

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.

INTRAPERITONEAL NUTRITION IN DOGS: A POSSIBLE
ALTERNATIVE ROUTE FOR NUTRITIONAL SUPPORT

by

TODD R. HALSEY

A thesis submitted in partial fulfilment of the
requirements for the degree of

MASTER OF VETERINARY SCIENCE

AT

MASSEY UNIVERSITY

1999

The author would like to dedicate this thesis to the faithful and loving dogs that contributed to the many important findings of this study.

ABSTRACT

Prolonged protein-energy malnutrition in dogs and other species has serious and wide-ranging adverse effects on organ systems. Separating the effects of poor nutritional status from those of the underlying disease mechanism is difficult and has made documenting a significant improvement in the long-term survival of patients frustrating. Despite these problems, nutritional support continues to be an important component of the treatment in critically ill or injured patients. In Chapter 1 the consequences of prolonged malnutrition throughout the body are discussed and the methods of providing nutritional support assessed. For patients who have a functioning gastrointestinal tract (GIT), nutritional support should be provided so that as much of the GIT as possible is used. In companion animals, enteral nutrition can be provided in various ways, ranging from forced oral feeding to an indwelling jejunostomy tube. Enteral nutritional support is a more physiological route for nutrient absorption, is less likely to result in serious adverse effects and is cheaper than parenteral nutrition. However, there are circumstances under which the GIT cannot be utilised as the primary route for nutritional support. This prompted the development of intravenous parenteral nutrition. Administration of total daily caloric requirements necessitates the use of a central venous line and a continuous 18 to 24 hour infusion. Unfamiliarity with central venous catheters, expensive and the inability to provide 24 hour monitoring for critically ill patients has precluded the use of intravenous parenteral nutrition in many veterinary hospitals. For these reasons alternative routes of nutritional support have been examined.

Chapter 2 examines the work achieved over the past 20 years by workers who have investigated the peritoneal cavity as an alternative route for parenteral nutritional support. The peritoneum is capable of absorbing electrolytes, dextrose, complex carbohydrates, amino acids, intact plasma proteins, lipids and particulate matter. Previous studies using experimental animal models have demonstrated that intraperitoneal nutritional support is feasible.

The pilot study (Chapter 3) in this experimental series examined the physical, haematological, biochemical and peritoneal cytological response in dogs receiving a total nutrient admixture (TNA) comprised of dextrose, amino acids and a lipid emulsion administered into the peritoneal cavity by a repeat puncture technique. This study identified a number of significant adverse effects associated with intraperitoneal nutrition (IPN) when using a TNA given in sufficient quantities to meet 100% of daily energy requirements (RER). Acute non-septic peritonitis, hypoalbuminaemia, mild anaemia, electrolyte and glucose derangements, and sudden fluid shifts from the vascular space into the peritoneal cavity were the problems recognised.

The study reported in Chapter 4 was undertaken to investigate the cause of the marked peritoneal inflammatory response and to pursue possible explanations for the clinicopathological changes that occurred in the pilot study. This was achieved by administering the components of the TNA as individual nutrients and comparing the peritoneal response over a 5 day period. This study demonstrated that the lipid component of the TNA was responsible for the majority of the peritoneal inflammation seen in the pilot study, causing a 13 fold greater increase in peritoneal total white cell count (TWCC) compared to a 5% amino acid solution and a 10% dextrose solution. Although there was a significant increase in peritoneal TWCC in dogs receiving the lipid emulsion, there were no signs supportive of a clinically significant peritonitis at the dose administered. The mild anaemia, hypoproteinaemia (particularly hypoalbuminaemia) and electrolyte disturbances noted in the pilot study were again seen in the study described in this chapter.

Although well tolerated by the peritoneal cavity, 10% dextrose in the volume administered in Chapter 4 failed to supply enough calories on a daily basis to make this route of nutritional support feasible. It was decided to try and increase the percentage of daily caloric requirements supplied by using a dextrose polymer. This nutrient solution allowed more calories to be provided for a given osmolality without the risk of fluid shifts into the peritoneal cavity because of its isotonic nature. The study presented in Chapter 5 identified that a 21.5% dextrose polymer solution caused an initial significant increase

in peritoneal TWCC, which then declined to near baseline concentrations by the end of the study.

It was concluded that twenty percent of resting energy requirements can be safely given to clinically healthy dogs in the form of a 10% dextrose solution, 5% amino acid solution, 10% lipid emulsion and a 21.5% dextrose polymer solution via a repeat abdominal puncture technique. Further studies are required before this form of nutritional support can be widely recommended to the veterinary profession.

ACKNOWLEDGEMENTS

The author wishes to thank McGaw BioMed Ltd. for their generous support of this project. In particular I would like to acknowledge Mr Melville Killip whose willingness to help in the formulation and production of the nutrient solutions was surpassed only by his attention to detail.

I would like to thank Ms Roz Power and Ms Sheryl Bayliss for their invaluable help with the processing of the clinical laboratory specimens and the many hours of interrupted work they endured when I muscled-in on the use of their microscope. Dr Phillip Clark is acknowledged for his expert help and critiquing of the interpretation of the diagnostic peritoneal lavage cell morphology and his help with ensuring the photomicrographs in this thesis were of the highest quality.

Mr Duncan Hedderley is thanked for performing the statistical analysis on the data and the creation of the graphs throughout the thesis.

To my three supervisors, Professor Grant Guilford, Roz Machon and Sandra Forsyth, I extend my deepest gratitude for your painstaking review of this manuscript and the endless positive and encouraging suggestions in writing this thesis despite suffering from several episodes of "apostrophe rage".

To my wife Lucy and our darling daughter Emma, thank you for your patience and undying love during those many hours spent locked away in the study.

TABLE OF CONTENTS	Page
Acknowledgements.	vi
Table of Contents.	vii
List of Tables.	x
List of Figures.	xii
 Chapter 1.	 1
□ Introduction.	2
□ Consequences of Malnutrition.	4
□ Protein-energy Malnutrition.	4
□ Micronutrient Deficiencies.	7
□ Methods of Providing Nutritional Support.	9
Enteral Nutrition.	9
Intravenous Parenteral Nutrition.	15
Components Used in Parenteral Nutrition.	18
Complications of Parenteral Nutrition.	25
 Chapter 2.	 36
□ Introduction.	37
□ Peritoneal Anatomy.	38
□ Peritoneal Physiology.	44
□ History of Intraperitoneal Nutrition.	45
□ Absorption of Carbohydrates from the Peritoneal Cavity.	46
□ Potential Complications of Rapid Glucose Absorption.	48
□ Absorption of Protein from the Peritoneal Cavity.	49
□ Absorption of Lipid from the Peritoneal Cavity.	50
□ Absorption of Water and Electrolytes from the Peritoneal Cavity.	51
□ Absorption of Total Nutrient Admixtures from the Peritoneal Cavity.	52
□ Histopathology of Abdominal Organs and the Peritoneum following Intraperitoneal Nutrition.	54
□ Complications Associated with Intraperitoneal Nutrition.	55
□ Summary of Intraperitoneal Nutrition.	59

Chapter 3.	61
□ Pilot Study of Intraperitoneal Nutrition in Dogs Using a Complete Nutritional Admixture via a Repeat Abdominal Puncture Technique.	62
Introduction.	62
Objectives.	62
Materials and Methods.	63
Results.	68
Discussion.	79
Summary and Conclusions.	83
Chapter 4.	85
□ A Comparison of Peritoneal Cytology in Dogs Given 10% dextrose, 5% Amino acids or a 10% Lipid Emulsion into the Peritoneal Cavity by Repeat Abdominal Puncture.	86
Introduction.	86
Objectives.	86
Materials and Methods.	87
Results.	90
Discussion.	102
Summary and Conclusions.	105
Chapter 5.	108
□ A Comparison of Peritoneal Cytology in Dogs Given 0.9% Sodium Chloride, 10% Dextrose or a 21.5% Dextrose Polymer Solution into the Peritoneal Cavity by Repeat Abdominal Puncture.	109
Introduction.	109
Objectives.	110
Materials and Methods.	110
Results.	113
Discussion.	128
Summary and Conclusions.	131

Chapter 6.	133
□ Summary and Conclusions.	134
□ Future Developments.	137
References.	138

LIST OF TABLES

Number	Page
1-1. Commercially available glucose preparations.	19
1-2. Properties of catheters for intravenous use.	27
2-1. Factors known to influence the spread and absorption of particulate matter and solutes within and from the peritoneal cavity of dogs.	45
3-1. Volume of nutritional solution administered to Dog 1 and Dog 2 on each day.	64
3-2. Schematic representation of the planned experimental protocol.	67
3-3. Changes in bodyweight throughout the study.	68
3-4. Comparison of the Day 1, 3, 5 and 7 values for the complete blood count in Dog 1 and Dog 2.	72
3-5. Comparison of the Day 1, 3, 5 and 7 values for the serum chemistry and electrolyte data in Dog 1 and Dog 2.	74
3-6. Comparison of the Day 1, 3, 5 and 7 values for the urinalysis results in Dog 1 and Dog 2.	75
3-7. Total white cell count and differential of the diagnostic peritoneal lavage fluid in Dog 1 and Dog 2.	76
4-1. Volume of nutrient solution infused in each group on Days 1 - 4.	88
4-2. Schematic representation of the experimental protocol.	90
4-3. Day 1 versus Day 5 bodyweights in the dextrose, amino acid and lipid groups.	91
4-4. Day 1 versus Day 5 values for the complete blood count in the dextrose, amino acid and lipid groups.	94
4-5. Day 1 versus Day 5 values for the serum chemistry panel with electrolytes in the dextrose, amino acid and lipid groups.	95
4-6. Day 1 versus Day 5 values for the DPL fluid total white cell count and differential in the dextrose, amino acid and lipid groups.	96
5-1. Volume of nutrients infused in each group on Days 1 - 4.	111
5-2. Schematic representation of the experimental protocol.	112
5-3. Day 1 versus Day 5 bodyweights in the saline, dextrose and dextrose polymer groups.	114

5.4. Day 1 versus Day 5 values for the complete blood count in the saline, dextrose and dextrose polymer groups.	120
5.5. Day 1 versus Day 5 values for the serum chemistry panel and electrolytes in the saline, dextrose and dextrose polymer groups.	121
5.6. Day 1 versus Day 5 values for the DPL fluid in the saline, dextrose and dextrose polymer groups.	122
6.1. Comparison of the Day 1 and Day 5 peritoneal total white cell count of the pilot study, second study and third study.	136

LIST OF FIGURES

Number	Page
1-1. Percutaneous gastrostomy tube associated with septic peritonitis in a dog.	12
1-2. Perforation of the jejunum by migration of a jejunostomy tube in a dog.	14
2-1. Human peritoneal mesothelium.	39
2-2. Human mesothelial cell and microvillus.	40
2-3. Appearance of peritoneal stomata at different anatomic sites in the rat.	42
3-1. Total nutrient admixture bag prior to mixing the lipid and dextrose / amino acid components.	64
3-2. 12-G over-the-needle catheter used for the diagnostic peritoneal lavage and nutrient infusion.	65
3-3. Sequential insertion of the over-the-needle catheter for diagnostic peritoneal lavage and nutrient administration.	66
3-4. Chest radiographs of Dog 1 on Day 7.	71
3-5. Cytological appearance of the diagnostic peritoneal lavage fluid on Day 3 of the study.	77
3-6. Lipid-laden mononuclear cells and neutrophils seen in the DPL fluid on Day 7.	77
3-7. Comparison of the total white cell count, neutrophil count and large mononuclear cell count on Day 1, 3, 5 and 7 in Dog 1.	78
3-8. Comparison of the total white cell count, neutrophil count and large mononuclear cell count on Day 1, 3, 5 and 7 in Dog 2.	78
3-9. Abdominal wrap bandage to minimise subcutaneous leakage of nutrient infusion from the peritoneal cavity.	80
4-1. Diagnostic peritoneal lavage fluid.	97
4-2. Mean peritoneal total white cell count \pm SEM vs. time graph in the dextrose, amino acid and lipid groups.	98
4-3. Mean peritoneal total white cell count \pm $\frac{1}{2}$ LSD bars vs. time graph in the dextrose, amino acid and lipid groups.	98
4-4. Mean peritoneal neutrophil count \pm SEM vs. time graph in the dextrose, amino acid and lipid groups.	99
4-5. Mean peritoneal neutrophil count \pm $\frac{1}{2}$ LSD bars vs. time graph in the dextrose, amino acid and lipid groups.	99
4-6. Mean peritoneal large mononuclear cell count \pm SEM vs. time graph in the dextrose, amino acid and lipid groups.	100

4-7. Mean peritoneal large mononuclear cell count \pm $\frac{1}{2}$ LSD bars vs. time graph in the dextrose, amino acid and lipid groups.	100
4-8. Baseline (Day 1) DPL cytology.	101
4-9. Mature hypersegmented polymorph neutrophils.	101
4-10. Vacuolated large mononuclear cells, lymphocytes and neutrophils in the DPL fluid from the lipid group.	102
5-1. Mean heart rate \pm $\frac{1}{2}$ LSD bars vs. time graph in the saline, dextrose and dextrose polymer groups.	115
5-2. Mean body temperature \pm $\frac{1}{2}$ LSD bars vs. time graph in the saline, dextrose and dextrose polymer groups.	115
5-3. Mean peritoneal total white cell count \pm SEM vs. time graph in the saline, dextrose and dextrose polymer groups.	123
5-4. Mean peritoneal total white cell count \pm $\frac{1}{2}$ LSD bars vs. time graph in the saline, dextrose and dextrose polymer groups.	123
5-5. Mean peritoneal neutrophil count \pm SEM vs. time graph in the saline, dextrose and dextrose polymer groups.	124
5-6. Mean peritoneal neutrophil count \pm $\frac{1}{2}$ LSD bars vs. time graph in the saline, dextrose and dextrose polymer groups.	124
5-7. Mean peritoneal large mononuclear cell count \pm SEM vs. time graph in the saline, dextrose and dextrose polymer groups.	125
5-8. Mean peritoneal large mononuclear cell count \pm $\frac{1}{2}$ LSD bars vs. time graph in the saline, dextrose and dextrose polymer groups.	125
5-9. Baseline (Day 1) diagnostic peritoneal lavage fluid cytology.	126
5-10. A group of large mononuclear cells surrounded by several mature, non-degenerate, hypersegmented polymorph neutrophils.	127
5-11. Diagnostic peritoneal lavage fluid: Large mononuclear cells in the dog receiving the dextrose polymer.	127