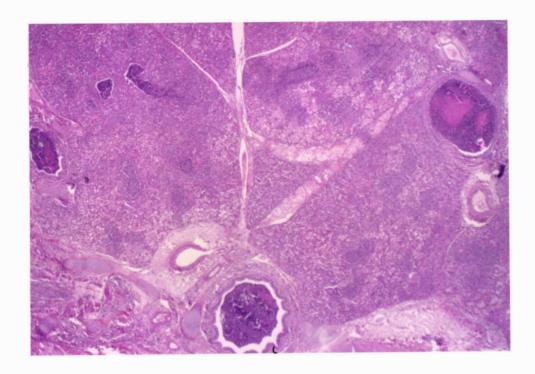


Figure 5.7: High power of the above showing intense neutrophilic accumulation in a bronchiole. H&E x 100.

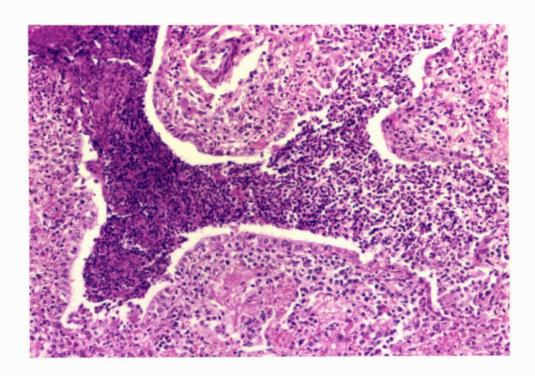


Figure 5.8: Bobby calf lung with fibrinous bronchopneumonia containing an area of coagulation necrosis. There are bacterial colonies in the centre. H&E, x40.

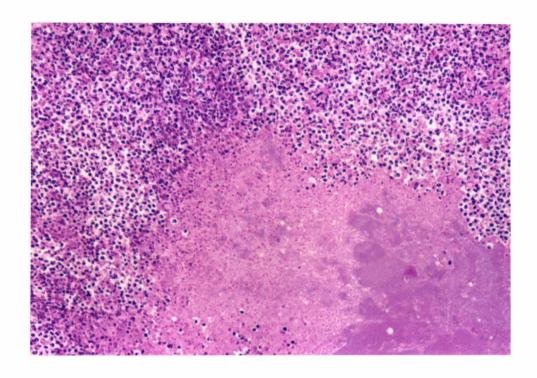


Figure 5.9: Bobby calf lung showing severe distension of the interlobular septa with fibrin and necrotic material. H&E, x10.

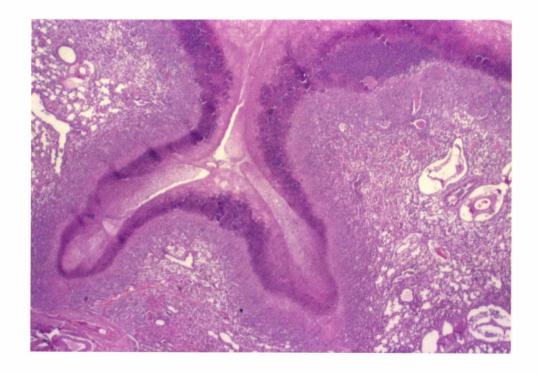


Figure 5.10: Bobby calf lung containing streaming macrophages ("oat cells") typical of infection with *Pasteurella haemolytica*. H&E, x200.

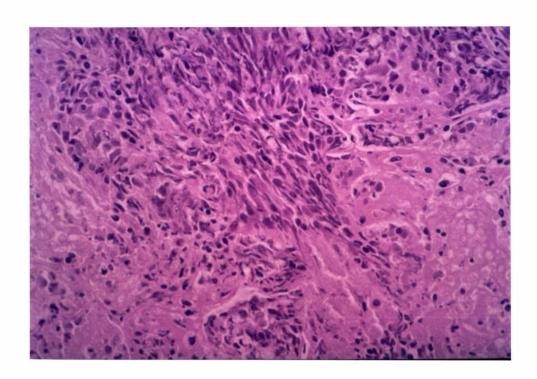
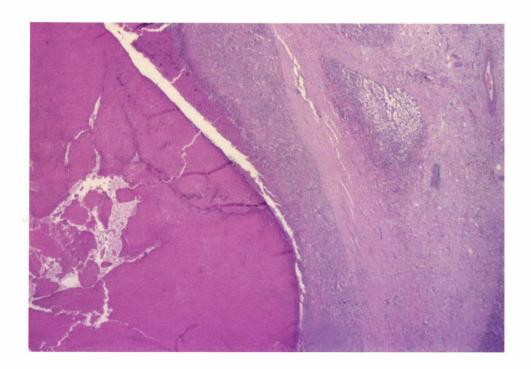


Figure 5.11: The edge of an old abscess in a bobby calf lung. There is encapsulation of the necrotic material and extensive destruction of the normal lung tissue. H&E, x10.



# Histological lesions

Three major types of lesion were present in the tissues sampled.

- (1) Acute suppurative bronchopneumonia consisting of extensive areas of inflammatory cells filling and destroying the architecture of the bronchi and bronchioles (Figures 5.6 and 5.7). Not all bronchi in an affected area were necessarily affected. In many cases, areas of recent, focal coagulation necrosis were also present. The necrotic centres were surrounded by inflammatory cellular and sometimes fibrinous exudate. In some cases, large gram positive cocci were clearly visible in the centres of the necrotic foci. Most lesions, depending on their stage of development, had at least some degree of fibrosis occurring around the lesions.
- Acute, necrotising, fibrinous bronchopneumonia (Figure 5.8). The necrotic areas were again recent, commonly lobular, and dominated the lesions. Although there was a cellular exudate, it was not the dominant feature of the lesions. Rather, extensive fibrinous exudation was present, the exudate often dilating the interlobular septa (Figure 5.9). Streaming macrophages typical of a *Pasteurella*-induced bronchopneumonia (Figure 5.10) were often present at the margins of the necrotic areas. These lesions presented a picture very similar to that described in the literature as typical of "shipping fever" in cattle (Rhemtulla & Thomson 1981). Large numbers of bacteria could be found amongst the inflammatory cells surrounding the necrotic areas of some lesions. Fibrosis was not prominent in these lesions.
- (3) In two cases the edge of an old, amorphous abscess had been sampled. There were no features remaining to allow the classification of these lesions as centred on the bronchi or the interstitial tissues. There was cellular exudate, but no fibrin present, and both lesions were walled off by a thick capsule of fibrous tissue (Figure 5.11).

# Microbiological Isolates

The lungs of three carcasses condemned for pneumonia were sampled for microbiological purposes. *Staphylococcus aureus* was recovered from two of these, and an isolate most resembling *Pasteurella haemolytica* was recovered from the other.

# (4) Arthritis

#### Gross lesions

Table 5.8 summarises the gross features of the arthritic lesions in 31 calves condemned for arthritis at the six slaughterhouses visited. Approximately half of the calves had localised arthritis, which is currently not differentiated from polyarthritis in terms of judgements passed on the carcass by the inspectors. In eight (57%) of the localised arthritic joints the character of the joint fluid (bright yellow, clear) and the periarticular structures (bruising and oedema) suggested that the lesions were the result of trauma. The remainder of the localised lesions probably result from an initial polyarthritis which has resolved in all joints except for the one detected by the inspector.

Only one (7%) of the carcasses with localised arthritis had other gross lesions indicative of a bacteraemia, while six (33%) of the carcasses with polyarthritis had such lesions, which included hypopyon, hepatic and myocardial necrotic foci, and FIN.

Only two, or 11% of the polyarthritic cases appeared to be due to trauma, presenting with similar lesions as described above. The remainder had joint fluid which varied from thin and cloudy to thick and greenish-yellow. In some, there was the development of many fibrinous plaques in the joint space, ranging in size from a few millimetres to one centmetre on the longest axis. Unopened joints ranged from slightly "puffy" to severely distended, the purulent material escaping under high pressure on opening the joint. There were often a large number of joints involved, including the carpi, hip, shoulder and elbow joints as well as the larger stifle and hock joints. These lesions are likely to be due to the localisation of an often pyogenic bacterial infection following a bacteraemia.

TABLE 5.8: GROSS FEATURES OF THE JOINTS OF 32 CARCASSES CONDEMNED FOR ARTHRITIS BY MEAT INSPECTORS IN SIX OF THE SLAUGHTERHOUSES PROCESSING CALVES.

	Localised arthritis		Polyarthritis		Other tissue involvement	
	Infectious	Traumatic	Infectious	Traumatic		
	6	8	16	2	1	
Synovial fluid:						
normal		6	-	1	1	
turbid	6	1	5	-	-	
purulent	-	-	5	-	-	
fibrinous	-	1	14	1	-	
sanguineous	-	-	3	-	-	
Chronic joint distortion	-	1	1	1	-	
Bursal thickening	1	-	2	-	-	
Concurrent bacteraemia	1	1.1	6	-	-	

# (5) Wounds and Bruising

The national rate of condemnation for "extensive" bruises is 0.14%, but during the period of the study, when 30,017 calves were inspected, the rate of condemnation was 0.07% This suggests that there exists considerable variability in the interpretation of the term "extensive".

Nineteen calves were condemned for bruising which was considered "extensive". The judgement resulting in the condemnation of carcasses with "extensive" bruising appears to stem from a similar requirement in the USA Federal Regulations (Anon. 1989). Only one of those condemned had lesions which extended more than 2-3 mm into the carcass musculature. All other lesions were as follows:

Superficial bruises (5), involving largely the superficial fascia. Very little of the underlying muscle was affected.

Bruising which only affected large boney areas (such as the stifles and elbows) and the lower limbs (7).

Extensive intra-peritoneal haemorrhage from poorly contracted umbilical arteries (4) There was no involvement of the underlying muscle tissues.

Bruising over the thoracic cage which resulted in a small area of fibrous pleurisy (1).

Granulation tissue on the point of an elbow (1).

Bruising that damaged the carcass so badly as to result in it being uneconomic to recover the meat was not seen by the author.

# (6) Miscellaneous Conditions.

There were a number of conditions which were, for ticketing purposes, considered "miscellaneous" and categorised under one or other of the non-specific tickets of OC, PYO, and SAL. They are, with the exception of omphalophlebitis, listed in Table 5.9. The number and percentage of these carcasses which also had gross lesions indicative of a bacteraemia are tabulated.

Individually the diseases listed do not constitute a problem, but as a group, significant numbers are condemned. The most important of the lesions in numerical terms are peritonitis, jaundice, "fever", hepatic abscesses and enteric hyperaemia.

TABLE 5.9: DISEASES DETECTED BY MEAT INSPECTORS WHICH RESULTED IN CONDEMNATION OF CARCASSES USING THE TICKETS OCS, PYO AND SAL.

Disease	N <sup>O</sup> affected carcasses	N <sup>O</sup> bacteraemic (percent)
Peritonitis (Figure 5.13)	9	5 (55)
Jaundice	13	-
"Fever"	7	6 (86)
Hepatic abscesses (Figure 5.12)	6	2 (33)
Enteric hyperaemia	5	1 (20)
Myocardial necrotic foci	2	2 (100)
Intra-pelvic haematoma	2	-
Other:		
Colonic stasis	1	
Pancreatitis	1	
Abnormal kidney colour	1	
Pericarditis	1	
Fistulating wound,		
lumbar region	1	-
Neoplasia	1	
Hepatic node calcification	1	N
Injection site lesion	1	1 (100)
Umbilical hernia	1	
Congenital abnormality	1	•
Carcass odour	1	1 (100)
White muscle disease,		
myocardium	1	-

Figure 5.12: Bobby calf liver with one encapsulated abscess in the left lobe.



Figure 5.13: Generalised fibrinous peritonitis in a bobby calf.



# **DISCUSSION**

# PART I: COMPARISON OF THE GROSS LESIONS, HISTOLOGICAL LESIONS, AND MICROBIAL FLORA OF CARCASSES FROM BOBBY CALF COHORTS WITH AND WITHOUT OMPHALOPHLEBITIS ("NAVEL ILL")

# Study Design

This study was not performed using the classical cohort design, since the cohorts were not followed forward in time (prospectively) to determine the rate of development of disease in each group. However, the fundamental requisite of a cohort study is not so much that the members of the two groups are studied prospectively, but rather that they are chosen on the basis of the presence or absence of a suspected predisposing factor for the disease of interest. In this study, the groups were chosen on the basis of the presence or absence of "navel ill". While a disease entity in itself, "navel ill" is also considered a predisposing factor for the development of septicaemia or bacteraemia, which in this study were the disease states of interest. If a case-control study had been undertaken, the two groups would have been selected on the basis of the presence or absence of bacteraemia/septicaemia, and subsequently assessed for the presence or absence of the suspected causal factor, "navel ill".

Because of the study design, the relative risk of septicaemia/bacteraemia in calves with gross lesions of "navel ill" was able to be calculated. Although the calculation of the odds ratio is also applicable in a cohort study, this is generally used to measure the strength of the association between the factor and the disease in a case-control study, where the relative risk is meaningless (Martin *et al* 1987, Thrusfield 1986).

# The Lesions of "Navel III" In Bobby Calves at Slaughter

The concern of international regulatory authorities such as the USDA (Anon. 1989) with regard to umbilical infections in young calves centres on the development of phlebitis of the umbilical vein and the subsequent potential for systemic disease in the animal at the time of slaughter. It seems incongruous that this concern does not extend to infections affecting umbilical vessels other than the vein. The term "omphalophlebitis" may be

being used as a collective term to imply infections involving any or all of the umbilical vessels, or the rationale is that urachitis or arteritis are much less likely to result in systemic infections of the carcass. While this rationale could be accepted in the case of urachitis, it is entirely possible that most bacteria from the umbilical arteries could enter the general circulation after passing through the capillary bed in the hind legs if there are no lesions affecting the integrity of that vascular bed.

The umbilical arteries are contiguous with the distal aorta, and are less commonly infected due to the strong retraction of this vessel into the abdomen. Infection of the umbilical arteries was cited by Bouckaert and de Moor (1965) as more likely to occur if the arteries do not retract fully following parturition. It was noted during the present study that infected arteries extended further down the midline towards the umbilicus than non-infected arteries, but it was unclear whether this enlargement and lengthening of the vessel was a result of the infection, or a contributing factor towards the development of the disease.

The clinical sequelae of infection of the urachus in calves which range in age from four days to 16 months is cited in the literature as relating more to urinary disease and subsequent ill-health rather than bacteraemia or septicaemia (Trent & Smith (a)&(b) 1984, Hylton & Trent 1987). It was difficult to ascertain whether infections had reached the bladder of calves examined during this study, since at Feilding, the bladder was removed prior to the inspection point. The lesion regularly seen involving the arteries and urachus (i.e.adhesions to the abomasum or abomasal omentum) was only found referred to in the literature in one paper by Bouckaert and de Moor (1965) in connection with the surgical implications of such adhesions accompanying arterial infections.

All of the literature which was reviewed discussed infections of the umbilical vessels in terms of abscessation. The infections seen during the present study were most frequently necrotic in nature, with purulent infections of the vessels being less common. This difference may be due to the younger age of the subjects in this study and thus the shorter duration of the infection. Further, the majority of umbilical infections in this study appeared to be dominated by organisms not generally regarded as pyogenic, such as  $E.\ coli$  and  $\alpha$  or non-haemolytic streptococci, rather than those species more commonly considered to be pyogenic, like  $A.\ pyogenes$ .

No data were generated from this study which would allow an assessment of the number of carcasses with "navel ill" which were missed by the inspectors. It is possible that variation in the judgements does occur, especially in the case of carcasses with infections of the umbilicus, but no involvement of the umbilical vessels.

# The Baseline Test for Septicaemia

The definitive way of determining the presence of a bacteraemia or septicaemia in an animal is to isolate micro-organisms from the blood-stream. All literature reviewed which was concerned with the microbiological determination of septicaemia recommended that serial blood cultures be taken over 24 hours to increase the probability of detecting bacteria in the blood-stream, since the presence of organisms in the blood, even in septicaemia, is regarded as intermittent in nature (Cruickshank 1973, Dow and Jones 1989). For reasons outlined in Appendix I, and the fact that the only practical time to sample the calves was when they were dead and only one sample could therefore obtained, serial sampling could not be successfully employed in this study.

To minimise the possible effect of this perceived deficiency in microbiological testing on the sensitivity of the test used to determine the presence or absence of disease, it was decided to rely on a combination of histopathological and microbiological examinations in the liver, kidney, and musculature coupled with gross lesions to determine the presence of septicaemia/bacteraemia in the animal at the time of slaughter. This combination of changes was then used as the "gold standard" against which the validity of the inspection procedure could be assessed. It was found that only one carcass had a combination of lesions which was indicative of an acute septicaemia. The combined findings in the other carcasses did, however, support the presence of bacteraemia in many of the carcasses examined, since minor, localised inflammatory or degenerative changes in the hepatic or renal tissues were the usual histopathological finding. There was a much higher prevalence of histological lesions indicative of systemic infection than isolation of bacteria from the key tissues. This was consistent with either the presence of bacteraemias which were being successfully dealt with by the immune system, or bacteraemias involving fewer bacteria than were detectable by the microbiological method used.

The lowest detectable number of bacteria in each sample can be estimated from the dilutions performed in preparing the samples:

One colony on an agar plate inoculated with the standard loopful of 0.001 ml of broth corresponds to 1,000 colony forming units (cfu) per ml. On average, 20 grams of tissue

was homogenised in 100 ml of saline. The lowest number of cfu's detectable was therefore

1,000 cfu/ml x 100 ml/20 g =5,000 cfu's per gram of tissue examined.

The sensitivity and specificity of the "gold standard" test were unknown. Since, also, the carcasses were selected on the basis of the presence or absence of the factor ("navel ill"), not the disease (bacteraemia/septicaemia), the apparent prevalence of "navel ill" was artificially elevated, and the sensitivity and specificity of the inspection procedure could not be calculated. Instead, the rate of agreement between the meat inspection test and the 'gold standard' was calculated, and found to be very low (0.22). In the usual circumstances where an assessment of the degree of test agreement is used, i.e. in the comparison of two serological tests, there would be uncertainty as to which is the more valid test. In this case, however, it would be biologically sensible to assume that the "gold standard" was the more valid, and that routine inspection procedures were not evaluating what they were purported to. It is thus contended that the presence of "navel ill" in a calf carcass is not a good indicator of the presence of septicaemia/bacteraemia in the carcass.

# The Relationship between "Navel III", Bacteraemia, and Septicaemia

The umbilicus is considered by most authors as the source of most neonatal infections in calves, including septicaemia and bacteraemia (Blood & Radostits 1989, Jubb & Kennedy 1985, Roy 1990 (e)).

Neither omphalitis nor omphalophlebitis are included in the list of third and fourth schedule of diseases in this country (Anon. 1969 (a)). Despite this omission, omphalitis is cited in the MAF meat manuals (Anon. 1977) as a possible source of organisms capable of producing food poisoning in humans. As such, its presence warrants the condemnation of the entire carcass. This study attempted to more critically define "navel ill", and in doing so evaluate the necessity for these requirements.

There is a high level of awareness and concern in developed countries of the health risk involved in the consumption of meat derived from animals which were septicaemic or toxaemic at the time of slaughter. The effect on public health of low numbers of bacteria in the bloodstream (bacteraemia) of animals at slaughter is largely unknown, but bacteraemia is considered to be a risk by one author (Franco 1988), since he considers the organisms causing the bacteraemia are "...a potential source of

pathogens that could cause food-borne disease in people." Others (Gracey 1979, Nottingham 1982) consider bacteraemias in food animals to be of little public health importance. General literature on pathology considers bacteraemia a common occurrence of little importance to the animal concerned, as the bacteria are rapidly cleared by the lymphoid system (Walter & Israel 1975).

This is in direct contrast to septicaemia which, from a public health point of view, is considered among the most serious of the conditions which may affect an animal at slaughter. This is due to the large numbers of pathogenic bacteria present in a septicaemic carcass, and the fact that the signs can be difficult to interpret by meat inspectors using traditional inspection procedures (Gracey 1979, Franco 1988). The diagnosis of septicaemia is therefore based either on a combination of non-specific pathological lesions, or extrapolated from the presence of a likely predisposing condition, such as "navel ill".

If it is argued that septicaemia in the calf results in severe clinical signs as outlined earlier (ref. Diseases of the Bobby Calf), then septicaemia will occur very rarely in calves presented for slaughter, as the majority will be unable to be presented at the farm gate, or will be found to be unacceptable at ante-mortem inspection in the yards prior to slaughter. Bacteraemia, on the other hand, since there are no or few clinical signs of the infection, will occur more commonly in calves at slaughter. This study found one case in the 54 carcasses with umbilical infections which were examined which could be considered septicaemic. This calf had generalised, non-specific gross lesions which were readily recognisable on post-mortem inspection. The prevalence of bacteraemia was found to be significantly higher in calves with "navel ill" which involved the umbilical vessels than in calves with no umbilical abnormality. Except for the umbilical infection, there were no other gross lesions in these carcasses which would enable the identification and removal of these bacteraemic carcasses from the consumer. There was inconclusive evidence that calves with localised infection of the umbilicus were no more likely to be bacteraemic than "normal" calves. The actual hazard to the consumer from meat derived from calves which were bacteraemic at or just prior to slaughter remains largely unquantified, but the results of this work would indicate that the carcass meat presents little microbiological hazard. The liver and the kidneys, as organs where bacteria in the bloodstream are most likely to be arrested, appeared to be more of a risk than carcass meat. Consideration of the pathogenesis of bacteraemia would support this contention.

# The Risk of Bacteraemia in Caives with "Navel III"

The usual test of significance for a relative risk calculation is either the calculation of the appropriate confidence interval for the rate, or the calculation of the appropriate  $\chi^2$  statistic (Schwabe 1979). It was decided to calculate both figures, since they should both result in the same conclusion. Although the actual figure for the relative risk of category (3) was greater than unity, the 95% confidence limits included unity, and so the figure could not be considered significant. The  $\chi^2$  test was in agreement with that interpretation. Unfortunately, the confidence limits for each category overlapped, indicating that larger samples of the two cohorts would be needed for any real difference to be detected.

Of more interest to the meat hygienist is the difference in the rate of detection of bacteraemia for each of the three categories, and the comparison of the calculated detection rates with the non-detection rate found for grossly normal carcasses. The detection rates were calculated with no knowledge of the rate at which inspectors fail to detect gross cases of "navel ill" as defined in the MAF manual. Again, due to the small sample size, the 95% confidence limits for the detection and wastage rates are wide. The non-detection rate in normal carcasses was calculated as  $0.06\pm0.08$ . The detection rates in all categories except (3) exceed this value sufficiently to preclude considering the possibility of exclusion of these categories from the condemnation criteria. These findings are aligned with the results of the relative risk calculation (ref. Results Part I), where all categories of "navel ill" except (3) show a significantly higher risk of bacteraemia than in normal calf carcasses.

The current inspection system condemns carcasses falling into all three categories in the majority of export slaughterhouses in New Zealand. Since umbilical lesions are readily detectable at inspection it could be assumed that 100% of carcasses with such lesions would be detected, and any difference in detection rates between the current system and one attempting to reduce the wastage rate may be assessed using that assumption (Table 4.8).

Although not specifically investigated, observations made during the study indicated that the inspection system was consistent in the application of judgements to carcasses with umbilical lesions which either involved the vessels or which showed concurrent carcass lesions such as polyarthritis. However there was between-slaughterhouse and within-slaughterhouse (between inspectors) differences in the consistency of the judgement applied to carcasses with umbilical lesions alone, such that many cases of localised

umbilical infection were ignored. The rate of detection of bacteraemia by the current system therefore cannot be assumed to be 100% if all cases of localised "navel ill" are not detected on the chain.

#### **Hazard Identification**

The potential human pathogens which may be present in the carcass of calves with "navel ill" are basically the *Salmonella spp* (ref. Diseases of the Bobby Calf). No other bacterial species found, or likely to be found, in New Zealand present a serious foodborne risk to the consumer of bobby veal.

Animals represent a major reservoir of salmonellae, which may be introduced to carcass meat directly from the animal by faecal contamination, or via the hide, contaminated personnel, equipment, or contact points in the meat processing area (e.g. tables, rails, walls). The most likely risk to the consumer from bobby calf meat itself is the consumption of inadequately cooked meat derived from a carcass heavily contaminated with salmonellae (Bensink 1980, Genigeorgis 1981, Ireland 1987). Salmonellae are sensitive to heat, and do not survive an adequate cooking regime, but raw product contaminated by salmonellae is a major source of cross-contamination of cooked products in food preparation are as.

Salmonellae were not recovered from any samples taken during the study. The technique used for culture of salmonellae was not the accepted standard technique used in the meat industry (MIRINZ publication 757 1980), but because the samples were from recently slaughtered animals which had not been subjected to any chilling procedures, the viability of the aerobic mesophilic microflora in or on the carcass should have been optimal. As a result, the selective medium used should have been adequate to support the growth of salmonellae, if they were present.

Using standard microbiological methods for the meat industry (MIRINZ publication 757 1980), Hathaway sampled 100 calves with omphalophlebitis, and salmonellae were isolated from the umbilicus, liver and hepatic lymph node of one calf (Hathaway, unpub. data). This result would indicate that there could be a risk of bacteraemia or septicaemia due to salmonellae in calves with omphalophlebitis. Similar work has not been done with calves with localised omphalitis, but from the preliminary work done in this study, there was no evidence that carcasses from such calves represent such a risk.

The relative impact of calves with omphalophlebitis on the total risk of salmonellae in the product can be calculated using Hathaway's results (Hathaway unpub. data), and data generated by the Meat Industry Research Institute of New Zealand:

The worst likely rate of infection of calves with omphalophlebitis by salmonellae is 3% (1%±2%), based on Hathaway's work (Hathaway unpub. data). The number of animals condemned for "navel ill" was about 4367 (0.5% (estimated prevalence) x 873362 animals slaughtered). Three percent of those are potentially bacteraemic with salmonellae, i.e. 131 animals.

In contrast, 0.54% of bobby calves slaughtered are contaminated on the meat surface with salmonellae (pers. comm. MIRINZ). This percentage of the 873362 animals slaughtered in 1989 represents 4716 animals with surface contamination with salmonellae.

Although only estimates, the figures serve to illustrate the relatively small impact in numerical terms that calves with omphalophlebitis are likely to have on the overall risk from salmonellae on or in calf carcasses. This risk is even less significant in calves with localised umbilical infection only: 28% of calves condemned for "navel ill" were in this category, with the result that only 33 animals, at worst, would be affected by salmonellae.

The relative numbers of salmonellae involved in surface contamination and bacteraemia/septicaemia must also be considered. Unfortunately, such is the concern with salmonellae in the meat industry that present/absent information is usually cited rather than data on the estimated numbers present on carcasses. Further, the enrichment methods used are designed to enhance the detection of presumably small numbers of salmonellae, and are unable to provide a numerical estimate of the numbers present in the sample. Similarly in attempting to enumerate the bacteria present in a bacteraemic or septicaemic carcasses, vague terms such as "fewer" and "large" are the norm. Dow and Jones (1989) comment that in an intermittent bacteraemia arising from a chronic source such as a hepatic abscess, there will be fewer than ten bacteria per ml of blood. The article does not specify how many bacteria could be present in an acute, overwhelming "bacteraemia", but implies later in the article that it could be around 104 organisms per ml of blood. Despite the very few numbers of carcasses which could be affected to this degree in the slaughterhouse environment, that number represents an extremely high potential infective dose. It is highly improbable that a carcass would sustain surface contamination of that order by salmonellae. The infective dose required to produce clinical salmonellosis in humans has estimates varying from "low" to 10<sup>5</sup> (Silliker & Gabis 1975, Ireland 1987), but it is probable that the minimum infectious dose is affected by the nature of the food vehicle and the health status of the host (Silliker & Gabis 1975). Thus it can be seen that the animal septicaemic with an infection due to salmonellae represents a very real risk to human health. The bacteraemic carcass, however, may represent no more risk than that which has been surface contaminated during processing.

# The Cost of Condemned Carcasses to the Producer

It is possible that to retain market access for bobby veal and offals to the USA or EC (offals only), the current *post-mortem* judgements must be retained in New ?ealand, despite the lack of similar requirements in the importing countries. The cost of this to the producer can be estimated from a partial budget using figures obtained from Dairy Meats New Zealand Ltd (DMNZ).

a.	Revenue from a passed carcass:		\$98.25
b.	Costs over a passed carcass (approx):	\$47.25	
C.	Revenue from a condemned carcass:		\$29.00 (skin)
d.	Costs over a condemned carcass (approx):	\$36.25	

The economic outcome resulting from the condemnation of a calf carcass would equal the product of the following four items:

- 1. Additional returns from the implementation of the changes.
- 2. Returns no longer obtained if the changes are implemented.
- Additional costs incurred in implementing the changes.
- 4. Costs no longer incurred if the changes are implemented.

Estimated change in income = 
$$(1+4)-(2+3)$$
.  
=  $(0+(b-d))-((a-c)+0)$   
=  $$11.00-$69.25$   
=  $$-58.25$ 

i.e. every condemned calf costs \$58.25. Since killing, inspection and processing charges do not depend on whether calves are passed or condemned, the only additional cost to DMNZ of saved carcasses are those of freight and insurance of the cartoned product to market destinations. In addition, returns from condemned calves in

the production of meat meal or blood and bone are retained by the company slaughtering the calves and are not realised by DMNZ. At a prevalence of 0.5%, there are approximately 4,367 calves condemned nationally for "navel ill" at a cost of \$58.25 each. This amounts to \$254,377.75 per season, or 29 cents per calf presented for slaughter. If calves with omphalitis only were not condemned, the additional returns would be \$69,826 per annum, since the proportion of calves with localised omphalitis is approximately 0.28. This would reduce the cost to \$184,551.60, or 21 cents per calf presented for slaughter.

#### Conclusions

While it is accepted that carcasses from calves with septicaemia are a risk to the consumer, very few such calves are presented for slaughter. The results of the investigation conducted during 1989 indicate that there is a high probability that a carcass with omphalophlebitis with or without grossly visible changes to the carcass may be bacteraemic but not septicaemic. The purpose for condemning calves with "navel ill", i.e. the removal of carcasses with septicaemia from the food chain, is not being met, but if carcasses with bacteraemia are also considered a serious risk to the consumer, very few are missed by the current inspection system. However, "navel ill" as presently defined is a poor indicator for the presence of bacteraemia in carcasses.

The predictive value of the positive test declines sharply as the test includes more general criteria in the definition of "navel ill". This trend reflects the decrease in the prevalence of bacteraemia as the criteria expand and the subsequent declining ratio of truly diseased animals relative to truly healthy animals within those classified as "diseased". This characteristic results in the condemnation of an increasing proportion of carcasses which are not in fact bacteraemic, and lost revenue to the producers of bobby veal. From the results of this study it seems that there would be little increase in risk associated with the exclusion of carcasses with localised omphalitis from the inspection judgements, but this needs validating by additional studies involving larger numbers of affected calf carcasses.

In summary, despite the methodological limitations of this work, the results obtained, coupled with a knowledge of the epidemiology of food-borne infections in humans, would support the continued application of carcass condemnation in cases with omphalitis where infection extends to involve any of the umbilical vessels and/or the carcass until such time as the risk to human health from bacteraemia is adequately investigated. There is doubt that there is significant improvement in the prevention of

carcasses with bacteraemia, much less septicaemia, reaching the consumer by the condemnation of carcasses with omphalitis alone. The wastage rate, with its inherent loss to the producer, increases markedly with the condemnation of such cases.

# PART II: A STUDY OF THE LESIONS PRESENT IN CALVES CONDEMNED BY MEAT INSPECTORS

# (1) Focal interstitial nephritis

No difference was detected between the histological lesions observed in kidneys classified as "acute" and those classified as "chronic" by the inspectors on the basis of their gross appearance. This suggests that carcasses from calves with lesions of FIN in the kidneys, whether "acute" or "chronic", but without gross systemic lesions, could be saved. A microbiological examination of these kidneys and a composite of carcass tissues would help to more precisely define the significance of these lesions.

# (2) Pleurisy

Of the calves condemned for pleurisy, 70% had histological lesions consistent with enzootic pneumonia. In most cases less than 1/3 of the lung volume was affected. The infection was usually associated with bacterial invasion, as evidenced by the histopathological lesions present, and the isolation of bacteria (*P. haemolytica* and *S. aureus*) from a limited number of lesions. Neither of these bacteria are of public health importance: human cases of staphylococcal food poisoning result from the contamination of meat with staphylococi of human origin, not animal origin (Genigeorgis 1981). Carcasses from calves with lesions of this type represent little apparent risk to the consumer. Current international requirements demand that carcasses from any class of livestock be condemned if acute pneumonia is present in the lungs (Anon. 1982, anon. 1985(a), anon. 1989), and a major research effort would be required to alter that fact. It was noted, however, that there were a minority of pneumonias which had progressed to being chronic in nature. Judgements accounting for these more longstanding conditions would bring the bobby calf judgements in line with requirements for all other classes of livestock.

There were a number of cases of pneumonia which involved large, discrete abscessating lesions which could have been interpreted as having arisen as a result of aspiration pneumonia. Unfortunately there was no direct evidence that these lesions contained milk or gut contents. They were predominantly suppurative bronchopneumonias rather than necrotising and fibrinous, and not all bronchi and bronchioles were affected in the lesions. There was no evidence on gross examination that there

was bacteraemia or septicaemia in the carcasses of these animals, but the presence of toxaemia, particularly in the more extensive cases, was highly likely. In the absence of generalised carcass lesions, it was considered that chronic, well-encapsulated pneumonias of this type could be considered localised in nature and the affected part only condemned.

Van der Mei and van den Ingh (1987) reported that the majority of lesions seen in their study were exudative and/or cuffing in nature. Lesions of this type were not seen in the samples taken, but those examined in this study were a very biased sample of the total spectrum of lesions seen in the slaughterhouse. A more representative sample of the pneumonic lesions present in bobby calves at slaughter would help elucidate the pathogenesis of the disease.

Traumatic lesions of the thoracic cage, i.e. broken and bruised ribs can result, in time, in pleural adhesions, for which the carcass is condemned. In some cases, such calves were condemned with pleural bruising in the absence of any adhesions. Two cases had developed osteomyelitis of the fractured ribs, while no other lesions were present in the carcass. While there is the possibility that there may have been salmonellae present in the osteomyelitis, this lesion alone is not, in the author's opinion, sufficient to warrant the condemnation of the entire carcass. Calves with pleuritic adhesions, whether acute and fibrinous or chronic and fibrous, resulting from trauma to the thoracic cage did not show any other lesions indicative of systemic infection. The continued condemnation of carcasses for traumatic pleurisy represents what the author considers an unnecessary annual wastage of approximately 229 calves.

In carcasses condemned for pericarditis, there was evidence for both haematogenous spread and direct extension as infective routes discussed by Jubb & Kennedy (1985) and Smith & Jones (1966). Again, the issue of the degree of localisation and chronicity of the lesion should be, but is not, assessed in the judgement of these lesions in calves.

In the three cases of pneumonia with a lesion distribution indicating haematogenous spread there were other grossly detectable carcass lesions supportive of a diagnosis of recent bacteraemia.

#### (3) Arthritis

As with other disease entities in bobby calves, there is no recognition in the New Zealand judgements that there may be cases of localised arthritis. Localised arthritis

was generally due to either trauma of the periarticular region or an infectious process which had infected only one joint or resolved in all except the affected joint. Cases which showed clear evidence of a traumatic origin, such as periarticular bruising, could be trimmed and saved for human consumption rather than condemned as a public health risk.

# (4) Jaundice

In performing detailed examinations of calves condemned for jaundice, it appeared that many of these calves had yellow discolouration rather than true jaundice. There was yellowing of the carcass and visceral fat, but seldom was there also yellowing of the conjunctivae, tendons or arterial intima. No gross abnormalities of the liver could be found in any of the cases examined. It was hypothesised that many of these cases resulted from the breakdown of foetal haemoglobin rather than obstructive disease of hepatic origin. For aesthetic reasons this colouration is, however, unacceptable to the markets for which this product is destined. For this reason, condemnation of such carcasses will continue.

# (6) Enteritis

There were a number of carcasses in this study which were condemned for the presence of hyperaemia of the intestines. Equally, there were also similar viscera sets which were ignored by meat inspectors. There is therefore confusion as to what constitutes gross lesions of "enteritis" in calves. The perceived risk for the consumer is of contamination of the carcass meat with faeces containing salmonellae or strains of E. coli pathogenic to humans. Strains of E. coli of animal origin are of doubtful public health significance. Outbreaks of enteritis in humans are usually directly or indirectly associated with human faecal contamination of food products in the home kitchen, fastfood outlets, hospitals, restaurants and specialty foods outlets (Genigeorgis 1981). There has recently been some concern about a "new" strain of E. coli, O157:H7, which is associated with cases of haemorrhagic colitis and haemolytic uraemic syndrome in humans and has been recovered from ground beef and from dairy cattle. Work by Doyle and Schloeni (1987) has shown that this organism has also been recovered from pork, chicken, turkey, lamb and milk as well as beef. This indicates that the source of contamination of foodstuffs is not exclusively bovine in origin, but is the result of inappropriate handling of many different products during food preparation. In a study investigating the risk factors for infection by E. coli O157:H7 in southern Alberta, Bryant et al (1989) concluded that proper handling and cooking of animal products rather than avoidance of particular food products (particularly hamburger meat and barbecued foods) was the preventive measure of choice.

None of the intestines examined in this study had lesions consistent with those cited in the literature as indicative of enteritis due to salmonellae or other enteric pathogens. A few examples of hyperaemic intestines were sampled for histological examination, and were found to be normal. It therefore seems likely that most cases were due to a passive hyperaemia. Further work is necessary to clarify this condition and determine whether or not meat from calves with hyperaemia of the intestines is a public health risk.

# (6) Wounds and bruising

Bruising is the result of stressors which have been imposed on the animal and which may result in a product with unsatisfactory quality parameters due to an increase of the ultimate pH of the carcass meat. However, bobby veal (particularly that which has been mutilated by trimming) is processed in such a manner, and its ultimate use is such that quality parameters altered by elevation of the ultimate pH are irrelevant. In fact, the increase in, for instance, water holding capacity of the meat as a result of high ultimate pH is a bonus for meat destined to be used in specialty reconstituted meat products.

There are, however, no data available as to the ultimate pH values of either normal bobby veal, or veal derived from a bruised carcass. This lack of information requires addressing before legitimate changes to the definition of "extensive bruising" as a lesion requiring carcass condemnation can be initiated.

It was noted that carcasses with the more severe examples of haemorrhage from the umbilical arteries, but without adhesions between the lesion and other abdominal organs, were also condemned as "bruised". There was no evidence of bruising of the underlying muscle, and the condemnation of these cases was in the author's opinion an unneccessarily harsh judgement on the carcass.

#### Conclusions

Due to historical misconceptions about the pathogenesis of disease in the bobby calf, inspection procedures have evolved which are severe, and result in significant wastage to the producer. MAF has an obligation to ensure that the products released for consumption by the public do not constitute a health hazard, but the judgements currently used in New Zealand are only partially successful in meeting the needs of the

consumer, and are at the same time a source of product loss to the industry. Virtually every other international regulatory body concerned with public health recognises that very young calves can be presented for slaughter with localised pathological lesions in the carcass or viscera which do not necessitate the condemnation of the carcass in its entirety.

The improved understanding of the epidemiology of many disease processes means that re-evaluation of traditional judgements could be appropriate at this time. There has been a world-wide reduction in the prevalence of many zoonotic and production-limiting diseases. New methods of animal husbandry and food handling techniques which increase the risk of animal products constituting human health hazards have also developed. These new risks are not as readily detected as those hazards (such as tuberculosis) for which traditional meat inspection techniques were originally instituted.

The results of this study show that the prevalence of disease in the bobby calf is low. There is also evidence that there is sufficient time for infections to become chronic and localised in nature. Consideration of this needs to be taken, and calf carcasses inspected in a more discriminatory manner. There is a need to revise the current judgements given the improvements in our understanding of the epidemiology of the diseases affecting these calves, and their relative importance compared to the risks resulting from the subsequent handling procedures applied to the product.

#### APPENDIX I

#### DEVELOPMENT OF BACTERIOLOGICAL MATERIALS AND METHODS

#### I.1 Pilot trial A

#### Introduction

The aim of the sampling regime used in the study (Ref. Materials Methods) was to identify as accurately as possible the presence of septicaemia in calves from each group. A sampling technique for the microbiological aspects of the study needed to be developed such that there could be confidence in the significance of the cultures obtained from the samples.

The bacteria of particular interest to this study were those of likely public health importance, and those likely to cause systemic disease or gross pathological lesions in calves submitted for slaughter. These included:

Actinomyces pyogenes

Escherichia coli

Staphylococcus spp

Streptococcus spp

Pasteurella spp

Erysipelothrix rhusiopathiae

Salmonella spp

Clostridium spp

Fusobacterium necrophorum

A pilot trial was carried out at the Feilding slaughterhouse to ensure that the author's aseptic technique was satisfactory and to compare two methods of surface sterilisation prior to sample collection.

#### **Materials and Methods**

The following samples were collected from ewes being slaughtered at Feilding during April, 1989:

- (i) Dry swab of liver parenchyma, transferred immediately to blood agar plates.
- (ii) 0.1 ml of blood taken from the right ventricle of the heart using sterile pasteur pipettes, and immediately spread onto blood agar plates.
- (iii) Control blood agar plates which were swabbed with a sterile dry swab, or "spread" with the tip of a sterile pasteur pipette.

N.B. Umbilical swabs are unable to be obtained from this class of stock.

Plates were incubated aerobically at 37°C for 48 hours, since the majority of the bacteria of interest were aerobic or facultative anaerobic mesophiles (Ingram & Roberts 1979).

Two methods of surface sterilisation of the sample tissues were compared:

- (1) Searing with a hot spatula. A metal spatula was heated until red-hot in the flame of a portable butane burner. The spatula was then applied to the surface of the tissue to be sampled.
- (2) Flaming with 95% ethanol, where ethanol was applied to the surface of the tissue to be sampled with a syringe, and the alcohol lit.

Subsequent incisions into the tissues were then made with a sterile scalpel through the sterilised area.

#### Results

The results of the trial are shown in Table I.1

TABLE I.1: COMPARISON OF TWO METHODS OF SURFACE STERILIZATION: HOT SEAR WITH A SPATULA VS. SEAR WITH AN ALCOHOL FLAME.

	Hot	Sear	Al∞hol F	Totals	
SAMPLE	c*	NC**	С	NC	
Liver	7	23	9	21	60
Blood	5	25	5	25	60
Control	1	5	1	5	12
TOTALS	13	53	15	51	132

C contaminated.

## Conclusions

At the end of the trial it was concluded that:

- (1) Searing with a hot spatula and flaming with alcohol were equally satisfactory in terms of the sterilisation achieved, but the searing method was more timeconsuming and clumsy in the environment of the slaughterhouse. The use of alcohol flaming as the method for surface sterilisation of samples was therefore adopted.
- (2) Although no difficulty was experienced in obtaining sterile blood samples, it was found that sterile pasteur pipettes were inclined to break if there was any need to manipulate the pipette to obtain the sample. A more reliable alternative was the use of sterile 1 ml disposable tuberculin syringes (Terumo), without the

NC not contaminated.

needles. These could easily be inserted into the heart after stabbing a point of entry with a sterile scalpel blade, and there was no problem with breakage if manipulation of the syringe was required.

(3) The aseptic technique needed improvement, and the slaughterhouse environment presented no significant source of contamination.

#### I.2 Pilot trial B

#### Introduction

The original series of samples proposed to be used included the umbilicus itself, rather than the umbilical vein.

On 25-5-89, Weddel Crown's Aoteoroa slaughterhouse at Cambridge began slaughtering bobby calves. The slaughterhouse was visited on 25-5-89, 8-6-89, and 14-6-89, when a limited number of calf umbilici and livers were sampled, allowing a preliminary trial of the proposed methodology to be used at Feilding.

#### **Materials and Methods**

- (1) Dry transport swabs were taken of liver and umbilicus on the initial visit, with all swabs processed in the evening after sampling.
- (2) On the subsequent visits, dry transport swabs of the umbilical vein were attempted, again with all samples processed on the evening after sampling.

Blood sampling was not attempted, since the technique used at Feilding appeared to be satisfactory.

#### Results

(1) The umbilicus itself was a non-specific sampling site, since the tissue was almost always harbouring a heavy, mixed growth of aerobic and anaerobic organisms, whether or not the tissue was grossly inflamed or infected. (2) In cases with a very small venous lumen, sampling of the umbilical vein with a dry swab was a near-impossible task to accomplish without contaminating the swab. This problem would occur when sampling virtually all normal cohorts, and a number of condemned cohorts.

#### Conclusions

(1) The cost, time and laboratory requirements for anaerobic culture of the samples exceeded the resources of the study. It was therefore decided to discontinue this aspect of the investigation.

A more appropriate sampling site for the determination of significant umbilical infections was the umbilical vein close to where it entered the liver. Sampling at this site would identify organisms which had penetrated further than the calf's umbilicus, and would yield much less complex cultures.

(2) A sampling technique which has been shown to produce no significant difference in microbial counts when compared with the dry swab technique (Newton, Harrison & Suisted 1974) is that of sterile recovery of a sample of the tissue required followed by homogenisation in a Colworth Stomacher 400. This technique eliminated the sampling problems encountered using dry swabs, assuming that surface sterilisation standards were maintained. Consequently, 15 to 20 grams of liver tissue, and a 2-3 cm length of umbilical vein were used in the study.

It was further decided to sample skeletal muscle in addition to blood in order to help determine the presence of a current septicaemia/bacteraemia caused by circulating bacteria at the time of slaughter. This is because in the event of a systemic infection in the animal at the time of slaughter, it is the carcass meat that represents a risk to the consumer, due to the possible presence of bacteria in the muscle microvasculature, extracellular spaces of skeletal muscle and possibly intracellularly in the muscle cells (Vimini et al. 1983).

# I.3 The Effect of the Sticking Procedure on the Sterility of Heart Blood In Bobby Calves

Once sampling of calves began at Feilding, it became obvious that there were difficulties in obtaining sterile heart blood samples. In the first few weeks, when most of

the calves sampled were passed, i.e. "normal" calves, eight out of the first nine calves sampled were contaminated, the heart blood yielding a heavy, mixed growth. Since no difficulty was experienced in obtaining sterile samples of liver and muscle, it was likely that the heart blood was being contaminated before collection. It was observed that animals slaughtered at Feilding were slaughtered in compliance with Halal requirements. As discussed previously (ref. The Bobby Calf Industry in New Zealand), calves receive the additional thoracic stick to satisfy the requirements for animal welfare in this country after the head-only stun and bilateral severance of the carotids. The ewes were also slaughtered by the Halal method, but do not receive the thoracic stick. The thoracic stick is effective if the brachiocephalic trunk and/or the heart itself is incised by the knife. The knife used for sticking was the same as that used to open the skin along the ventral midline of the neck, and it was very rarely cleaned between the two operations. It was hypothesised that the sticking knife was inoculating bacteria into the heart chambers and that the contaminated blood was unlikely to be delivered anywhere else in the body because of the elimination of normal vascular pressure gradients as a result of the sticking operation. This hypothesis would appear to be borne out by the lack of similar heavy and mixed growths from vascular organs such as the liver.

In an attempt to test the hypothesis that blood was being contaminated by the sticker's knife, it was decided to take blood samples from 10 calves in the yards and collect blood from the hearts of the same calves at the inspection stand.

#### Materials and Methods

The samples taken in the yards were collected aseptically into 10 ml heparinised vacutainers (Nipro Neotube) and mixed. Seven drops from each sample were spread onto blood agar plates in the processing area within 15 minutes of collection. These calves were slaughtered at the end of the day, and blood collected in the normal way from the hearts on the viscera table.

#### Results

The results are shown in Table I.2 below:

TABLE I.2: COMPARISON OF CONTAMINATION RATES OF DUPLICATE BLOOD SAMPLES FROM 10 CALVES TAKEN IN THE YARDS AND AT THE INSPECTION STAND. (Expected distribution of values for  $\chi^2$  are subscripted.)

=	N <sup>O</sup> contaminated	N <sup>O</sup> sterile	Total	
Bood at slaughter.	6(4.5)	<sup>4</sup> (5.5)	10	
Blood at ante-mortem.	<sup>3</sup> (4.5)	<sup>7</sup> (5.5)	10	
Total	9	11	20	

The  $\chi^2$  statistic was calculated as 1.8, which is not significant at the 95% confidence level.

#### Conclusion

No valid conclusion could be drawn about the effect of the sticking knife on the contamination rate of the heart blood. The degree of asepsis obtained when blood sampling calves in the yards was inadequate.

Because the problem of contamination of heart blood could not be adequately resolved and the fact that up to 80% of calf hearts were incised by the sticker's knife, resulting in further contamination, the microbiological examination of heart blood was removed from the protocol.

Other sources of carcass blood, such as the femoral or mesenteric veins, were investigated for their merits as sampling sites for blood. These were found to be unsatisfactory because there was insufficient blood in these vessels to provide a suitable sample before the vein collapsed. Because arteries did not collapse, there was rarely sufficient blood present in their lumina for sampling purposes.

## I.4 Investigation of the Effect of Heparin on the Viability of a Broth Containing Escherichia coli

Heparinised tubes were used for the *ante-mortem* collection of whole blood for microbiological purposes as described above. Although anticoagulants other than sodium polyanetholesulphonate (SPS) are considered unsuitable for the collection of whole blood for microbiological purposes because of their possible inhibitory properties on bacteria present in the blood (Cruikshank *et al* 1973, Isenberg *et al* 1985), heparin is not specifically mentioned as one such anticoagulant (Cruikshank *et al* 1985). It was therefore decided to test the effect of heparin on the total viable counts (TVC) of an *E. coli* broth in order to determine whether the contamination rate of the blood samples collected *ante-mortem* could have been affected by the presence of heparin.

#### **Materials and Methods**

Twenty millilitres of two 24 to 25 hr heart infusion broth cultures of *E. coli* were obtained on two separate occasions. Ten millilitres of the broth was added to each of two heparinised vacutainers (Nipro) and mixed thoroughly. Serial dilutions from 10<sup>-1</sup> to 10<sup>-9</sup> of both the heparinised and non-heparinised cultures were prepared 15 minutes after adding the broth to the vacutainers. It was anticipated that the dilutions from 10<sup>-5</sup> to 10<sup>-8</sup> would provide cell densities on the agar plate most suitable for enumeration. Triplicate samples of these dilutions measuring 0.1 ml were taken and spread uniformly over blood agar plates and incubated at 37°C for 12 hours. All plates were counted, and plates with counts between 100 and 500 were used for TVC comparisons.

#### Results

Both trials showed a highly significant difference between the means of heparinised and non-heparinised cultures.

TABLE I.3: TOTAL VIABLE COUNT (TVC) OF AN E. COLI BROTH CULTURED WITH AND WITHOUT HEPARIN.

	Non-heparinised	Heparinised
	511x10 <sup>6</sup>	200 406
TVC 1 a		336x10 <sup>6</sup>
b	507×10 <sup>6</sup>	362x10 <sup>6</sup>
С	552x10 <sup>6</sup>	334x10 <sup>6</sup>
mean	523x10 <sup>6</sup>	344x10 <sup>6</sup>
pooled s.d. 2.0x1	0 <sup>7</sup>	
<i>t</i> -value=10.8, 5 d.f. p<0.	.001	
TVC 2 a	235×10 <sup>6</sup>	144×10 <sup>6</sup>
b	243×10 <sup>6</sup>	134×10 <sup>6</sup>
С	289x10 <sup>6</sup>	164x10 <sup>6</sup>
mean	255x10 <sup>6</sup>	147×10 <sup>6</sup>
pooled s.d. 2.3x1	10 <sup>7</sup>	
$s.dp = \sqrt{(n_1-1)s.d1 + (n_2)}$	-1)s.d. <sub>2</sub> /n <sub>1</sub> +n <sub>2</sub> -2	
$t=x_1-x_2/\sqrt{s.d.p^2(1/n_1+1/s)}$	n <sub>2</sub> )	
<i>t</i> -value=5.7, 5 d.f. 0.001	I0.005	

#### Conclusions

The results indicated that heparin is likely to have affected the total viable count of the samples taken from the calves *ante-mortem*, since the counts of the heparinised plates were only about 60% of those of the non-heparinised plates. At low cell densities, as could be expected from the contaminated heart blood samples, this depression of cell viability could have confounded and further invalidated the results of the comparative trial. Although no other organisms were tested, it was assumed that bacteria other than *E. coli* would be similarly affected by the presence of heparin.

#### **APPENDIX II**

## DETAILS OF MICROBIOLOGICAL AND HISTOLOGICAL METHODS AND MATERALS

#### (1) Microbiological Materials and Methods

### (a) Gram stain

A differential stain for bacteria based on the inability of certain bacteria to be readily decolourised by alcohol once they have been stained with a combination of crystal violet and iodine. Gram negative bacteria readily decolourise, and subsequently pick up the colour of the counterstain, usually saffranin, a red dye. Gram positive bacteria, on the other hand, are not readily decolourised, so they retain the violet to blue-black colour of the crystal violet-iodine dye complex.

## (b) Oxidase test

This test differentiates bacteria which possess the oxidase enzyme from those which do not. Oxidase promotes an oxidation reaction indicated by the colonies changing colour to black.

#### (c) API 10 S

La balme les grottes, 38390 Montalieu vercieu, France.

This is a standardised identification system for common *Enterobacteriaceae* and other gram negative rods. It uses 12 miniaturised biochemical tests, the results of which are recorded on a result sheet provided with the kit. The results are coded into a four-digit profile number, which is interpreted by the identification table also provided.

#### (d) API 20 NE

This test kit extends the use of the API 10 S by a further ten tests. The principles and general methodology are exactly the same as for the API 10 S system.

## (e) Sugars, fermentative base

Fermentation of the appropriate sugars is indicated by a colour change to pink.

## (f) T.G.Y (tryptone, glucose, yeast) slopes

This medium is used for temperature studies, usually at 25°C, 37°C and 42°C, on bacterial colonies. It is also a suitable medium on which to perform the catalase test, since there are no body fluids which can produce false positive reactions.

#### (g) Catalase test

Up to 3 ml of fresh 3% hydrogen peroxide is added to a TGY slant of the bacterium to be tested. The presence of catalase is indicated by the active liberation of oxygen from the peroxide (fizzing).

#### (h) Litmus milk

#### Acid reactions:

- (i) Pink-red colour caused by the fermentation of carbohydrates, glucose and lactose.
- (ii) The production of lactic acid results in a casein curd in clear fluid.
- (iii) A 'stormy' clot results from gas formation in the curd.

#### Alkaline reactions:

- (i) Blue colour due to basic amine formation from proteolysis.
- (ii) Alkaline coagulation, with the formation of a soft, blue clot.
- (iii) Peptonisation results in complete digestion of the clot. The medium becomes transparent.

#### Redox reactions:

The medium is decolourised and similar in appearance to fresh litmus milk. This is caused by reductase enzymes.

#### (m) Loeffler's slope

This medium, very rich in animal serum, is used for the cultivation of fastidious organisms such as *Corynebacterum spp*, plus the demonstration of proteolysis by the development of pitted areas of liquified medium around the colonies. Eventually the slope may be liquified with the production of a putrefactive odour.

Prepared Loeffler's slopes supplied by Fort Richards Laboratories, Box 22-172, Auckland New Zealand, were used in this work.

#### (n) DNase agar

There is a close correlation between DNase activity and coagulase production of staphylococci, providing the basis for a laboratory test for the identification of potentially pathogenic organisms.

Enzymes (DNases) which are produced by such organisms hydrolyse DNA present in the agar into a mixture of mono and poly-amines. After incubation of DNase agar inoculated with a heavy streak of the suspect organism at 37°C for about 18 hours, the plate is flooded with normal HCI. This reacts with nucleic acid to produce a cloudy precipitate. There is a clear zone around DNase positive colonies, however, due to the inability of the nucleotide fractions to precipitate.

#### (o) Coagulase test

This may be performed as either a slide test, or a tube test. The tube test is described.

To 0.5 ml of rabbit plasma in a tube, a loopful, a colony, or 0.5 ml of a broth of the suspect colony is added. The tube is placed in a water bath at 37°C and examined at 6 and 24 hours. Known positive and negative controls are always put up simultaneously. A positive result is one where a clot of any sort is formed.

## (p) Aesculin agar

Certain streptococci may be differentiated on the basis of their ability to hydrolyse aesculin. Hydrolysis of aesculin is demonstrated when the agar turns black.

#### (r) Simmons citrate agar

This agar medium is used for the differentiation of the family Enterobacteriaceae. A positive growth occurs when bacteria are able to use citrate as the sole source of carbon, and results in a colour change in the medium from green to bright blue. A negative test results in no colour change.

#### (s) Salmonella-Shigella agar

This is a differential, selective medium for the isolation of *Salmonella* and *Shigella spp*. from specimens. Salmonellae form transparent colonies, usually with black centres, while shigellae form transparent colonies. Lactose fermenting organisms develop colonies with pink centres after 48 hours.

#### (t) Decarboxylase tests

Some members of the enterobacteriaceae may be differentiated by the production of lysine, arginine, ornithine and glutamic acid decarboxylase enzymes. These enzymes form alkaline products which show a purple or violet reaction. Negative tests are a definite yellow.

#### (u) Christensen's urea

This urea agar was devised for the differentiation of enteric bacilli. Bacteria capable of splitting urea produce ammonia, resulting in a purplish or bluish red colour change to the slant.

## (v) Nitrate broth

This determines the ability of bacteria to reduce nitrate. When nitrate positive organisms reduce the nitrate to nitrite, a pink colour develops after a few drops

of the reagents suphanilic acid and alpha-naphthylamine are added to the bacterial broth.

#### Choice of selective and differential agar plates for the study

The agar plates used were chosen for the following purposes (Anon. 1982(b)):

#### **Blood Agar**

A non-selective medium containing animal blood (sheep blood in this instance) to enhance the growth of slow-growing bacteria, or those that will not grow at all on simple media. Certain bacteria also produce haemolysis of the red blood cells around their colony. Bacteria may therefore be non-haemolytic, alpha-haemolytic (incomplete, with the production of a greenish opaque zone around the colony), or beta-haemolytic (complete, where a clear zone is produced around colonies). Some bacteria may produce both types, resulting in alpha-beta-haemolysis.

#### MacConkey's Agar

A selective and differential agar used for the selective isolation of gram-negative bacteria. It contains lactose, neutral red indicator and bile salts. The bile salts prevent the growth of some bacteria, resulting in selectivity.

Lactose fermenting bacteria produce acids which react with the indicator, resulting in pink or red colonies. Those which do not ferment lactose do not react with the indicator, producing pale, non-pink colonies. Initial differential information is thus provided about the colonies.

#### Xylose-Lysine-Deoxycholate (XLD) Agar

Another selective and differential agar used for the isolation and presumptive identification of Salmonella spp and Shigella spp.

- (a) Shigella spp. are Xylose negative bacteria, thereby producing an alkaline medium. This is indicated by phenol red indicator as red colonies.
- (b) Salmonellae, on the other hand, are Xylose positive. Lysine is added to the medium, and is decarboxylated by Salmonellae, thus changing an initial acid

reaction to alkaline and the bacterial colonies to red. In addition, some *Salmonellae* produce hydrogen sulphide from iron salts also incorporated in the medium, resulting in black centres to the colonies- a feature which aids in differentiating *Salmonellae* from *Shigellae*.

- (c) Any Lysine positive Coliforms present are prevented from reverting the pH to acid by the addition, to excess, of lactose and sucrose, as well as xylose. These bacteria grow as yellow colonies on XLD.
- (d) Sodium deoxycholate is added to the medium at concentrations sufficient to suppress the growth of Coliforms and non-enteric bacteria.

Direct plating of samples onto XLD was the method used to provide the initial presumptive identification of any *Salmonellae* which were causing systemic disease in the calves. The far more sensitive methods incorporating pre-enrichment were not used because the objective was not to isolate the low numbers of *Salmonellae* which may be present in the organs of otherwise healthy, carrier animals. Although these serve as an important source of contamination of the meat processing environment, the intent was to detect *Salmonellae* which were producing systemic disease and should have therefore been present in high numbers in the tissues sampled.

## (2) Histological Materials and Methods

## (a) Haematoxylin-Eosin stain

Haematoxylin is most commonly used as a nuclear stain, but is also used for the demonstration of myelin, elastic fibres, fibrin, neuroglia and muscle striations. It usually precedes the use of eosin, which stains cytoplasm and connective tissue. Nuclei are stained blue-black; cartilage usually light blue to dark blue; calcium and calcified bone purplish blue; basophilic cytoplasm purplish; red blood cells and eosinophilic granules bright orange-red; cytoplasm shades of pink; muscle fibres, thyroid colloid, thick elastic fibres, and decalcified bone matrix deep pink; collagen and osteoid tissue light pink (Drury & Wallington 1967).

#### (b) Gram-Twort stain

Gram positive bacteria stain dark blue, gram negative bacteria pink. Nuclei stain red, cytoplasm light green, and red blood cells green (Drury & Wallington 1967).

## **APPENDIX III**

## **RECORDING FORM**

## GROSS PATHOLOGICAL DETAILS OF BOBBY CALF CARCASSES CONDEMNED FOR NAVEL ILL, AND THEIR UNAFFECTED COHORTS

Calf No.:			
Chain:			
Time:			
Sex:			
Age:	4 days		
	8-10 days		
UMBILICAL ABNORM	ALITY:		
No		Complete resolution  Limited cellulitis and/or scarring	
Yes		Inflammation/bruising	
		Cellulitis	
		Unencapsulated abscess	
		Necrotic	
		Encapsulated abscess	
		Gangrenous	

Involvement	of:	Umbilical vein	
		Arteries/urachus	
		Both	
		Neither	
LIVER:			
	Normal		
	Hyperaemic		
	Petecchiated		
	Pale		
	Cirrhotic		
	Miliary abscesses		
	Peritonitis: adhesions - a	cute hronic	
	Infarcts		
	Extraneous abscesses		
	Neoplasia		
	Congenital abnormality		

## LYMPH NODES:

	pt				
	Normal	Enlarged	Haemorrhagic	Abscessed	Oedematous
Liver					
Mediastinal					
Mesenteric					
Head					
Forequarter		ON THE BOARD AND A PART AND A PAR			
Hindquarter					
Pelvic					

## KIDNEYS:

	Single	Both
Normal		
Hyperaemic		
Swollen	THE PERSON OF THE PERSON MANAGEMENT AND ADDRESS OF THE PERSON AD	
Petecchiated		
Pyelonephritis		
Shrunken		
Pale		
Congenital abn.		
Infacrcts		
**Embolic Nephritis		
Extraneous Abscess		

- \*Pyelonephritis ascending infection of
  kidneys from l.u.t.
  Necrosis of pelvis and
  papillae. <u>+</u> necrosis of
  parenchyma.
- \*\*Embolic Nephritis subsequent to bacteraemia.
  Cortical abscessation and
  fibrosis.

#### LUNGS:

Embolic lesions	Acute Chronic		
EMDOIIC lesions	Abscessated		
	Acute		
Lobar lesions	Chronic Abscessated		
	Acute		
Adhesions	Chronic Abscessated	-	
		Serous	
leuritic Effusions	Massive	Sanguineous	
		Fibrinous Pustular	
		Serous	
	Minor	Sanguineous	
		Fibrinous	
		Pustular	

HEAR!	r:								
	Norma	1					. [		
	Petec								
	Peric								
	Myoca								
	Valvu	lar les	sion	ıs					
	Conge	nital o	defe	ects					
	Emaci	ated fa	at c	leposits					
THOR	ACIC C	AVITY:					ſ		
	Norma	1					1		
	Acute	pleur	isy						
	Chron	ic ple	uris	зу					
	Seros	al pete	ecci	niations			1	-	
	Broke	n ribs						M. dan harata bertakan tilai ara s	
	Bruis	ing							
							,		
GUT	SET:								
								ı'	
								A	dhesions
		Nor	mal	Hyperaemic	Haemorrhagic	Necrotic	Neoplastic	Acute	Chronic
	nasum								
Jeju Ileu									
Colo	n								
Caec	um								
						1			
		0		11					
		congen	ıla.	l abnormali	cies				
SPLE	EN.								
SPLE		Normal							
		Hypera							
		Haemor				-			
				nlarged					
		Neopla	Sti						

PERITONEA	L CAVI	TY:			t						
	Norma	1		1							
	Petec	chiated			The second of th						
	Adhes	ions - a				20 ° 10 ' 10 ' 10 ' 10 ' 10 ' 10 ' 10 ' 1	-				
			chronic								
	Bruis						_				
	Emaci	ation				****	i				
JOINTS:											
		-		*		1		1			
		Enlar	rged	Distor	rted	Contra		Injury			
		1	2	1	2	1	2	1	2		
Hock											
Stifle				1							
Scapular				•		The state of the s					
Carpal		de de la companya de	-	•					7,700,000,000		
JOINT FLU	JID:										
	Norma	ıl									
	Exces	sive									
	Turbi	.d									
	Sangu	ineous									
	Pustu	ılar					-				
MUSCULAT	URE:										
							i				
	Norma	al									
	Emaci	ated									
	Bruis	sed/wound	d								
	Seros	sal/fat	petecchia	ations							
	Trocu	lation	aheceeeo	2							

Embolic abscesses

#### **APPENDIX IV**

#### **RANDOM TIMES TABLE**

To eliminate selection bias on the normal carcasses selected as cohorts for the calves with "navel ill", 12 random times tables generated by the Department of Biotechnology, Massey University, (Figure IV.1), were used.

To use these tables proceed as follows:

- (1) Blindly pick a random times table.
- (2) Block off all the times of day during which carcasses are not flowing past the inspection stand (i.e. before and after the kill day, during breaks and lunch).
- (3) Total the number of times of day during which carcasses are flowing past the inspection stand (Y).
- (4) Determine the number of carcasses which will be sampled during the day (X).
- (5) Calculate the sampling interval (Y/X), dropping all fractions.
- (6) Randomly select a starting point, N, from the list of numbers at the left of the table. The number must be equal to or less than the sampling interval.
- (7) Select the required number of times of day from the table:  $N + Y_1/X_1 + Y_2/X_2 + .... Y_i/X_i$

RAN	DOM	NO.					RANI	DOM T	IMES					
32	57	57	702	756	841	954	1049	1157	1258	149	249	333	431	551
37	22	54	704	802	845			1157	1259	149	250	339	442	553
38	10	22	705	804	846	955	1052	1159	103	151	250	340	444	558
63	2	1	706	804	848	956		1205	104	152	251	341	451	603
34	34	49	709	806	849	959	1055	1210	105	159	251	345	452	605
63	17	21												
42	17	64	710	806	852	1003	1100	1212	107	200	254	345	453	610
10	64	56	710	806	853	1004	1105	1212	107	205	256	350	456	611
12	23	35	711	807	902	1009	1108	1213	117	207	256	353	456	620
7	45	13	712	808	903	1013			119	208	259	354	458	621
21	62	20	716	808	903	1016	1115	1217	121	209	306	355	504	624
40	67	51												
69	23	16	718	813	909	1017		1220	121	211	308	356	<b>507</b>	625
29	70	60	720	814	909			1221	122	211	309	357	514	630
35	37	34	721	814	914	1021		1222	124	213	310	359	514	633
38	21	63	721	815	915	1021	1123	1222	126	216	312	403	515	634
69	20	58	722	816	915	1024	1125	1231	126	216	313	403	517	638
10	66	1												
27	8	13	725	818		1025	1131	1232	130	218	314	405	532	641
12	66	33	727	818	931	1025	1131	1234	134	223	314	406	533	641
29	15	41	728	827		1027			136	233	316	410	534	643
6	53	7	731	828	933	1027			139	233	316	415	535	643
6	45	14	739	829	935	1034	1140	1247	139	235	318	415	538	645
58	20	5												
10	64	51	745	830				1248	140	242	325	417	540	648
5	60	59	746	830	936	1037	1147	1248	142	243	326	422	542	652
6	42	70	749	831	944			1249	144	246	328	425	548	654
47	45	28	750	832		1044			145	248	329	429	551	658
11	31	59	755	837	949	1049	1123	1257	147	248	332	431	<b>5</b> 51	700

## REFERENCES

- Alley M.R. The Pathogenesis of Pneumonia in Sheep. PhD Thesis, 1975.

  c/o Department of Pathology & Public Health, Massey University, Palmerston North, New Zealand.
- Amstutz H.E. Chapter 20: Neonatal diarrhoea (white scours, colibacillosis, calf septicaemia, infectious diarrhoea), pp. 689-695.
  In Bovine medicine, surgery and herd health management, 1970.
  Gibbons, W.J., Catcott, E.J., Smithcors, J.F., editors.
  - American Veterinary Publications, Ltd., 114 North West st, Wheaton, Illinois.
- In Proceedings of a course of dairy cattle medicine, 1983. Slaughter, R.E., convenor.

Foundation for the Continuing Education of the N.Z. Veterinary Association, Massey University, Palmerston North, New Zealand.

- Anon 1955. Bobby Calf Marketing Regulations of New Zealand.

  Government Printer, Wellington, New Zealand.
- Anon 1969 (a). Meat Regulations of New Zealand.

  Government Printer, Wellington, New Zealand.

Anderson D.C. Chapter 1: Calf rearing, pp. 6-20.

- Anon 1969 (b). The Slaughter of Stock, Game and Poultry regulations of New Zealand.

  Government Printer, Wellington, New Zealand.
- Anon 1977. Meat manual number 16: post-mortem inspection procedures.
  Enquiries to: Chief Meat Veterinary Officer, MAFQual, PO Box 2526, Wellington New Zeraland.
- Anon 1980. Salmonellosis data. Surveillance, 7 (4), pp. 4-5, 1980.

  Enquiries to: Stuart C. McDiarmid, Consultant Editor, Surveillance (MAFQual),

  Ministry of Agriculture and Fisheries, PO Box 2526, Wellington.

Anon 1982(a). Manual of Procedures for the Meat Hygiene Division of the Food Production and Inspection Branch of Agriculture Canada. Chapter 4: Red Meat Species, pp. 41-44, 1982.

Government Printer, Canada.

Anon 1982 (b). The Oxoid Manual of Culture Media, Ingredients and other Laboratory Services, 5th edition.

OXOID Limited, Wade Road, Basingstoke, Hampshire RG24 OPW, UK.

Anon 1983. Article 20(f), Official Journal NO L59, p. 42, 1983.

In: Official Journal of the European Communities, council directive 83/91/EEC. Enquiries to: The Chief Meat Veterinary Officer, MAFQual, P.O. Box 2526, Wellington New Zealand.

Anon 1985 (a). Codex Alimentarius Commission, volume C.

The Recommended International Code of Practice for *Ante* and *Post-mortem* Judgement of Slaughter Animals and Meat.

Issued by: Secretariat of the Joint FAO/WHO Food Standards Programme, FAO, Rome.

Anon 1985 (b). Australian Export Meat Manual, Section 20. c/o Department of Primary Industry, Canberra, Australia.

Anon 1986. History of tb control scheme. Surveillance 13 (3), pp. 4-12, 1986.

Enquiries to: Stuart C. McDiarmid, Consultant Editor, Surveillance (MAFQual),

Ministry of Agriculture and Fisheries, PO Box 2526, Wellington.

Anon 1987. Inspection disposition form for cattle.

United States Department of Agriculture Government Printing Office, 1987-169-404, USA.

Anon 1988. Cattle Tb- an update on the situation in New Zealand. Surveillance, <u>15</u> (1) pp. 3-7, 1988.

Enquiries to: Stuart C. McDiarmid, Consultant Editor, Surveillance (MAFQual), Ministry of Agriculture and Fisheries, PO Box 2526, Wellington.

Anon 1988/89.

Annual Report 1988/89. Livestock Improvement Corporation, Ltd., Private Bag 3016, Hamilton, New Zealand.

Anon 1989. United States Meat and Poultry Inspection Regulations, Book 1: Meat Inspection Regulations, Title 9, Chapter III, Subchapter (a): Code of Federal Regulations, 1989 revision.

United States Government Printery, USA.

Anon. 1990 (a). Clostridial Diseases of Animals in New Zealand. Surveillance 17 (4), pp. 19-20, 1990.

Enquiries to: Stuart C. McDiarmid, Consultant Editor, Surveillance (MAFQual), Ministry of Agriculture and Fisheries, PO Box 2526, Wellington.

Anon 1990 (b). Anderson's Pathology, 9th edition, volume 2 (index). John M. Kissare, editor. C.V. Mosby Co, 11830 Westline Ind. Dr, St. Louis, Missouri 63146, USA.

Baxter G. M. Umbilical Masses in Calves: Diagnosis, Treatment and Complications.

The Compendium for Continuing Education 11 (4): 505-513, 1989.

Bensink J.C. Review of Meat-borne Pathogens, pp. 49-61.

In Advances in Veterinary Public Health, vol 1, 1981.

The Australian College of Veterinary Scientists, AVA House, PO Box 34, Indooroopilly, Quensland 4068, Australia.

Blackmore D.K. and Humble M.W. Campylobacteriosis, p 15.

Zoonoses in New Zealand, 1987.

Occcasional publication 112, Foundation for the Continuing Education for the New Zealand Veterinary Association, Massey University, Palmerston North...

Blakiston's Gould Medical Dictionary, 4th edition.

McGraw-Hill Book Company, New York.

Blood D.C. and Radostits O.M.

- (a) Chapter 3: Diseases of the Newborn-neonatal infection, pp.107-119.
- (b) Omphalitis, omphalophlebitis and urachitis in newborn farm animals ("navel ill"),p. 121.
- (c) Pericarditis, pp. 329-331.
- (d) Diseases caused by Escherischia coli, pp. 619-637.
- (e) Diseases caused by Salmonella spp., pp 643-657.
- (f) Diseases caused by *Pasteurella* spp., pp. 657-673.
- (g) Tuberculosis caused by *Mycobacterium bovis*, pp. 710-720.
- (h) Diseases caused by *Clostridium* spp., pp. 612-614.
- (i) Aspiration pneumonia, p. 373.
- (k) Arthritis, p. 469.

In Veterinary medicine, 7th edition, 1989.

Balliere Tindall, 24-28 Oval rd, London W1 7DX.

Bouckaert J.H. & de Moor A. Surgical Treatment of Umbilical Infections in Calves. The Veterinary Record, 77 (27): 771-774, 1966.

Brown W.A.B., Christofferson P.V., Massler M., Weiss M.B. Postnatal tooth development in cattle.

American Journal of Veterinary Research, (1): 15-16, 23, 28-31.

Bryant H.E., Athar M.A., and Pai C.H. Risk Factors for *Escherischia coli* O157:H7 Infection in an Urban Community.

The Journal of Infectious Diseases, 160 (5): 858-864, 1989.

#### Cheville N. F.

- (a) Aspiration and gangrenous pneumonia, Chapter 12, p. 544.
- (b) ETEC, Chapter 11, p. 522.
- (c) Clostridium perfringens, Chapter 11, p. 524.
- (d) Septicaemia/bacteraemia, Chapter 6, pp. 286-291.

Cell pathology, 2nd edition, 1983.

Iowa State University Press, Ames, Iowa 50010, USA.

Cruickshank R., Duguig J.P., Marmion B.P., Swain R.H.A.

Medical Microbiology: the practice of medical microbiology, 12th edition, vol 2, p. 162, 1973.

Churchill Livingstone, 23 Ravelston tce, Edinburgh EH4 3TL.

- Dairy Meats New Zealand Ltd, Level 3, 114 Dominion Road, Mt. Eden, Auckland 3, New Zealand.
- de Jong H, and Ekdahl M.O. Salmonellosis in calves-the effect of dose rate and other factors on transmission.

New Zealand Veterinary Journal, 13: 59-64, 1965.

Doige C.E. Chapter 11: Skeletal System, pp. 495-498.

In Special Veterinary Pathology. R.G. Thomson (editor) 1988.

B.C. Decker Inc, 3228 South Service Road, Burlington, Ontario L7N 3H8, U.S.A.

Dow S. W., and Jones R. L. Bacteraemia: pathogenesis and diagnosis.

The Compendium for Continuing Education, 11 (4): 432-443, 1989.

Doyle M. P. and Schoeni, J. L. Isolation of *Escherischia coli* O157:H7 from Retail Fresh Meats and Poultry.

\*\*Applied and Environmental Microbiology, 53: 2394-2396, 1987.

Drury R.A.B. and Wallington E.A. Chapter 7: General Staining Procedures, pp. 129-130.

In Carleton's histological technique, 2nd edition, 1967 Oxford University Press, Ely house, London 1.

Dyce K.M., Sack W.O., Wensing C.J.G. Chapter 7: The Cardiovascular System: the Circulation in the Foetus and the Changes After Birth, pp. 244-246.
 In Textbook of veterinary anatomy, 1987.
 W.B. Saunders & co, The Curtis Centre, Independence Square West,

W.B. Saunders & co, The Curtis Centre, Independence Square West, Philadelphia, PA 19106.

Firth E.C., Kersjes A.W., Dik K.J., Hagens, F.M. Haematogenous Osteomyelitis in Cattle.

The Veterinary Record 120: 148-152, 1987.

Franco D. A. Micro-organisms in the Blood: the Public Health Implications. *Veterinary Medicine*, <u>83</u> (8): 832-835, 1988. Frank G. H. Symposium on Pasteurella haemolytica.

When *Pasteurella haemolytica* Colonises the Nasal Passages of Cattle. *Veterinary Medicine*, 83 (10): 1060-1064, 1988.

Frank G. H. Chapter 9: Pasteurellosis of Cattle, pp. 207-219.

In Pasteurella and Pasteurellosis. Adlam, C. and Rutter, J.M. (editors), 1989.

Academic Press Ltd., 24/28 Oval rd, London NW1 7DX.

Gibson E.A. 1.-Salmonellosis in calves.

The Veterinary Record, 73 (48):1284-1296, 1961.

Genigeorgis C. A. Flecent Trends in Foodborne Diseases of Bacterial Origin.

Eighth International Symposium of the orld Association of Veterinary Food Hygienists. Collins, J.D. & Hannan, J. (editors) 1981.

E.T.A. Publications Ltd, Department of Veterinary Preventive Medicine and Food Hygeine, University College, Dublin 4, Ireland.

Gracey J.F. Chapter 1: Pathology, pp. 279-304.

In Meat Hygeine, 8th edition, 1986.

Balliere Tindall, W.B. Saunders, 1 St. Annes rd, Eastbourne, East Sussex, BN21 3UN, England.

Greene H. J. and Dempsey D. Bovine Neonatal Salmonellosis: an Outbreak in a Dairy Calf Unit.

Irish Veterinary Journal 40: 30-34, 1986.

Harrison R. Chapter 5: Veal Calves, pp. 62-88.

In Animal Machines-the new Factory Farming Industry, 1964.

Vincent Stuart Publishers Ltd, 45 Lower Belgrave st, London SW1.

Hathaway S.C. Leptospirosis in New Zealand: an ecological view.

New Zealand Veterinary Journal, 29: 109-112, 1983.

Hylton W. E., and Trent, A.M. Congenital Urethral Obstruction, Uroperitoneum, and Omphalitis in a Calf.

Journal of the American Veterinary Medical Association 190 (4): 433-434, 1987.

Ingram M., and Roberts T.A. The Microbiology of the Red Meat Carcass and the Slaughterhouse.

Journal of the Royal Society of Health 96 (6): 270-276, 1976.

Ireland J.A. Death Survival and Dispersion of Salmonellae in Food Production, Processing and Storage, pp. 53-88.

In Advances in Veterinary Public Health, vol 3, 1987.

The Australian College of Veterinary Scientists, AVA House, PO Box 34, Indooroopilly, Quensland 4068, Australia.

Isenberg H.D., Washington J.A. II, Balows A., Sonnenwirth A.C.

Clinical Microbiology, 3rd edition, 1980: p. 54. Lennette E.H., (chief editor).

American Society for Microbiology, 1913I St, NW Washington DC 20006.

Jones E.W., Hamm D., Corley L., and Bush, L. Diarrhoeal Diseases of the Calf:
Observations on Treatment and Prevention.

New Zealand Veterinary Journal, 25: 312-316, 1977.

Jubb K.V.F., Kennedy P. C., and Palmer Nigel.

- (a) Focal interstitial nephritis: vol 1, p. 115; vol 3, p. 135.
- (b) Pneumonia: vol 2, pp. 470, 452-458, 489.
- (c) Arthritis: vol 1, pp. 104-116.
- (d) Congenital tuberculosis: vol 3, p. 498.
- (e) Pericarditis: vol 3, pp. 13-15.

In Pathology of Domestic Animals, 3rd edition, 1985.

Academic Press Inc, Orlando, Florida 32887.

Keatinge W.R. Blood vessels- Responses to Injury, pp. 252-253. British Medical Bulletin, 35 (3): 252-253, 1979.

Leigh P., and Delaney M. Use of "Thoracic Stick" in Halal Slaughter of Bobby Calves.

New Zealand Veterinary Journal, 35: 124-125, 1987.

Manktelow B.W. The Veterinary Handbook, 1st edition, pp. 55-56, 1984.

The Foundation for the Continuing Education of the N.Z. Veterinary Association,
Massey University, Palmerston North.

Martin S. W., Meek A. H., & Willeberg P.

- (a) Chapter 6: Surveys & Analytic Observational Studies.
- (b) Chapter 3: Measurement of Disease Frequency & Production, pp. 73-76.
   In Veterinary Epidemiology- Principles & Methods, 1987.
   lowa State University Press, Ames, Iowa, 50010, U.S.A.
- Matthewson J.A., and duPont H.L. Chapter 6: *Escherichia coli*, pp. 85-100.

  In Infectious diarrhoea. Gorbach, Sherwood C. (editor) 1986.

  Blackwell Scientific Publishers, 52 Beacon st, Boston, Masachusetts 02108, USA.
- Maximov A.A, and Bloom W. Chapter 10, The Cardiovascular System, pp. 109-135.

  In A textbook of histology, 7th edition, 1957.

  W.B. Saunders & Co, Philadelphia.
- McFarlane D. Perinatal Lamb Losses I. An Autopsy Method for the Investigation of Perinatal Losses.

  New Zealand Veterinary Journal, 13: 116-135, 1965.
- Mims C. A. Chapter 5: The Spread of Microbes through the Body, pp. 68-93.

  In The pathogenesis of Infectious Disease.

  Academic Press, 24/28 Oval rd, London NW1.
- Meat Industry Research Institute of New Zealand. Publication 757: Microbiological Methods for the Meat Industry. Lowry, P.D. (editor) 1980.

  Enquiries: The Director, MIRINZ, P.O. Box 617, Hamilton, New Zealand.
- Morgan A., Hall G.A., and Reynolds D. J. The Association of *Campylobacter* Species with Calf Diarrhoea.

XIV World Congress on Cattle Diseases, vol 1, pp. 325-329, 1986.

Proceedings of the World Association for Buiatrics, c/o M. Monaghan,

Veterinary Medical Faculty, University College of Dublin, Ballsbridge, Dublin 4,

Veterinary Medical Faculty, University College of Dublin, Ballsbridge, Dublin 4, Ireland.

Nazer A.H.K. and Osborne A.D. Salmonella Infection and Contamination of Veal Calves: a Slaughterhouse Survey.

British Veterinary Journal, 132 (2): 192-200, 1976.

Newhook J.C. and Blackmore D.C. EEG Studies of Stunning and Slaughter of Sheep and Calves Part 2- the Onset of Permanent Insensibility in Calves during Slaughter.

Meat science 6: 295-300, 1982.

Newton K.G., Harrison J.C.L., and Suisted J.R. An evaluation of the Colworth Stomacher and Droplette as Aids for Microbiological Counting of Meat and Meat Products.

MIRINZ publication 393, 1974.

Enquiries: The Director, Meat Industry Research Institute of New Zealand, P.O. Box 617, Hamilton, New Zealand.

Nottingham P.M., and Urselmann A.J. Salmonella Infection in Calves and Other Animals.

New Zealand Journal of Agricultural Research, 4: 449-460, 1961.

Nottingham P.M., Penney N., and Wyborn R. Salmonella Infection in Calves and Other Animals III. Further Studies with Calves and Pigs.

New Zealand Journal of Agricultural Research, 15: 279-283, 1972.

Nottingham P.M. Chapter 2: The Microbiology of Carcass Meats.

In Meat Microbiology. Brown, M.H. (editor), 1982.

Applied Science Publishers Ltd, Ripple rd, Barking, Essex, England.

O'Connor B.C. A History of the New Zealand Bobby Calf Pool Movement: pp. 7, 40, 41, 53, 1979.

Enquiries to: The Central Executive, c/o Dairy Meats NZ Ltd, P.O. Box 2240, Auckland 1, New Zealand.

Osburn B.I. Chapter 14: Ontogeny of Immune Responses in Cattle, pp. 252-260.

In The Ruminant Immune System in Health and Disease. Morrison, W. Ivan, (editor) 1986.

Cambridge University Press, The Pitt Building, Trumpington st, Cambridge CB2 1P.

Omar A.R. The Aetiology & Pathology of Pneumonia in Calves. *The Veterinary Bulletin*, (36) 5: 1-15, 1966.

Petrie L., Selman I.E., Grindlay M., and Thompson H. Salmonellosis in Young Calves due to *Salmonella enteritidis*.

The Veterinary Record, 101: 398-402, 1977.

Radostits O.M., and Acres S.D. Chapter 2: Disease Prevention in Calves, Diarrhoea in the Newborn, pp. 43-54.

In Bovine medicine and surgery, volume 1, Amstutz, H.E. (editor) 1980.

American Veterinary Publications Ltd, 114 North West st, Wheaton, Illinois.

- Rehmtulla A.J., and Thomson R.G. A review of the Lesions of Shipping Fever in Cattle. Canadian Veterinary Journal, 22: 1-8, 1981.
- Robinson R.A. Ante-mortem Condemnation of Young Calves.

  New Zealand Veterinary Journal, 13: 77 (correspondence) 1965.
- Robinson R.A. Salmonellosis in Young Calves.

  New Zealand Veterinary Journal, 14: 33-39, 1966.

## Roy J.H.B.

- (a) Chapter 3: Neonatal Calf Diarrhoea (excluding salmonella) pp. 53-117.
- (b) Chapter 4: Salmonellosis pp. 118-131.
- (c) Chapter 5: Respiratory infections pp. 132-153.
- (d) Chapter 2: Immunity to disease pp.17-52.
- (e) Chapter 7: Miscellaneous infections, metabolic disorders, nutritional deficiencies and poisoning pp. 197-198.

In The Calf- Management of Health, vol 1, 5th edition, 1989. Butterworths, Boston.

Schlesselman J. J. Chapter 7: Basic Methods of Analysis, pp. 171-181.

In Case-Control Studies: Design, Conduct, Analysis, 1982. Oxford University Press, New York.

Schwabe C. W., Riemann, H. P., Frank, C. E. Chapter 11: Identification of Disease Determinants: Use of Case-Control Studies, pp. 177-181.

In Epidemiology in Veterinary Practice, 1977.

Lea and Feabiger, Philadelphia, USA.

Silliker J.H., and Gabis D.A. Salmonella: Chapter 7, pp. 212-213. In: Advances in Meat Research vol. 2, 1986.

- Slauson D. O. and Cooper, B. J. Chapter 8: Nature and Causes of Disease-Interactions of Host, Parasite and Environment, p. 403.
  In Mechanisms of Disease- A Textbook of Comparative General Pathology, 1982. Williams & Wilkins, 428 East Preston st, Baltimore London.
- Smith H.A. and Jones T.C. Veterinary Pathology, 3rd edition, p. 996, 1966.

  Balliere Tindall and Cassell, 7 & 8 Henrietta st, WC2 London.
- Thomson G.W., McSherry B.J., and Valli V.E.O. Endotoxin Induced Disseminated Intravascular Coagulation in Cattle.

  Canadian Journal of Comparative Medicine, (38): 457-466, 1974.
- Thomson R.G General Veterinary Pathology, p. 198, 1978.

  W.B. Saunders co, West Washington Square, Philadelphia PA 19105, USA.
- Thrusfield M. Chapter 13: Demonstrating association, p. 145.
  In Veterinary Epidemiology, 1986.
  Butterworth & co, England.
- Trent A.M., and Smith D.F. (a) Surgical Management of Umbilical Masses with Associated Umbilical Cord Remnant Infections in Calves.

  Journal of the American Veterinary Medical Association, 185 (12): 1531-1534, 1984.
- Trent A.M., and Smith D.F. (b) Pollakuria due to Urachal Abscesses in two Heifers.

  Journal of the American Veterinary Medical Association 184 (8): 984-986, 1984.
- Tzipori S. The Relative Importance of Enteric Pathogens Affecting Neonates of Domestic Animals.

  \*\*Advances in Veterinary Science, 29: 103-206, 1985.\*\*
- Urquhart J. Chapter 5: Cattle, pp. 78-113.

  In Animals on the Farm: Their History from the Earliest Times to the Present Day, 1983.

McDonald & Co, Maxwell House, 74 Worship st, London EC2A 2EN.

van der Mei J., van den Ingh T.S.G.M. Lung and Pleural Lesions of Veal Calves at Slaughter and their Relationship with Carcass Weight.

The veterinary Quarterly, 9 (3): 203-207, 1987.

Walter J.B., and Israel M.S. Chapter 8: The Body's Response to Infection: Spread by the Bloodstream, pp. 99-100.

In General pathology, 4th edition, 1975.

Harper & Rowe, New York & Evanston.

Wray C., and Morris J.A. Aspects of Colibacillosis in Farm Animals. *Journal of Hygiene, Cambridge*, <u>95</u>: 577-593, 1985.

#### Woolcock J.B.

- (a) Chapter 10: Normal bacterial flora- intestinal tract (1), pp. 102-104.
- (b) Chapter 16: Immune mechanisms in the intestinal tract, pp. 158-160.
   In Bacterial Infection and Immunity in Domestic Animals, 1979.
   Elsevier Scientific Publishing co, 335 Jan van Galenstraat, P.O. Box 211, 1000AE Amsterdam, The Netherlands.

Yates W. D.G. Respiratory System: Enzootic Pneumonia, pp. 99-100.
In Special Veterinary Pathology. R.G. Thomson (editor) 1988.
B.C. Decker Inc, 3228 South Service Road, Burlington, Ontario L7N 3H8, U.S.A.