



University  
of Glasgow

<https://theses.gla.ac.uk/>

Theses Digitisation:

<https://www.gla.ac.uk/myglasgow/research/enlighten/theses/digitisation/>

This is a digitised version of the original print thesis.

Copyright and moral rights for this work are retained by the author

A copy can be downloaded for personal non-commercial research or study,  
without prior permission or charge

This work cannot be reproduced or quoted extensively from without first  
obtaining permission in writing from the author

The content must not be changed in any way or sold commercially in any  
format or medium without the formal permission of the author

When referring to this work, full bibliographic details including the author,  
title, awarding institution and date of the thesis must be given

Enlighten: Theses

<https://theses.gla.ac.uk/>  
[research-enlighten@glasgow.ac.uk](mailto:research-enlighten@glasgow.ac.uk)

PARENTERAL FLUID THERAPY STUDIES  
IN THE DOG

Alexander McKechnie Clark, B.V.M.S., M.R.C.V.S..

A thesis submitted for the degree of  
Doctor of Philosophy to the University of  
Glasgow, based upon research carried out  
in the Department of Veterinary Surgery, in  
the Faculty of Veterinary Medicine.

May 1978



ProQuest Number: 10662263

All rights reserved

INFORMATION TO ALL USERS

The quality of this reproduction is dependent upon the quality of the copy submitted.

In the unlikely event that the author did not send a complete manuscript and there are missing pages, these will be noted. Also, if material had to be removed, a note will indicate the deletion.



ProQuest 10662263

Published by ProQuest LLC (2017). Copyright of the Dissertation is held by the Author.

All rights reserved.

This work is protected against unauthorized copying under Title 17, United States Code  
Microform Edition © ProQuest LLC.

ProQuest LLC.  
789 East Eisenhower Parkway  
P.O. Box 1346  
Ann Arbor, MI 48106 – 1346

Dedication

To

HONEY, PENNY and ROBBIE

{three dogs whose recovery prompted this study}

## CONTENTS

	Page
Acknowledgements	1
Summary	2
List of figures	4
Glossary and List of abbreviations	5
INTRODUCTION	7

## PART 1 - EXPERIMENTAL

Section 1.	Venous Pressure Study	12
	Materials and Methods	27
	Results	34
	Discussion	42
Section 2.	Experimental Study	47
	Materials and Methods	68
	Results	74
	Discussion	86

## PART 2 - CLINICAL

Section 1.	Historical Background	104
	Survey of Fluid Therapy Usage in Veterinary Practice	112
Section 2.	Body Fluid Physiology	124
	Pathophysiology	140
	Clinical Examination and Assessment	161
	Ancillary Aids to Diagnosis of Fluid Imbalance	172
	Parenteral Fluids /	

Parenteral Fluids	179
Treatment	193
Section 3. Study of Clinical Cases	202
Materials and Methods	204
Results	220
Discussion	281
<u>PART 3 - GENERAL DISCUSSION</u>	282
Bibliography	295
Appendices	326

## ACKNOWLEDGEMENTS

I would like to record my sincere thanks to Professor Donald D. Lawson for his time and advice in the supervision of this study. I am indebted to those members of the Surgery Department of the University of Glasgow Veterinary School who were involved in the experimental and clinical studies.

Many other departments have helped in different ways and I would like to thank Professor T. A. Douglas and the staff of the Clinical Biochemistry Department for their untiring assistance in the analysis of blood samples. Thanks are also due to the Haematology Laboratory staff for their cooperation.

For the study of overinfusion in the dog, I am grateful to Dr. I. McA. Ledingham of the Western Infirmary, Glasgow for his advice, and to Dr. A.M. Harper for the use of the Wellcome Surgical Research Institute and the cooperation of his staff.

I am most grateful to the animal nurses of the Glasgow University Veterinary School for their unlimited cooperation in the treatment and investigation of the clinical cases. I would also thank Mr. A Finnie and the Photography Department staff for their help.

The manuscript was read and scrutinised by my parents and Miss A. Ritchie to whom I am exceedingly grateful for their time and effort. Thanks are due to Mrs. H Rodrick for the advice regarding the typing of the manuscript and for typing some of the tables of results.

Finally, I should like to express my gratitude to the Horserace Betting Levy Board for their financial assistance without which this study would not have been possible.

## SUMMARY

The subject of this thesis has been an investigation of body fluid imbalances arising from spontaneous disease that occur in the dog. The range of diseases that can give rise to body fluid balance disturbance is extensive and examples are intestinal foreign body, pyometritis and hypovolaemic circulatory failure.

A detailed study of the benefits of both central venous pressure and peripheral venous pressure measurements gave an indication that these had little if any value in clinical studies in the dog. One parameter that proved to be valuable was the monitoring of the output of urine, since it was found that once adequate replacement infusion had been administered, the urinary output returned to normal.

An experimental study of the effects of different rates of fluid infusion on vital functions such as urine output, venous pressures and respiratory gas exchange was conducted and the effects of overinfusion were observed in the dog. This study indicates the rate and volume of fluid infusion that may be employed in the dog and reports on the value of the use of urine output as a practical method of monitoring the adequacy of infusion and prevention of overinfusion.

A preliminary survey of the use made by general practitioners of parenteral fluid therapy revealed that although most claimed to practise such treatment, few cases were treated.

A large series of clinical cases were investigated to determine their fluid and electrolyte status and to institute care and therapy. The results have been quantified where possible and a considerable/

considerable improvement in both survival rate and in the rate of recovery of cases was observed.

The methods of fluid administration in the dog were closely studied and successful designs of equipment selected and recommended for routine use. A list of fluids for infusion was also discussed.

This study was intentionally clinical since it was designed to promote the use of parenteral fluid therapy in general veterinary practice.

LIST OF FIGURES

Figure

1. CVP PVP Pressure Recording
2. Radiograph - CVP line
3. Venous Pressure Manometer
4. CVP PVP Measurements
5. CVP PVP Measurements
6. PVP Measurements
7. Experimental Dog - Pressure Recordings
8. Radiograph - Pulmonary Oedema
9. Radiograph - Intestinal foreign body
10. Radiograph - " " "
11. Radiograph - " " "
12. Radiograph - " " "
13. Radiograph - " " "
14. Radiograph - " " "
15. Radiograph - Pyometra
16. Radiograph - "



GLOSSARY AND LIST OF ABBREVIATIONS

Kilogram	Accepted unit of weight
Litre	Accepted unit of volume
Mole	A mole of a substance comprises a gram weight of the substance equal to its molecular weight
Millimole (mmol.)	Represents one-thousandth of a mole
Osmol	This represents the osmotic pressure or force exerted in a solution by a mole of a substance
S.I.Units	The metric system now widely adopted, (Douglas 1977), and the units are laid down by the Systeme Internationale d'Unites as approved by the Conference General des Poids et Mesures, (1960).
HECAscore	A clinical scoring system based on History, Examination, Clinical Assessment and the Ancillary aids.
A.C.D.	Acid citrate dextrose
B.S.A.V.A.	British Small Animal Veterinary Association
C.R.T.	Capillary refill time
C.V.P.	Central venous pressure
E.C.F.	Extracellular fluid
E.D.T.A.	Ethylene diamine tetraacetic acid
I.C.F.	Intracellular fluid
I.P.P.V.	Intermittent positive pressure ventilation
PaO <sub>2</sub>	Partial pressure of oxygen in arterial blood
PaCO <sub>2</sub>	Partial pressure of carbon dioxide in arterial blood
P.C.V.	Packed cell volume
P.V.P./	

P.V.P.	Peripheral venous pressure
S.W.G.	Standard wire guage
T.B.W.	Total body water
T.S.P.	Total serum protein
W.B.C.	White blood cells
E.U.A.	Examination under anaesthesia

## INTRODUCTION

## INTRODUCTION

There is an increasing awareness within the veterinary profession that parenteral fluid therapy is a valuable method of treatment and support for patients which have suffered large losses of body fluids. In the prevailing economic environment of veterinary medicine, intensive therapy is largely limited to small animals such as the cat and the dog, and only in certain clear cut circumstances would it be employed in the horse, ox and sheep. Ethically, all animals should receive the best possible treatment and no patient should be denied the opportunity of therapy now readily available, but not always routinely and actively administered.

After considering many aspects of knowledge derived from veterinary and medical sources, most in fact of the latter using experimental cats and dogs, it was decided to investigate fluid therapy in dogs as a gross subject, with particular interest in parenteral fluid administration and the prevention or early detection of overinfusion. It was intentionally a clinical study of fluid therapy considering the advantages and disadvantages, and methods of administration and monitoring, with a simultaneous experimental study to evaluate the prevention of overinfusion. The presentation of the thesis is in two parts, although they combine to form a complete investigation and assessment of parenteral fluid therapy in the dog.

The experimental studies are considered first. Peripheral venous pressure was measured in two groups of dogs, one conscious and the other undergoing surgery. It was found to be an interesting area of investigation/

investigation and the two groups were expanded to include a comparative study of peripheral and central venous pressure. The results of this study are discussed and the methods of peripheral and central venous pressure measurement described.

Central venous pressure has been advocated as a reliable measurement for the prevention and detection of overinfusion. Since information from clinical cases was limited by equipment availability and Home Office regulations regarding experimental studies in the dog, a license was obtained to permit further experimental work into venous pressures, with a view to discovering a practical and economical method of preventing accidental overinfusion. An experimental model was designed using the dog. Infusions of commercially produced fluids were given intravenously at fixed rates under anaesthesia and various parameters, including venous pressures, were monitored. The maximum infusions were given in excess of those used clinically in the hope that safe rates for the intravenous administration of certain fluids could be derived from the results.

The second part of the thesis is concerned with the clinical aspects of parenteral fluid therapy. Many veterinary technologists have defined the daily fluid and electrolyte requirements of various species, and this information is of import when there is presented a dehydrated, vomiting patient which should receive parenteral fluids to replace losses and promote recovery. The problems then arise - which fluid is needed ? - how much is required ? - which is the best route of administration ? - and how can the progress of treatment be monitored ? It is hoped that answers to these questions are contained within the text of/

of the clinical study since from the outset the intention was that any results and recommendations should be directly advantageous to the general veterinary practitioner. The foundation of this study was the treatment of dogs and cats suffering from body fluid imbalances. The major part of the work was performed on cases admitted to the University of Glasgow Veterinary School where the facilities for examination, diagnosis and treatment together with the ancillary aids of radiography, blood analysis and pathology were routinely accessible. A survey was instituted as a preliminary study in early 1976 to obtain information from general practitioners regarding parenteral fluid administration in small animal practice. The results are contained in the appendices and are discussed in the text. It was noted at this time that in the veterinary literature fluid therapy had been largely neglected except in some general textbooks of surgery and a few sophisticated small volumes. A short section deals with the historical background of parenteral fluid therapy derived mostly from experimental studies performed in animals and humans.

There is discussion of the normal physiology of dogs outlining the composition of body fluids, the normal requirements for survival, the control mechanisms affecting these body fluids and an examination of the function of water and electrolytes in the body. Thereafter the common body fluid abnormalities found in small animals are discussed. Following consideration of the theory of fluid therapy, the clinical applications are outlined and discussed. There is a detailed assessment of body fluid imbalances, a description of the common clinical signs seen most frequently in dogs and an indication of which body fluid losses are most relevant/

relevant. The use of ancillary aids to diagnosis in veterinary surgeries is in general increasing, with radiography and a limited selection of blood analysis tests being routinely available. These aids are described and the advantages and disadvantages of their use outlined.

The final stage is the selection of treatment, and a short section is devoted to the types of fluid required for the replacement and maintenance types of therapy. The adjuncts to fluids for therapy such as antibiotics and vitamins are briefly discussed.

A review of cases presented to the Surgery Department of the University of Glasgow Veterinary School was made and two common conditions were selected for fuller investigation - intestinal foreign body and pyometritis. To permit a comparison of the advantages and disadvantages of fluid therapy, cases of the two conditions were divided into two clinically chosen groups, one to receive parenteral fluid therapy and the other to be left untreated. The dogs underwent surgery to either remove the foreign body from the intestine or to perform a total ovarohysterectomy, and to allow a comparison of the cases pre-operatively and post-operatively, a scoring system, the HECAscore, was devised. This system and the results are discussed.

A section of the appendices describes and illustrates many types of equipment used in this study from cannulae to infusion sets and monitoring aids. An investigation using the various devices was carried out during the course of the clinical study. Monitoring and recording are important parts of intensive therapy and are considered within the investigation into equipment.

It is hoped that the results of this study will be considered in context providing the veterinary practitioner in small animal practice/

practice with a further weapon in the treatment of patients which have suffered large losses of body fluids.



## PART 1 - EXPERIMENTAL

## SECTION 1

### VENOUS PRESSURE STUDY

## VENOUS PRESSURE STUDY

### INTRODUCTION

In 1628 Harvey described the basic circulation in mammals, determining from the arm veins that blood flowed within vessels driven by a pump. The replacement of fluid within this recognised body circulation awaited Sir Christopher Wren who performed intravenous injections in 1656 and was succeeded by Lower in 1660 who successfully administered blood transfusions to humans. This was the first stage of parenteral fluid therapy which remained suppressed as a subject due to religious scruples until the nineteenth and twentieth centuries.

With the advent of fluid therapy by routes other than oral and rectal, methods of assessing the blood volume have been sought by many technologists. Overinfusion became a problem when the replacement of blood volume and body fluid deficiencies was performed by the intravenous route and pulmonary oedema was a serious side effect. Many clinical methods have been used to detect overinfusion, the most commonly used one being Central Venous Pressure, (CVP).

Starling in 1910 cannulated the inferior vena cava of the dog in removed heart lung preparations supported in Ringer lactate solution to permit measurement of the venous pressure before the return of the blood to the heart. This measurement was the CVP. As venous pressure increases, cardiac output rises until a certain point when any further increase in venous pressure causes a fall in the cardiac output due to overdistension of the heart. To measure venous pressure, Starling used a water manometer, the method still used today to monitor CVP in cases undergoing intensive therapy./

therapy. It was stated that venous pressure could be used with reliability to detect pathological changes when infusing fluids, (Bedford and Wright 1924). These workers measured venous pressure in an arm vein, Peripheral Venous Pressure, (PVP), using a water manometer, having the arm at the same level as the right side of the heart. The pressures measured were consistently between 5 and 15 cm. of water, but it was found that low readings were unreliable.

If repeated readings with a water manometer from a CVP line were taken during the infusion of fluids, it was possible to assess the ability of the circulation to tolerate the increase in blood volume, (Caughey 1935). Patients with a poor heart action showed immediate increases in CVP when infused with fluids. Caughey felt that further work was necessary if any relationship between the venous pressure and the blood volume was to be established. Much of this work was confirmed in studies which showed that the venous pressure did rise with the rapid infusion of fluids intravenously (20 cc. per minute to a man), but despite increase in the blood volume at low infusion rates, the venous pressure did not alter, (Altschule and Gilligan 1938). When the CVP rose rapidly with infusion, it returned to normal on cessation of administration perhaps due to vasodilatation during the infusion.

It was noted that when infusing fluids to elderly or cardiac patients, that the increase in blood volume could cause pulmonary oedema, (Landis, Brown Fauteux and Wise 1945). These workers demonstrated that CVP rose in cardiac disturbances shortly before death and that cardiac tamponade increased the pressure. PVP was found to increase with the infusion of salt solutions, but it was thought that this might be due to tissue pressure on small veins and was not indicative of/

of increased auricular pressure, It was shown that increases in blood volume consistently caused rises in atrial pressure, but despite large increases in CVP, these were not indicative of heart failure, (Warren, Brandon, Weens and Stead 1948). The infusion rates in humans in this study varied from 32 to 77 ml. per minute using normal saline and human serum albumin. The CVP was measured with a water manometer. Similarly in dogs given large volumes of saline, there was generalised oedema but rarely dyspnoea. It was thought by Warren and colleagues that pulmonary oedema seen in animals and humans was due to predisposing factors such as occur in cardiac failure patients when input exceeded output. These workers concluded that increasing the blood volume and right atrial pressure caused little embarrassment to normal patients, but that pulmonary oedema could occur if the predisposing factors were operative.

It was demonstrated in surgical patients in shock that CVP decreased before the arterial blood pressure fell or the pulse rate increased as a result of vasoconstriction, (Pierce, Boyan and Masterton 1953). This fact was already known by Moon and colleagues in 1942. It was shown by Pierce and colleagues that venous pressure did not increase until a volume equivalent to that lost was replaced. The depth of anaesthesia in these surgical patients also varied the venous pressure, deep anaesthesia causing a fall in CVP. They concluded that CVP monitoring required experience.

The possibility of the continuous recording of venous pressure using the forearm was investigated, (Gauer and Sieker 1956). They used a water manometer, checking for pulse movements in time with respiration before taking readings, and found that the method was fairly straightforward, reliable and accurate for measuring trends of venous pressure. Numerous/

Numerous advantages over CVP were put forward because of the simplicity and relative lack of equipment. Continuous comparative CVP and PVP studies were performed by Gauer and Sieker showing identical trends in the venous pressures. The resting PVP varied from 3 to 13 cm. of water.

In the same year it was demonstrated that with moderate haemorrhage or infusion, pressures at all locations from the great veins to the left atrium changed in unison by approximately equivalent amounts, (Gauer, Henry and Sieker 1956). It was also demonstrated that the right ventricle anatomically separates the systemic venous system from the pulmonary circulation, imbalances in blood volume having so little effect on its activity from the point of pressure relationships, that the systemic veins and the left atrium could be considered part of the same functional unit. The pressures in the venous system, both atria and the pulmonary arterioles rose and fell in unison with moderate changes in blood volume.

The relationship between the CVP and the blood volume was investigated, and measurements of blood volume by dilution methods were found reliable if some inherent problems were accepted, (Hughes and MacGovern 1959). Dilution techniques depend on the ability of the heart, reliability of the single blood volume determination, over-transfusion, residual material held in the circulation and repeated venipuncture. It was decided by these two authors from their work with blood volume estimations that CVP was not an accurate guide to blood volume, but was a good indicator of possible circulatory overload with fluids. Peripheral venous pressure had similar problems, but the veins also showed properties of venopression shut down which rendered readings meaningless.

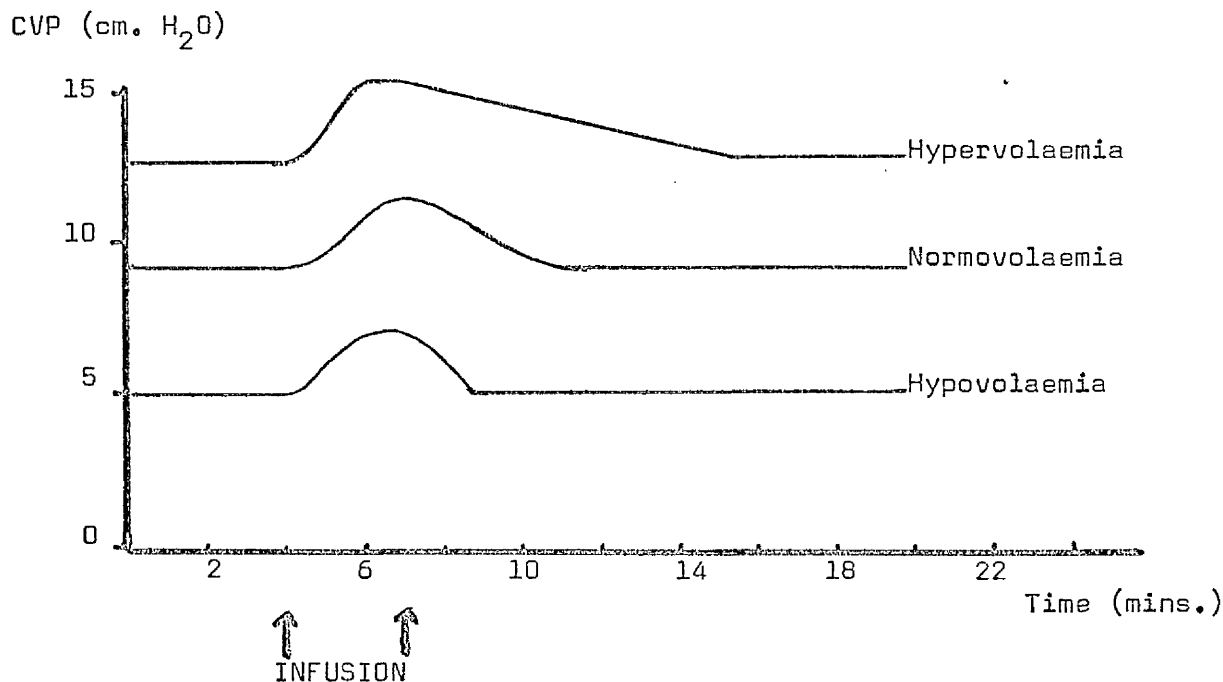
Investigation/

Investigation of the use of central venous pressure monitoring in optimal blood volume maintenance revealed that readings had to be viewed in proper perspective with the overall clinical situation. The trends of both CVP and the response to treatment were noted to be of greater significance than the actual level of any single determination, (Wilson, Grow, Demong, Prevedel and Owens 1962). The CVP served as a useful index of circulatory blood volume relative to cardiac pump capacity. This group of doctors also found that CVP was a useful measurement during circulatory failure or massive blood or fluid infusion, but they determined that the level at which pulmonary oedema was imminent was variable.

The value of CVP monitoring in oligoemic shock and as an indication of overload with fluids was reviewed, (McGowan and Walters 1963). It was demonstrated that CVP was reasonably accurate when treating shock patients and permitted the safe and rapid replacement of fluid. Monitoring CVP allowed one to estimate the degree of oligoemic circulatory failure, treat the imbalance and attempt to restore CVP to normal. CVP was also used to assess blood volume instead of other methods, since it was found that arterial blood pressure was inaccurate in shock patients due to vasoconstriction, hypotension and vasodilation. The paper concluded by stating that all patients being given fluids rapidly should have their CVP monitored regularly, the only disadvantage being with gasping respirations when readings were difficult to assess, and it was decided that CVP was an accurate method for the determination of blood volume.

CVP monitoring was accepted during the early 1960's by most medical technologists as a method of assessing the success or otherwise of fluid /

of fluid replacement. The use of CVP as an indicator of adequacy of transfusion was investigated, and it was found that the measurement was quick, easy and reliable if one accepted the readings in context, (Sykes 1963). A low venous pressure was indicative of hypovolaemia, but it should be remembered that the venous system can accommodate up to a 10 % change in blood volume due to venomotor tone, hence the changes in venous pressure. Sykes found that tone depended on various stimuli, and it was reduced by warmth, sleep, thiopentone, histamine and ganglion blockers. Tone was increased by skin stimulation, hyperventilation, I.P.P.V., catecholamines and cardiac failure. Emphasis was placed on the use of CVP measurement as a means of assessing venous return and cardiac output, and was performed by a rapid infusion of some 200 to 300 ml. of fluid in 1 to 3 minutes in a human. The response is shown below.





Sykes restated the need to zero the manometer at the manubrium sterni, and only accept readings if there was a respiratory fluctuation. The manometer and drip apparatus should be as near to the patient as possible to reduce resistance within the plastic tubing.

As much as one litre of blood can be removed from a man without a change in CVP and it was stated that the measurement of CVP was accurate in determining venous pressure return to the heart, but not blood volume, (Jones 1963). CVP was used successfully by this author to prevent overtransfusion, and although a gradual rise was accepted as normal, a sudden large increase in CVP might indicate the chance of pulmonary oedema.

CVP was monitored in shocked patients after it was found that arterial blood pressure was of no value in patients receiving treatment for hypovolaemia, whereas CVP was a reliable method of determining organ perfusion or the lack of it, (MacLean 1964). CVP was also claimed to be useful as an indicator of overtransfusion and pulmonary oedema. MacLean measured both arterial blood pressure and CVP in shocked patients and found that in some CVP rose above the acceptable normal with no change in arterial blood pressure. He stated later, (MacLean 1966), that any patient with a low CVP should receive fluids regardless of the clinical picture, since overtransfusion rarely occurred if the CVP was maintained below 15 cm. of water.

CVP was measured in shocked and dehydrated patients, and it was found that there was no relationship between blood volume and venous pressure, but that CVP was an indicator of the heart's ability to handle fluids. PVP was not as accurate as CVP due to venous constriction in shock and dehydration, (Weil, Shubin and Rosoff 1965). It was further demonstrated/

demonstrated that CVP was more accurate than PVP when determining the capability of the heart, and that venous pressure monitoring was accurate as a guide to hypovolaemia since the pressure varied directly with the circulating volume, (Borow, Aquilizen, Krausz and Stefanides 1965). Serial venous pressure measurements are more accurate than one reading, a fact stated by many authors and recommended by Borow and colleagues.

CVP was monitored regularly in shocked patients and it was found that it was accurate in the comparison of blood volume and cardiac capacity at any particular time. Its use was invaluable in preventing fluid overload, (Artz 1966).

In the latter half of the 1960's, some doubt was cast regarding the reliability of CVP monitoring in the prevention of body fluid overload, since it was found that large infusions of fluid caused interstitial oedema of the lungs without an increase in CVP, (Fieber and Jones 1966). A diuresis induced using 5% dextrose solution caused the CVP to fall whereas Ringer lactate caused the reverse. However when Ringer lactate was given to overinfusion level, CVP did not rise accordingly, there being only generalised oedema and diuresis. The hazard of interstitial pulmonary oedema was restated when it was noted that CVP did not increase during the infusion of crystalloids in some cases, and overloading of the pulmonary circulation with "wet lung" formation occurred, (Frank 1969). Frank found that CVP did rise when the blood volume was increased or cardiac function altered, but not when the interstitial spaces of the lung were overloaded. Chest radiography and the alveolar arterial oxygen gradient were reliable methods of pulmonary oedema detection.

These latter findings by Frank were at the time when in North America,

America, Shires et al had demonstrated the so-called " third space " or shift of the extracellular fluid in surgical patients. This hypothesis resulted in the infusion of massive quantities of crystalloids to cases undergoing surgery, the end result in many instances being pulmonary oedema, especially when cardiac action was impaired. In an article entitled " Moderation " it was stated that the replacement of body fluid losses should only restore normal body physiology, a process which could not be achieved by inundation. It was also stated that balanced salt solutions were only an adjunct to surgical trauma and not a replacement for blood loss, (Moore and Shires 1967),

The CVP was shown not to alter when animals were infused to twice their blood volume within one hour, (Berman and Spencer 1972). Rales were not often heard on auscultation, but in a study by Berman and Spencer chest radiography was performed at set intervals during the infusion and changes were noted before the clinical signs of overinfusion or an increased CVP were observed. The main findings in overinfusion were dyspnoea, lung oedema and arterial hypoxaemia.

The blood volume was determined using radio-isotope dilution techniques and compared with CVP in shocked patients given more than five litres of fluid, (Wilson, Sarver and Birks 1971). There was no correlation between CVP and blood volume, and there was no consistent correlation between the blood volume, CVP and the clinical impression with the fluid load administered. It was suggested that CVP alone could not be used to regulate fluid infusion in patients, and that many factors affected the CVP including anaesthesia, I.P.P.V. and lung disease.

It was found that CVP was a valid monitoring method to assess the/

the fluid needs if both ventricles were working efficiently, (James and Myers 1972). Unfortunately one death was recorded due to air embolus aspirated via the subclavian vein needle at insertion of the needle. These workers found that pulmonary artery pressure was a more accurate measurement in preventing overinfusion, since the right heart compensated and showed a normal CVP. This work has recently been confirmed, (Gilbertson 1978).

Another report stated that CVP was more useful in shocked patients than those with electrolyte imbalances, and that crystalloids caused interstitial oedema of the lungs without a CVP increase, (Dougan and Finlay 1973).

The jugular vein pressure was monitored rather than CVP because of the inherent difficulties in establishing a central line, and was found to be directly related to the CVP. It was suggested that the easier catheter insertion routine might make this method more acceptable, (Briscoe 1973). He also investigated PVP finding a difference of 7 cm. of water between CVP and PVP. The position of the body played an important part in the taking of readings.

The complications of CVP lines became more apparent when it was noted that the use of wide bore needles was more likely to cause air embolus aspiration than when narrow gauge tubes were used, (Ordway 1974). Two cases of atrial perforation by polyethylene CVP catheters were recorded, (Friedman and Jurgeleit 1968), and it was stated that the complications arising from the use of CVP monitoring lines, including air embolus and vena cava perforation, outweighed the advantages of the technique, (Adar, Mozes 1971 and Adar 1976).

The complications of CVP lines and the technique in general were/

were reviewed, and doubts as to the accuracy of CVP monitoring in any form of patient, shocked or otherwise were cast, MacGovern 1976).

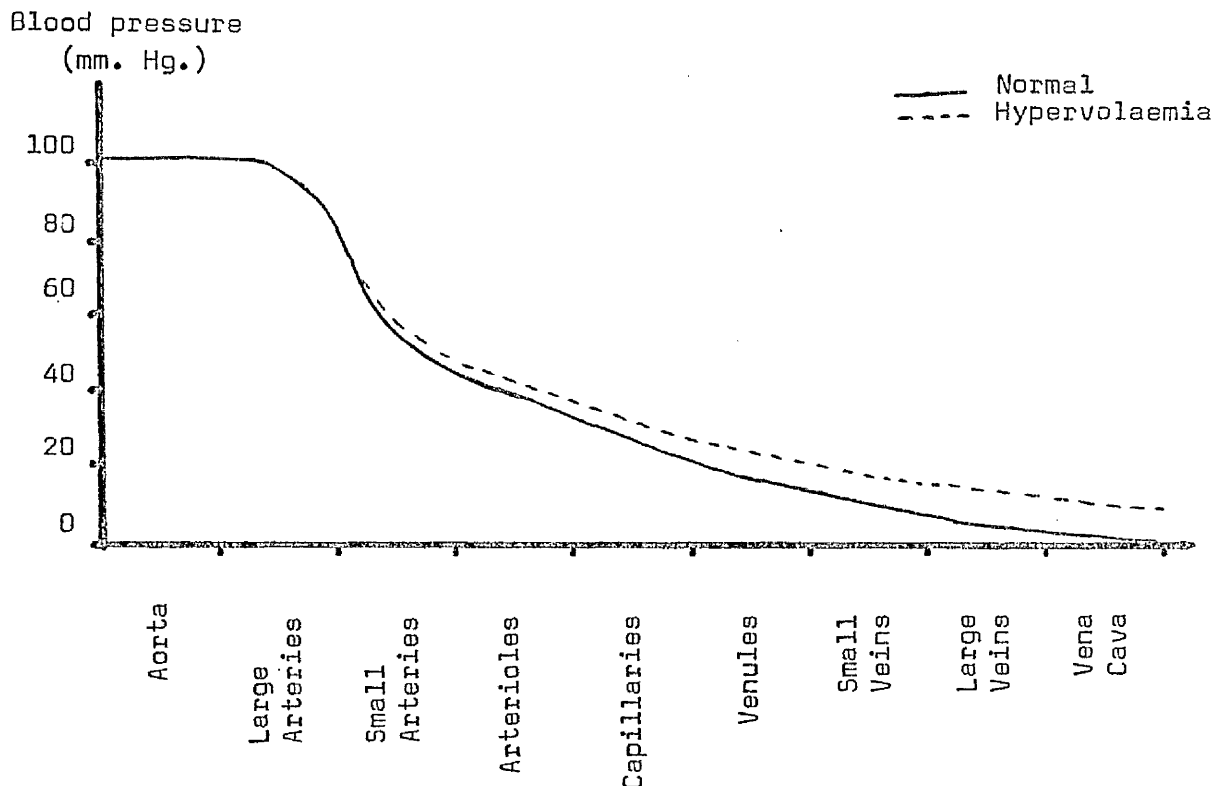
In the veterinary literature some authors have mentioned the use of CVP monitoring, and it has been recommended in cases receiving fluids to permit assessment of therapy and prevention of overinfusion, Hall 1967). It was also stated that the measurement of CVP had become an invaluable aid in estimating the fluid requirements of seriously ill patients, (Burrows 1976).

In modern medical units CVP monitoring is used routinely in most patients undergoing intensive therapy or major surgery, but doubts as to its efficiency and accuracy exist. In view of these reports, it was decided to further investigate the use of venous pressures in veterinary practice.

CVP measurements in the dog may be made via the external jugular vein using a wide bore needle through which a cannula is inserted to the level of the right atrium within the great veins, (Guyton 1961, Bell, Davidson and Scarborough 1963, Hall 1967, Burrows 1976). In my opinion this method can be difficult and this led to attempts to measure venous pressure at another body site. The common difficulties were isolation of the jugular vein in obese animals, particularly when recumbent, fractious animals where it was necessary to monitor CVP without sedation, the insertion of larger bore needles into relatively small jugular veins leading to haematoma formation and occasionally secondary infection, re-penetration of the jugular vein if the animal rotated its head, removal of the apparatus by the animal, the narrow bore of the measuring cannula permitted only minor infusions to be made, and the time required to establish a central line. The routine administration of fluids was via the cephalic vein in my trials, and it was decided to monitor venous pressure at this level rather than centrally.

## VENOUS PRESSURE

Venous pressure is that hydrostatic pressure existing within the vessels of the circulation, between the capillary beds and the right side of the heart, and which permits the flow of blood back to the pump and the systemic circulation. A pressure gradient necessarily exists between the venules of the peripheral tissues and the great veins within the thorax adjacent to the right atrium. Guyton (1961) states that the gradient appears to depend on the central venous pressure, that being the pressure within the great veins at the level of the right atrium, and this, CVP, affected the total venous system. As may be visualised in the following graph, an increase in pressure at the right atrium causes increased pressure throughout the venous system and capillary bed.



Therefore the pressure in the peripheral veins is dependent on CVP, and any factor affecting right atrial pressure will affect the total venous system pressure. When right atrial pressure increased, Guyton showed that blood was confined within the great veins until all the spaces were occupied before the PVP rose, which happened once the right atrial pressure had risen by 4 to 6 mm. of Hg.. This would seem to indicate that the measurement of PVP is not an accurate method of assessing CVP or blood volume, but that it might be useful in the prevention of overinfusion.

Bell, Davidson and Scarborough (1961), discuss venous pressure stating that a zero point should be established when measuring venous pressure with a water manometer since different postural positions markedly affect the readings. They suggest that zero point should be the level of the right atrium. One other consideration is that the pressure in the right atrium and great veins is not constant, varying with the cardiac and respiratory cycles. The flow of blood in the great veins at rest is continuous except near the heart, and variations in right atrial pressure usually pass back a short way into the great veins near the heart since they are not equipped with semi-lunar valves. This defines the venous pulse which is seen when recording CVP via a pressure transducer and a pen recorder, (Fig. 1 ). When monitoring CVP one is advised to ensure a respiratory fluctuation in the water manometer column before accepting a reading. This fluctuation occurs due to changes in pressure within the thorax when a subject breathes.

It is interesting to note that despite the fact that semi-lunar valves exist in the cephalic vein of the dog, both venous pulse and respiratory fluctuation may be recorded when taking PVP measurements, (Fig. 1 )/



Fig. 1 • Central and Peripheral Venous Pressure Recordings



Further investigation of PVP was performed to determine if it was a valid measurement with reference to CVP and if it alone could be used as a monitoring aid to prevent overinfusion.

CLINICAL EXPERIMENTAL STUDY OF CENTRAL AND PERIPHERAL VENCUS PRESSURE

The peripheral venous pressure and the central venous pressure were measured in conscious and anaesthetised dogs. The selected animals were approximately normovolaemic, having been starved pre-operatively for a period of not less than three hours and not greater than twelve. They had no clinically detectable abnormalities of the cardiovascular or respiratory systems, and were of random age, breed, weight and sex.

MATERIALS AND METHODS

The measurement of peripheral venous pressure was performed using a plastic over-the-needle cannula inserted into the mid-antebraial segment of the cephalic vein. This procedure was performed under aseptic conditions, the area of insertion being close clipped, scrubbed with " Savlon " and water and finally douced with a pevidine alcohol mixture. A light dressing was applied over the point of cannula insertion, and using a three-way stopcock attached to the cannula, fixation to the limb was achieved with adhesive tape. An administration set incorporating a three-way stopcock and graduated water manometer connected to a container of Ringer lactate was attached to the intravenous unit. To maintain cannula patency, 2000 I.U. of heparin was added to each litre of solution. The graduated manometer was filled with this solution prior to each reading, thus preventing the reflux of blood into the cannula and unit should the pressure have increased from one reading to the next. The Ringer lactate was infused at 0.5 ml, per kg, per hour controlled by a " dial-a-flo " flow regulator unit.

Central/

\* I.C.I. - Cetrinide

Central venous pressure was monitored by means of a long intravenous cannula inserted into the opposite cephalic vein and advanced into the anterior vena cava. The design used incorporated an introducer of the over-the-needle type of cannula, through which the measuring cannula was fed into the vein. Placement of the cannula was checked by the reading on the water manometer and by lateral chest radiography, (Fig 2 ). Once in position a plastic locking device ensured stability.

Prior to insertion, aseptic precautions were taken.

The cannula was connected to a graduated water manometer and a container of heparinised Ringer lactate, which was infused at a rate of 0,5 ml, per kilogram per hour.

Prior to the recording of measurements, both manometers were connected directly to the dog and allowed to stabilise, and readings were only taken when a distinct respiratory fluctuation was observed. The fluctuation ranged between 2 and 10 mm, of water, rising on inspiration and falling on expiration. Readings were always taken during the expiratory pause phase of the respiratory cycle.

The water manometer unit is illustrated, (Fig. 3 ).

Fig. 2. Central venous line check radiograph

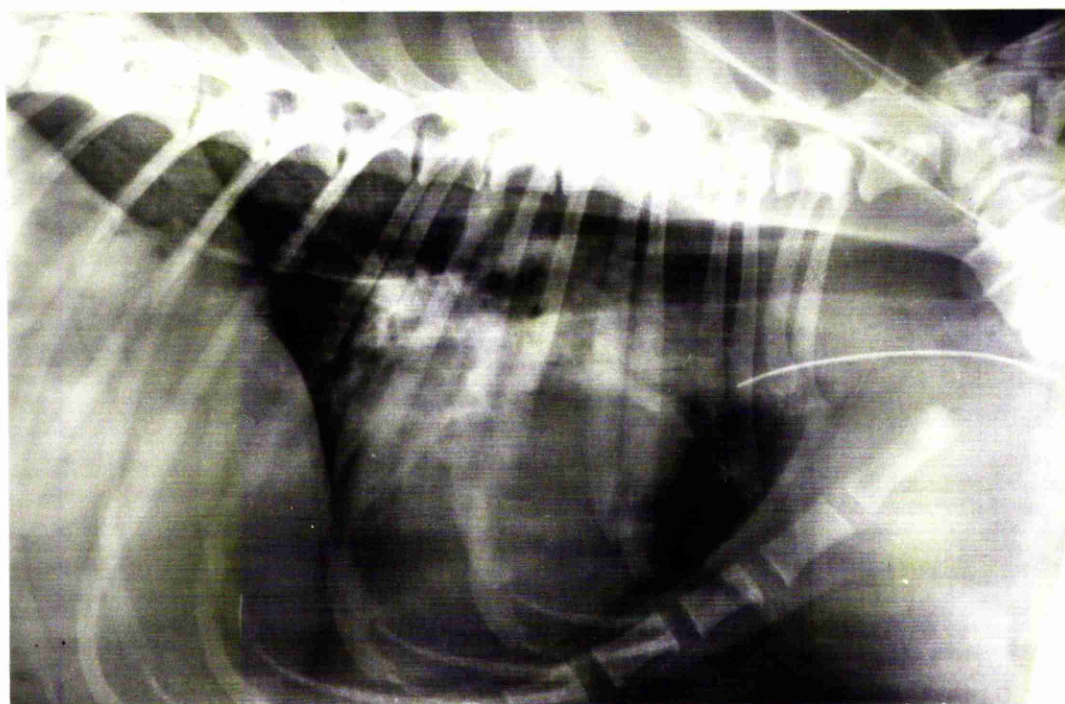
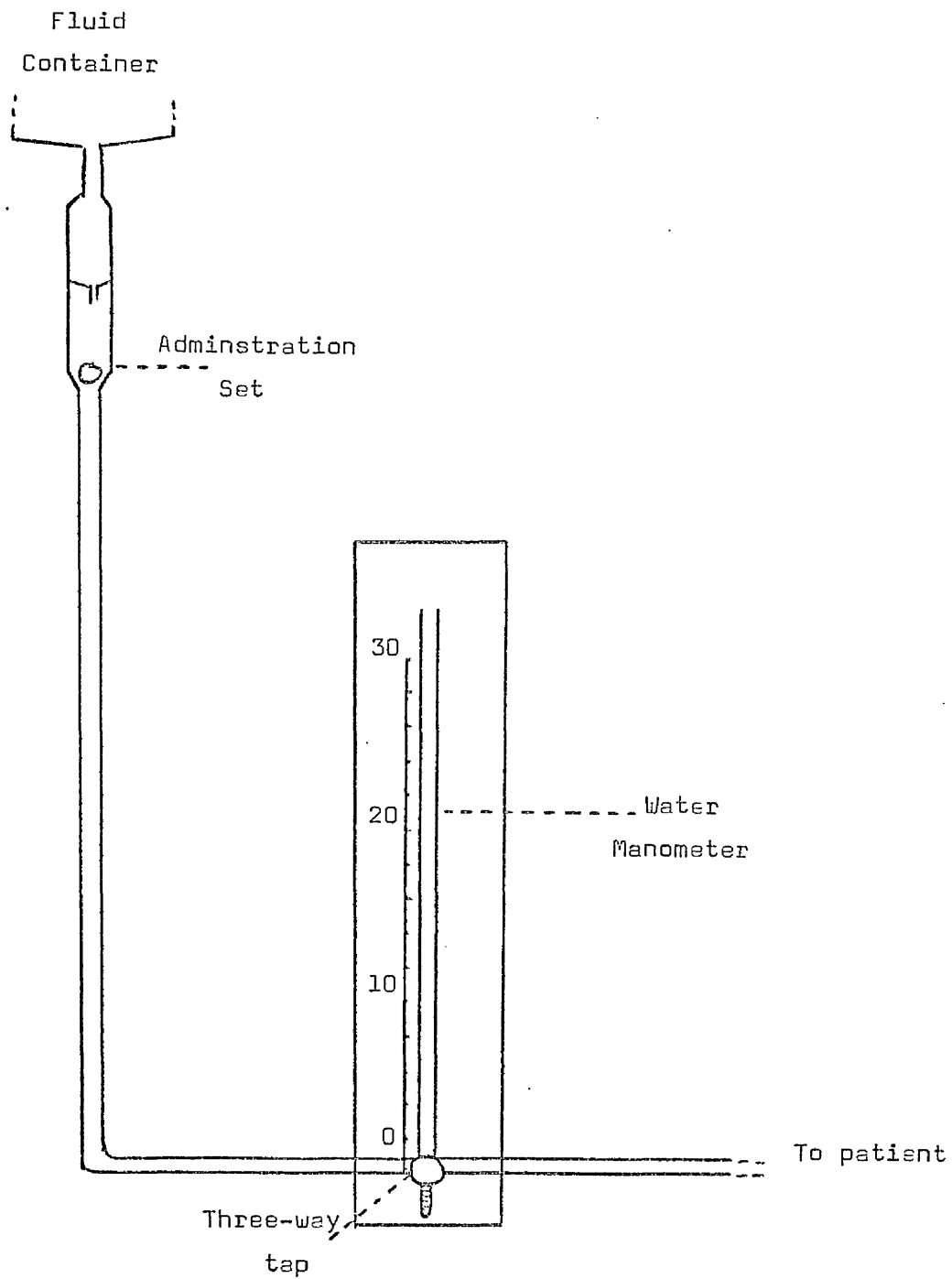


Fig. 3.



Graduated Water Manometer Unit

The postural position of the dog was carefully controlled, since any change in position was found to alter readings. The PVP changed dramatically when limbs were raised or lowered relative to the zero point on the manometer. For this reason set postural positions and relative zero points were established.

The zero point has been described as the level of the right atrium of the heart, but it was found more convenient to standardise this using set reference points determined by posture.

A. Standing, Dorsal recumbency and Sternal recumbency.

Zero point was the mid point of a dorso-ventral line from spine to sternum at the level of  $T_8$  and the xiphisternum.

B. Lateral recumbency.

Zero point was the medial line in a horizontal plane from the spinal column to the sternum, with the sternum elevated to be in line with the spine.

For the purposes of this study four postural positions existed.

1. Standing
2. Dorsal recumbency
3. Sternal recumbency
4. Lateral recumbency

1. Standing            all four legs were upright with the feet on a horizontal plane.

2./

2. Dorsal recumbency
3. Sternal recumbency                      both the forelegs were extended forward parallel to the flanks and parallel to the horizontal surface of the table. The hindlegs were extended behind parallel to the horizontal surface.
4. Lateral recumbency                      the sternum was elevated to a line horizontal with the spine and the forelegs were allowed to lie extended forward, the upper limb on top of the lower limb.

One group of dogs were monitored conscious, and the other group were anaesthetised. The groups were,

Group 1,	a)	Standing	PVP
	b)	Sternal recumbency	PVP
Group 2,	a)	Lateral recumbency (upper limb)	PVP
	b)	Lateral recumbency (lower limb)	PVP and CVP/PVP
	c)	Dorsal recumbency	PVP and CVP/PVP

Anaesthesia was routine in all dogs, Premedication was with acepromazine maleate intramuscularly, ( 0.1 mg./kg. ) thirty minutes prior to intravenous induction with sodium thiopentone, ( 10 mg./kg. ).

A/



A cuffed, rubber Magill endotracheal tube was inserted, the cuff inflated and the dogs maintained on a semi-closed circuit with a carbon dioxide absorber in a circle or to-and-fro unit. Spontaneous respiration with 2 % halothane in oxygen was permitted.

The surgery performed was variable and to permit a comparison of the procedures undertaken, the scale of surgical trauma was established indicating the degree of severity in each case. It was recorded by Shires (1961) and others that the requirement for fluids during the period of surgery was dependant on the degree of surgical trauma indicated by a reduction in the extracellular fluid volume, and in some instances, a decreased central venous pressure. In the light of these comments, the degree of trauma in the cases used in this study was assessed and compared with the respective PVP and CVP recordings, to determine if there was any relationship.

The scale used was based on an idea described by Moore and Bull (1952) and adapted for veterinary purposes by Annis (1961) who graded the metabolic response to surgical trauma. The criteria established by these authors were considered and a new surgical trauma scale devised. Annis stated that the greater the magnitude and duration of surgical trauma, the greater will be the depth of response.

The surgical trauma factor scale is,

5. Gross abdominal surgery  
Gross fracture fixation  
Multiple fracture fixation
4. Gastro-intestinal surgery  
Organ or tissue removal
3. Fracture fixation
2. Ovarohysterectomy  
Castration
1. Examination under anaesthesia

The disciplines of surgery were also noted to indicate the type of surgery performed.

O	Orthopaedic
A	Abdominal
N	Neurological
S	Soft tissue
X	Others including E.U.A.

The cannulation of veins was performed immediately after the induction/

induction of anaesthesia, before preparation of the operation site, To permit the monitoring of CVP and PVP, the two manometers were used set side by side, zeroed with a spirit level and fixed approximately level to the dog.

#### RECORDING OF RESULTS

In each case the breed, age, sex and weight of the dog were recorded. The nature and severity of surgery were assessed and noted. The duration of anaesthesia and surgery was recorded. Measurements of venous pressures were made every 15 minutes during the time from venous cannulation to extubation.

When monitoring venous pressures some important factors were noted which could affect readings on the manometers. The dog and the surface upon which it lay had to be maintained in a horizontal plane. The movement of limbs above or below the zero point also altered the readings. Venous pressures rose if the cannulated leg dropped below the suggested level, and fell when the limb was elevated.

The operating table was found to sink during some procedures and this caused changes in the venous pressures read, but this could be overcome by fixation of the manometers to the table. Surgical interference affected the PVP in all cases especially when the legs were raised or lowered or there was movement of the abdominal contents.

When taking readings the surgical interference was always at a minimum. Respiration caused changes in pressure which were normal.

## RESULTS

The following groups of dogs are considered,

Group 1,	a)	Conscious standing	PVP
	b)	Conscious sternal recumbency	PVP
Group 2,	a)	Anaesthetised lateral recumbency (upper limb)	PVP
	b)	Anaesthetised lateral recumbency (lower limb)	PVP
	c)	Anaesthetised dorsal recumbency	PVP
	d)	Anaesthetised lateral recumbency (lower limb)	PVP
			CVP
	e)	Anaesthetised dorsal recumbency	PVP
			CVP

There were 25 dogs in group 1., 25 dogs in each of groups 2, a), b) and c) giving a total of 100 peripheral venous pressure measurements. There were 25 dogs in each of group 2, d) and e) and hence a total of 50 central venous pressure peripheral venous pressure comparative measurements were made.

The tables referred to in the following pages can be found in Appendix 1.,

Group 1, a) Conscious standing PVP

Table 1.

The peripheral venous pressure in conscious standing dogs on a horizontal plane ranged from 3 to 11 cm. of water. The readings were constant in most dogs over the 15 minutes of recording. There was no apparent relationship between age, weight or breed in respect of the PVP. Any movement of the dog caused minor changes in the readings obtained, if for example,

- i) The animal sat down
- ii) The limbs were elevated
- iii) There was tilting of the horizontal surface
- iv) The head was raised or lowered.

Group 1.      b) Conscious sternal recumbency PVP

Table 2.

The PVP in conscious sternally recumbent dogs with the forelegs extended ranged from 7 to 15 cm. of water. The recordings were constant in most dogs over a period of 15 minutes. There was no apparent relationship between PVP and age, breed or weight. Movement of the dog caused minor changes in the readings, especially when the limbs were elevated or lowered relative to the zero point.

Group 2.      a)    Anaesthetised lateral recumbency (upper limb) PVP

Table 3.

The peripheral venous pressure in the anaesthetised laterally recumbent dogs ranged from 5.5 to 14.5 cm. of water. The readings varied in most dogs during the period of surgery, but there was no apparent relationship between the duration of surgery, the degree of surgical trauma, age, breed or weight with respect to PVP. Four cases required parenteral fluids post-operatively and these showed a decrease in PVP during the period of observation.

Group 2.      b) Anaesthetised lateral recumbency (lower limb) PVP

Table 4.

The PVP in anaesthetised laterally recumbent dogs ranged from 9.5 to 22.5 cm. of water. The measurements varied in most dogs during the period of observation, but there was no relationship between the PVP and the duration of surgery, the degree of surgical trauma, age, breed or weight. Four dogs required parenteral fluids post-operatively and all showed a decrease in PVP during the period of monitoring.



Group 2.      c)    Anaesthetised dorsal recumbency PVP

Table 5.

The PVP in anaesthetised dorsally recumbent dogs varied from 4 to 14.5 cm. of water. The readings varied in most dogs during the period of observation, but there was no apparent relationship between the PVP and the duration of surgery, the degree of surgical trauma, age, breed and weight. Six cases required post-operative parenteral fluids and they showed a decrease in PVP during the period of surgery.

Group 2. d) Anaesthetised lateral recumbency (lower limb) PVP/CVP

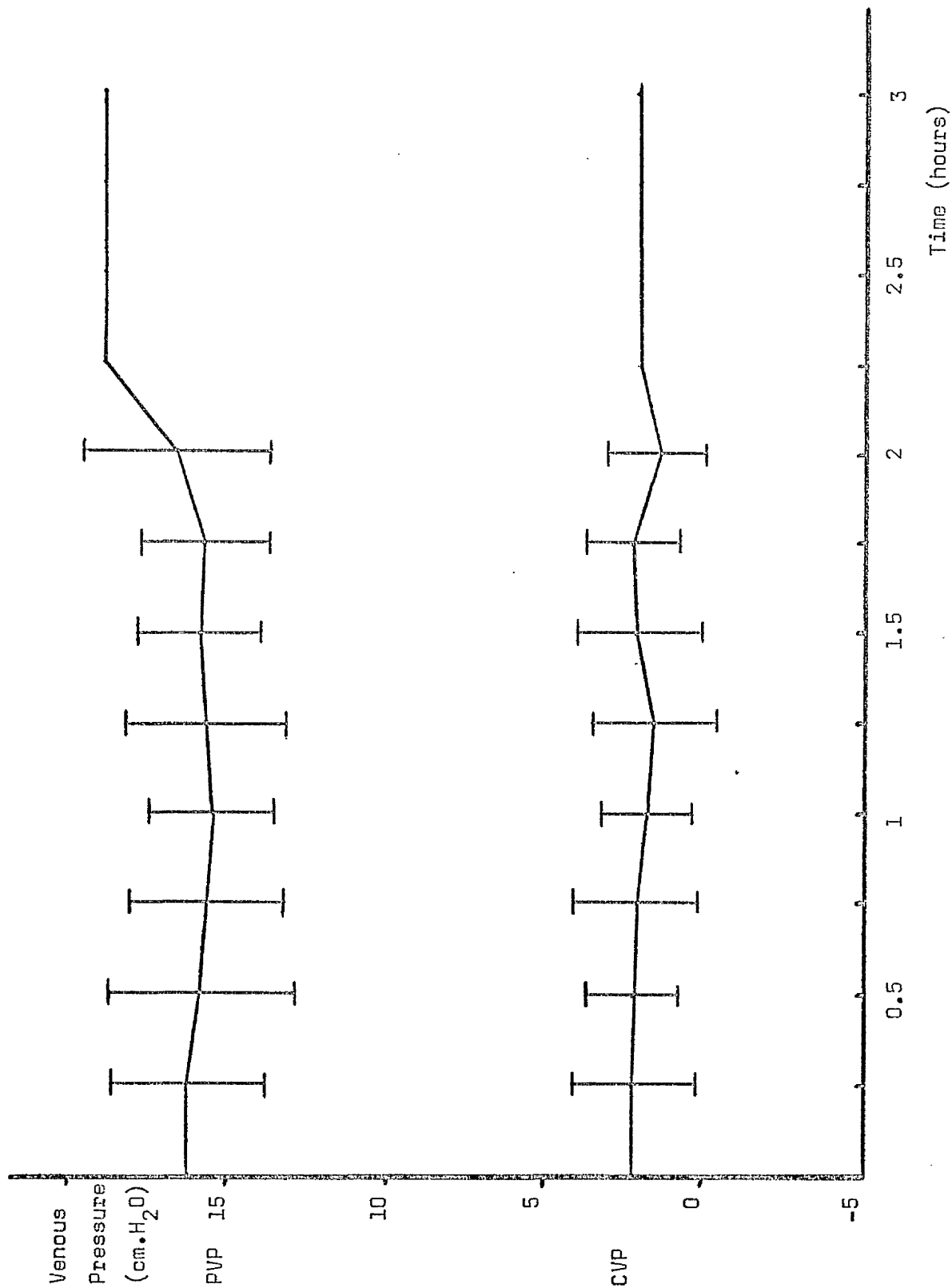
Table 6.

The PVP in this group ranged from 10 to 22.5 cm. of water which compares favourably with Group 2 b) - (9.5 to 22.5 cm. of water). The CVP in this group ranged from -3 to 6 cm. of water. There was no apparent relationship between the venous pressures and the duration of surgery, the degree of surgical trauma, age, breed and weight.

Two cases required post-operative parenteral fluids and these showed a decrease in both PVP and CVP during the period of surgery.

When the mean values for CVP and PVP are calculated and illustrated against time in a graph with one standard deviation, a paralleling effect is seen, ( Fig. 4).

Fig. 4 . Group 2. d) Anaesthetised Lateral Recumbency PVP/CVP - mean and S.D.



Group 2.      e) Anaesthetised dorsal recumbency PVP/CVP

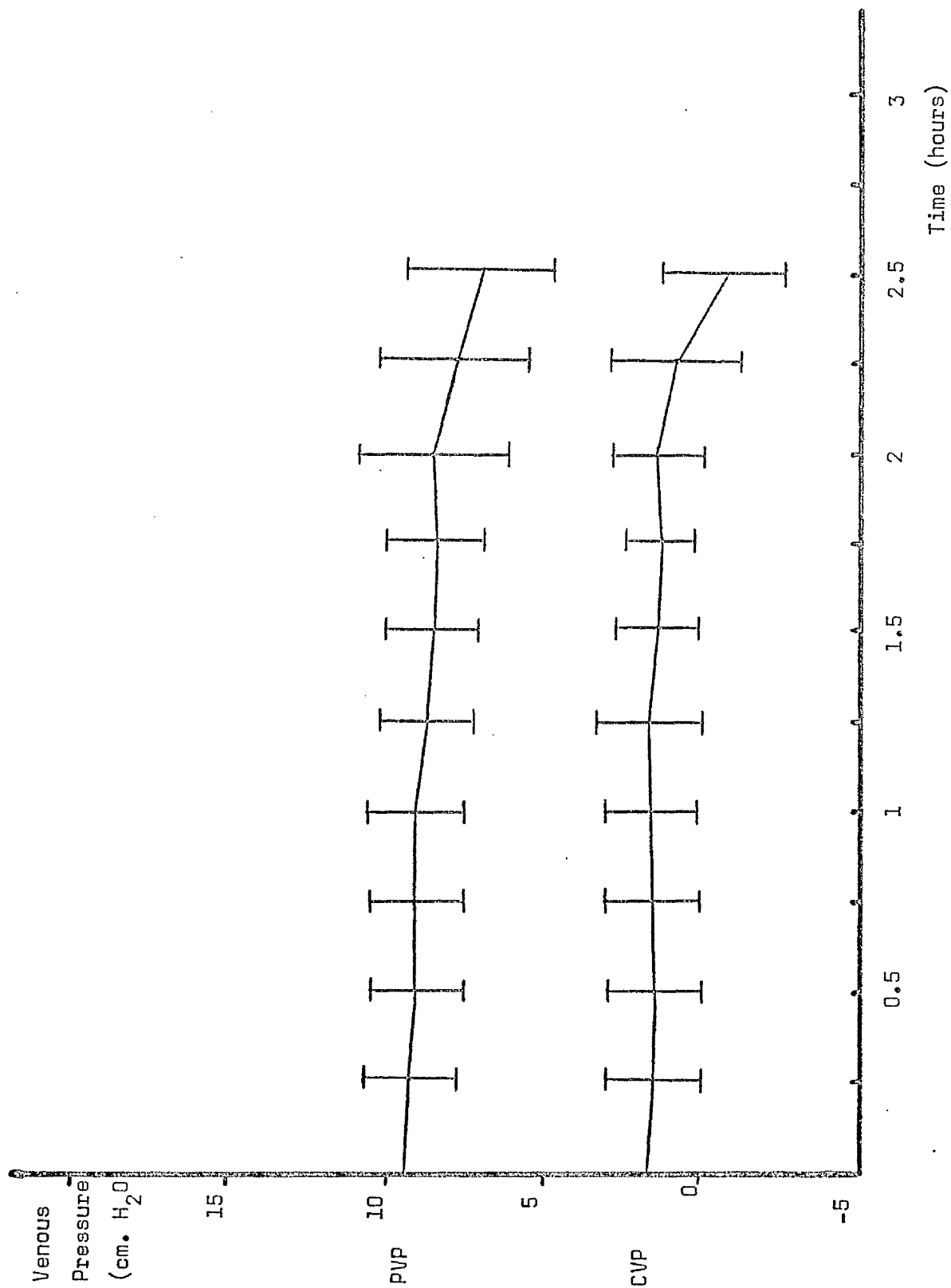
Table 7.

The PVP in this group of dogs ranged from 4.5 to 15.5 cm. of water which compares favourably with Group 2 c) - (4 to 14.5 cm. of water). The CVP ranged from -2 to 6.5 cm. of water which is similar to Group 2 d). There was no apparent relationship between the duration of surgery, the degree of surgical trauma, age, breed and weight and the PVP or CVP.

When the mean of CVP and PVP are calculated for this group and illustrated in a graph with one standard deviation, there is seen to be a paralleling effect of the two venous pressures, ( Fig. 5).

Six cases required post-operative parenteral fluid therapy and these showed a decrease in CVP and PVP during the period of observation.

Fig. 5. Group 2. e) Anaesthetized Dorsal Recumbency PVP/CVP - mean and S.D..



## DISCUSSION

In the group of 25 normovolaemic conscious, non-sedated standing dogs on a horizontal surface, the peripheral venous pressure varied from 3 to 11 cm. of water. There was no apparent relationship between the PVP and the age, breed or weight, and most of the recordings were constant over the period of 15 minutes. A respiratory pulse was noted in every case and with the results obtained, it proved that it was possible to measure PVP in dogs using the cephalic vein. It was also possible to define a range for this position of PVP. When the same 25 dogs were placed in sternal recumbency, and the water manometer repositioned to the respective zero point, further PVP measurements were found to be possible and these ranged from 7 to 15 cm. of water. Most subjects showed an increase above the standing level.

From these initial measurements it appeared that there was no age, breed or weight correlation with PVP. It was shown that PVP monitoring was possible in all the dogs and that ranges for readings could be established. It should be stated that no average value is given for any of the groups of PVP measurements since a range is more accurate when monitoring venous pressures and that trends in readings are of more importance than one single value. An ascending or descending trend in readings is clinically more important in PVP monitoring.

The second group of PVP measurements were recorded in 75 approximately normovolaemic anaesthetised dogs undergoing surgery of varied trauma. Dorsal and lateral recumbency were found to be the most common operating positions, and this allowed the monitoring of PVP in both positions, the laterally recumbent group being divided into two using/

using either the lower or the upper limb.

The 25 dorsally recumbent dogs under standard premedication and anaesthesia gave a range in PVP of 4 to 14.5 cm. of water. When monitoring these dogs it was necessary to have the forelimbs extended forward and parallel to the horizontal surface of the operating table and the flanks of the dog. Only then were readings possible which demonstrated a satisfactory respiratory pulse in the water manometer column, and were thus acceptable in this study. The range of pressure readings for individual dogs varied by different amounts during the period of observation, the majority changing by only one or two centimetres of water. It was demonstrated that in this group it was possible to measure PVP, but that factors such as surgical interference or external movement of the animal could alter the readings. The actual process of taking readings was short and did not interfere in any way with the surgery in hand or the anaesthesia.

The monitoring of PVP in laterally recumbent dogs undergoing surgery provided an immediate problem in deciding which limb to use. Two groups were established to investigate the PVP in both the upper and the lower limb. The readings taken from the upper limb ranged from 5.5 to 14.5 cm. of water whereas the readings from the lower limb ranged from 9.5 to 22.5 cm. of water. This showed that measurements could be taken from both limbs but that they were significantly different.

The peripheral venous pressure was measured in 75 dogs undergoing surgery and it was found that three distinct groups existed depending on the postural position of the dog. It is therefore important to decide which position the animal is in, and then to remember that single readings are of little value. Trends in a series of readings should be observed since these/

these are more accurate in any determination of the physical status of the animal. In all these dogs there was apparently no relationship between the PVP and the age, breed or weight. It was shown that there was no consistent relationship between the PVP and the duration of surgery or the degree of surgical trauma. In this group of dogs, 14 required post-operative parenteral fluid therapy and all of these dogs demonstrated a fall in PVP during the period of observation. It was noted, however, that some other dogs showed decreases in PVP similar to the group clinically in need of supportive fluid therapy, but these dogs were not deemed in need of support. This is only an observation and further investigation is indicated.

The ranges for PVP are illustrated in Fig. 6.

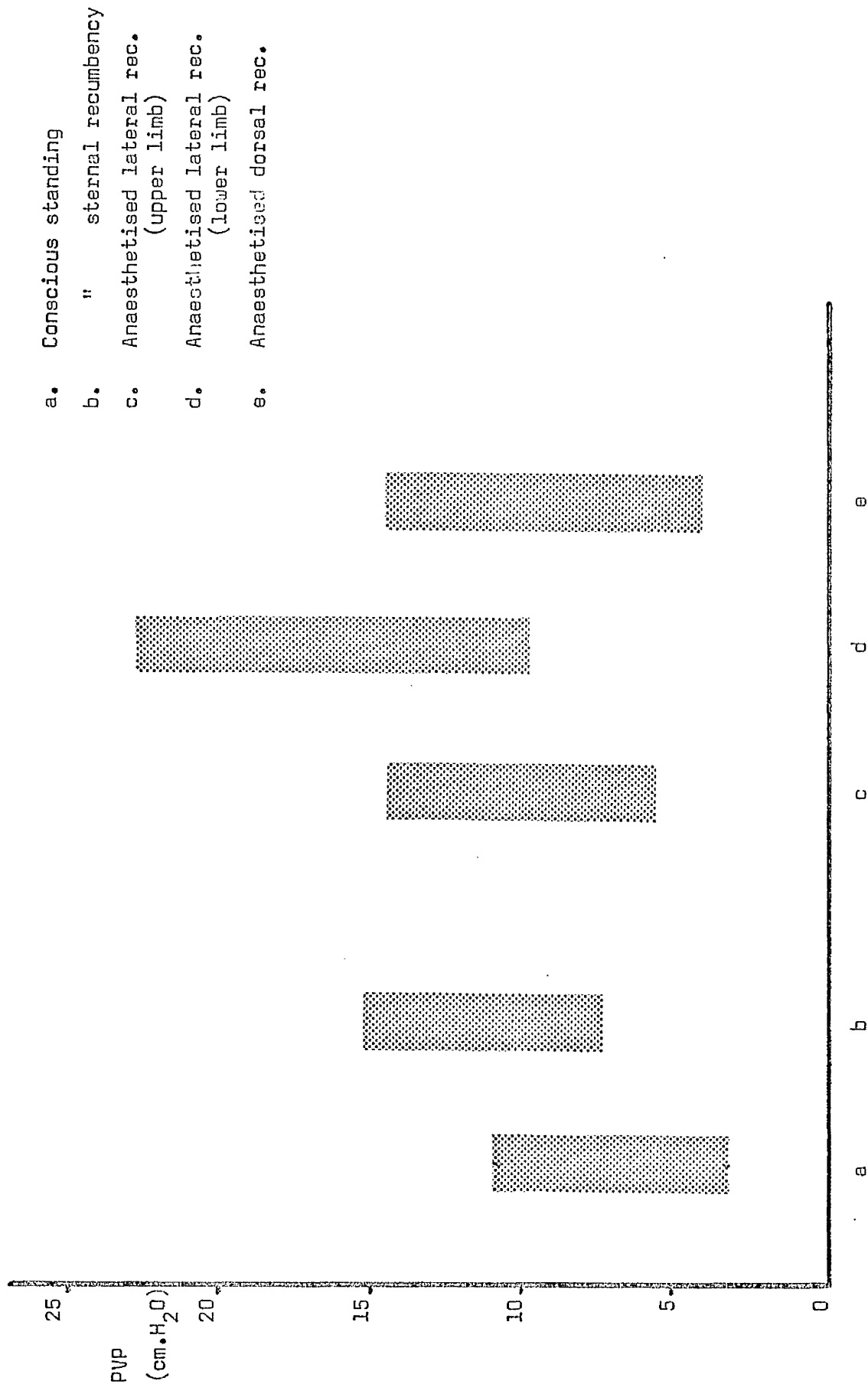
In the second group a comparison of PVP and CVP was made in 50 dogs undergoing surgery and it was found that the PVP readings were similar to those in similar postural positions described previously. The CVP measurements ranged from -3 to 6 cm. of water in the laterally recumbent group and from -2 to 6.5 cm. of water in the dorsally recumbent group. It therefore appears that as has been stated in the literature, CVP is relatively constant and is not dependable on postural position. It has been shown that after statistical analysis, graphs of CVP and PVP demonstrate a paralleling relationship between the two venous pressures.

The eight cases requiring post-operative supportive fluid therapy all demonstrated a decrease in CVP and PVP, but the significance of this was doubtful since other dogs showed similar decreases but did not appear clinically to need treatment.

In these two groups there were no apparent relationships between the/



Fig. 6 Peripheral Venous Pressure Ranges



the venous pressure measurements and age, breed, weight, duration of surgery or the degree of surgical trauma.

It therefore appears that one may use PVP monitoring to estimate the venous pressure in dogs undergoing surgery where fluid infusion is required.

The problems associated with the introduction of central venous catheters and the doubts regarding the reliability of CVP monitoring may warrant the use of the easier but perhaps equally unreliable PVP method. The ease with which a PVP line may be established to permit the monitoring of venous pressure makes CVP monitoring seem unnecessary, provided one remembers that PVP changes with the postural position. The use of venous pressure monitoring in clinical practice is in the determination of trends which may indicate that there is a requirement for supportive fluid therapy should the venous pressure decrease. It appears that in the 22 dogs requiring fluid therapy post-operatively, that either PVP or both PVP and CVP decreased during the period of surgery. In none of these dogs did the pressure remain static or increase. Further work is required to determine if there is a relationship between the venous pressures and the requirement for supportive fluid therapy in the event of a decrease in the pressure. The other important use of CVP monitoring in veterinary and medical cases is to detect overinfusion. If CVP rises with a relative fluid overload, would the PVP act in a similar way ? To provide this information, an experimental study was performed to overinfuse dogs and measure various parameters including CVP and PVP.

In conclusion, it has been demonstrated that the measurement of peripheral venous pressure is possible in dogs and that there is some correlation/

correlation between the readings and the postural position. It was demonstrated that PVP parallels CVP in anaesthetised dogs undergoing surgery. There appears to be no relationship between PVP and age, breed, weight, duration of surgery and the degree of surgical trauma. In 22 dogs which required post-operative supportive fluid therapy, there was a decrease in the venous pressure during the period of observation.

## SECTION 2

### EXPERIMENTAL STUDY

## EXPERIMENTAL STUDY

### INTRODUCTION

Overinfusion, sometimes referred to as overtransfusion or overhydration, is the state that exists when an animal receives fluid to excess, causing an overload of the tissues and the circulation. The state can be described best as the position occurring when input exceeds output.

When the blood volume is increased by moderate or large infusions, the low pressure vascular system accomodates most of the increase due to the distensibility of the veins which is believed to be one to two hundred times that of the arteries, (Gauer and Henry 1963). Hence the initial overload may be sequestered within the venous system which normally contains 80 % of the total blood volume. Minor increases in blood volume are controlled by renal activity and there is little change in the vascular systems since extra fluid is excreted. Gauer and Henry explained that as the blood volume increases and therefore increases the intrathoracic volume, a diuresis was prompted by an increase in the cardiac output.

Excess body fluid is excreted via the kidneys and if haemodilution exists, there is triggering of the osmoreceptors in the supraoptic nucleus of the hypothalamus to inhibit the release of anti-diuretic hormone from the posterior pituitary gland. If the blood volume is further increased, the renal output is maintained dependent on the cardiac output which presents blood to the kidney tubules at an increasing rate until, according to Starling's Law, the raised venous return reaches a point beyond which the cardiac output decreases due to cardiac distension. If the blood volume continued/

continued to increase and the venous return caused the cardiac output to fall, then overinfusion would result. At this point, fluid would leave the circulation and enter the tissues causing oedema. This is of greatest importance when it affects the pulmonary tissues causing pulmonary oedema which will result in hypoxia if persistent and severe.

The situation described would exist in the normal animal, but any disease of the kidneys, heart or lungs could alter the picture dramatically. Renal disease could result in a decreased output of urine which would allow circulatory overload to occur more readily. Defects in the action of the heart would cause the overload situation to occur more readily, especially if the venous pressure increased above that which the heart's action could deal with. Decreased left heart function would readily lead to pulmonary oedema in the fluid overloaded animal. These factors are worthy of note before the administration of fluids by the intravenous route.

Pulmonary oedema has been discovered in patients with certain predisposing factors, such as lung disease and cardiac failure, (Warren, Brannon, Weens and Stead 1948). They demonstrated that normal dogs given saline intravenously developed generalised oedema, but rarely dyspnoea.

The aetiology and clinical appearance of pulmonary oedema are described, (Harpster 1974), and it is stated that it is due to an upset in the balance between the hydrostatic and osmotic forces across the pulmonary capillary walls. Overinfusion is a form of haemodynamic pulmonary oedema seen most commonly in small animals or patients with underlying renal or cardiac insufficiency. The mechanism is a combination of increased pulmonary capillary hydrostatic pressure and decreased pulmonary capillary oncotic pressure secondary to haemodilution. Decreased pulmonary capillary oncotic/

oncotic pressure is a reduction in the colloidal osmotic pressure of the pulmonary capillaries, favouring the movement of fluid into the alveolar-capillary space. This, according to Harpster, is commonly seen in animals with hypoalbuminaemia or hypoproteinaemia. The only clinical signs of note are hypoxaemia due to the impaired diffusion of oxygen across the alveolar membrane and perhaps tachypnoea. Radiographically there is evidence of fine diffuse increased alveolar densities with little evidence of vascular prominence or airway involvement. Interstitial pulmonary oedema is difficult to diagnose clinically.

A review of the literature, (Staub 1974), regarding pulmonary oedema revealed that the interstitial oedema seen as a result of overinfusion made the lungs more rigid and the breathing laboured, but did not cause crepitations on auscultation, expectorated fluid or a fall in the arterial oxygen saturation or the partial pressure of oxygen in arterial blood. Radiographically, Staub claimed that reports as to the detection of interstitial pulmonary oedema were conflicting.

The ease with which fluid may be administered to animals using the intravenous route caused some concern at the start of this study. The patients most likely to suffer were the small dogs and cats which could be accidentally infused with 500 ml. of fluid without the total awareness of the practitioner. The problem facing the veterinary practitioner who treats many sizes of patient is how to prevent overinfusion or to detect it before it becomes clinically significant. To be of use clinically such a technique would have to be cheap and easily used.

Another problem which is commonly seen when treating cases of body fluid imbalance is to determine when the patient is satisfactorily rehydrated/

rehydrated. The experimental study investigated both of these problems, the latter problem being considered secondarily to the state of overinfusion, although the two problems are directly linked.

The literature regarding the administration of fluids to both animals and humans was reviewed to determine if there was an available method other than CVP monitoring which could be used to assess the fluid requirements of the patient. CVP monitoring is gradually losing favour in the medical world due to some complications in the use of the intravenous lines and the doubt regarding the reliability of such monitoring. This review is not considered chronologically but in a more logical order of facts and opinions.

The administration of large volumes of fluid, particularly isotonic saline, to normal, healthy surgical patients undergoing anaesthesia and operation was investigated, (Stewart and Rourke 1942). These workers found that the E.C.F. volume increased during the infusion and that 80 % of the volume administered was retained for up to four days post-operatively. These doctors concluded that the E.C.F. compartments could be flooded with fluid without any functional disturbance. When 5 % glucose solution was infused instead of isotonic saline, an immediate diuresis was evidenced thus preventing haemodilution. Stewart and Rourke also postulated that there was a reserve of plasma protein which entered the circulation when the saline was infused but not when the dextrose was given. It was advised that glucose should not be given to replace E.C.F. losses due to its dehydrating effect.

It was noted that the administration of large volumes of physiological saline to dogs, up to an equivalent amount to body weight, caused/



caused generalised oedema and a loss of protein from the plasma, (Warren, Merrill and Stead 1943). The plasma volume in these dogs remained normal and therefore it was determined that the E.C.F. volume was important in the control of plasma volume.

Further work revealed that physiological saline solution caused a temporary increase in plasma volume which disappeared in four hours taking little if any protein with it, (Ashworth, Payne and Jester 1944). However when 5 % dextrose solution is infused it causes little increase in plasma volume and the total infusion was excreted within two hours. There was no loss of protein from the circulation when dextrose was infused. If plasma was infused, there was a rapid and sustained increase in the plasma volume.

The problem of post-operative salt retention attracted much interest and it was found that when isotonic saline solution was infused, 53 % of the sodium, 46 % of the chloride and 19 % of the water was retained for 30 hours, necessitating a reduction in the I.C.F. to compensate the hypertonicity of the E.C.F., (Coller, Iob, Vaughn, Halder and Moyer 1945). However when one-fifth normal saline solution was administered, only 27 % of the sodium, 32 % of the chloride and 39 % of the water was retained which caused little change in the E.C.F. and it was concluded that the infusion of 0.18 % Na Cl solution was preferable to 0.9 % Na Cl post-operatively.

It was against this background of surgical thinking that the E.C.F. volume increased during the infusion of balanced electrolyte solutions during anaesthesia, that it was stated in North America that the E.C.F. volume fell isotonically during surgery in response to the degree of surgical trauma, (Shires, Williams and Brown 1961). Even when fluids/

fluids were administered at the accepted rates, the E.C.F. volume decreased, and it was deduced that a third body space existed whereby, during anaesthesia and surgery, the E.C.F. was redistributed in the body tissues adjacent to the surgical wound, in the splanchnic bed and into the I.C.F.. This loss of E.C.F. could not be accounted for in blood loss and there was found to be no correlation between the E.C.F. volume and the whole blood loss in patients. It was thus advocated that large volumes of balanced electrolyte solution be administered to patients undergoing surgery to compensate for this loss.

It was suggested that the post-operative oliguria seen commonly in surgical patients was due to the decrease in the E.C.F. volume in response to surgical trauma, (Boba and Landmesser 1961). They also stated that the reduced E.C.F. volume caused the release of anti-diuretic hormone and constriction of the renal vasculature. They advised that the use of electrolyte solutions and mannitol be promoted to prevent post-operative oliguria.

The initial findings were re-emphasised in a further study, and the use of balanced salt solutions during surgery was strongly advocated, (Shires and Jackson 1962). The requirement was directly related to the degree of surgical trauma.

After a study of post-operative oliguria, it was concluded that Ringer lactate administered intra-operatively prevented the renal shutdown more effectively than 5 % dextrose solution, (Miller, Stoetling and Paradise 1963). It was stated that the oliguria was directly due to a transfer of the E.C.F. to the I.C.F. when insufficient fluid was given during surgery.

The third space was profounded as late as 1968, (Hayes), and it was stated that after any injury there was an obligatory fall in tissue perfusion which could be compensated for by the administration of salt solutions/

solutions..

Grave doubts were cast on the reasoning of Shires and his colleagues when many cases of overinfusion pulmonary oedema were seen at autopsy, (Anderson, Simmons, Collins, Bredenberg, James and Levitsky, 1969). Prior to this report, the changes in the E.C.F. volume during surgery were further investigated and it was found that there was no change in the E.C.F. volume during operative trauma, and that it was clearly prudent to limit fluid administration, (Roth, Lax and Maloney 1967). The problems of the pulmonary oedema or wet lungs were further outlined in two reports which questioned the ability of CVP monitoring to detect overinfusion, (Frank 1969, Spencer and Berman 1972). Both these authors stated that new methods of preventing overinfusion were needed urgently to replace CVP monitoring.

It was because of these findings that a report titled "Moderation" was published which stated that balanced salt solutions in preference to normal saline or 5 % dextrose solution should only be administered to patients undergoing major surgical procedures to aid in the reduction of wet lung cases, (Moore and Shires 1967). The use of Ringer lactate was as an adjunct to surgical trauma and not a replacement for blood loss. Careful assessment of the cases was required and it was advised that the patient should only be returned to physiological normal, a situation which could not be accomplished by inundation.

Attempts were made to quantitate the requirement for fluid intra-operatively and it was found that although the E.C.F. volume fell during and immediately post-operatively in man, it was not in any way associated with the degree of surgical trauma, (Stahl 1967) A recommended rate of 5 to 15 ml. per kg. body weight per hour of Ringer lactate was advocated/

advocated for all human surgical patients. Stahl stated that the renal response of post-operative oliguria was related to the duration and severity of surgery.

It was found that the estimation of the E.C.F. volume was difficult when isotope dilution techniques were used and that the volume did not markedly decrease despite trauma or shock in dogs, (Gutelius and Shizgal 1968). The changes seen were accounted for by the withdrawal of blood samples for analyses.

Many studies were performed in Vietnam using combat victims to determine if the E.C.F. volume changed during surgery or trauma. The studies recorded no deficit in the E.C.F. which could not be accounted for by dehydration or blood loss, (Anderson et al 1969). These authors reported many cases of pulmonary oedema following Shires type treatment, and they concluded that there was a need for intense monitoring of patients to avoid overinfusion when attempting to compensate for the supposed E.C.F. loss.

A comprehensive report stated that the isotope dilution techniques used by Shires and his colleagues were doubtful and using other proven methods it was found that contrary to the previous belief, the E.C.F. volume did not change at all during the period of surgery, (Roth, Lax and Maloney 1969). It was found that up to 50 % of surgical cases in Vietnam had degrees of post-operative pulmonary oedema as a direct result of overinfusion. It was concluded that more care should be taken when administering large volumes of balanced salt solutions to patients undergoing surgery, and that the technique of large fluid infusion be discontinued.

Further to the reports of 1969, the Lancet published a report on the recent findings stating that 40 years of human fluid therapy had seen advances/

advances from the barely adequate rectal infusion to intravenous saline in moderate quantities to massive infusions and pulmonary oedema and back again to the more moderate approach to clinical treatment of fluid loss in surgical patients. The inherent pulmonary oedema complication due to massive infusion far outweighs any benefit derived from the balanced salt solutions. The time had now come for moderation and common sense in fluid therapy.

Despite the work of Roth et al, two reports during this decade still refer to the third space and the need for intra-operative fluid therapy. One report from Vietnam stated that massive quantities of whole blood and Ringer lactate were required to maintain body function, but it was found that some overinfused patients had very high outputs of urine, (Doty, Hufnagel and Moseley 1970). The third space was reported in patients undergoing prolonged major surgery and it could only be compensated for by the infusion of massive quantities of fluid, (Bevan, Dudley and Horsey 1973). The use of CVP monitoring and the regular assessment of the haematocrit were satisfactory methods of preventing overinfusion.

Within the veterinary profession, little controversy has existed regarding what method of fluid administration should be used, primarily because of the apparent lack of use of parenteral fluid therapy in animals. It is perhaps only within the past ten years that any interest has been raised among small animal practitioners to routinely use parenteral fluid therapy during surgery and hence the problems of overinfusion have rarely if ever occurred. With the increase in the teaching of body fluid physiology and the practical techniques of fluid administration as part of the anaesthetic support, more use will undoubtedly be made in the future of correct/

correct fluid therapy. Once the intravenous route is being used routinely, there may be problems associated with overinfusion. CVP has long been the established method of detecting fluid overload within the body, (Hall 1967, Burrows 1976), but the complications and the poor results obtained from this technique in routine clinical work prompted a study to evaluate the problems of overinfusion.

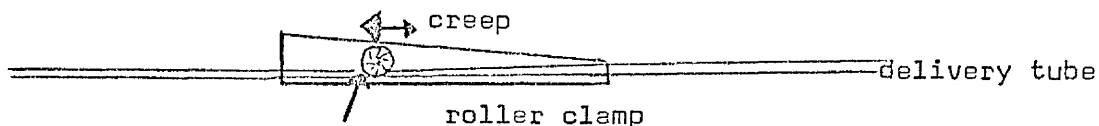
A clinical and experimental study were instituted to determine if there was a means by which the veterinary practitioner might detect or prevent overinfusion, and at the same time assess the success or failure of therapy. The clinical study involved the observation of patients undergoing fluid infusion and an assessment of the equipment available. The experimental study involved the infusion of large quantities of fluid to dogs, monitoring various parameters, in an attempt to detect subclinical pulmonary oedema or overinfusion.

## CLINICAL STUDY

The clinical study involved the monitoring of patients undergoing infusions of crystalloids and colloids to replace fluid or whole blood loss. Attempts were made to evaluate the equipment available for the administration of fluids and also to study the methods by which overinfusion might arise.

With the use of plastic cannulae and butterfly needles it has become possible to inject fluids with ease and to administer large volumes rapidly. The advantages are obvious, but the possible disadvantages are the accidental overinfusion of patients and the lack of control of the flow rate. Flow control has already been discussed and it is important when attempting to prevent overinfusion. The other major point of interest was in the size of the fluid container that could be attached to any size of animal. Finally the question of monitoring was most relevant to this study and also the method of case assessment.

Flow control is a major factor in the rate of administration of fluid and the various designs of regulator have been described and illustrated. One major problem in the design of the plastic roller clamp is that the plastic delivery tube may attempt to regain its former shape and diameter. This causes the roller to "creep" and this in turn directly affects the flow rate, which commonly increases.



This could prove dangerous in a small animal if a large volume of fluid was allowed to infuse rapidly and accidentally.

One other problem is the difficulty in setting the roller clamp flow regulator such that it will deliver a constant but small volume each hour to a small animal. This problem has partly been overcome by the introduction of a simple device which sets the flow rate accurately. This is illustrated (Appendix 4, Fig. 13) and is called a "Dial-a-flow" flow regulator. It is possible to set flow rates as slow as 5 ml. per hour as well as permitting rapid infusions should the need arise. The unit does not creep or alter with time and it therefore appears that this device could be used to prevent overinfusion. The only factors which may alter the flow rate are the height of the fluid reservoir above the patient and the occlusion of the intravenous line by clotted blood when slow flow rates are being used.

Another cause of overinfusion is the size of fluid container attached to the patient. Fluids are commonly presented in 500 ml. or 1000 ml. containers, and it is obviously disadvantageous to connect one or other of these to a small animal whose requirement is for 100 ml. of fluid. A method of reducing this volume without interfering with the container is to use a graduated burette as part of the administration set. This is illustrated (Appendix 4, Fig. 11), and commonly there are 1 ml. graduations on the side of the burette. This allows very accurate control over the amount of fluid that may be administered at any one time.

Despite these precautions overinfusion may still occur due to the lack of supervision.

The recommendations are therefore,

a)/



- a) Use a satisfactory, well designed flow regulator
- b) Use a moderate size of needle or cannula
- c) Use a graduated burette chamber as part of the administration apparatus
- d) Monitor patients regularly
- e) Monitor the infusion equipment regularly

By following these recommendations, moderate or rapid infusions are possible, and control is perhaps better than when using a "flutter valve" type arrangement which will allow fluid flow, but with little accuracy.

## EXPERIMENTAL STUDY

In human medicine and intensive therapy units the methods used for the prevention or detection of overinfusion are CVP monitoring, respiratory function monitoring using the blood gas levels, chest radiography, the output of urine and the overall clinical appearance. Which of these along with other parameters would be of most value and accuracy to the general veterinary practitioner ? Following the considerations of Staub (1974), who reviewed pulmonary oedema, an experimental model was established under Home Office regulations to infuse various fluids intravenously in dogs at set rates to determine if one of a series of parameters was of most value. Since the study was to have clinical significance, it was necessary to restrict the various parameters to those which could be monitored routinely, although some measurements were taken for experimental reasons.

The study was performed using dogs within the Wellcome Surgical Research Institute, Bearsden, Glasgow.

In this experimental study various parameters were considered and although many more and more complex measurements could have been made, there were restrictions with equipment availability and other economic demands. Nevertheless the following groups of parameters were monitored in each dog.

- A. Fluid input and urine output
- B. Blood pressures
- C. Blood gases
- D. Chest radiography
- E/

- E. Haematocrit and Total serum protein
- F. Serum electrolytes
- G. Clinical signs

A. Fluid input/urine output

The fluid input was set at different rates in dogs over a period of time followed by a test dose of fluid to assess the capabilities of the circulation. The rates of fluid infusion were based on the clinical infusion rate used in this study when treating patients with a body fluid deficiency undergoing surgery. This rate was calculated from the blood volume ( 90 ml. per kilogram ) one third of this volume being administered during the period of surgery. This was based on the work of Stahl (1967) who recommended an infusion rate of 15 ml. per kg. per hour to patients undergoing surgery which was prolonged or complex. Most of Stahl's patients were normovolaemic pre-operatively and it was accepted that a similar infusion rate in dogs would not be detrimental since many were hypovolaemic. However, it was also shown that this rate of infusion could cause an increase in the incidence of post-operative pulmonary oedema, (Anderson et al 1969).

The rates of infusion were based on these former findings and were,

- a) 20 ml. per kg. per hour
- b) 30 " " " " "
- c) 40 " " " " "

This it was hoped would give some indication as to the time at which/

which overinfusion was most likely to occur, and at what time each of the measured parameters would show changes. The rates were maintained experimentally over 4 hours.

Following the 4 hour infusion, a test dose of fluid was administered in 15 minutes to assess the capabilities of the body systems. It was hoped that this would stress the circulation, and a preliminary experiment was performed to show what changes occurred in the parameters when rapid infusions of fluids similar to those used in the clinical part of this study were given to normovolaemic dogs. The results of this experiment are recorded later. The fluids were divided into two groups,

1. Crystalloids
2. Colloid

1. Crystalloids
  - a) 0.9 % Na Cl
  - b) 5 % dextrose solution
  - c) 0.18 % Na Cl + 4.3 % dextrose
  - d) Ringer lactate

These were selected because of their widespread use in veterinary practice for the replacement of body fluid losses. The crystalloids were infused at the three rates over 4 hours.

2. Colloid
  - e) Dextran 10 % in 0.9 % Na Cl

This/

This was the principal colloid in use at the time of the experimental project and it is hypertonic in nature, its use being as a plasma expander. The colloid was infused at 4 rates over 1 hour followed by Ringer Lactate at 20 ml. per kg. per hour for 1 hour. The infusion rates were 10, 20, 30 and 40 ml. per kg. per hour.

The addition of crystalloid to the dogs after the infusion of colloid was to evaluate the effect of such a procedure.

The urine output was monitored at regular intervals during the fluid infusion in an attempt to evaluate the renal capabilities of the normovolaemic anaesthetised dog. The rate of urine output is commonly proportional to the glomerular filtration rate and is therefore a measure of renal perfusion and indirectly arterial blood pressure. The output of urine is also controlled by the osmolarity of the body fluids and any reduction will result in an increased output in an attempt to regain the correct osmolarity. Changes in blood volume and body fluid balance also affect renal output and in the state of overinfusion, the output is increased due to changes in osmolarity and body fluid volume. The normal daily output of urine in the dog is 20 ml. per kilogram body weight, which is approximately 1 ml. per kg. per hour, and theoretically any increase in this rate is indicative of overinfusion. A urinary output in humans in excess of 1 ml. per kg. per hour is a sign of overhydration, (Doty, Hufnagel and Moseley 1970). It has been shown that the peak urine output in humans infused with Ringer Lactate occurred at the end of the infusion, and that any degree of surgical interference or anaesthesia prolonged the period of fluid retention, (McKenzie and Donald 1969). The output of urine was calculated for/

for each dog to determine if there was any correlation between the rate of infusion and the output.

B. Blood pressures

The pressures monitored were

- a) Arterial blood pressure
- b) Central venous pressure
- c) Peripheral venous pressure

a) Arterial blood pressure This was monitored from the carotid artery which was cannulated and connected to a pressure transducer pen recorder unit, to determine the changes seen with the infusion of fluid. It was also used to determine if there was any correlation between it and other parameters, especially CVP, PVP and urine output. It has been demonstrated that when humans are infused with fluids the arterial blood pressure rarely changes, (Altschule and Gilligan 1938). It was also demonstrated in shocked patients that the venous pressure changed before the arterial pressure when fluids were infused, (Pierce, Boyan and Masterton 1953). It was intended to evaluate dextran 40 in the dog since it has been claimed that dextran causes a rapid rise in blood pressure which is maintained after infusion, (Bull et al 1949, Ricketts 1973). It has been reported that although dextran re-establishes the blood volume, a supplementary infusion of crystalloid is often necessary to ensure a satisfactory renal output, (Ricketts 1973). Dextran is hypertonic and without the addition of isotonic or hypotonic fluid to the vascular system/

system, the I.C.F. will be depleted to the detriment of the patient. The infusion of crystalloid with colloid ensures a renal output without depletion of the I.C.F..

b)      Central venous pressure                      This was monitored via a jugular line extended into the chest to lie near to the right atrium. CVP is used routinely in human hospitals to evaluate blood volume and to detect overinfusion, and its use in the veterinary field has been advocated, (Hall 1967, Burrows 1976).

c)      Peripheral venous pressure                      This was monitored via a cephalic cannula and the pressure has already been shown to relate directly to CVP. It was therefore equally important to evaluate this parameter in this study.

#### C.      Blood gases

The gases measured were,

- a)      Arterial partial pressure of oxygen
- b)      "                      "                      "                      "                      carbon dioxide

It is recorded that the arterial oxygen level in overinfusion changes, especially when pulmonary oedema is demonstrable, (Staub 1974), but it has been stated that the level changes little if at all in the presence of pulmonary oedema, (Staub 1974). It has been claimed that hypoxaemia/

hypoxaemia is often the only sign of pulmonary oedema in an overinfused dog, (Harpster 1974). Two other authors advise that the Pa O<sub>2</sub> should be monitored regularly in patients undergoing massive body fluid replacement, (Frank 1969, Berman and Spencer 1972).

The arterial blood gases were monitored at regular intervals in the dogs in this study to determine if any changes occurred.

#### D. Chest radiography

Lateral chest radiographs were taken every hour to determine the degree of pulmonary change with the rate of infusion. It has been recorded by some authors that X-ray plates were of immense use in the diagnosis of overinfusion pulmonary oedema, (Staub 1974). It was also claimed that routine serial radiographs of the chest were of more value than CVP monitoring, (Frank 1969, Spencer and Berman 1972). It is recorded that the changes seen are fine and diffuse in nature, (Harpster 1974). The effects are seen in the alveolar regions and there is little evidence of vessel thickening or airway change. It was therefore thought that lateral chest radiography would be a method of determining when pulmonary oedema was present.

#### E. Haematocrit and Total serum protein

These were estimated to determine the degree of haemodilution caused by the infusion of fluids. It has been claimed that progressive haemodilution occurs if whole blood loss is replaced with crystalloid solutions, and there is a decline in the P.C.V., (Wright 1976).

F/



#### F. Serum electrolytes

Sodium, potassium and chloride were monitored hourly to determine if any changes occurred other than dilution during the infusion of fluids. A number of different solutions were used and it was intended to discover if any of these were responsible for any of the changes seen. It was important to determine which fluid caused the least changes since this would be of clinical significance.

#### G. Clinical signs

The signs monitored were	i)	Heart rate
	ii)	Respiratory rate
	iii)	Thoracic auscultatory sounds
	iv)	Core temperature
	v)	Mucous membrane colour and consistency
	vi)	General body oedema
	vii)	Extra-renal fluid loss

The reported clinical signs of pulmonary oedema are coughing, laboured breathing and of overinfusion cyanosis and venous distension, (Hall 1967, Harpster 1974, Staub 1974). It has been reported that little is heard on chest auscultation and only rarely are crepitations heard, (Staub 1974). The value of changes in the clinical signs could have clinical significance when monitoring cases undergoing fluid infusion.

## MATERIALS AND METHODS

### EXPERIMENTAL MODEL

Dogs of 15 to 30 kilograms body weight with an average body fat content were selected and were deprived of food and water for no longer than two hours prior to anaesthesia.

Pre-anaesthesia            The dogs were checked clinically for abnormalities of the pulmonary and cardiovascular systems. The general condition and physical status of the dog were also assessed prior to use.

Anaesthesia            Premedication was with acepromazine maleate (0.1 mg. per kilogram) intramuscularly 30 minutes pre-induction with sodium thiopentone (10 mg. per kg.) intravenously in the right cephalic vein. A cuffed, red rubber Magill endotracheal tube was inserted and the cuff inflated. Spontaneous respiration permitted maintenance with 1 % halothane in oxygen from a Fluotec mark III vapouriser administered through a modified Magill circuit, (illustrated later). A flow rate of 10 litres of oxygen per minute was used. This high flow rate was necessary to allow rapid filling of the rebreathing bag to a set pressure to allow chest radiographs to be taken. The fluotec was used out of circuit and was accurate in delivering 1 % halothane at flow rates of 1 to 10 litres per minute at 18 to 36°C. over prolonged periods.

Surgery            The left carotid artery and left external jugular vein were exposed and cannulated using plastic cannulae connected to three-way stopcocks. Both the left and the right cephalic veins were cannulated with plastic over-the-needle cannulae connected to three-way stopcocks. The position/

position of the central venous pressure cannula inserted through the jugular vein was checked by later chest radiograph. The PVP was monitored using the upper limb. A plastic catheter was inserted into the bladder and connected to a graduated plastic bag to permit recording of urine output.

Recording Continuous recordings were made of heart rate, arterial blood pressure, CVP and PVP using Bell and Howell 221 (0-75 cm. Hg) physiological transducers connected to the respective cannulae, and to Devices M2 heated stylus recorders. Short connecting lines were used between the cannulae and the transducers to increase accuracy. All lines were maintained patent with heparinised saline. The transducers were calibrated at atmospheric pressure and 100 mm. Hg prior to recording each day.

The heart rate was recorded using a Devices Neilson heart rate meter connected to the M2 recorder measuring left carotid pressure allowing continuous recording with a Devices M2 heated stylus pen recorder.

Respiratory rate was recorded manually as too were core temperature and the clinical signs.

Radiography Lateral chest radiographs were taken during the infusions using a portable Philips Practix X-ray machine set at,

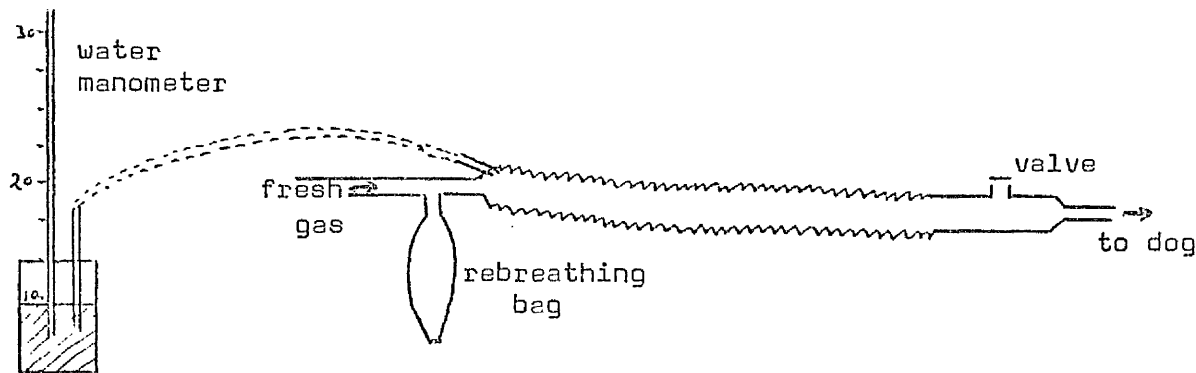
65 KV

0.25 seconds

20-25 mA.

A Lysholm aluminium cased grid measuring 15" by 12" and with a grid ratio of 12:1 was used. X-Omat H film (18X24 cm.) XH1 within a regular screen cassette was used for the chest radiographs and to ensure similarity of pulmonary inflation, a pressure system was attached to the Magill anaesthetic circuit at the time of radiography allowing films to/

to be taken at a set pulmonary inflation pressure.



When taking films, the valve was closed and pressure was exerted by squeezing the rebreathing bag until a pressure of 30 cm. of water was recorded. This pressure appeared to give the best degree of pulmonary inflation radiographically. Once the film had been taken the valve was re-opened. To enable these radiographs to be taken without disturbing the position of the dog, a perspex box was laid beneath the dog which permitted the cassette to be placed underneath the chest.

Films were taken at regular intervals and stored in a light proof box until being developed simultaneously at the end of each experiment. Development was performed using a Williamson R.P. automatic film processor which utilises Matalex A X-ray developer and Parafix A fixer mixed 1:4 with water.

Blood gases                      These were determined using an Eel Corning blood gas analyser.

Blood analyses                      The haematocrit estimation was performed using microhaematocrit tubes and a microhaematocrit centrifuge, the readings being made with a slide rule. Haemoglobin was assayed by the Cyano-methaemoglobin method using a colorimeter.

The total serum protein assay was performed by a modified biuret/

biuret reaction done in an Autoanalyser (Technicon). Sodium and potassium assays were performed with an Eel 22 integrating flame photometer, with a stock lithium solution for the high and low dilutions standard. Chloride was estimated with the Eel chloride meter which depends on the amount of silver left after a serum sample is added to silver nitrate.

Fluid infusion

Commercial fluids were chosen, the list being,

- a) 0.9 % Na Cl
- b) 5 % Dextrose
- c) 0.18 % Na Cl + 4.3 % Dextrose
- d) Ringer Lactate
- e) Dextran 40 10 % in 0.9 % Na Cl

The infusion of fluid was performed using a burette and administration set with a flow regulator of the " dial-a-flo " type which was accurate to 10 ml. per hr.

Euthanasia

Pentobarbitone, 20 ml. of 25 %.

Following the induction of anaesthesia, a period of one hour was allowed before the commencement of the infusion for the stabilisation of the model. Resting blood samples were taken for biochemical and haematological analysis. A lateral chest radiograph was taken to check the position of the CVP line.

The resting blood results are given in Appendix 2 along with the general information regarding each dog.

## EXPERIMENTAL PROTOCOL

Single measurements were made at different intervals before, during and after the infusion. The following scale will outline the fluid infusions for the two groups of dogs. The groups are,

## I. Crystalloids

## II. Colloids



I.	Fluid	Infusion	Test dose	Recovery
----	-------	----------	-----------	----------

II. Colloid infusion	Lactate infusion	Test dose	Recovery
----------------------	------------------	-----------	----------

The parameters monitored were as listed and the recording of results was made at the interval indicated in the following table.

	Stabilisation	Infusion	Test dose	Recovery
A. Fluid input	15 min	15 min	15 min	15 min
Urine output	"	"	"	"
B. Art. Blood pressure	"	"	5 min	"
CVP	"	"	"	"
PVP	"	"	"	"
C. Arterial O <sub>2</sub>	1 hour	1 hour	15 min	15 min
" CO <sub>2</sub>	"	"	"	"
D. X-ray	"	"	"	"
E. Blood samples	"	"	"	"
F. " "	"	"	"	"
G. Clinical signs	"	"	"	"

The groups of dogs were,

I. Crystalloids

Dogs 1 - 3    0.9 % Na Cl  
Dogs 4 - 6    5 % Dextrose  
Dogs 7 - 9    0.18 % Na Cl + 4.3 % Dextrose  
Dogs 10-12    Ringer Lactate

2. Colloid

Dogs 13-16    Dextran 40

The infusion rates were

a)    20 ml per kg per hour  
b)    30   "   "   "   "   "  
c)    40   "   "   "   "   "

when infusing Dextran, an additional infusion rate of 10 ml. per kg. per hour was used.

## RESULTS

The experimental data from each dog is recorded in Appendix 2 and these results are analysed in full in this section. Each group of parameters is discussed individually and there is a general discussion later.

An experiment was performed prior to the above series to determine if the model would work, and in this dog infusions of Ringer lactate were made over periods of 15 minutes, the rate of infusion being 20 ml. per kg. per infusion. The results are recorded in the following table and when the CVP, PVP, arterial blood pressure and the heart rate are illustrated in a graph, it is seen that the venous pressures rise and fall in time with the infusion whereas the heart rate and the arterial blood pressure remain stable, (Fig. 7). The PCV and the total serum protein levels altered as expected, decreasing during the infusions and recovering later. The urine output in this dog was markedly increased over the period of the infusion,

Hour 0 - 1	59.3	ml./ kg.
" 1 - 2	50.6	"

It would therefore appear that this dog was overinfused and that the renal function was satisfactory under anaesthesia. Within 30 minutes of the end of the infusion, 89 % of the infused fluid had been excreted. This does not compare with the work performed in humans by Stewart and Rourke/



Rourke (1942) who noted that 80 % of infusions were retained in the body for 4 days.

Table - Preliminary experimental dog results

Preliminary experimental dog

Ringer Lactate infusion 600 ml.

Time (hrs)	0	15	30	45	1	15	30	45	2	15
Pulse /min	130	130	140	135	135	135	130	135	140	135
Resp. /min	8	8	8	8	8	8	8	10	8	8
Syst. B.P.	110	110	110	110	110	110	110	110	110	110
Diast.B.P.	65	70	70	70	70	70	70	70	70	70
Mean B.P.	85	90	90	90	90	90	90	90	90	90
mm.Hg										
PVP mm.Hg	10	14	11	16	11.5	15	11.5	14	11	12
CVP mm.Hg	-1	2	0	4	0	3	1	4	1.5	1.5
Urine output	0	80	320	660	890	1010	1240	1370	1650	1870
Fluid infusion	0	600	600	1200	1200	1800	1800	2100	2100	2100
PCV ml%	37	30	33	28	32	28	30	26	30	33
TSP	60	47	49	40	41	37	40	37	53	57
Oral MM	M	M	M	M	+	+	+	+	+	+
Eyes MM	M	M	M	M	M	+	+	++	++	++
Nose MM	M	M	M	M	M	M	M	M	M	M
Feet Oedema	--	--	-	-	-	-	-	-	-	-
Flank Oedema	-	-	-	-	-	-	-	-	-	-
Auscultation	-	-	-	-	-	-	-	-	-	-

Fluid

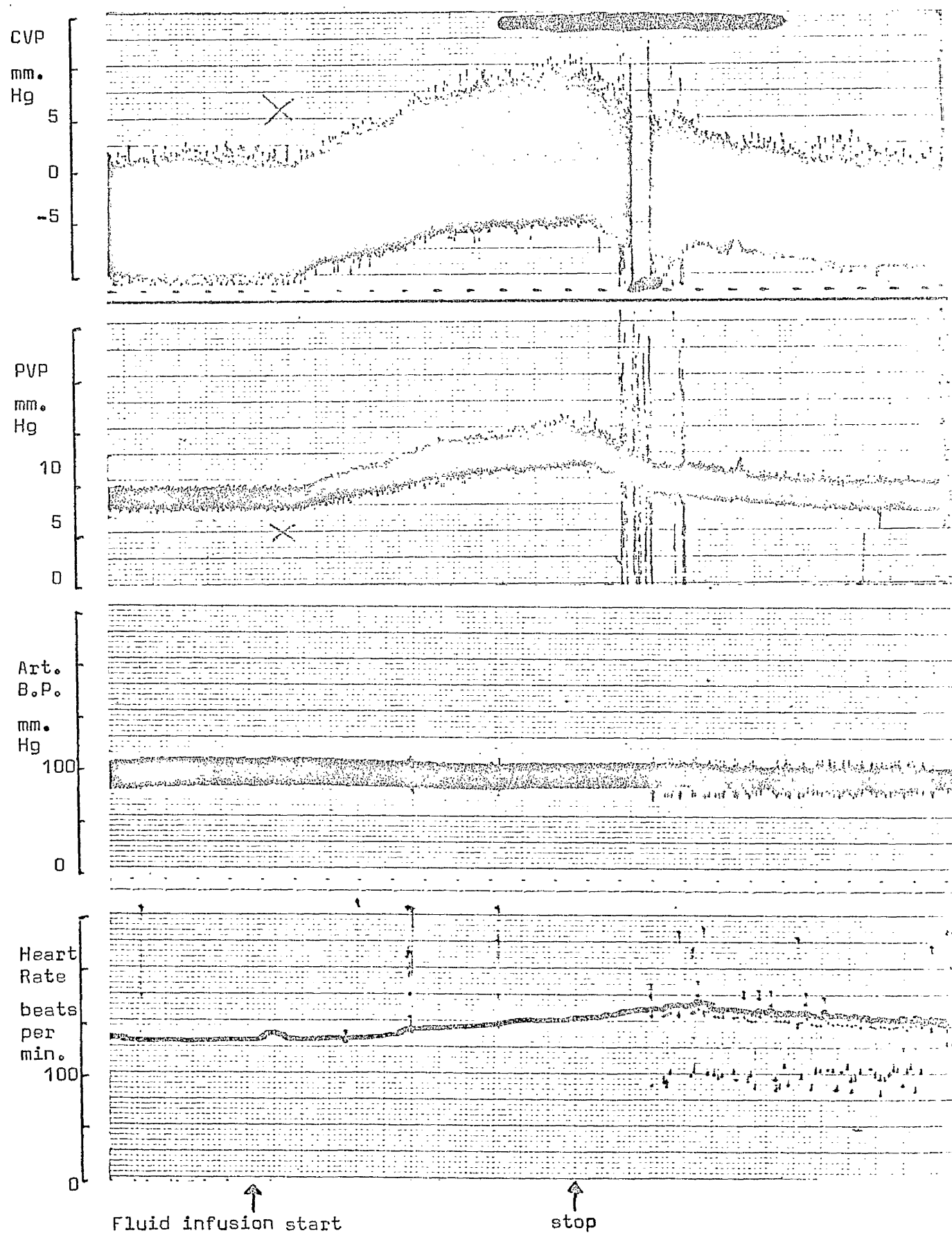
Fluid

Fluid

Fluid

Fig. 7.

Pressure recordings from dog.



( In the following section, all graphs and tables of results referred to in the text may be located in Appendix 2. )

A. Fluid input/urine output

The fluid input in dogs 1 to 12 was constant over the four hour period of infusion, followed by a test dose in 15 minutes of the hourly dose. The fluid inputs and urine outputs were recorded and the output was calculated as a percentage of the input and illustrated in graphs for the 12 dogs, ( Graphs 1 - 4 ).

The urine output rate was also calculated over the period of the infusion and the results are given in a table, ( Table 1 ).

The input in dogs 13 to 16 was with dextran 40 followed by Ringer lactate and the urine output was monitored during these procedures. The output as a percentage of the input is illustrated in a graph, ( Graph 5 ).

The urine output rate was calculated and is recorded in a table, ( Table 2 ).

The output of urine from all the dogs increased above the normal 1 ml. per kg. per hour. The rate increases in all the dogs within the first hour of crystalloid infusion and by the four hour point most dogs had excreted some 40 - 50 % of the fluid administered.

It would appear from the figures that except in the infusion of colloid, the kidneys are capable of a rapid increase in output. In most instances the higher outputs were associated with the higher inputs.

There appeared to be little relationship between the output of urine and the infusion of crystalloid, all types of fluid being excreted fairly/

fairly rapidly. The order of rapidity was

- |    |                 |            |
|----|-----------------|------------|
| a) | Ringer lactate  | most rapid |
| b) | 5 % dextrose    |            |
| c) | dextrose saline |            |
| d) | normal saline   | slowest    |

When dextran 40 was administered there was a lower than normal output of urine until the infusion of Ringer lactate commenced. This is possibly due to the hypertonicity of the solution, although the increase in arterial blood pressure might have been expected to produce an increase in the output of urine.

Theoretically any increase above an urine output rate of 1 ml. per kg. per hour is a sign of overinfusion and hence one may deduce that all the dogs involved in this experiment were overinfused.

#### B. Blood pressures

The systolic, diastolic and mean arterial blood pressure, the central venous pressure and the peripheral venous pressure were monitored every 15 minutes. From the results graphs were completed demonstrating the trends in pressure changes, ( Graphs 6 - 10 ).

During the four hour infusion, when infusing crystalloid the PVP and CVP either remained static or increased slightly, there being little correlation between the rate of infusion and the changes. In most dogs the PVP and CVP paralleled each other and the type of fluid infused did not appear to cause any specific changes. The increases in both/

both pressures were of the order of 3 mm. to 8 mm. Hg. The significance of such increases will be discussed later.

During the infusion the arterial blood pressure increased in the majority of dogs and most of the increases were within the first hour of the infusion.

During the test dose period the CVP and PVP showed an increase directly associated with the rapid infusion and there was an immediate return to normal after the infusion stopped. The increases in both venous pressures were parallel and there was some relationship between the rate of infusion and the changes recorded, since the largest changes were seen at the higher rates. It would appear that the body circulatory system had some reserve to handle the final assault since the pressures rapidly returned to normal. The arterial blood pressure during the test dose period altered little if at all. The changes in the arterial blood pressure appear to be of little significance when infusing crystalloids.

The infusion of colloid caused changes in all the pressures apparently related to the rate of infusion. In all the dogs the rise was most rapid during the first 30 minutes. The largest increases were in the arterial blood pressure which rose on average from 80 mm. to 140 mm. Hg. The changes in PVP and CVP paralleled each other and were related to the rate of infusion.

In the period of crystalloid infusion to those dogs after the infusion of colloid, the arterial blood pressure remained static. The CVP and PVP remained stable or decreased and there was no relationship between the rate of infusion and the changes since during the test dose, the venous pressures showed little change.

The/

The administration of colloid appeared to cause a retention of fluid within the body and a stabilisation of pressures at a level above the resting point. The addition of crystalloid did not appear to alter this, and the pressures remained relatively constant.

In the prevention of overinfusion it appeared that arterial blood pressure was not a useful guide and that the venous pressures were doubtful indicators.

#### C. Blood gases

The results were obtained from arterial samples taken hourly during the initial infusion, and every 15 minutes during the test dose and recovery phases. The partial pressure of arterial oxygen and carbon dioxide are considered.

The  $\text{PaO}_2$  varied from 400 mm. to 500 mm. Hg at the start of the infusions of crystalloids to only as low as 380 mm. Hg at the end of 4 hours at the highest rate. There was a fall in the level in all the dogs over the four hour period but the size of the decrease is of doubtful significance. When the circulatory test dose was administered, there was always a fall in the  $\text{PaO}_2$  but none of the levels was below 360 mm. Hg. The levels always rose after the test dose to the previous level.

When infusing colloid at various rates, the  $\text{PaO}_2$  fell slightly and then stabilised with the infusion of crystalloid, except in dog 16 where the level fell dramatically to 170 and then 105 mm. Hg and did not rise post-infusion. This dog showed marked respiratory embarrassment clinically.

The  $\text{PaCO}_2$  level in all the dogs varied little and this value did not appear to be significant in the determination of overinfusion.

D Chest radiography

Lateral chest radiographs were taken at regular intervals during the infusions. The films were viewed and assessed for the signs of pulmonary oedema. A scale of 1 to 5 was used, 5 being the most severe form seen. The nature of the oedema seen is illustrated, ( Fig. 8).

At low rates of infusion the occurrence of pulmonary oedema appears to be slight in development whereas at high rates there is often radiographic evidence of lung changes. The low infusion rate dogs were used as controls to determine if there were radiographic changes associated with the prolonged period of lateral recumbency. It was demonstrated that there were few if any changes and the conclusion was that posture did not increase the risk of pulmonary oedema in dogs of this weight and size.

There did appear to be a relationship between the type of fluid infused and the occurrence of oedema. When infusing 5 % dextrose solution, there were signs of oedema earlier than with other fluids.

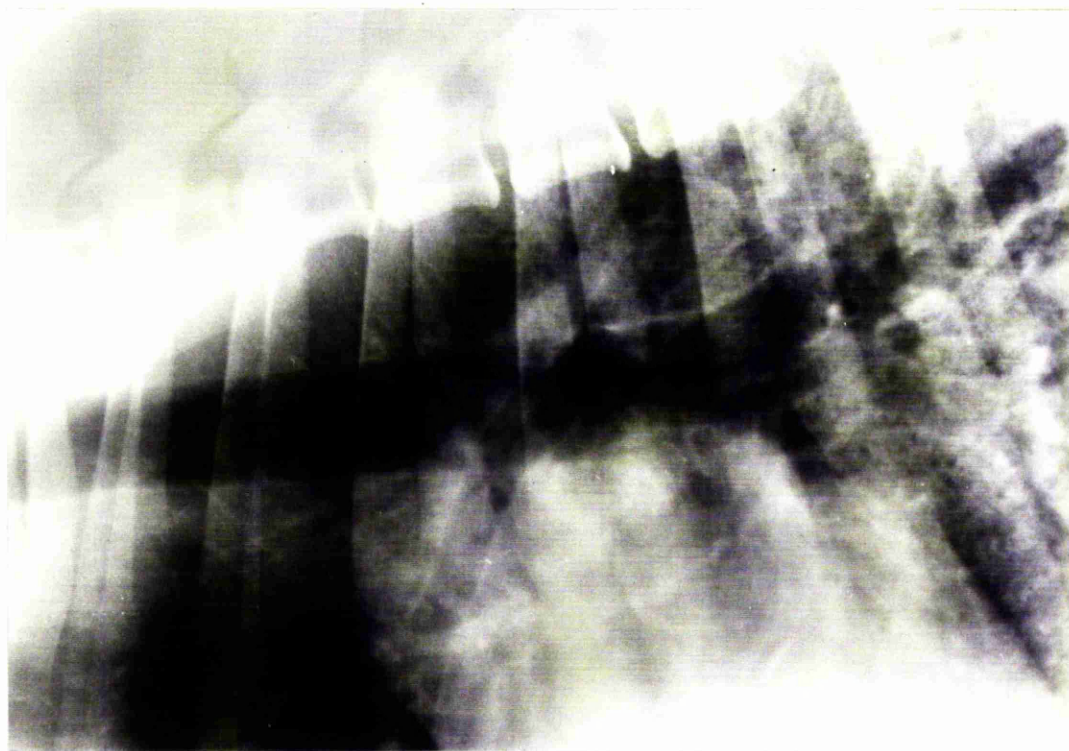
During the period of the test dose the incidence of oedema increased, but the changes were transient and the oedema resolved rapidly once the infusion had ceased.

When infusing colloid 2 of the 4 dogs showed mild changes in the lungs radiographically, and when Ringer lactate was infused, oedema readily occurred, it being most severe at the highest rate of dextran 40 infusion. It is postulated that the increased circulating blood volume in these dogs causes fluid to be expelled from the circulation into the tissues, especially the pulmonary interstitial spaces.

From this investigation it appears that chest radiography has an important part to play in the detection of overinfusion pulmonary oedema,



Fig. 8. Pulmonary oedema - experimental dog number 12.



E. Haematocrit and total serum protein

These assays were performed to determine the degree of haemodilution occurring in these dogs undergoing fluid infusion.

When infusing crystalloids at set rates the degree of haemodilution is directly related to the rate of infusion, and in these dogs it appears that there is an initial large decrease followed by a slower decrease over the following period. The test dose caused a fall in both the PCV and the total serum protein but this was transient, the levels returning to the previous level within 15 minutes.

When infusing colloid, the values fell initially and then stabilised with the infusion of Ringer lactate. The degree of haemodilution was marked in all the dogs receiving colloid.

The degree of haemodilution was expected in all the dogs, although it was of interest to note that the decrease associated with the colloid was marked and that it did not further decrease with crystalloid infusion. This may be because of the persistence of the colloid in the circulation and the hypertonicity of the solution used.

In the detection of pulmonary oedema, these two parameters are of doubtful clinical significance because of the availability of analysing equipment and the retrospective view that the technique permits.

F. Serum electrolytes

Sodium, potassium and chloride levels were estimated during the infusions to determine the changes occurring and to determine which of the solutions/

solutions best maintained the serum electrolyte levels.

The crystalloid which best maintained the serum levels of the electrolytes were normal saline and Ringer lactate, the latter being more capable. When normal saline was administered, the levels of sodium and chloride increased in the blood, whereas when Ringer lactate was infused, the levels remained relatively constant.

When dextrose solution and dextrose saline solution were infused, the levels fell dramatically, especially with dextrose alone. These changes could be attributed to the degree of haemodilution since the greatest decreases were seen at the highest flow rates. These changes were transient and there was some degree of recovery during the recovery phase of the experiment.

When infusing dextran and Ringer lactate there was little change in the serum electrolyte levels.

The assay of serum electrolytes has little place in the detection or prevention of overinfusion. The point of interest in this study was that Ringer lactate did not affect the levels when infused, even at high flow rates, whereas the sugar containing solutions caused dramatic changes which could be attributed to the degree of haemodilution.

#### G. Clinical signs

The clinical signs were monitored at regular intervals and changes were seen in most. The rectal temperature varied little during the infusions of fluid. The heart rate initially increased with the infusion of all the fluids and stabilised after one hour of fluid administration. The/

The greatest increases were with Ringer lactate, but the changes were not related to the rate of infusion. Its use as a method of preventing overinfusion is doubtful.

The respiratory rate in most dogs receiving crystalloid infusions increased slightly and stabilised. During the test dose the rate increased in all the dogs but returned to normal within the 15 minute recovery phase. The changes were not related to the rate of infusion.

When infusing colloid, the respiratory rate increased initially and at the higher rates of infusion on the addition of Ringer lactate, there was a dramatic increase in respiratory rate, and in two of the dogs the respirations became laboured.

The use of the respiratory rate as a means of preventing overinfusion is possible since a dramatic increase in respiratory rate occurs when severe pulmonary oedema is present.

The auscultatory sounds were few even when there was radiographic evidence of pulmonary oedema. The use of a stethoscope as a method of detecting overinfusion is doubtful, and it certainly could not be used to prevent overinfusion.

The severity of the signs exhibited by the mucous membranes were directly related to the rate of fluid infusion. The common signs were of increased moisture of the eyes and mouth and eventually oedema. There was also generalised oedema present in some of the dogs, seen especially in the prepuce, feet and flanks.

When infusing fluids it would appear that the clinical signs could be used as a method of assessing the body fluid status since the changes seen in this study were related to the rate of fluid infusion.

The/

The areas of maximum change were the mucous membranes of the eyes and the mouth. The respiratory rate could be used, and if it increased associated with the other changes and perhaps generalised oedema, overinfusion could be diagnosed.

## DISCUSSION

The object of the experimental study was to devise a clinically applicable method of detecting and preventing the state of overinfusion. The work was based on information from literature regarding studies into pulmonary oedema and was to investigate the condition of subclinical pulmonary oedema as a method of predetermining the occurrence of overinfusion.

The experimental model was successful in that it did produce adequate degrees of overinfusion which could be detected by radiography and permitted an assessment of other parameters. These dogs were relatively normovolaemic initially and the rates of infusion were designed to cause overinfusion, thus determining at what rate fluid may be administered without causing the recipient embarrassment. The parameter most suited to normal veterinary practice was sought since this is principally a clinical study and any information derived must be of use to practitioners.

Pulmonary oedema was caused by the infusion of crystalloids at rates greater than 20 millilitres per kilogram per hour. The infusion of fluid at 20 and 30 millilitres per kilogram per hour could be maintained for three hours in all but one dog without causing radiographically detectable pulmonary oedema changes. The exception was 5 % dextrose solution at 30 millilitres per kilogram per hour which caused changes within one hour of the commencement of the infusion. The most dramatic oedema seen radiographically when infusing crystalloid was with Ringer lactate at 40 millilitres per kilogram per hour over four hours and 5 % dextrose solution over four hours. In most of the dogs the test dose of fluid caused some degree of oedema which resolved within 15 minutes of the end of the infusion. It thus appears that Ringer lactate, 0.9 % Na Cl and 0.18 % Na Cl + 4.3 % dextrose/

dextrose solution can be infused at up to 30 millilitres per kilogram per hour for 4 hours without causing pulmonary oedema. This amount, 120 ml. per kilogram, would account for 3 days total requirement of fluid for a dog and it is unlikely that greater quantities of fluid would be administered within this time intentionally. Clinical cases would most probably be deficient in body fluid and therefore could more readily accept the infusion of fluid if all body systems were functional. Renal and cardiac incompetence could provide problems and in these cases more care should be taken. At 40 millilitres per kilogram per hour one may infuse fluid for up to one hour without causing more than minimal oedema. It is more advantageous to use lower flow rates over longer periods since greater quantities of fluid may be administered overall.

Using colloid and crystalloid infusions, pulmonary oedema occurred more readily, especially when large quantities of dextran 40 were infused followed by Ringer lactate. A maximum rate of 10 millilitres per kilogram per hour followed by 20 millilitres per kilogram per hour for one hour is recommended for colloid and crystalloid respectively. It is important to be aware of the effect of hypertonic colloid and how crystalloid infused after colloid may cause pulmonary oedema which is severe at high flow rates of infusion.

The other parameters were then considered to determine which would best indicate the changes seen radiographically or show signs of deterioration and overinfusion earlier.

The methods used most commonly in human medicine and veterinary practice for the prevention of overinfusion and pulmonary oedema when infusing/

infusing large quantities of fluid intravenously are central venous pressure monitoring and blood gas analysis. Radiographs are also commonly used in human intensive care units, but as this study shows, X-rays only become useful once there is something to visualise, and by the time there is evidence of pulmonary oedema, overinfusion has occurred. A negative radiograph merely indicates that the pulmonary clearance mechanisms are still functioning adequately. The test doses of fluid used to assess the competence of the body circulation demonstrate how rapidly pulmonary oedema can occur and be detected radiographically. One would be ill advised to use radiography as a routine method of monitoring since to be effective plates are necessary at least every 15 minutes. If one is infusing fluids to patients there is often no indication for lateral X-ray plates of the chest and hence rarely is there a base line film for comparison. In human intensive care and therapy units all patients have initial chest X-rays taken, firstly to give a base line, secondly to check for pulmonary pathology and thirdly to check the position of the central venous cannula if one is inserted. This procedure in veterinary practice is uneconomic and since it is decided that CVP monitoring is of doubtful use, the need for radiography is negated, other than in cases of suspected pulmonary pathology.

The use of radiography, therefore, is to determine the presence or absence of pulmonary oedema in cases where overload with fluid is suspected. It has been demonstrated that in these normal experimental models that if pulmonary oedema is radiographically obvious, the discontinuation of infusion when infusing crystalloids results in rapid resolution. When the blood pressure is increased due to the infusion of colloid, and then crystalloid is infused causing pulmonary oedema, resolution/



resolution takes longer.

This study aimed at the detection of sub-clinical pulmonary oedema, and since radiography demonstrates only the existence of oedema, other methods were investigated. It is interesting to note that the detection of respiratory incompetence by thoracic auscultation is apparently a poor method of assessment since abnormal sounds were only heard when there was quite severe oedema of the lungs.

Central venous pressure is widely used in human hospitals in intensive care and therapy units and operating theatres to determine the circulatory capabilities during intravenous infusions. Patients with cardiac incompetence show marked changes in CVP with the infusion of fluid, and therefore overinfusion could readily occur. The techniques used are numerous and all have inherent problems, mostly associated with the placement of the cannula within the great veins of the chest.

In veterinary medicine similar procedures are recommended by some authors, but the monitoring of CVP has never become accepted in general veterinary practice. The changes in CVP in the experimental dogs were compared with the radiographic presentation. Although with the infusion of fluid, particularly crystalloids, there is a general increase in CVP over the four hours investigated, the increases were not related to the flow rate, whereas the radiographic appearance was directly related to the infusion rate. In some instances the rise in CVP was not an indication of pulmonary oedema, and there is no clear relationship between a certain value of CVP and overinfusion. This questions the validity of CVP monitoring when attempting to prevent overinfusion. A marked rise in CVP seems to be related to pulmonary oedema with all the crystalloids infused, but/

but there was no apparent relationship between the size of the increase, the severity of the radiographic appearance and the infusion rate. When infusing colloid there is a marked increase in CVP but no change in the radiographic appearance. When crystalloid infusion was added after the colloid to the same dog, the CVP tended to fall or remain stable, whereas the radiographic appearance changed dramatically indicating oedema, especially at the higher rates of colloid infusion. The use of CVP monitoring in these models was proved unsatisfactory in the determination of pre-clinical pulmonary oedema.

The use of CVP monitoring is still possible in animals receiving fluids intravenously, but one must consider many factors before deciding that overinfusion has occurred. It is impossible to state that a certain increase in venous pressure would be indicative of overinfusion pulmonary oedema. When infusing crystalloids and colloid, the CVP fell when oedema was apparent radiographically, although the high level of infusion of colloid caused no appreciable oedema initially without the crystalloid infusion. The use of CVP monitoring in these cases is not recommended.

It has been demonstrated that peripheral venous pressure almost exactly parallels CVP in dogs. It was therefore decided that PVP measurement could be used instead of CVP monitoring. Since the techniques involved in establishing a PVP monitoring system are reasonably straightforward, it is recommended that if venous pressure be monitored at all, PVP be used. Any animal receiving intravenous fluid therapy invariably has a needle or cannula inserted into a peripheral vein and this can be used to permit the measurement of PVP as well as the infusion of fluid. This means that should the possibility of overinfusing a patient arise, the manometer unit can be connected/

connected to the already present administration set without any disturbance of the patient and little inconvenience to the staff. The only problem in this system is to set the correct zero point dependent on postural position.

There is a need in patients which are suspected of having cardiac incompetence to monitor venous pressure when infusing large volumes of fluid intravenously and it would perhaps be of more benefit in these patients to administer fluid slowly and with care thus avoiding any unnecessary strain on the cardiovascular system.

This study supports the beliefs of Frank (1969) who stated that interstitial pulmonary oedema or generalised diffuse pulmonary oedema can occur without change in CVP when infusing crystalloids. Frank stated that this is more important in older patients or those with an already incompetent circulation. Wet lung formation has been recorded in many patients without appreciable rises in CVP. Berman and Spencer (1972), stated that an animal can be infused to twice its blood volume in one hour without an appreciable rise in CVP but overinfusion pulmonary oedema was evident radiographically. These two workers rarely detected harsh sounds or rales by auscultation even when marked wet lung formation had occurred, a fact recorded regularly in this study.

It is my recommendation that the use of CVP monitoring be carefully assessed before increasing the risk to patients, (Adar 1976, MacGovern 1976). These authors, and others, recorded fatalities and serious potentially fatal sequelae due to the insertion of CVP measurement lines. The risk involved does not appear to be warranted in any patients of any species. If it is desirable to monitor venous pressure, PVP can be monitored without much risk to the patient.

The/

The monitoring of arterial blood pressure was performed in the experimental model by a direct method. Various indirect methods are possible in the dog using cuff systems on the tail or limbs. In this study the arterial blood pressure varied little in association with the infusion of fluid. In most dogs there was an increase in pressure unrelated to the rate of infusion, and the significance of arterial blood pressure measurements when attempting to determine pre-clinical pulmonary oedema is doubtful. It would appear from these experimental dogs receiving crystalloids that the circulatory system was capable of handling large quantities of water and electrolyte. The output of urine was increased and this was partly due to the increase in the arterial blood pressure. It is interesting to note that in the dogs which were infused with colloid, the arterial blood pressure rose markedly, but the output of urine did not until the infusion of crystalloid. The colloid used, dextran 40, was in a 10 % hypertonic solution and appeared through tissue dehydration to make the whole body hypertonic, thus preventing an output of urine due to the change in osmolarity. The osmolarity increase would cause the increased release of antidiuretic hormone from the posterior pituitary. It is only when isotonic fluid is added that urinary flow commences, the rate being high presumably due to the increased arterial blood pressure and increased glomerular flow. The blood pressure in the dogs infused with colloid remained high and stable during the infusion of crystalloid demonstrating the ability of dextran to remain in the circulation and maintain the circulatory volume. The venous pressure in these dogs increased, presumably due to the increase in the blood volume and venous return to the heart.

Therefore it is concluded that the measurement of arterial blood pressure/

pressure has little place in the clinical prevention of overinfusion.

It has been stated previously in this study that some authors have shown changes in the level of oxygen in the arterial blood with over-infusion. Arterial blood gases are monitored in human patients in intensive therapy units to assess respiratory function and it was thought of value to monitor these gases in the experimental model. The partial pressure of oxygen in the arterial blood,  $\text{PaO}_2$ , was found to decrease with the increasing presence of wet lungs, (Frank 1969, Sykes, Adams, Finlay, Wightman and Munroe 1970, Berman and Spencer 1972). It was found in this experimental model that there was a correlation between the radiographic findings and the  $\text{PaO}_2$  in most dogs. This agreed with other workers, and it can be stated that as overinfusion pulmonary oedema of a generalised form occurs, there is interference with the alveolar arterial oxygen gradient such that oxygen uptake is reduced. The effect was minimal with the infusion of crystalloid alone and no particular fluid was found to be responsible for the creation of pulmonary oedema. The effect of colloid infusion followed by crystalloid caused the most interference with pulmonary function at the higher rates of colloid infusion. The  $\text{PaO}_2$  fell markedly in these dogs. It is useful to know that the radiographic plates taken in this study show some correlation with this parameter. This helps to confirm that the experimental models were overinfused and that they might be creating pre-clinical pulmonary oedema.

From the experimental models it appears that after the test dose of fluid, the  $\text{PaO}_2$  rose again in most instances during the recovery phase, indicating that the changes were only transient. This means that under normal circumstances the discontinuation of infusion will result in some degree/

degree of oedema resolution. In conscious dogs, the degree of interference experienced by these dogs might lead to cyanosis.

$\text{PaO}_2$  is the level of oxygen within the plasma and the percentage saturation of the blood with oxygen depends on the haemoglobin level. The oxygen content of the blood is the total amount of oxygen contained in the blood and plasma. It is therefore noted that animals which were already anaemic would not be greatly affected unless the percentage saturation was altered which would only occur if there was a large decrease in oxygen uptake. The reason for the fall in  $\text{PaO}_2$  is the presence of pulmonary oedema due to overinfusion. Overinfusion with crystalloid fluids interferes with the fluid clearance system of the lungs which is controlled by the hydrostatic and osmotic pressures across the capillary walls in the alveoli. The hydrostatic pressure is determined by the action of the heart and the venous pressure in the body, since an increase in the amount of fluid present within the venous system means the movement of more fluid by the right side of the heart through the lungs. Therefore the hydrostatic pressure would increase in an overinfusion state.

The lungs have an elaborate fluid clearance system based on lymphatics which depends on the osmotic pressure within the alveolar capillaries. This osmotic pressure appears to be due to the presence of a proteinaceous substance, (Harpster 1974). Normally the alveolar membrane is impermeable due to the presence of surfactant, but this may be overcome by changes in osmotic pressure. When crystalloid overinfusion occurs, there is haemodilution which causes a reduction in osmotic pressure throughout the body. This change is more apparent where the balance between hydrostatic pressure and osmotic pressure is fine, a situation which occurs at all times/

times in the lungs. If the hydrostatic pressure in the pulmonary capillaries increases as the osmotic pressure decreases, especially if the decreased pulmonary osmotic pressure is secondary to haemodilution, and the protein content of the fluid in the capillaries is decreased, fluid will cross the alveolar capillary membrane. This results in free fluid in the alveoli.

The effect of surfactant would be overcome by this change in pressures and the presence of free fluid in the alveoli interferes with oxygen uptake. This situation would be more likely to occur when colloid is infused increasing the arterial blood pressure and the pulmonary hydrostatic pressure, followed by the infusion of crystalloid causing reduced osmotic pressure secondary to haemodilution.

As the surface area of the alveoli is reduced by the presence of free fluid due to overinfusion, there is interference with the alveolar arterial gradient which in turn would affect the V/Q ratio, where V is the ventilation of the lungs and Q is the perfusion of the lung tissue. The effect created by overinfusion would be of reduced ventilation since free fluid in the alveoli causes a disturbance in V and this would then result in a decreased uptake of oxygen. Perfusion would remain the same in these dogs since there would be no factor present affecting blood flow other than the additional fluid.

Hypercapnia did not occur since carbon dioxide is twenty times more diffusible than oxygen.

The use of the measurement of  $\text{PaO}_2$  in clinical veterinary practice is nil and although it would be a useful method of preventing overinfusion, it is not generally possible or even economic to do so.

In this progression, or apparent regression, of monitoring aids, the blood "picture" demonstrated by the serum electrolytes, the total serum/

serum protein and the haematocrit is now assessed. These measurements have no place in general veterinary practice in the monitoring of patients to attempt to prevent overinfusion. Their use for this purpose even in human hospitals is doubtful due to the cost and the time requirement to perform the assays, except the haematocrit which can be estimated rapidly by the micro-haematocrit method.

Haemodilution was expected in the experimental dogs due to the infusion of fluid which would fill the cardiovascular system, and it was shown that the degree of haemodilution was directly related to the rate of infusion. The haematocrit fell in all cases, the greatest decrease being within the first hour of the infusion and with the test dose of fluid. It was noted that following the test dose, recovery was rapid. The total serum protein fell evenly over the period of four hours and gave a better estimate of haemodilution in most dogs. The type of crystalloid used caused little difference in the changes seen, but it appears that the infusion of colloid caused a more dramatic fall in the total serum protein, again associated with the rate of infusion.

Although they are accurate methods of assessing the degree of haemodilution, their use as a means of preventing overinfusion is extremely doubtful.

The assay of serum electrolytes revealed that although dilution took place, 0.9 % Na Cl solution and Ringer lactate solution best sustained the levels of sodium, potassium and chloride in the plasma. Ringer lactate achieved the best results presumably due to its close approximation in content to plasma. 5 % dextrose solution and 0.18 % Na Cl in 4.3 % dextrose solution caused decreased levels of sodium and chloride which would be considered/



considered as deficiencies in clinical cases.

There is no place for these assays in the prevention of overinfusion.

The output of urine in the normal, healthy dog should be approximately 1 millilitre per kilogram body weight per hour, and any increase in this value is either due to renal disease, systemic disease or overinfusion. The experimental anaesthetised dogs gave marked increases in urinary output which were compared with the radiographic appearance to determine if there was a relationship between the rate of infusion, the output of urine rate and the presence of pulmonary oedema.

The higher outputs of urine were associated with the higher rate of infusion, and there appeared to be a relationship between the urine output and the radiographic appearance. The high outputs were found when there was pulmonary oedema demonstrable on the radiographs. The relationship was not a linear one, but rates over 20 millilitres per kilogram per hour were associated with marked oedema. Theoretically any rate above 1 millilitre per kilogram per hour for the output of urine is a sign of overinfusion, and perhaps one could monitor patients suffering from deficiencies of the body fluids by their output of urine. Talbot et al (1953) stated that any excess water administered to patients with normal kidney function was excreted and was a sign of the adequacy of infusion. It is suggested that an output of urine of greater than 2 millilitres per kilogram per hour be set as an upper limit, and that any animal exceeding this limit be deemed as overinfused. The cases seen clinically would have to be carefully assessed since any interference with cardiac function or renal function could upset the rate of urine output. If one was satisfied that the renal and/

and cardiac systems were competent, the monitoring of fluid infusion by determining the output of urine would give not only an indication of overinfusion, but also of the adequacy of the infusion. It seems that the output of urine rate increased before any of the other valid parameters, and since the study was to determine a method of preventing or detecting overinfusion, renal output would seem a likely candidate.

The process of bladder catheterisation in the dog is relatively straightforward and the complications are few, cystitis being perhaps the most apparent. Strict adherence to hygiene when passing a catheter and careful aseptic maintenance of the catheter and the collection unit should limit the incidence of bladder and urinary tract infections. The use of urinary output as a means of preventing overinfusion in animals without nephropathies which decrease the renal outflow is recommended, and the use of other clinical signs in conjunction with the renal flow will enable one to determine the kidney function in dogs where oliguria or anuria persist.

In conscious dogs the clinical signs of overinfusion recorded in the literature are venous distension, cyanosis, pulmonary oedema and elevation of CVP. In the experimental dogs venous distension was not appreciated in any of the animals despite the high flow rates and the radiographic appearance of pulmonary oedema. If venous distension occurs after the severe phase of pulmonary oedema recorded in these dogs, it is a clinical sign of doubtful use in determining overinfusion. It was not possible to visualise cyanosis since the dogs were breathing 99 % inspired oxygen, and there requires to be a fall in oxygen saturation of haemoglobin before the blue colouration is created.

The/

The heart rate, although increased in most of the dogs, was not related to the rate of infusion or the degree of oedema visualised radiographically and clinically. The heart rate did increase markedly with the infusion of colloid, but since these were normovolaemic dogs initially, this increase was due to direct overloading of the circulation and this might not occur in hypovolaemic dogs receiving dextran therapeutically.

The relationship between the respiratory rate and the radiographic appearance of the lungs was not dependent on the infusion rate. There was an increase in the respiratory rate in the dogs once there was evidence of pulmonary oedema. As a method of detecting subclinical pulmonary oedema, respiratory rate does not appear satisfactory.

The auscultatory sounds heard in these dogs were not satisfactory in determining the presence of wet lungs. In most instances quite marked oedema was radiographically present before even increased respiratory noise was heard. Rales were only recorded in the dog infused with 40 millilitres per kilogram of colloid followed by Ringer lactate solution. It therefore appears that if increased respiratory noise can be detected by auscultation, oedema is present. It is also important to know what respiratory sounds were evident prior to the commencement of the infusion.

When infusing crystalloids the severity of general body oedema was related to the rate of infusion. The common sites for oedema were the oral mucosae which became soft, spongy and very wet. The eyes became bulbous due to oedema of the conjunctivae and there was increased tear production. The external nares became soft and spongy with pitting oedema on digital pressure in some cases. There was a nasal discharge of fluid in dogs receiving the high rates of infusion. The other sites where oedema was/

was evident were the feet, especially the toes which became splayed and the prepuce. The lower flank of the abdomen was noted to be oedematous in certain cases, once again related to the infusion rate. The presence of foot or flank oedema is a definite sign of overinfusion and is to be avoided. The signs exhibited by the mucous membranes were related to the flow rate, and once the eyes became bulbous, one could consider that overinfusion had occurred.

The general assessment is worthwhile when attempting to detect the presence of overinfusion, or when trying to prevent its occurrence.

After consideration of the previous comments, it is possible to discuss what conclusions might be reached regarding the prevention or detection of overinfusion pulmonary oedema.

The infusion of fluids to animals by the intravenous route is now an essential part of veterinary therapeutics. It is important to be able to assess this treatment and to prevent any iatrogenic side effects at the same time. Overinfusion is a risk which can be avoided by the use of certain pieces of equipment, monitoring and by not exceeding safe flow rates in the administration of fluids. The methods of all three have been discussed and it appears that there are safe means of administering fluids to animals, particularly the dog.

Careful clinical assessment and accurate history taking are primary considerations in cases with fluid deficiencies and these can enable one to pre-determine the fluid requirement, the selection of the correct fluid and the choice of infusion equipment. The use of a graduated burette chamber as part of the administration set is strongly advised, and/

and with proper flow control is efficient in preventing accidental overinfusion. A flow controller which does not alter in setting is advised to give some accuracy in the flow rate.

Once the correct equipment has been selected, the risk of overinfusion is reduced. The next important factor is the rate of infusion and from the information in this study, some maximum rates can be determined which in normal dogs should not lead to overinfusion pulmonary oedema.

The recommended maximum rates are,

- i) For crystalloids 30 ml. per kg. per hour for 4 hours
- ii) For colloid 10 ml. per kg. per hour for 2 hours

The infusion of colloid and crystalloid should be controlled and monitored to prevent the formation of pulmonary oedema, and the formation of tissue dehydration when infusing dextran.

The monitoring aids considered in this study were those used routinely and others. It appears that the monitoring of CVP is of doubtful value and if such pressures are required, the measurement of PVP would appear more satisfactory, but still of doubtful use in preventing overinfusion. The monitoring of  $\text{PaO}_2$  values would be of some value but expense is a major drawback. Radiographic methods are positive once overinfusion pulmonary oedema is present. For this reason it seems that the close monitoring of renal output against fluid infusion is of value when considered as a whole with the clinical signs. Most cases of body fluid deficiency which require the intravenous administration of fluids have reduced outputs of urine and dry, congested mucous membranes. When fluids are/

are infused one attempts to replace the losses and not to inundate the body with water and electrolyte.

Hence, if one can re-establish a satisfactory renal output of less than 2 millilitres per kilogram per hour and the mucous membranes are wet, then satisfactory rehydration is in progress and eventually other signs such as a decreased tissue turgor and the sunken eyes should disappear. At a renal output of 2 millilitres per kilogram per hour the risk of overinfusion should be minimal unless there is a disruption of renal function. In cases of nephropathy, PVP could be monitored during fluid infusion, and the administration of diuretics is advisable to promote a diuresis.

Overinfusion did not become detectable by any other of the methods used in the experimental dogs at urinary flow rates below 5 millilitres per kilogram per hour. The normal figure of 1 millilitre per kilogram per hour is to be aimed for, since any output above this is theoretically an indication of overinfusion. Once a steady urine output of 1 to 2 millilitres per kilogram per hour is achieved over a period of four hours, rehydration of the correct form could be deemed complete. The type of fluid used is also of importance since the addition of water to an electrolyte deficient animal will result in an output of urine to maintain osmolarity. For this reason it is best to use electrolyte solutions for the hydration of patients.

It is concluded that overinfusion can be prevented by,

1. The use of a graduated burette chamber as part of the administration set.

2. The use of an accurate flow regulator.
3. Not exceeding 30 ml. per kg. per hour with crystalloid solutions.
4. Not exceeding 20 ml. per kg. per hour with colloidal solutions.
5. Monitoring the output of urine.
6. Monitoring the clinical signs.
7. Monitoring PVP when indicated.

## PART 2 - CLINICAL



## SECTION 1

## HISTORICAL BACKGROUND

During my undergraduate studies, I was fortunate enough to have the opportunity to observe activities in both human and veterinary hospital wards and operating theatres, and although there were many similarities, there were noted some differences. One of these differences was the parenteral fluid therapy given to support patients undergoing surgery. Nowadays it is commonplace to find body fluid disturbances in many human surgical patients being corrected by basic parenteral fluid therapy, whereas, in my own experience, this seems to be the exception in general veterinary practice.

As a student "seeing practice" , I was given encouragement and support by two practitioners to administer some supportive fluid therapy to many cats and dogs. In this initial study, isotonic saline, dextrose solution, whole blood and a proprietary electrolyte-amino acid-vitamin complex infusion were used. Fluid was administered pre-operatively or during operations and it was noted that animals receiving such treatment were more willing to eat and moreso to drink post-operatively, than those denied such treatment. The intravenous route was used whenever practicable but the less satisfactory subcutaneous and intraperitoneal routes were used in some small dogs and cats. These initial thoughts formed the background for a further study of fluid therapy.

In human hospitals, supportive fluid therapy is used routinely in the correction of body fluid imbalances and to maintain those patients, who for some reason cannot accept their daily requirements orally. The intravenous route is used almost exclusively being the one most satisfactory to patient and staff. The reasons for fluid therapy are/

are numerous, including the replacement of massive blood loss in accident victims, the correction of gastro-intestinal losses and the maintenance of the unconscious patient who is unable to eat or drink. The fluids used vary from a basic saline solution to modern parenteral nutritional complexes which provide fluid and nutritional requirements to sustain normal life.

In veterinary practice few animals receive an intravenous infusion of fluid since the use of parenteral fluid therapy seems to be the exception. However, increasing use of supportive fluid therapy is being made, and perhaps practitioners will accept the concept of parenteral fluid therapy now that equipment and fluids are readily available and easily used.

Fluid therapy is not a recent innovation in medical or veterinary practice. Harvey (1628) described the circulatory system of mammals thereby facilitating the advance of techniques of injection into the body circulation. Potter (1650) attempted blood transfusions with varied success, but Sir Christopher Wren (1656), who incidentally was not medically trained, improved intravenous techniques, thus enabling Lower (1660) to give many successful blood transfusions to humans. During the eighteenth century religious beliefs were more readily accepted than medical opinions, causing a temporary halt in the furtherance of fluid administration techniques. It was not until the early nineteenth century that the infusion of fluid and blood into human beings was accepted, partly due to the success of a young Edinburgh general medical practitioner who treated cases of cholera at Leith by infusions. This practitioner, Thomas Aithchison Latta, wrote of his success/

success describing the method of treatment and giving lucid details of the techniques and fluids used, (1831). Latta was the first doctor recorded who administered saline intravenously to many patients near to death due to cholera, and his success rate was astonishing. The saline solution he used consisted of "water saturated with the protoxide of nitrogen and a solution of the muriate and bicarbonate of soda in the proportion of half a drachm of the former and eight grains of the latter to the pound of water drained through shammy leather". Latta found that the fluid had to be given at a temperature between 98<sup>o</sup>F. and 115<sup>o</sup>F., otherwise at lower temperatures the patient complained of cold rigors and at higher temperatures the heart rate increased with the patient assuming a flushed appearance. Fluid was administered till urine was voided and the patient was able to eat and drink normally. The technique used by Latta was reviewed by Masson (1971) who discovered that the Leith doctor was indeed the pioneer of saline infusions given intravenously, using the methods of O'Shaughnessy described in London, (1831). Gold or ivory tubes were inserted into the external jugular vein, Latta finding that prior penetration of the skin by a lance made insertion of the cannula easier - perhaps the first cut-down technique. Infusion was made using an enema syringe and glass reservoir.

Wheatcroft (1858) successfully treated cases of uterine haemorrhage by blood transfusion, using a technique of pumping blood directly from donor to recipient, by means of a syringe pump with unidirectional valve, and the blood was kept warm by a vessel containing hot water. Wheatcroft used a cut down technique onto the basilic vein, inserting a silver nozzle attached to a rubber pipe from a syringe. It was stated that it was important to have the nozzle pointing towards the/

the heart for correct infusion. Problems arose with this method when blood was caused to flow in the wrong direction or air was introduced into one or other person involved if a mechanical rather than a hand pump was used. It was not in the best interests of the medical profession to have donors dying from air embolism.

Richardson (1866) praised Latta for his treatment of cholera using intravenous infusions and furthered the knowledge of the medical profession by using a new type of replacement fluid which restored blood volume and fluidity. He also indicated the necessity to reduce the fluid losses from patients and used opium as the new drug of choice. This author experimented with many parenteral fluids, discovering that milk was not recommended due to its poor mixing with blood and rapid decomposition. Water alone did not sustain the patient satisfactorily, but saline did, the latter being the fluid of choice. Richardson's own fluid which he used with success was so-called Artificial Serum made from the following :-

- 4 ounces of white of egg
- 1 drachm of salt
- 1 scruple of bicarbonate of soda
- 1 ounce of clarified animal fat
- 2 ounces of pure glycerine

made up to one pint with water. A pressure system with reservoir and hand pump was used for infusions.

Accompanying the advances in fluid therapy, the search for improved methods of infusion continued and Messers. Salt and Son of Birmingham/

Birmingham (1875) developed a pump and syringe unit which contained a ball to prevent air embolism by occluding the end of the fluid reservoir exit pipe. Messers, Charles Traux and Co. of Chichago (1888) demonstrated a hand roller pump which was effective for infusions or aspirations, being operated for either procedure by means of a large wooden handle. This was called the Allen Surgical Pump after its American inventor. As recently as 1941, MacIntosh and Pask described a pressure system for intravenous infusion, using oxygen from a cylinder through a reducing valve and flowmeter to provide the necessary pressure. They also described a new steel cannula which passed through a slightly wider but shorter introducing needle, thus preventing repenetration of the vein. Pumps were still being used to infuse fluids prompting a report by Simpson (1942) outlining the side effects of air embolism, resulting in many fatalities. The most common cause of air embolism was faulty equipment or lack of nursing supervision during infusions. Blood donors occasionally died from air embolism, one case being recorded in 1941 where the electric pump action was reversed, filling "the kind and thoughtful donor who was aiding the war effort" with air intravenously.

From the end of the Second World War in 1945, fluid therapy and its accompanying technology progressed rapidly in medicine reaching the sophistication of today when most equipment is plastic, complex, yet easy to use and is disposable. Fluids are prepared in sterile units for easy use, and technology continues to advance with increasing knowledge of the physiology and pathophysiology of body fluids.

The veterinary profession has followed the medical profession in many aspects of fluid therapy, and it was not until the second half of the twentieth century that authors seriously suggested the routine administration/

administration of fluids to domestic and sometimes wild animals in captivity. Fortunately much of the research carried out to further medical knowledge involved the use of cats and dogs, thereby providing a direct benefit to the veterinarian. Many physiology books provide information regarding the normal dog and cat as experimental animals, hence a reasonable understanding of their body fluids is possible. Pure veterinary fluid therapy is only briefly described in the literature.

Hollis (1952) presented a paper at a scientific conference in North America, stating that parenteral fluid therapy had come to play an important role in the practice of veterinary medicine. His conclusions were that the manufacture of sterile, pyrogen free, disposable equipment along with the selection of fluids available would make fluid therapy safer and convenient, and be of considerable therapeutic benefit to veterinary patients. The use of fluid therapy was mentioned in "Canine Surgery" by Hewitt (1952), where the administration of salt solutions and whole blood was advocated in small animals to replace losses and to compliment other therapeutic considerations, such as post-operative starvation. Armistead (1954) stated that the use of fluids, particularly blood transfusions and electrolyte infusions in cases of pyometra in the bitch were of great benefit to patients, especially when shock was either present or was to be anticipated during surgery or in the post-operative period. The recommended dose of electrolyte solution was 10 to 15 cc. per pound body weight to sustain renal function.

In "Canine Surgery" (1965), Kirk stated that there was a need for fluid therapy in acutely ill patients to support and maintain life. Hall (1967) describes the simplicity of fluid therapy giving instances of common deficiencies, and indicates the need for monitoring, especially/

especially central venous pressure, (CVP). In "Canine Surgery" (1974), Sattler, MacDonnell and Presnell gave accounts of treating acutely ill patients where support was imperative and involved the monitoring of C.V.P., arterial blood pressure, electrocardiograph (ECG) and body temperature. The treatment of shock by various techniques using fluids and drugs such as vasoactive agents, corticosteroids, antibiotics and bicarbonate was described. Finally the correction of electrolyte and water losses and acid/base imbalances pre-operatively and post-operatively were illustrated. "Current Veterinary Therapy", edited by Kirk (1974), contains articles on basic fluid therapy and shock, stating that the greatest need for the parenteral administration of fluid was in acutely ill patients, and that the monitoring of various parameters was imperative during such treatment.

Various articles have been written by a number of authors regarding fluid therapy in all species and most state similar opinions to those reported above. Indeed the majority of veterinary papers have been written in the last twenty-five years and they emphasise the need for the administration of fluids parenterally to all animals where deficiencies exist or shock prevails.

As the use of fluid therapy has increased in both medical and veterinary practice, methods of monitoring have been sought to estimate blood volume and infusion success or failure. Central venous pressure has been accepted by the medical profession in the past thirty years as being reliable in evaluating fluid therapy, but despite advice given to veterinary surgeons, little use of C.V.P. monitoring has been made in small or large animal practice. The reliability and value of C.V.P. monitoring is discussed further in this study, but one must consider why fluid therapy is not fully utilised by veterinarians, let alone monitoring/



monitoring techniques. More use is now being made of parenteral fluid administration, but only when the intravenous route is used almost exclusively, is it possible to monitor venous pressure satisfactorily. The skill required to insert intravenous apparatus allowing fluid infusion takes little time to acquire, and it is only one step further to institute some form of monitoring. Burrows (1976) stated that the measurement of C.V.P. had become an invaluable aid in estimating the fluid volume requirements of the seriously ill patient, and more importantly, the shocked patient. If the technique of assessing fluid therapy success or failure was even simpler than C.V.P. measurement, perhaps more use of this alternative technique would be made by veterinary surgeons.

SURVEY OF FLUID THERAPY USAGE IN VETERINARY PRACTICE

To determine the use made of parenteral fluid therapy in general veterinary practice, a survey of practitioners domiciled in the main areas of population in Scotland was carried out in January 1976. The survey was in the form of a questionnaire containing seven questions with multiple choice answers, the results being recorded and analysed with a short discussion.

From the initial analysis it seems that some practitioners made varied use of the available parenteral fluids, but few treated many cases or even saw animals in a state of dehydration or shock frequently. A large proportion of veterinary surgeons do not appear to be aware of the wide range of parenteral fluids available and how to use them fully.

The survey results are recorded in Appendix 3. The following short discussion assesses these results.

## MATERIALS AND METHODS

Questionnaires were sent to 125 general veterinary practitioners domiciled in Scotland, mostly in populated areas where small animals would be seen as part of everyday practice. Many of the practitioners were full-time small animal veterinary surgeons within the central belt of Scotland. By controlled mailing an attempt was made to ensure that only one questionnaire was returned from each practice.

Names and addresses of the practitioners were obtained from the British Small Animal Veterinary Association register of members within the Scottish region, and additional names were located in the register of the Royal College of Veterinary Surgeons under the geographical location section for Scotland.

All questionnaires were posted in the second week of January 1976 and had to be returned by the 1st. of March 1976 if the replies were to be considered. A stamped addressed reply envelope was included with each questionnaire and this seemed to add to the success of the survey in that the number of replies compared more than favourably with other surveys, (Dodman, 1976, Onamegbe, 1976). A letter of introduction and explanation accompanied each questionnaire, and to increase the validity of the analyses, all practitioners were urged to reply, whether or not they used fluids parenterally in treating small animal cases.

The letter and questionnaire were :-

Dear Practitioner,

As part of a study designed to clarify the  
current/

current position of fluid therapy in small animal practice,  
the enclosed questionnaire has been constructed.

I would be grateful, therefore, if you could complete and return the questionnaire whether or not you use fluids, as the success of the survey depends on the returns made by practitioners.

Yours Sincerely,

Another sheet contained seven questions laid out with multiple choice answers to enable rapid and easy replies. Answers were made by ticking ✓ the appropriate box, or in some cases, specifying a technique or substance.

1. Do you treat dehydrated or shocked patients  
by the parenteral administration of fluids ?      Yes.....  
No.....
2. Indicate which routes you would use.
- a) Intravenous.....  
b) Subcutaneous.....  
c) Intraperitoneal.....  
d) Other (specify).....
3. Indicate fluids used,
- a) NaCl (0.9%) Normal saline...  
b) Dextrose (5%).....  
c) Ringer Lactate/  
Hartmann's Solution.....  
d) Dextran - plasma substitute.  
e) Whole Blood.....  
f) Other (specify).....

4. Approximately how many dehydrated or shocked patients do you see ?

- a) 1 per week,.....
- b) 1 - 5 per week,.....
- c) 5 - 10 per week,.....
- d) 10 - 20 per week,.....

5. What proportion of these receive fluids parenterally ?

- a) 0 - 25 % .....
- b) 25 - 50 % .....
- c) 50 - 75 % .....
- d) 75 - 100 % .....

6. When do you administer fluids ?

- a) On admission,.....
- b) Pre-operatively,.....
- c) During Operation,.....
- d) Post-operatively,.....

7. How do you assess the degree of dehydration or shock ?

- a) Clinically,.....
- b) Packed Cell Volume,.....
- c) Blood Urea,.....
- d) Other (specify),.....

## DISCUSSION

The number of replies returned in this survey was excellent and covered most of Scotland's small animal practitioners. Some of these were members of the B.S.A.V.A. and others were domiciled in populous areas. It was hoped to give a cross section of ages of practitioners, differences of opinion in the assessment of cases, in treatment and the timing thereof, in methods of administration and in the type of small animal practice as defined by locality. This, it was believed, would represent the feeling of general practitioners towards fluid therapy.

This survey is intended to show that some interest exists in general practice for parenteral fluid therapy but that there is a lack of information as to when, how and what to infuse in cats and dogs. These prime factors may determine the overall success of therapy and an understanding thereof is essential.

From the replies obtained, 95% of general practitioners claim to use fluids parenterally in small animals and this is surprising considering the available information on veterinary fluid therapy. It is noted that this high percentage is from a 73.6% return and in fact may represent the use of fluids parenterally in practice. Despite the letter of introduction requesting information from all, only four replied indicating that they did not use fluid therapy. It is felt that this figure might have been greater had the other 26.4% of practitioners replied. This stems from my belief that a failure to reply probably implies non-involvement in the use of parenteral fluid therapy. Nevertheless it is enlightening to learn that so many practitioners are taking some interest in the problem of fluid balance in intensive care cases. With increasing discussion /

discussion, problems will inevitably arise, but solutions will undoubtedly be found.

The routes for the input of fluids are numerous - oral, intragastric, rectal, intraperitoneal, subcutaneous and intravenous. In this study only parenteral administration is discussed and from the survey results it became clear that the subcutaneous route is most popular presumably because of technical ease, speed and lack of necessity for continual supervision, in contrast to intravenous infusion. Despite the problems of intravenous infusion in small animals, 78% of practitioners do use this route. Only a small percentage (37%) use the intraperitoneal route. The last choice in the answers sought information regarding other routes, but none were described.

It is interesting that only 6% of practitioners use intravenous infusion exclusively, 15% subcutaneous infusion exclusively and 1% intraperitoneally exclusively. This implies that most practitioners employ at least two of these routes, 39.5% use intravenous and subcutaneous administration and 25.5% use all three routes. The reason is that some animals can be infused intravenously with ease and especially when under anaesthesia for surgery, whereas others require physical restraint and often an alternative method of administration.

The intravenous route is preferable for all parenteral fluid therapy, and with the methods of administration now available it is relatively easy. It permits accurate control of the flow rate and is mandatory for certain fluids which cannot be given by other routes, ( whole blood, plasma expanders, plasma substitutes and hypertonic electrolyte solutions ). In addition, where certain fluids can be infused/

infused by alternative routes, the intravenous route almost always results in earlier or more satisfactory response. Its use is necessary in all emergency therapy as in haemorrhage, acute anaemia or dehydration with or without circulatory failure, and the group of practitioners (22%) who do not use this route are possibly at a disadvantage.

Most practitioners use the subcutaneous route which is suitable for the administration only of certain fluids for maintenance purposes, but is not suitable for the resuscitation of cases of massive haemorrhage or acute dehydration accompanied by compensatory peripheral vasoconstriction. Chemical irritation is likely to occur in the subcutaneous tissues when this route is used, unless hyaluronidase is added to the infusion. The subcutaneous administration of some fluids such as dextrose or "Duphalyte" causes pain and irritation. This route may be used in small cats and dogs where there is difficulty in the location of veins and the maintenance of cannula therein.

The intraperitoneal route is used by few practitioners for the administration of some fluids, but it is not recommended in view of the possible complications and sequelae such as peritonitis, mechanical damage to the body wall and internal organs, and later, formation of peritoneal adhesions. Abdominal discomfort may be caused when large volumes of fluid are given intraperitoneally.

A wide range of fluids is available commercially. From the results only 84% of practitioners do use electrolyte solutions ; that this figure is not 100% is disappointing since electrolyte solutions are often necessary when correcting body fluid imbalance. The main ions present in the body are sodium, potassium and chloride, hence one must supply these when/



when undertaking therapy, 67% of general practitioners use 0.9% NaCl which is the simplest form of electrolyte solution. In many instances a more physiological solution is compound sodium lactate (Ringer lactate) which supplies sodium, potassium, chloride, calcium and bicarbonate. Only 16% of practitioners use this fluid. Dextrose solution is a source of water and a limited quantity of energy, (790 k.joules per litre) and 50% of practitioners use 5% dextrose in water.

The ability to treat shock, massive haemorrhage and anaemia were analysed. Dextran is a plasma volume expander which is readily available in different molecular sizes (40,70,110,150) and may be used to replace blood loss. It also has properties such as the prevention of platelet agglutination and blood cell sludging as can occur in shock. Only 9% of practitioners use this useful fluid. The other fluid of use in the treatment of haemorrhage, shock and anaemia is whole blood which can be obtained from other cats and dogs fairly readily and be transfused without real difficulty. 24% of practitioners use whole blood.

48% of practitioners use other solutions such as "Duphalyte", "Ionalyte", "Aminosol", Darrow's solution and laevulose. "Duphalyte" is popular and 31% of practitioners use it, 13% being exclusive users. The recommended volume of this preparation to be administered daily is small and should not be exceeded. "Duphalyte" contains electrolytes, amino acids, vitamins and some sugar, but the quantities are insufficient for fluid and nutritional replacement. It must also be stated that as is the case with "Aminosol", energy in the form of fat or carbohydrate are essential for amino acid utilisation. Hence infusing "Aminosol" on its own is of little value as there is no calorific source, Darrow's solution contains/

contains additional potassium and should be infused with care to avoid cardiac disturbance ; however only 3% of practitioners use it. Laevulose is fructose and a similar calorie value to glucose, but does not require breakdown in the liver prior to utilisation.

It is surprising that of the 95% of practitioners using parenteral fluid therapy, only 5% use all the fluids listed in question 3. 84% of general practitioners use electrolyte solutions and 30% use whole blood or plasma expanders or substitutes. Clearly some practitioners use both these groups of fluids, but it is felt that more use could be made of the available fluids in the treatment of differing fluid requirements. There is little point in having at hand only one or two types of fluids since individual cases require individual treatment and there will be a suitable fluid for each, be it in shock, anaemia or dehydration. With newer methods, the administration of fluid has become relatively easy and can be embarked upon with confidence.

The total number of intensive fluid therapy cases seen in practices in Scotland is small considering the common events like road traffic accidents, abdominal catastrophes and pyometras. Some road accident cases will have recovered from the initial "shock" where there is no haemorrhage by the time they are presented to the practitioner, and will not be considered to be intensive therapy cases. Many accident cases improve without treatment or at least do not deteriorate in general condition over the first 24 to 48 hours, and the necessity for fluid therapy may not exist.

Most of the acute surgical cases would recover sooner if given parenteral fluids, but some practitioners, however, anticipating spontaneous recovery, may not consider these cases as meriting intensive fluid/

fluid therapy. The types of surgery performed in a practice may also determine the parenteral fluid therapy requirements. For instance, in oesophageal and abdominal surgery there is a greater emphasis on fluid therapy including parenteral nutrition.

On analysis it would appear that at present practitioners do not recognise many body fluid imbalance cases, but with increasing knowledge and practical application thereof, this number will certainly decrease.

On consideration of the cases receiving fluids, 34% of practitioners treat 75% to 100% of the cases seen, whereas 29% of practitioners treat only 0 to 25% of the cases seen. Broadly, 50% of practitioners treat less than half their cases and 50% treat more than half. No specific group of practitioners treat all their cases, as might be expected where a practice sees a large number of cases per week and may be more proficient in treatment. This is not the situation, and the groups of practitioners seeing large and small numbers of cases fall into all the "number treated" groups. The reason for this is not clear and would require fuller investigation. One explanation may be that where large numbers of cases are seen, fluid therapy is instituted only after careful assessment and those cases not requiring therapy are better defined. In contrast, the practitioner seeing one occasional case may or may not treat it depending on available equipment and fluids.

The timing of treatment is important and many cases will withstand surgery better and have a more satisfactory post-operative period if fluid imbalance is corrected on admission or pre-operatively. 74% of practitioners treat animals on admission and presumably these patients/

patients are in need of urgent treatment. The largest percentage (81%) of practitioners treat cases post-operatively suggesting poor recovery of cases due to surgical interference or a failure to restore fluid balance pre-operatively. This large number of practitioners do treat cases post-operatively, but 9% of practitioners treat patients only during this phase. This would suggest a failure in initial assessment and the early institution of parenteral therapy as may be evidenced by a prolonged post-operative recovery.

A problem existing in general practice is the shortage of trained staff to monitor intensive therapy cases. Animals on such therapy should have 24 hour attention, but this is not always possible and for this reason fluids tend to be administered at times suitable to the staff. Unless dogs or cats are totally collapsed or are tranquillised, which is not always advisable, some form of restraint may be necessary during fluid administration. As newer methods of administration are discovered, it may be possible to give fluids parenterally over a 24 hour period which would be of greater benefit than intermittent fluid therapy.

It would be of interest to know which methods of restraint and which types of administration units are used in conscious animals in general practice.

The clinical assessment of dehydration and shock is important but some ancillary aids are now available. It was hoped that some practitioners might mention methods other than the estimation of the packed cell volume (PCV) and blood urea, but only one did and this practitioner stated that he hoped to use C.V.P. monitoring soon. Clearly clinical/

clinical assessment is important with particular reference to pulse volume, pulse rate, colour and consistency of mucous membranes, capillary refill time, respiratory rate and nature, urinary output, tissue turgor and overall appearance. History is also important and owners should be questioned meticulously for details of the incident.

P,C,V. estimation is a useful measurement and can be obtained by the micro-haematocrit method used by some practitioners. Blood urea level can be determined by a commercial stick test or, better still, by the "Unitest" method. The results of the survey suggest a lack of facilities in general practice provision of which must be aimed for in the future.

## SECTION 2

## BODY FLUID PHYSIOLOGY

In simple terms, two-thirds of the world's surface area is covered by water and the remaining one-third comprises minerals, proteins and fats. It is of interest that the animal body is similar in construction, and this is now described indicating the position of these substances, the quantity present and the controlling mechanisms affecting them.

An understanding of an animal's body fluid physiology is essential if any form of fluid therapy is to be correctly and safely managed. Many different techniques have been used to determine body fluid composition ranging from exsanguination and dessication (Bischoff 1863, Robertson 1938, Hardy 1958) performed on dogs and executed criminals, to complex dilution techniques using radio-isotopes and tracer dyes, (Krieger, Storaasli, Friedall and Holden 1948, Black 1953, Williams and Fine 1961, Shires, Williams and Brown 1961, Miller, Stoetling and Paradise 1963, Roth, Lax and Maloney 1967, Stahl 1967, Gutelius and Shizgal 1968, Roth, Lax and Maloney 1969, Anderson 1969, Doty, Hufnagel and Moseley 1970). The majority of these studies were performed on humans in intensive care units and on dogs under experimental conditions.

As yet a complete picture is not available of the total watery environment of the cells or the total quantity of substances in solution with their varying functions, but it is hoped in this section to outline and explain some of the knowledge available to the veterinary profession.

A normally functioning body is in a state of homeostatic equilibrium which is only too easily disturbed by extraneous demands and insults varying from external environmental conditions to the total/

total dysfunction of certain internal organs. These changes will become evident in the section on pathophysiology.

In all animals between one-half and two-thirds of the body weight is represented as water, described as the Total Body Water, (T.B.W.), which is used as a base line in the determination of other parameters. This value, although relatively constant, theoretically changes every time an animal breathes since water is lost to the external environment. Nevertheless it is sufficiently constant to permit its use as a base line.

Body fat has a lower content of water and hence the percentage of the body weight that is present as water falls as the degree of obesity rises. This largely accounts for the variation in the percentage of the body weight that is accounted for by water in different individuals. It also explains the higher water content of young animals compared with most adults. Experimental work, (Liebman and Edelman 1959), has shown that young animals have little if any storage fat. An assessment of body fat content can be made on body conformation or with the use of calipers, (Durnin and Rahaman 1967).

There are two types of fat found within the animal body,

- i) Structural Fat
- ii) Storage Fat

The structural fat or permanent fat is located in all bodies surrounding the nerves, in joints and in the brain. Storage fat is the remaining fat found to a variable degree in animals and it is this that determines the total body water as is seen below, (from Wilkinson 1955).



	<u>Body Weight</u>	<u>Body Solids</u>	<u>Storage Fat</u>	<u>T.B.W.</u>	<u>T.B.W.</u>
	kg.	kg.	kg.	l.	% body weight
Basic					
Structure	20	6	0	14	70
Average	25	6	5	14	56
Obese	30	6	10	14	46

It has been noted that in older humans the total body water content is less than expected, (Wilkinson 1955, Garret 1970).

For convenience the T.B.W. is considered as being located within body "compartments", although these communicate freely with each other to maintain osmolarity and the body homeostatic equilibrium.

The compartments are, (Elkinton, Gilmour and Wolff 1939, Edelman and Liebman 1959, Horsey 1974),

- A. Intracellular Fluid (I.C.F.) which is contained within the body cells and represents 55 % of the T.B.W..
- B. Extracellular Fluid (E.C.F.) which comprises 45 % of the T.B.W. and is further divided into,
  - 1) Interstitial Fluid (20% T.B.W.) which is located between the cells and acts as a medium for the transport of electrolytes, cell nutrients and metabolic waste products.
  - 2) Non-functional E.C.F. (15% T.B.W.) contained in dense connective tissue and is so-called for the purposes of fluid therapy because its movement/

movement is so much slower than the E.C.F. at other sites, (Elkinton, Gilmour and Wolff 1939).

- 3) Intravascular Fluid (7% T.B.W.) which is the major component of plasma. It is located within the cardiovascular system and it permits the transport of nutrients and excrements in the body. This fluid is in constant equilibrium with the interstitial fluid which is its most common neighbour.
- 4) Transcellular Fluid (3% T.B.W.) which is found within the synovial spaces, as cerebrospinal fluid , aqueous in the eye and in the bladder, (Hall 1967, Horsey 1974). Also considered by other authors as being transcellular fluid are free fluid in the abdomen and chest, and respiratory water vapour, (Wilkinson 1955, Bell, Davidson and Scarborough 1962, Garret 1970). It has been suggested that saliva, gastric and intestinal secretions be considered as transcellular fluid, (Selkurt 1966). These fluids are generally manufactured at the site of function and are reabsorbed by complex filtration mechanisms.

The interstitial fluid, the intravascular fluid and the transcellular fluid are sometimes termed the functional E.C.F..

The/

The body fluid compartments have been determined mostly by dye or isotope diffusion techniques, the E.C.F. being relatively accessible and the I.C.F. relatively inaccessible. The non-functional E.C.F. has been found to be the most difficult to determine, (Hardy 1958, Horsey 1974). The I.C.F. is commonly calculated by subtracting the E.C.F. from the total body water, (T.B.W.), (Wilkinson 1955, Hardy 1958).

The intravascular fluid or plasma volume has been determined reasonably accurately. The blood volume in many species is relatively constant and is related to the T.B.W., (Garret 1970). The size of this compartment has been defined by various methods which are not discussed, (Ashworth and Tiegert 1940, Krieger, Storaasli, Friedell and Holden 1948, Penny 1953, Black 1953, Prentice, Olney, Artz and Howard 1954, Edelman, Liebman, O'Meara and Birkenfield 1958, Williams and Fine 1961). The intravascular fluid is usually 7 % of the total body water, and if the cellular component can be determined, the blood volume is known. The accepted volume in the dog is 80 ml. to 100 ml. per kilogram body weight.

Plasma can be described as the non cellular component of blood and is composed of water, electrolytes, protein, nutrients and excretory products. The protein is the component most responsible for the intravascular osmotic pressure which permits the reabsorption of fluid from the tissues.

In all the body fluid compartments electrolytes exist in ionic form either as cations or anions. These ions must balance at all times to maintain the state of chemical electroneutrality. The distribution of the electrolytes in the body compartments is set out in the following table. The electrolytes tend to remain in their set compartments/

compartments, though some can move freely from one space to another and this is known as the exchangeable or non-exchangeable property of the ions, (Garret 1970).

<u>Cations</u>	<u>Anions</u>	<u>I.C.F.</u>	<u>E.C.F.</u>	
			<u>Interstitial Fluid</u>	<u>Intravascular Fluid</u>
Sodium $\text{Na}^+$		3 %	95 %	90 %
Potassium $\text{K}^+$		90	3	4
Calcium $\text{Ca}^{++}$		2	1	3
Magnesium $\text{Mg}^{++}$		5	1	2
	Chloride $\text{Cl}^-$	1	70	70
	Bicarbonate $\text{HCO}_3^-$	4	20	15
	Phosphate $\text{HPO}_4^{--}$	50	6	2
	Sulphate $\text{SO}_4^{--}$	10	1	1
	Proteinate	35	0	8

It is concluded therefore that the principal ions of the I.C.F. are potassium and phosphate, and the primary electrolytes of the E.C.F. are sodium and chloride. The intravascular electrolytes can be determined by direct blood sampling and analysis. The accepted serum or plasma electrolyte levels in the dog are contained in Appendix I.

The transcellular fluid varies in composition depending on the location and the function.

The principal electrolytes are now described outlining their location/

location, function and estimated content in the normal body.

Sodium Sodium is located mostly in the extracellular fluid, there being a cellular mechanism to prevent it entering the cells, (Hardy 1958). Within the body it is found in two moieties - exchangeable and non-exchangeable, 75 % being normally exchangeable and is involved in the continuing process of homeostasis. 50 % of the body sodium is located in bone and half of this is non-exchangeable. The functional E.C.F. contains 45 % of the body sodium and the I.C.F. the other 5 %. The total content has never been accurately determined, (Wilkinson 1955). Sodium is reabsorbed in the distal convoluted tubules of the kidneys at the expense of hydrogen and potassium ions. Sodium occupies a leading role in any body fluid imbalance, (Hall 1967).

Potassium Potassium is located predominantly in the cells, mostly in skeletal muscle, and it is the principal ion of the I.C.F.. It is almost totally exchangeable, especially when there is cell destruction or injury. Only 2 % of the total body potassium is located in the E.C.F. and it is readily excreted by the kidneys. High blood levels of potassium are toxic to the myocardium.

Calcium Calcium is found predominantly in bone and it is the most common mineral in animals. There is only a small blood content which is present in an ionised and non-ionised or protein bound form, the latter accounting for two-thirds of plasma calcium. It has important functions in the blood clotting mechanism and also muscle movement, (Garret 1970). It may have a sedative effect on the cells of the nervous system/

system, (Wilkinson 1955). Vitamin D controls its utilisation in the body, but unless there is adequate protein in the blood, uptake from the bowel is not possible. The role of calcium in fluid therapy is unknown in dogs other than in the treatment of eclampsia.

Magnesium            Magnesium is found in small quantities in the body, 65 % being intracellular and 35 % extracellular with 30 % of the plasma magnesium being protein bound, (Wilkinson 1955, Garret 1970). Its functions are in neuromuscular activity and it is known to be an important factor in the maintenance of normal muscle contractility and the excitability of neural tissue. High levels are found in the cerebrospinal fluid where neither its presence nor its function is understood, (Garret 1970).

Chloride            Chloride exists almost totally in the extracellular fluid. It is found in the gastric secretions as hydrochloric acid which has important sterilising properties. It is found in erythrocytes acting as a buffer in oxygen and carbon dioxide exchange, where it passes into the plasma when red blood cells are oxygenated, and this is known as the chloride shift, (Garret 1970). Its role in fluid therapy is important since it has an equilibrating effect on the quantities of sodium and potassium in the body.

Phosphate            Phosphate is the major anion of the intracellular fluid and very little is located in the extracellular fluid where it is controlled by vitamin D and parathormone. Its main location is in bone.

Sulphate/

Sulphate Sulphate is found principally in the cells associated with protein.

Protein Protein is found in the plasma and cells and is of importance in osmosis and in buffering cations. It is polyvalent in nature and is able to equilibrate with extra cations as necessary.

The maintenance of body fluid equilibrium in normal animals depends on the preservation of a state of balance between input and output - intake and excretion - both of which may vary. In discussing the daily requirements of an animal, it is easier to determine the losses. The normal water and electrolyte losses from the body are relatively constant, and can be divided into two groups, (Wilkinson 1955),

- A. Urine sometimes termed the sensible loss
- B. Inevitable loss which comprises
  - 1. Respiratory water vapour
  - 2. Faecal water
  - 3. Sweat

The term inevitable is used in preference to extra-renal or insensible loss since it best describes the loss. The losses are now discussed.

A. Urine The normal output of urine in a 24 hour period in the dog is 20 ml. per kilogram body weight, and this volume is required for excretion/

excretion of natural toxic metabolites such as urea. The kidneys achieve this normal loss by the filtration of the blood volume many times each day, and excretion depends on the osmotic load determined by the total water content and the concentration of toxic solute, (Wilkinson 1955). The kidneys have powers of concentration of urine to conserve body water, and this ensures the necessary excretion of solute, but there are limitations in disease.

Renal loss takes second place to the inevitable losses which determine the amount of fluid available for excretion as urine. The production of urine also depends on the glomerular filtration rate which in turn depends on arterial blood pressure and cardiac function. The production of urine, therefore, is a useful measure of body function with respect to renal and cardiovascular systems, (Wilkinson 1955, Black 1957, Garret 1970).

In a dog experimentally infused with Ringer Lactate solution and given twice the daily requirement of fluid, intravenously in 24 hours, it was shown that the output of urine trebled, the inevitable losses remaining unchanged, (Clark 1978).

B. Inevitable loss                      These losses are relatively constant and are primarily from respiratory water vapour in the dog, the loss in faeces and sweat being negligible. These losses amount to 20 ml. per kilogram body weight per day, and they determine the volume of urine in all circumstances, (Wilkinson 1955).

Obviously external environmental conditions such as humidity and temperature can affect the inevitable loss, but it is relatively constant.

The/



The daily requirement for water, therefore, is 40 ml. per kilogram body weight to maintain normal homeostasis and fluid equilibrium. The normal method or route of fluid gain is orally, and both water and electrolytes are consumed each day in food and as free water. Most foodstuffs contain 50 % to 95 % water and the oxidation of this food in the body releases further water and carbon dioxide. The electrolytes are contained in most foodstuffs and the exact quantities required each day are not fully known for the dog, but the following are estimated to be compatible with life in the dog, (Harrison, Sussman and Pickering 1960, Finco 1974),

Sodium	1.5	mmol.	per	kg.	per	day
Potassium	1.2	"	"	"	"	"
Chloride	1.0	"	"	"	"	"

Calcium is of importance in body homeostasis, particularly in young animals and lactating bitches where the need for the element is increased. Vitamin D is required for calcium uptake from the bowel and there must also be sufficient protein in the plasma for utilisation.

Phosphorus and magnesium are required, and are found in most foodstuffs.

The excretion of electrolytes is in both urine and faeces, the quantities for each being variable, dependant on intake, (Wilkinson 1955, Garret 1970).

The control of chemical electroneutrality, body pH and fluid equilibrium are now discussed.

The maintenance of the water and electrolyte content of the body/

body depends on the preservation of equilibrium between the input and the output, and this is controlled by various organs and systems in the body, (Wilkinson 1955, Hardy 1958). The kidneys are the main regulatory organs where exchange takes place, although the pulmonary alveoli act to a minimal degree, (Wilkinson 1955, Garret 1970). In functioning adequately, the kidneys have an inbuilt integrity, but they also require the coordinating control of other organs such as the pituitary, adrenal and parathyroid glands, the heart, the lungs and the blood vessels, (Garret 1970).

Electrolytes play an important physiological role in water distribution, osmotic pressure, muscular action and pH regulation which are all controlled in different ways. The methods of water and electrolyte movement are vitally important in maintaining equilibrium and electroneutrality. There are four basic mechanisms,

- A. Diffusion
- B. Active transport
- C. Osmosis
- D. Hydrostatic pressure .

A. Diffusion                      Diffusion is generally along a concentration gradient by ions to establish electroneutrality between two body spaces, and the movement is from a high to a low concentration, being achieved without the expenditure of energy.

B. Active transport                      Active transport is the opposite of diffusion, and ions are transported against the concentration gradient which/

which requires the expenditure of energy at the site of transport.

C. Osmosis                      Osmosis is the movement of water across cell membranes when there is a high concentration of solute on one side, and a low concentration on the other. The water moves from the lower to the higher concentration of solute, and the osmotic "pull" exerted is proportional to the concentration of particles in solution, (Garret 1970). Within the vascular system this factor causes the reabsorption of fluid into the circulation from the venous side of the capillary bed. Osmosis also takes place between the cells and the interstitial fluid since living cells are generally freely permeable to water and semi-permeable to some electrolytes.

D. Hydrostatic pressure                      Hydrostatic pressure is the force exerted by the heart through the arterial system which forces fluid into the interstitial spaces and thence into the cells. This is essential for the maintenance of the life support services to the tissues.

The principal body fluid regulatory organ is the kidney and once the inevitable loss is excreted, excess water is excreted in the urine under the control of hormones and the glomerular blood flow. Initially the arterial blood pressure must be such that glomerular filtration is adequate. Secondly sufficient water is required to permit excretion of toxic metabolites such as urea and associated products, and potassium, which if allowed to accumulate would endanger life. The output of urine depends on the amount of excess/

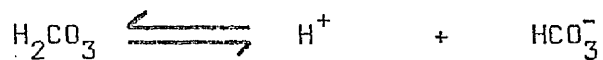
excess water in the body and also the concentration of solute, (Garret 1970).

The tonicity of body fluids in the dog is held between 280 and 300 mosmols. per litre by increased or decreased reabsorption of water via the mechanism of the kidneys and the permeability for water of the distal convoluted tubules and the collecting ducts. The permeability is determined by antidiuretic hormone (ADH) released by the posterior pituitary gland in response to stimuli from the supra-optic and paraventricular nuclei found rostrally in the hypothalamus, where blood tonicity is monitored. If there is haemodilution, that is the state of hypotonicity, ADH release is inhibited and dilute urine is excreted. In a state of hypertonicity, ADH secretion increases and the permeability of the tubules increases, allowing better water reabsorption. The anterior pituitary gland releases growth hormone which appears to be a controlling factor in maintaining glomerular filtration and kidney tubular function, (Fisher 1978).

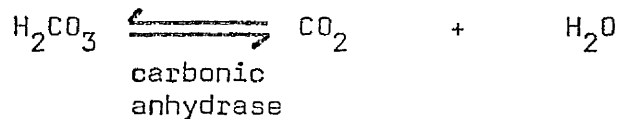
The volume of the extracellular fluid is controlled mainly by the sodium content, and this is determined by renal tubule reabsorption which is under the influence primarily of aldosterone. Aldosterone secretion is controlled by the secretion of renin from the juxtaglomerular apparatus. Factors affecting glomerular filtration pressure effect the secretion of renin. Aldosterone is a hormone released by the adrenal glands in response to adreno-cortical trophic hormone (ACTH) from the anterior pituitary gland under the control of stimuli from the hypothalamus in response to the sodium concentration in the blood. Aldosterone causes the reabsorption of sodium and the water absorbed with it. Chloride is also reabsorbed, but potassium is excreted. ACTH promotes/

promotes the release of aldosterone which conserves sodium, but its long term effectiveness is unknown, and appears to decline, (Garret 1970, Fisher 1978).

The pH of the body is maintained relatively constant by a number of buffer systems, the principal one being the carbonic acid/bicarbonate system. The plasma proteins also act as effective buffers due to the polyvalency of the proteinate ion, but this is not further discussed. The concentration of hydrogen ion in the body depends on the concentration of proton acceptor, principally bicarbonate. The products of body metabolism in the dog have an excess of hydrogen ions and the bicarbonate system buffers the pH, maintaining a balance prior to renal excretion. The kidneys regulate the pH by excreting bicarbonate or hydrogen ions according to the body needs, (Fisher 1978). In normal life the number of proton donors and proton acceptors should be equal, but should one or other alter, either bicarbonate is produced from carbonic acid or vice versa.



The lungs can affect the rate of excretion of carbon dioxide which is formed from carbonic acid.



This pulmonary function permits the excretion of carbon dioxide which reduces the proton donor content of the blood.

Metabolic/

Metabolic disturbances of the pH regulatory systems are due to many varied causes such as increased production of substances within the body, decreased elimination by the kidney, increased elimination by the kidney, increased absorption from the alimentary tract or increased loss from the alimentary tract. These can result in a metabolic acidosis or a metabolic alkalosis.

Respiratory disturbances of the pH regulatory systems are due to the increased or decreased excretion of carbon dioxide by the lungs. This can result in a respiratory acidosis or a respiratory alkalosis.

Respiratory defects are corrected by metabolic alteration and bicarbonate is excreted or retained by the kidney. Metabolic defects are compensated by respiratory alteration, carbon dioxide being expelled or retained, (Finco 1975).

## PATHOPHYSIOLOGY

" Adequate management of the surgical patient today demands a thorough knowledge of the changes in fluid and electrolyte balance incident to the operation itself ", (Shires, Williams and Brown 1961).

Having discussed normal body fluid physiology, certain pathophysiological imbalances are now discussed, which for many years have created intense interest among human and veterinary clinicians. There are numerous possible body fluid imbalances, but only those of major and of known significance clinically are mentioned. These may be divided into three groups,

- A. Water imbalance
- B. Water and electrolyte imbalance
- C. Blood volume imbalance

Some terms require definition. Losses or excesses may be either absolute ( where there is a true alteration in the body content ) or relative ( where there is a change in concentration but not in total body content ). The word imbalance is used to describe losses or excesses.

Imbalances may arise as a result of a change in one substance, for example, water, or in a group of substances, for example, gastrointestinal secretions or blood. The imbalances described are those most commonly seen during this study and are discussed under the three headings listed above.

A, Water imbalance

1. Adipsia
2. Anorexia and oligodipsia
3. Pyrexia
4. Hyperpnoea and tachypnoea
5. Overinfusion

1. Adipsia This is a failure to consume fluid and results in body water loss which is absolute and only occurs singly when there are present changes in environmental conditions or some forms of pathology. Examples are, of the former, water deprivation and accidents, and of the latter, oesophageal obstruction, oropharyngeal lesions and collapse where the consumption of water is not possible. This last cause is often combined with other fluid imbalances.

A reduction in total body water occurs due to the inevitable losses of 20 ml. per kilogram per day resulting in body fluid hypertonicity. the output of urine falls and anuria may develop if adipsia continues. Thirst is stimulated and there is drying of the mucous membranes and a decrease in tissue turgor. In experimental dogs, it was found that prolonged adipsia and anorexia caused a decreased plasma volume and cardiac output and a lowering of the arterial blood pressure, but the signs of circulatory failure were not seen until 30 % of the total body water had been lost - equivalent to zero intake for eight days, (Elkinton, Danowski and Winkler 1946). These authors stated that there was no loss of protein from the plasma. Due to the reduction in urinary output, urea accumulates in the body, but this state is easily reversed by treatment, (Marriot 1950)./



1950). Where animals are eating but not drinking, the blood urea level will increase more rapidly due to the metabolism of food.

Haematologically there is an increase in the haematocrit and haemoglobin level. Biochemically there is an increase in the serum electrolyte and plasma protein levels. The plasma potassium content may remain static or even decrease rather than increase since renal absorption of sodium commonly results in the excretion of potassium. The blood urea level may also be elevated. These changes are due to the depletion of plasma water and are usually relative. Any urine produced will be of high specific gravity.

2. Anorexia and oligodipsia This is a failure to consume food along with a reduced water intake. It is commonly found in animals which have some form of infectious or inflammatory process, for example, pyometritis, peritonitis or intestinal foreign body. Initially these animals are willing to drink and may be polydipsic, but eventually become oligodipsic as the condition progresses. The symptoms are similar to those of adipsia, but usually electrolytes are lost simultaneously. This is discussed under water and electrolyte loss.

The blood changes are similar to those seen in adipsia, but may not appear to be so severe since some fluid is being consumed.

3. Pyrexia An increase in the body temperature results in an increased loss of water each day. The increase is equivalent to 3 ml. per kg. per 24 hours for each centigrade degree rise in body temperature, (Hayes 1968). This is of importance when supporting an animal parenterally/

parenterally.

Pyrexia can be due to injury or disease and is caused by the release of endogenous pyrogen, (Blood and Henderson 1963). This substance has a direct effect on the hypothalamus. Most cases of fever are due to an elevation of the thermoregulatory threshold in a centre of the hypothalamus. The alteration can be induced by many different agents most of which act via the intermediary substance, endogenous pyrogen. This substance increases body metabolism as well as affecting the thermoregulatory centre. There are five recognised methods of releasing pyrogen to induce a febrile reaction,

- i) Phagocytosis
- ii) Toxin release
- iii) Tissue necrosis
- iv) Inflammation
- v) Hypersensitivity

Common causal agents are bacteria, viruses, parasites and trauma which evoke phagocytosis, inflammation and toxin release and may lead to tissue necrosis. Tissue pyrogen is found in muscle, liver, kidney, heart, spleen and lung, and hence any damage to these organs will result in the release of pyrogen into the blood-stream. This adequately explains the increase in body temperature seen following surgery, and it is often related to the degree of surgical trauma. Such pyrexia normally resolves within 24 to 48 hours, (Wilkinson 1955). Persistence of pyrexia may be due to infection or continuing inflammation with pyrogen release.

4. Hyperpnoea and tachypnoea

These abnormalities may increase the normal inevitable respiratory water loss due to the increased depth and rate of respiration. Likely causes are pneumonia, thoracic lesions, metabolic acidosis with excretion of excess carbon dioxide and pyrexia. This increased loss has never been satisfactorily calculated, but may be of some significance when supporting a patient with parenteral fluid therapy.

5. Overinfusion

Overinfusion is an iatrogenic increase in body water which may occur during the replacement of fluid intravenously. It causes an increased E.C.F. volume of lowered tonicity and results in an increased output of urine usually of low specific gravity.

Biochemically and haematologically there is a relative reduction in electrolyte levels and the total serum protein, and also the haematocrit and the haemoglobin.

Water intoxication may occur due to overhydration or overinfusion resulting in an increase in cellular water and E.C.F. volume. It rarely occurs in dogs with normally functioning kidneys and a functional hypothalamic-hypophyseal system, (Hall 1967). The water overload leads to convulsions, seizures, ataxia and occasionally vomiting, but there may also be related electrolyte disturbances, (Zimmerman and Wangenstein 1952, Talbot, Crawford and Butler 1953, Wilkinson 1955).

B. Water and electrolyte imbalances

1. Anorexia and oligodipsia
2. Vomiting
3. Diarrhoea
4. Wound discharge
5. Organ discharge and effusions
6. Trauma
7. Overinfusion

1. Anorexia and oligodipsia                      These states occur in an animal which is able to drink a little, but is disinterested in food. They are seen in cases of pyometritis, peritonitis, in cases following surgery, road traffic accidents and in certain animals in strange surroundings. Some patients in collapse are adipsic and may become deficient in water and electrolyte, but largely remain deficient in water alone unless there are other fluid losses, for example, discharges, diarrhoea or vomiting.

This type of imbalance is the result initially of water depletion followed by an electrolyte depletion due to the lack of intake. The principal electrolyte affected is potassium which is preferentially excreted by the kidney to maintain body sodium. If the basic cause is left untreated, the animal eventually enters a state of hypovolaemic shock and circulatory failure, (Elkinton, Danowski and Winkler 1946).

If water alone is consumed to satiate the thirst stimulated by the reduction in E.C.F. volume, it is excreted to maintain body tonicity and hence it is better to consume electrolyte and water, for example/

example, milk, or to treat parenterally with an electrolyte solution.

2. Vomiting This is one of the most common causes of fluid imbalance where both water and electrolyte are lost and there is often a simultaneous pH regulation imbalance. ( The term pH regulation is used instead of the out-dated term, "acid/base balance".) Vomiting is an involuntary expulsion of gastric or gastro-intestinal content through the mouth or nostrils. As stated, it may be of gastric secretion ( mostly hydrochloric acid, sodium, chloride and water with a little potassium ), or gastro-intestinal secretions from the stomach, pancreas, liver and small bowel, ( sodium, chloride, potassium, bicarbonate, hydrochloric acid and water ). It can be considered that saliva ( if produced ) is also lost in vomit either directly from the mouth or indirectly from the gastro-intestinal tract. Sodium, potassium, chloride and bicarbonate are the electrolytes found in saliva.

The causes of vomiting are numerous, for example, gastritis, gastric foreign body, pyloric stenosis, enteritis, intestinal obstruction, whether due to foreign body or tumour, toxæmia, septicaemia as seen in pyometritis, nephritis, peritonitis and drug therapy. A form of vomiting is seen in oesophageal obstruction due to foreign body, tumour or congenital abnormality, but in these cases only saliva is lost from the body, and rarely some gastro-intestinal content. Such cases suffer more from anorexia and oligodipsia rather than vomiting and the symptoms are due to the inability to retain food and water. They may present in a state of collapse if the condition has progressed over a period of time, and the symptoms are more obvious in patients with foreign bodies than in congenital/

abnormalities.

Gastric vomiting due to a foreign body, ( particularly when it causes occlusion of the pylorus ), is an acute condition where large volumes of fluid are lost from the body. This results in a reduction in hydrogen ion content in the body, and a decrease in body sodium, chloride and water. This results in thirst as the body becomes hypertonic. Most commonly water is consumed resulting in dilution of the remaining electrolyte content with the excess water being excreted as urine. If the patient continues to vomit, more water, acid and electrolyte are lost, leading to a reduction in body sodium and chloride, (Wilkinson 1955), and body water culminating in the earlier manifestation of shock due to oligæmic circulatory failure than is seen in pure water loss, (Nadal, Pederson and Maddock 1941). In such cases the animal becomes collapsed with a rapid, weak pulse, poor capillary refill time, dry, sticky mucous membranes and a decreased tissue turgor. A reduced body chloride level can result in weakness, lethargy, vomiting and anorexia, (Coller, Bartlett, Bingham, Maddock and Pederson 1938).

Simultaneously there is a pH regulation imbalance due to the loss of acid from the stomach. This is due to renal retention of sodium which is buffered by bicarbonate ion released from carbonic acid and, therefore as hydrogen ion is lost, the body bicarbonate content increases causing a metabolic alkalosis. This situation leads to a decreased respiratory rate with shallow breathing in an attempt to conserve carbon dioxide. In gastric vomiting hydrochloric acid and sodium are lost and the body conserves the remaining sodium at the expense of potassium and hydrogen ions in the distal convoluted tubules of the kidneys. Since there is insufficient hydrogen ion for excretion, extra potassium is lost leading/

leading to a plasma deficiency greater than would be expected from vomiting alone, (Wilkinson 1955, Hardy 1958, Robinson 1962, Garret 1970). As this progresses still further and plasma potassium is depleted, hydrogen ion is eventually excreted to conserve sodium and potassium and this leads to a further increase in the body pH.

Biochemically there is a decrease in plasma sodium, chloride and a marked decrease in potassium. The total serum protein, haematocrit and haemoglobin levels will increase due to the water loss and these changes are all relative.

The more commonly encountered form of vomiting is gastrointestinal when both acid and alkali are lost along with sodium, chloride, potassium and bicarbonate in water. This can be due to obstruction of the bowel lumen or toxæmia as in cases of nephropathy, pyometritis or peritonitis. The exact reason for the vomiting in these so-called toxic patients is uncertain, but it may be due to impaired renal function and the accumulation of waste metabolites in the blood, (Armistead 1954). In peritonitis the bowel wall commonly becomes oedematous and peristalsis is reduced with resultant accumulation of fluid in the bowel lumen perhaps leading to vomiting, (Wilkinson 1955). In nephritic patients there may be a correlation between the raised blood urea level and associated products, the raised potassium level and vomiting. It has been suggested that in toxic or potentially toxic cases, there may be oedema of the intestinal cells ( cloudy swelling ) which can cause symptoms similar to those of ileus which is described later. This acts as an obstruction and secretion accumulates.

In intestinal obstruction due to foreign body, tumour or bowel stasis/

stasis ( ileus ), the intake of food or water may aggravate the condition and result in vomiting. This may also occur without oral intake since accumulating secretions may cause bowel irritation. This accumulation of intestinal contents proximal to the obstruction stimulates the continual secretion of juices and the passage of fluid into the intestinal lumen, (Wilkinson 1955). The higher the obstruction, the greater the degree of secretion and it follows that the signs of fluid imbalance will be more acute. These patients lose large quantities of electrolyte including sodium, chloride, potassium and bicarbonate as well as water, and this causes a total body depletion. The more acute the signs, the sooner will oligoemic circulatory failure ensue.

When the obstructing lesion is in the lower jejunum or ileum, the patient may simply vomit occasionally and show few other clinical signs. These dogs are commonly deficient in chloride, sodium, potassium and water depending on their intake. Most show signs of water depletion with haemoconcentration and occasionally raised blood urea levels. Some cases of pyometritis present in this manner where there is a copious vaginal discharge and they may be polydipsic usually consuming water alone. The cases of closed or partially open pyometra tend to be of the acute form with a reduced intake of food and oligodipsia showing signs of toxæmia such as pyrexia, hyperpnoea and marked lethargy.

Ileus is a state of bowel stasis acting similarly to an obstruction due to adhesions or paralysis of a segment and may cause vomiting accompanied by colic type pains. The severity depends on whether the site is proximal or distal and electrolyte changes are similar to gastro-intestinal vomiting. This apparent bowel stasis may result from a lowered body potassium or calcium level, since it has been found that if these/



these ions are given intravenously, the condition sometimes rapidly resolves, (Wilkinson 1955).

Vomiting associated with gastritis, gastro-enteritis or enteritis may be severe and result in the loss of large quantities of water and electrolyte with similar changes to those described in the previous types of vomiting. These conditions may be nutritional, bacterial, viral or be due to other infective agents and require immediate treatment of the causal agent.

Large doses of certain drugs can cause vomiting, for example, phenylbutazone. This may be due to gastro-intestinal irritation although the exact aetiology is unknown.

3. Diarrhoea This results in an increase in the faecal water and electrolyte loss, the major ions involved being potassium, sodium and chloride. The causes of diarrhoea are many, such as nutrition, metabolic imbalances, infections and tumours. Little sodium is found in normal faeces, but in cases of diarrhoea increasing fluidity is paralleled by a greater loss of sodium. The sodium content of mucinous discharges is high and these are commonly lost in diarrhoea. In the colon there is an ion exchange between potassium and sodium resulting in the excretion of potassium, and with the loss of large quantities in diarrhoea there is a reduced potassium content in the body. This can lead to generalised weakness and lethargy depending on the intake, (Hardy 1958). If diarrhoea is profuse and the intake of fluid is decreased, water and electrolyte loss can occur, but this rarely is acute in nature unless associated with infection.

Diarrhoea/

Diarrhoea commonly accompanies intussusception where there is bowel obstruction and resultant vomiting. These patients are sometimes deficient in electrolytes, commonly potassium, but since they are acute cases oligæmic circulatory failure may be of considerable importance.

If protein is lost from the bowel in diarrhoea with a decrease in plasma protein level, osmolarity disturbances will lead to generalised oedema and circulatory failure. This occurs due to the decreased osmotic pull at the venous end of the capillary bed.

4. Wound discharges                      These contain water, sodium, potassium, chloride and sometimes protein. They are due to trauma or infection and the degree of loss is highly variable. Large quantities of potassium are lost due to cellular breakdown and this could lead to a body deficiency if the intake is reduced. It is unlikely that a wound discharge would result in severe fluid imbalance, unlike organ discharge which can be fatal. Burns due to chemicals or heat may lead to a loss of plasma protein from the skin in a serous discharge and can result in circulatory failure.

5. Organ discharge and effusion                      Any discharge from an organ the peritoneum or the pleurae internally or externally to the environment constitutes a loss of fluid from the body. Most discharges are rich in potassium and sodium with some chloride and water, (Coller, Bartlett, Bingham, Maddock and Pederson 1938, Wilkinson 1955). The common forms seen in veterinary cases are pyometritis, peritonitis, hydrothorax and cystitis. There is often cell breakdown with the release of potassium, protein and water which will lead to a body depletion of these substances. Most discharges are dependent on the presence of sufficient body fluid for formation. Nevertheless/

Nevertheless they constitute a major fluid loss which requires to be accounted for in therapy.

6. Trauma Trauma in accidents, surgery and disease may cause some imbalance in body fluids with the loss of electrolyte and water when cells are destroyed. Pyrogen is also released and may result in pyrexia with increased water loss. The degree of surgical trauma has been related to the need for parenteral fluid, (Shires and Moore 1969).

7. Overinfusion If excess electrolyte and water are given intravenously, increases in electrolyte levels are possible. Water intoxication has already been discussed, but now the effects of excess electrolyte are described. Most sodium excess is excreted by the kidneys except in some nephropathies, but there do not appear to be any marked adverse effects due to a temporary excess of body sodium, other than oedema recorded by some authors, (Stewart and Rourke 1942, Moyer 1947). Oedema can also be due to a decreased plasma protein level. Excess potassium in the body, especially in the plasma, can interfere with cardiac function and cause death due to cardiac failure. Hyperkalaemia is often due to terminal tissue breakdown, since most animals are capable of excreting excess potassium in the urine.

The overinfusion of water and electrolyte can cause pulmonary oedema, oedema of the mucous membranes and an increased urine output. These effects are to be avoided whenever possible, but may be associated with nephropathies or cardiac pathology.

C. Blood volume imbalance

1. Haemorrhage
2. Shock
3. Plasma volume depletion
4. Overinfusion

1. Haemorrhage                This is the loss or escape of blood from its natural vessels to the internal tissues and body spaces or to the external environment, the former being termed internal haemorrhage and the latter external haemorrhage. Internal haemorrhage occurs into body tissue spaces and presents as haematomata, petechiae, ecchymoses, haemothorax, or free blood in the gastro-intestinal tract, bladder, lungs, central nervous system, pericardium, abdomen or eye.

The causes of haemorrhage are numerous and include trauma, both iatrogenic and accidental, poisons and infections.

Primary haemorrhage is the most common and obvious type of blood loss and its measurement has proved difficult to many technologists and clinicians. The accurate measurement of blood loss is essential in the treatment of cases. Many methods have been used in the past forty years since arterial blood pressure was found not to be a measure of the volume lost, (Robertson 1936). Robertson later found that indirect methods for the assessment of blood loss were reasonably accurate. The methods used were dye and radio-isotope dilution tests. Specific gravity of blood was found to be related to the haematocrit and could be used to determine blood loss, (Ashworth and Tiegerdt 1940), but unfortunately this method required the haematocrit prior to haemorrhage to be the same in all patients. These workers/

workers claimed that it was an accurate method of assessing shock and could be used as a guide to fluid therapy. The dye dilution methods were later assessed and were found to be a reliable means, or as reliable as the technique would allow, but a still better method was sought, (Coller, Iob and Crook 1944). In the same year another group of workers claimed that the clinical signs were of most use, (Shenkin, Cheney, Gouard, Hardy, Fletcher and Starr 1944), but this is doubtful in my opinion. More evidence against the dilution techniques was provided when it was found that certain parts of the body were not totally perfused and therefore did not allow the agent to penetrate, (Prentice, Olney, Artz and Howard 1954).

However in 1961, Williams and Fine used red blood cells coated with  $\text{Cr}^{51}$  and the haematocrit level to determine blood loss and found that the estimates by surgeons and anaesthetists of blood loss were commonly one-third less than the actual loss. The new Volemetron used by these workers was relatively accurate and inexpensive. The Volemetron is a semiautomatic device for measuring the red blood cell volume or the plasma volume by means of the radio-isotope dilution technique.  $\text{I}^{131}$  coated serum albumin and  $\text{Cr}^{51}$  coated red blood cells are best for the assessment of blood loss, and by having knowledge of the background and disintegration rate of the injected sample before and during the assessment, one may use the Volemetron to determine the blood volume. The determination can be repeated frequently and accurately. The Volemetron was used in operating theatres and it was found that blood loss estimates by surgeons were often 40 % inaccurate, (Cullen 1961). Gardner and Dudley (1962) compared the weighing of swabs and drapes with the Volemetron and found that this device was more/

more accurate. It was noted , though, that for accuracy prior knowledge of the initial haematocrit level and the total serum albumin/globulin level was needed. Interestingly one of these workers developed a technique of washing all the surgical instruments, drapes, swabs, gowns and packs and using a colorimeter was able to accurately determine the blood loss, (Gardner and Dudley 1962). This technique was written up and compared with other available methods, being proved relatively accurate, (Roe, Gardiner and Dudley 1962).

Many more authors have researched this subject, but it is not thought advantageous to this study to consider these further.

Secondary haemorrhage is due to a weakening of the blood vessels with resultant rupture and loss of fluid. Examples of causal agents are toxins and drugs such as cortisone. Another form of secondary haemorrhage would be the rupture of a tumour for example, a haemangiosarcoma.

Reactionary haemorrhage occurs when there is loss from what was thought to be a controlled bleeding site. This can occur following intensive fluid therapy when re-perfusion of the tissues opens up bleeding sites and further loss takes place. This is due to the increased blood pressure and a similar effect is seen following surgery when anaesthesia has depressed the blood pressure.

2. Shock            The definition of shock is, " inadequate perfusion of the tissues with oxygenated blood - or - failure of certain vital organs to utilise oxygen ", (Ledingham 1977). The second definition applies in particular to septic shock where there is a failure in oxygen uptake by organs prior to the loss of perfusion.

If/

If one considers shock to be a shift of fluid, it becomes easier to explain the terms neurogenic and cardiogenic shock. Various factors can be responsible for the production of shock.

- i) Haemorrhage
- ii) Body fluid loss - water and electrolyte
- iii) Plasma loss
- iv) Trauma
- v) Thermal injury
- vi) Sepsis
- vii) Toxins
- viii) Pulmonary insufficiency
- ix) Adrenal insufficiency
- x) Anaphyllaxis
- xi) Cardiogenic insult
- xii) Fright

The body response to all of these is similar. Some other factors may be considered,

- xiii) Pain
- xiv) Cold
- xv) Fear
- xvi) Asphyxia
- xvii) Anaesthesia

The last factor, anaesthesia, is recorded since it has been shown that poor or light anaesthesia may precipitate shock in the body, and it would/

would therefore appear that light anaesthesia is to be avoided for this reason. Conversely deep anaesthesia may also cause shock if overdose stages are reached.

All of the precipitating factors cause a decrease in the circulating blood volume by either the external loss of plasma or blood or a shift of fluid out of the circulation into the interstitial tissues. In neurogenic shock there is a vaso-vagal response which results in the pooling of blood in the splanchnic bed and other areas, thereby depriving the external and vital organs of blood and leading to the signs, seen commonly after a fright, of cold extrmitities, pallor and weakness.

The result of the previous causes may be a reduction in the blood volume which triggers body compensatory mechanisms, and it is usually once these mechanisms are functional that the clinical signs are apparent. These mechanisms can be summed up as follows,

Drop in blood volume  
Sympathetic nervous activity  
Vasoconstriction  
Clinical signs

The more severe the loss the more obvious are the compensatory mechanisms, (Shenkin, Cheney, Gouard, Hardy, Fletcher and Starr 1944).

The mechanism of compensation depends on a sympatho-adrenal response which increases the heart rate and causes vasoconstriction. The adrenal gland plays a vital role in the release of adrenaline and then aldosterone, both of which act to conserve the remaining blood volume and to increase the blood supply to the vital organs.

Part/




Part of the compensatory mechanism is the expulsion of plasma protein and red blood cells from the spleen which contracts in response to adrenaline and sympathetic nervous stimulation. This aids the maintenance of the colloid osmotic pressure within the intravascular compartment, (Coller, Crook and Iob 1944, Robertson 1938, Soma, Burrows and Marshall 1974).

Eventually when the compensatory mechanism is unable to cope and despite the attempts to direct blood back to the vital organs, a stage is reached where there is a pulse of reduced volume, tachycardia, cold extremities, tachypnoea, pallor of the mucous membranes and a poor capillary refill time. If this stage is left to progress, irreversibility occurs, (Armistead 1954, Wilkinson 1955, Hardaway 1962, Cohn and Luria 1964, Lillehei, Longerbeam, Bloch and Manax 1964, Bloch, Dietzman, Pierce and Lillehei 1966).

The mechanism of irreversible shock is explained in the following table derived from the work of the above authors.

## Irreversible shock

- 1      Oligaemic Hypotension
  - 2      Outpouring of adrenaline
  - 3      Increased sympathetic activity
  - 4      vasoconstriction - of the pre and post-capillary sphincters
  - 5      Ischaemia and anoxia
  - 6      Accumulation of tissue metabolites
  - 7      Deterioration of the smooth muscle function in blood vessels
  - 8      Destruction of the capillary endothelium
  - 9      Relaxation of the pre-capillary arteriolar sphincter
  - 10     Inflow of blood into the capillary beds
  - 11     Capillary stasis
  - 12     Decreased venous return
  - 13     Further hypotension
- 
- 14     Ischaemia of tissues
  - 15     Tissue necrosis
  - 16     Irreversible shock
  - 17     Death

If treatment is not started before point 10 , the chances of preventing irreversible shock are poor and commonly death results due to intravascular clotting, tissue hypoxia and haemorrhage. This is seen most commonly in the intestinal tract in the dog as a haemorrhagic sloughing of the mucosa and occasionally the excretion of blood and mucosa per rectum shortly before death.

3. Plasma volume depletion                      This is a form of shock and may be seen in cases of trauma where there is a sequestration of fluid at the site of injury.

4. Overinfusion                      Overinfusion is the state arising from the overload of the circulation with colloid and or crystalloid causing a series of clinical signs depending on the severity. Initially there is oedema of the mucosae with excessive secretion, followed by oedema peripherally and then pulmonary oedema with impaired oxygen uptake.

## CLINICAL EXAMINATION AND ASSESSMENT

Careful and systematic inquiry of the owner should yield sufficient information to base reasonably accurate fluid replacement therapy on. There should be enquiry into the consumption of food and water both before the onset of illness and during it, as well as additional loss, for example, vomiting, pyrexia, tachypnoea, discharge, diarrhoea or other obvious symptom. The duration and severity of the illness are determined and the additional loss estimated. It is relevant to state that all the information gained from the history and clinical examination should be recorded in a case record to permit consideration of the findings.

The animal's health status prior to the present illness is important in determining if the input and output were normal, and this can be used as a base line to assess recovery.

In peracute cases, it is often necessary to institute treatment immediately and the owner may be questioned later.

The clinical examination should be concerned with the whole animal. The important signs which may aid the diagnosis of body fluid imbalance are listed and considered.

- A. Appearance and weight
- B. Demeanour
- C. Mucous membranes - colour and consistency
- D. Capillary refill time
- E. Pulse - rate and volume
- F. Heart sounds
- G. Respiratory characteristics - rate, nature and sounds
- H./

- H. Body temperature - core and skin
- I. Tissue turgor
- J. Palpation
- K. Injuries

A. Appearance and weight                      The general appearance of an animal is important and may be an indicator of the home environment and previous health status. Acute illness should occur in normal looking dogs whereas chronic disease is often determinable by poor body condition. A loss of weight is common in body fluid imbalance cases, especially if the condition has been present for a number of days or weeks. It is good policy to weigh dogs on presentation since this allows a comparison post-treatment. Dogs with weight loss may have abnormal excess hanging skin, sunken eyes, dry coats, an ungroomed appearance and be generally dull. If there is abdominal pain present, the animal's back may be arched and free movement will be limited. They may also resent being handled in the lumbar, pelvic and abdominal area. There may be changes in the respiratory characteristics in these animals which are not attributable to lung pathology, but to abdominal pain. Respiratory changes should be noted on examination of the general appearance and it is often possible to determine if these changes are of thoracic origin. The abdominal size may lead one to suspect a mass or fluid.

Injuries and traumatic lesions are sometimes obvious, but often further examination is necessary to determine fractures, abrasions, internal compression or rupture of internal organs or hernias.

B. Deméanour /

B. Demeanour Demeanour can often change in a systemic disease or with the degree of severity in an accident case. The owner is the best judge of their pet's demeanour and the initial complaint may be that the animal is dull and "off colour". Patients with a loss of body fluid are commonly dull and disinterested in their surroundings, and may be weak. Losses of electrolytes, for example sodium and potassium, can cause skeletal muscle weakness which is sometimes seen in dogs related to vomiting, anorexia, diarrhoea or septicaemia. The presence of toxins or infective agents in the blood can cause depression.

Acute cases often appear dull and may be collapsed following road traffic accidents, haemorrhage or persistent vomiting.

C. Mucous membranes Both the colour and consistency of the oral and conjunctival mucous membranes should be noted. Normal mucous membranes are pink, smooth, moist and will have a capillary refill time of less than two seconds.

Darkening of the colour is commonly due to haemoconcentration seen in dehydration and body fluid loss. The colour can be dark red to crimson in animals which have been vomiting, diarrhoeic, anorexic or oligodipsic. In cases of haemorrhage, hypovolaemic shock, anaemia or circulatory failure the membranes may be either pink or white indicative of a lack of perfusion with red blood cells. In septic shock the membranes may be darkened ( hyperaemic ) but the other clinical signs will reveal the circulatory problem.

In animals with systemic disease there may be icteric changes or yellowing of the membranes, indicative of liver malfunction. Jaundice may be observed following blood transfusion if there has been incompatibility. Petechial haemorrhages may indicate poisoning, for example with warfarin, or/

or systemic disease. These haemorrhages have been recorded in dogs which have been on prolonged corticosteroid therapy causing the blood vessels to become fragile and to rupture spontaneously.

A blue colouration of the membranes is seen occasionally in animals which have respiratory difficulty or are grossly haemoconcentrated. It may indicate overinfusion due to pulmonary oedema and the failure of oxygen uptake. Another indication would be cyanosis due to cardiac pathology.

The consistency of the mucous membranes is useful as an aid to diagnosis. Dryness of the membranes occurs in body fluid imbalances especially when water is deficient. If the membranes are dry and sticky, this is commonly due to extracellular fluid loss as occurs with vomiting or diarrhoea, and there may be signs of impending circulatory failure. The dryness is due to the lack of saliva or lacrimal secretion and these should return with successful therapy.

In circulatory failure due to haemorrhage or severe body fluid loss, the membranes may be dull, grey, pale and clammy. This is indicative of hypovolaemic shock and commonly the patient requires emergency therapy.

D. Capillary refill time (C.R.T.) this is estimated by the use of digital pressure on the mucous membranes of the gum or lip. The normal effect is the return of blood to the membrane within two seconds. This is an indicator of tissue perfusion and is important in the assessment of circulatory function. If the time is prolonged, it is commonly due to poor perfusion of the capillary beds which may be due to/

to fluid depletion with resultant compensatory peripheral vasoconstriction. This is due to constriction of the pre- and post-capillary sphincters which occurs as part of the normal compensatory mechanism.

If treatment is successful the sphincters re-open and the C.R.T. should return to normal. In anaemic dogs it is sometimes difficult to assess the C.R.T. due to the lack of membrane colour.

E. Pulse            The assessment of the arterial pulse, commonly the femoral, is of vital importance in the clinical examination. The normal pulse is full in volume and the rate and rhythm that of the heart. In obese animals palpation of the femoral artery may be difficult. The normal rate varies with the size of the dog, but is usually between 80 and 160 per minute for large and small breeds respectively. An increase in rate occurs with exercise, fear, fright and pathophysiological changes. An increase is evident in animals with a reduced blood volume due to cardiac and circulatory compensation. The increase is often proportional to the degree of blood loss or fluid loss. A decrease in pulse volume may occur without a change in rate if there is adequate compensation. Changes in rhythm indicate cardiac malfunction and it is always advisable to auscultate the heart.

In dehydrated animals the pulse rate may be increased and the volume decreased, and in cases of circulatory failure there is a tachycardia with decreased volume pulse often described as rapid and thready.

An increase in pulse volume and rate may occur with overinfusion or chronic interstitial nephritis where there is hypertension.

If the femoral pulse is not palpable the reasons are numerous,  
for/



for example, cardiac failure, femoral artery occlusion, aortic thrombosis or severe cardio-vascular collapse.

F. Heart sounds                      The heart rate, rhythm and sounds heard on auscultation should be assessed in all patients, particularly where surgery is envisaged. An increase in rate occurs in those conditions which cause a rise in pulse rate as well as cardiac or respiratory abnormalities. Many conditions such as valvular or cardiac muscle disease or disturbances in electrophysiology can cause a tachycardia. Other conditions, for example, cor pulmonale, pericarditis, pneumothorax, hydrothorax, diaphragmatic hernia or lung disease may affect cardiac function.

Auscultation is therefore of importance in the differential diagnosis of these conditions and often body fluid imbalance cases will have other presenting signs. Nevertheless the heart function should be known before the administration of large volumes of fluid parenterally since animals with reduced cardiac competence are poor risk infusion cases.

G. Respiratory characteristics                      The respiratory rate and character should be noted since certain body fluid imbalances can cause alteration in them both. In a metabolic acidosis where there is excess carbon dioxide in the body, the rate will increase to excrete the excess.

An increased rate may be evident in accident cases or in animals with pneumothorax, fluid in the chest, fractured ribs, diaphragmatic hernia or lung contusion.

The auscultatory sounds are important indicators in assessing respiratory function. The presence of fluid in the lungs may be a contraindication/

contraindication to immediate fluid therapy. Increased respiratory noises in sick animals can be due to crusting of dried mucous or vomit in the nares causing respiratory embarrassment.

Overinfusion in a dog with a poor circulatory system may result in pulmonary oedema with increase in the respiratory rate and auscultatory sounds in a severe case.

H. Body temperature Alteration in the body temperature, both internal and external, can be a useful indicator of the physical status of a patient. In cases of body fluid deficiency and circulatory collapse, the external temperature is decreased due to compensatory peripheral vasoconstriction. The exception is septic shock where the body temperature may be elevated. An increased body temperature post-operatively is normal due to surgical trauma and inflammation, but it should regress within 24 to 48 hours. If it does not it may be indicative of infection or further inflammation.

The differential in temperature between the skin and the core is important in determining the degree of body perfusion and a decrease in the difference is often a sign of improvement if both are increasing.

All parenteral fluids should be preheated to avoid unnecessary body temperature disturbance. In shocked patients the infusion of warm fluids intravenously is often sufficient to restore the blood supply to the peripheral tissues.

I. Tissue turgor This is the ability of the skin to recoil when "tenting", and this occurs instantly when the E.C.F. volume is normal. Any increase in the time taken for recoil or the prolonged presence/

presence of tenting are signs of dehydration and reduced E.C.F. volume. The test is best performed at the point of the elbow or the hock where there is normally little subcutaneous tissue.

J. Palpation Palpation tends to be a neglected art among veterinary clinicians but is of importance in many cases. Intestinal foreign bodies, abdominal masses as well as the presence of fluid can be determined by palpation, although the routine palpation of cases with a pyometritis is not advisable since rupture with serious side effects may occur.

Other factors of note on abdominal palpation are the size of the liver, spleen, kidneys, the intestinal contents and the presence of pain.

Palpation of other areas of the body may reveal fractures, dislocations, wounds, tumours and the temperature and consistency of the skin.

K. Injuries These should be identified where possible before the commencement of treatment, but it is important in cases of shock to institute therapy immediately.

On completion of the clinical examination and the determination of relevant history, the body fluid status may be assessed. From the facts obtained, one should attempt to make a provisional diagnosis which might allow correction of the underlying lesion or cause of the body fluid imbalance. The symptoms seen in body fluid disturbances are often similar and some of the basic imbalances are now described.

Dehydration is the state of reduced body water and electrolyte with decrease in the E.C.F. volume and resultant dry, commonly congested mucous membranes, increased pulse rate of reduced volume, decreased tissue turgor and dry skin, but normal C.R.T. and body temperature. This may be due to reduced intake or increased output of fluid which disturb the homeostatic balance. The clinical signs appear more rapidly when there is extra electrolyte loss as in vomiting, diarrhoea or wound or organ discharge. Debilitated animals suffering from lesions of the kidneys, liver, spleen, gastro-intestinal tract or the uro-genital system may also present in a state of dehydration when input and output have been disrupted.

Acute cases arise due to persistent vomiting, starvation, water deprivation, the inability to eat or drink or the overuse of drugs such as diuretics. Persistent vomiting can occur in cases of oesophageal or gastro-intestinal obstruction, renal failure, pyometritis and liver failure. The inability to eat may be due to oesophageal obstruction or paralysis, oral tumours, paralysis of the oropharyngeal muscles or abdominal masses where there is gastric compression.

When electrolytes are lost or not gained, the signs of dehydration arise rapidly and the phase of circulatory collapse ensues.

If/

If sodium is lost in quantity, as occurs in vomiting, the clinical signs are of dehydration accompanied by dullness, weakness, dry and tacky mucous membranes, sticky saliva, marked loss of tissue turgor and the impending signs of shock such as slowing of the C.R.T. and tachycardia. In vomiting, chloride is lost and there is often a pH regulation imbalance, particularly a metabolic alkalosis due to the loss of acid from the stomach resulting in laboured, slow respirations.

In chronic or prolonged sub-acute vomiting, the dogs usually consume some food or water and appear thin with sunken eyes, dry mouths, loss of tissue turgor, congested mucous membranes and a poor volume pulse.

The depletion of body water and potassium occurs due to reduced intake, wound or organ discharge, effusions, vomiting or injury and results in dehydration with skeletal muscle weakness and fatiguability. This can cause further vomiting due to gastro-intestinal atony and shallow breathing due to muscle weakness. A potassium deficiency may also arise when an animal is maintained on potassium free parenteral fluids.

A deficiency of blood calcium may cause tetany or eclampsia, and calcium may have a part to play in gastro-intestinal tone, but this is not fully understood.

If the states of dehydration mentioned above are allowed to continue, oligæmic circulatory failure ensues. This is indicated by a rise in pulse rate and a decrease in pulse volume. The C.R.T. is prolonged and the mucous membranes are pale rather than congested. The external body temperature falls and the difference between the skin and the core temperature increases. These are the clinical signs of shock as seen in cases following accidents or acute hæmorrhage.

In/

In accident cases fluid can be sequestered at fractures and other sites of injury, and the signs of shock may be present.

In internal haemorrhage the diagnosis is more difficult and often the cause is not obvious. The patient must be judged by the clinical signs and the response or otherwise to treatment.

ANCILLARY AIDS TO DIAGNOSIS OF FLUID IMBALANCE

On completion of the clinical examination and assessment, ancillary aids may be used to determine or confirm the diagnosis.

Some of these tests are,

- A. Blood examination
- B. Urine examination
- C. Radiography
- D. Catheterisation
- E. Paracentesis
- F. Electrocardiography

A. Blood examination                      It is of use in most cases of body fluid imbalance to determine the various haematological and biochemical parameters of relevance. This is best performed prior to therapy where possible and also post-therapy to determine the degree of improvement or otherwise. It is advantageous to have analyses results from the time of admission for use as a base line for the subsequent examinations.

The examinations of most relevance in cases of fluid imbalance are,

Haematology

Packed Cell Volume

Haemoglobin

White Blood Cell Count

Biochemistry/

Biochemistry

Blood Urea

" Sodium

" Potassium

" Chloride

Total Serum Protein

Some additional estimations may be of use in specific cases,

Haematology

Red Blood Cell Count

White Blood Cell Differential Count

Biochemistry

Serum Bilirubin

" Alkaline Phosphatase

" Aspartate Transferase

" Alanine Transferase

" Albumin

" Globulin

" Creatinine

The collection of blood is discussed. Two samples are required for normal haematological and biochemical examination, 2 ml. in E.D.T.A. for the former and 10 ml. in heparin for the latter. The collection of blood in most patients is easiest from the external jugular vein which is usually patent even in cases of acute hypovolaemic shock. In my experience, a 20G. (SWG) one inch needle and a 10 ml. syringe is best. The cephalic and saphenous veins are often too small for the satisfactory collection of blood since clotting occurs before sufficient is obtained. After/



After collection, the needle is removed from the syringe and the blood is added to the sample bottles which are then gently rocked to prevent coagulation and haemolysis.

The analyses are now described.

1. Haematology                      The haematocrit or packed cell volume (PCV) is of use in determining the degree of haemoconcentration which commonly increases with dehydration. It is of use in cases of haemorrhage, anaemia and shock. The test is commonly performed using the microhaematocrit apparatus which involves the placing of blood in a microhaematocrit pipette tube, the end of which is heat sealed, (Schalm, Jain and Carroll 1975). The sample is spun in a centrifuge for five minutes at 7500G and the result is obtained by placing the tube in a microhaematocrit slide rule which permits one to read the percentage of cells to plasma.

Haemoglobin levels are obtained by the cyanomethaemoglobin method and the use of a colorimeter, (Dacie 1956, Schalm, Jain and Carroll 1975). This test is relatively complex and is commonly only available in laboratories. The level will increase with dehydration due to the relative increase in the red blood cell population per ml. of plasma. It decreases in patients which are anaemic.

Red blood cell counts and White blood cell counts (WBC) can be performed manually with a microscope and a haemocytometer. In most modern laboratories a Coulter Counter is used which gives a rapid and accurate count, (Schalm, Jain and Carroll 1975). The white blood cell differential count is performed by visual inspection of a blood smear. The white blood cell count increases in animals with an inflammatory process or infection.

2. Biochemistry                      The blood urea estimation is a measure of the urea present in the blood which is a reflection of renal function. Urea is the end product of protein metabolism and is formed principally in the liver. When the kidney is malfunctioning, the urea concentration in the blood increases. The test in this study was performed using a Technicon Autoanalyser by the diacetyl monoxime method, (Skeggs 1957, Oser 1968, Varley 1969). The common causes of blood urea level elevation are,

i)    Pre-renal                      Pre-renal uraemia is due to dehydration where there is a reduced renal blood flow and hence reduced glomerular filtration,

ii)   Renal                      Renal uraemia is due to nephritis, glomerulonephritis, pyelonephritis and conditions which impair the renal output and concentration effect,

iii)   Post-renal                      Post-renal uraemia is due to obstruction of the flow of urine, for example in ureter obstruction, ruptured bladder and urolithiasis.

The serum electrolytes, in particular sodium, potassium and chloride are important factors in body fluid balance, which are commonly estimated by flame photometry, (Jacobs and Hofmann 1931, Varley 1969 Oser 1968). The chloride estimation in this study was performed by the Chloride meter (Eel) which uses silver ions in a solution which form a salt when chloride is added, and the time taken for electrical neutrality to return is equivalent to the amount of chloride added, (Oser 1968).

The total serum protein , a measure of the plasma proteins, was estimated by the modified biuret reaction in the Technicon autoanalyser, (Oser 1968). The level increases in dehydration due to a reduced plasma water,

The/

The other tests of use in the confirmation of disease are now considered. The serum magnesium and calcium can be assayed by Flame Photometry. Their full significance in fluid balance in the dog is not understood.

The serum bilirubin level is present as the result of red blood cell breakdown, and it is conjugated in the liver. It is estimated by the diazo methanol method in the Technicon Autoanalyser. Increases may be due to the following,

i) Pre-hepatic bilirubinaemia which is due to increased degeneration of red blood cells, and the liver is incapable of handling the increased amount of unconjugated bilirubin. This may be due to an incorrect blood transfusion.

ii) Hepatic bilirubinaemia due to hepatic failure and there is an increase in both unconjugated and conjugated bilirubin in the blood.

iii) Post-hepatic bilirubinaemia which is due to obstruction of the flow of bile from the liver to the duodenum. In this case, there is an increase in the conjugated form.

The unconjugated form of bilirubin is sometimes termed the indirect bilirubin and the conjugated form the direct bilirubin.

The serum alkaline phosphatase, the alanine transferase and the aspartate transferase are measures of liver function. The alkaline phosphatase is also formed in the heart, bone, uterus and kidney and elevated levels will be seen with pathology in these organs.

The ratio of albumin to globulin is a measure of the immune response. The estimation of these two substances is performed by electrophoresis.

B./

B. Urine examination                      The collection of urine may be either by urinary catheter or by spontaneous excretion. It should be collected in a sterile bottle. The important assays are,

Urine protein

"    urea

"    pH

"    specific gravity

The urine protein may be estimated by the turbidimetric method and assessed in a colorimeter. The level will rise in cases of nephropathy where protein is lost after passing through the glomerular membrane, for example in glomerulo-nephritis.

The urine urea is estimated by the hypobromite method in the Technicon Autoanalyser. It will decrease in some renal conditions where there is failure of concentration in the renal tubules and collecting ducts. The urine urea level is of use in determining if a raised blood urea is renal or pre-renal. The normal ratio of urine urea to plasma urea is less than 5:1. If it is 1:1 to 5:1 the condition is renal in origin, whereas if the ratio is greater than 5:1 the condition is pre-renal.

The specific gravity is a measure of the concentrating ability of the kidney and the estimation is performed using a refractometer

The urine pH can also be monitored and may be of assistance in the diagnosis of certain pH imbalances.

The serum electrolytes may also be monitored to give a measure of body requirements, since only excesses are excreted.

Stick/

Stick tests are available for urine to estimate the content or presence of bilirubin, glucose, ketones, protein and the pH. They are relatively useful, but require careful interpretation.

C. Radiography This ancillary aid is most useful in the confirmation of diagnosis in many cases. It may be used in cases of pyometritis, intestinal foreign body, thoracic masses or fluid, abdominal masses or fluid and fractures which are usually visible on plain films. Other specialised techniques exist to permit interpretation of the initial findings. Positioning of the animals is important and both lateral and dorso-ventral views should be taken for correct analysis.

D. Catheterisation Catheterisation of the bladder is a useful aid to diagnosis in road traffic accidents if rupture is suspected. For fuller confirmation, air is injected into the bladder and radiography performed. Urine may also be collected and the output monitored by means of a catheter and a graduated container.

E. Paracentesis Paracentesis is a common technique which permits determination of the presence or absence of fluid or air within a body space, for example the chest or the abdomen. It is performed under sterile conditions using a plastic cannula rather than a needle. The substance aspirated can be analysed for colour, consistency, smell, cellular content and clotting ability.

F. Electrocardiography The E.C.G. may be used to determine cardiac abnormalities where suspected. It is useful to have a visual display during surgery on patients who are seriously ill.

## PARENTERAL FLUIDS

The commercially produced parenteral fluids of most relevance to veterinary practitioners are described. These fluids are produced in 500 ml. and 1000 ml. plastic and glass containers. It is useful to consider them according to type, function and substance content.

Two basic fluid types exist,

A. Crystalloids which are basically water and electrolytes or sugars. They are of various tonicities and supply calories, salts and water for the body.

B. Colloids which are fluids similar in nature to plasma containing a substance which is retained in a set body compartment, commonly the vascular system.

Parenteral fluids are produced in several different tonicities ranging from hypotonic through isotonic to hypertonic. Most of the fluids under consideration are isotonic, that is of similar tonicity to normal body fluids.

The final description of fluids involves their use which necessarily revolves around their substance content. A replacement fluid is used for the rehydration of the tissues and the correction of body fluid and electrolyte imbalances. A maintenance fluid is used to provide the daily requirement of water and electrolyte, thus enabling an output of urine after the inevitable loss.

Fluids/

Fluids for parenteral use must satisfy these requirements to be of use to animals. They should fulfill their purpose and provide water and electrolytes in the correct amounts or restore the blood volume and establish an output of urine.

The fluids are now described.

<u>FLUID</u>	<u>TONICITY</u>	<u>pH</u>	<u>ELECTROLYTE CONTENT</u>					
			MMOL. per L.					
			<u>Na</u>	<u>K</u>	<u>Cl</u>	<u>Ca</u>	<u>Mg</u>	<u>HCC<sub>3</sub></u>
<b>A. <u>Crystalloids</u></b>								
1. 0.9% Na Cl	Iso.	5.5	150		150			
2. 5% Dextrose	Iso.	4						
3. 0.18% Na Cl + 4.3% Dextrose	Iso.	4	30		30			
4. Compound sodium lactate	Iso.	6.5	134	5	111	2		29
5. Darrow's solution	Iso.	6.5	121	35	103			53
<b>B. <u>Colloids</u></b>								
6. Dextran 40 10% in 0.9% Na Cl	Hyper	5.5	150		150			
7. Haemaccel	Iso.	7.2	145	5	145	6.3		
8. Plasma	Iso.	7.4	148	4.4	110	2.6	0.9	24

A. Crystalloids

1. 0.9 % Na Cl also known as normal saline, ( that is physiologically normal ), isotonic saline or physiological saline. It is isotonic and contains sodium, chloride and water and is of use in the replacement of electrolyte losses. Its use in the replacement of blood loss is doubtful since it does not sustain blood pressure, (Blalock Beard and Thuss 1932). It can apparently be administered in large amounts - 2 litres subcutaneously to a 7 kilogram dog - without ill effect, changes in the E.C.G, or oedema, (Miller and Poindexter 1932). It is incapable of causing water intoxication, (Talbot, Crawford and Butler 1953). When used in the treatment of shock due to electrolyte depletion, it is relatively successful in the restoration of the plasma volume, (Danowski, Winkler and Elkinton 1946). It was noted by Danowski and colleagues that plasma protein levels improved best when 0.9 % sodium chloride was infused in preference to glucose solutions, even if they too contained electrolytes. In the treatment of haemorrhagic shock, 0.9 % Na Cl was found to be totally unsuccessful, (Dillon, Lynch, Myers, Butcher and Moyer 1966).

Its use as a general replacement solution has been advocated by numerous authors, (Coller, Bartlett, Bingham, Maddock and Pedersen 1938, Hall 1967, Garret 1970, Foster 1970, Finco 1975). Despite these recommendations, it has been reported as being of little use in the replacement of E.C.F. losses since it is not totally physiological, containing only sodium and chloride, unlike Ringer lactate, (Fox, Winfield, Slobody, Swindler and Lattimer 1952, Black 1953).

It is useful as a replacement solution and can be used to correct the losses due to vomiting or prolonged anorexia. In some cases, for/



for example, gastric vomiting, it may be the fluid of choice since it will not exacerbate the metabolic alkalosis present due to its lack of bicarbonate.

2. 5 % Dextrose is an isotonic solution containing water and glucose which is metabolised to water and carbon dioxide in the body. It is suitable for the replacement of water losses alone, (Coller and Maddock 1953, Coller, Bartlett, Bingham, Maddock and Pedersen 1938, Cooper, Job and Coller 1949, Armistead 1954, Wilkinson 1955, Fieber and Jones 1966, Hall 1967, Foster 1970). It has proved of little use in the re-establishment of a circulatory plasma volume, (Ashworth, Hutcheson, Payne and Jester 1944, Danowski, Winkler and Elkinton 1946, Dillon, Lynch, Myers, Butcher and Moyer 1966). Water intoxication due to its administration has been reported, (Talbot, Crawford and Butler 1953), but if electrolyte is added to the dextrose solution, this problem can be avoided. Electrolyte loss and dehydration can occur if dextrose solutions are infused continuously due to an increased output of urine, (Stewart and Rourke 1942). It has been discovered that it is of use in the support of patients with intestinal and abdominal trauma, (Page and Kohlstaedt 1947), and if added to Ringer lactate provides good rehydration without excess electrolyte infusion, (Fieber and Jones 1966).

Therefore it appears that 5 % dextrose solution can be used in the treatment of pure water loss alone as occurs in water deprivation, but that it may be advisable to infuse some electrolyte simultaneously to prevent water intoxication or depletion of the E.C.F.,.

3. 0.18 % Na Cl + 4.3 % Dextrose contains glucose,

water and one-fifth the sodium chloride found in 0.9 % Na Cl. The solution is isotonic and is suitable as a rehydrating solution, or a maintenance solution since it contains sufficient sodium and chloride in water to satisfy the daily requirement, (Harrison, Sussman and Pickering 1960). It is recommended in the replacement of water loss and to a lesser degree electrolyte loss, (Nadal, Pedersen and Maddock 1941, Stewart and Rourke 1942, Moyer 1950, Armistead 1954, Hall 1967, Foster 1970). It is of little use in the treatment of shock, (Talbot, Crawford and Butler 1953). It does not appear to cause water intoxication as readily as pure dextrose solution, (Talbot, Crawford and Butler 1953).

Therefore this solution can provide the daily maintenance requirement for water and sodium chloride when infused at 40 ml. per kg. per day. It is not satisfactory for the replacement of losses due to vomiting or diarrhoea which result in marked electrolyte depletion.

4. Compound sodium lactate called Ringer lactate,

lactated Ringer's solution or Hartmann's solution and contains sodium, chloride, potassium, calcium, bicarbonate ( as lactate metabolised in the liver to bicarbonate ) and water. It is isotonic and approximates to plasma without the protein element. It has been advocated in preference to 0.9% Na Cl as a replacement solution in patients suffering from an electrolyte loss, (Coller, Iob, Vaughn, Campbell and Moyer 1944, Moyer 1950, Armistead 1954, Shires, Coln, Carrico and Lightfoot 1964, Foster 1970, Miller, Stoetling, Paradise, Bevan, Dudley and Horsey 1973). This solution has been used with some success in the treatment of minor blood loss, (Trudnowski, Goel, Lam and Evans 1967, Baue, Tragus, Wolfson, Cary and Parkins 1967, Finco 1974). Ringer lactate has been recommended during/

during surgery to prevent shock and to maintain renal output, (Boba and Landmesser 1961, Shires 1961, Shires and Jackson 1962, Moore and Shires 1967, Trudnowski, Goel, Lam and Evans 1967, Roth, Lax and Maloney 1969, McKenzie and Donald 1969, Garret 1970, Finco 1973, Soma, Burrows and Marshall 1975, Rawlings, Wingfield and Betts 1976, Doty, Hufnagel and Moseley 1976).

Ringer lactate is capable of buffering in the acidotic patient since the lactate is metabolised to bicarbonate in the liver, (Moyer 1950, Roth, Lax and Maloney 1969). This process takes between two and six hours and is related to the hepatic perfusion and the degree of liver normality. The solution itself is acidic and may not act as a buffer as readily as sodium bicarbonate solution. In diabetic patients the administration of Ringer lactate causes a dramatic rise in the blood sugar level, (Thomas and Alberti 1978).

Therefore Ringer lactate is probably the universal replacement solution for electrolyte loss, water and electrolyte loss and initially for blood loss. It can overcome most acidotic situations and this is of importance in the dog which is a naturally acidotic species due to its metabolic products,

5. Darrow's solution or lactated potassic saline solution contains sodium, chloride, potassium, lactate and water. The concentration of potassium and lactate is greater than in Ringer lactate, and this solution is used basically to replace potassium loss. It is isotonic and can be used as a replacement solution in the presence of a diuresis. Excess plasma potassium is toxic to the myocardium and if there is oliguria, high plasma potassium levels result. It should only be administered by a peripheral venous route and certainly not via a central venous/

venous catheter since it would affect the heart directly, (Fox, Winfield, Slobody, Swindler and Lattimer 1952, Garret 1970),

Its use in veterinary practice is limited, except in the treatment of the chronic diarrhoeic animal with a proven low plasma potassium, (Berry, Iob and Campbell 1948, Finco 1974).

## B Colloids

The colloidal solutions are basically used to replace blood volume loss which is usually plasma or whole blood. A colloid is matter in which the individual particles consist either of single large molecules such as protein, or aggregates of smaller molecules such as sugars, uniformly distributed in a dispersion medium. They can be divided into two groups; Plasma substitutes should approximate to plasma in tonicity and have a similar constitution. Volume expanders are used to expand a volume of fluid, commonly the intravascular fluid.

The optimal volume expanders are plasma and whole blood, neither of which is readily available without a little effort on the part of the practitioner. The substitutes and expanders were developed for this reason, and have a long history from gum acacia to gelatin, globin, albumin, polyvinylpyrrolidone (PVP) and dextran. Gum, globin and PVP have become purely historical, but gelatin, albumin and dextran are commonly found in use, (Bull 1963, Foster 1970, Ricketts 1973, Finco 1974).

These fluids are described,

6. Dextran is a polysaccharide of glucose discovered in Sweden in 1940 by Ingleman, and it has come to have an important role in fluid therapy. It is produced in different sizes according to the molecular weight, ( 40, 70, 110, 150 ), the low molecular weight dextrans being of use in animals. Dextran 40 was used in this study as a volume expander.

Its principle use is in the replacement of loss due to haemorrhage or plasma depletion, and it is efficient and effective for up to twelve hours, (Gronwall and Ingleman 1944, Goldenburg, Crane and Popper 1947, Bull, Ricketts, Squire, Maycock, Spooner, Mollinson and Paterson 1949, Bloch, Pierce, Manax, Lyons and Lillehei 1965, Foster 1970, Ricketts 1973). Dextran is eventually metabolised to glucose in the body providing calories and water, although a small amount is excreted unchanged by the kidneys, (Goldenburg, Crane and Popper 1947). This group of workers claimed that although dextran caused transient changes in the renal tubules, there was no impairment of function. This has recently been disputed, (Feest 1976)

Further uses of the low molecular weight dextran were discovered when it was shown that it prevented blood sludging as occurs in shock and that it prevented platelet aggregation, both features which led to irreversible shock, (Bull 1963, Bennet, Dhall, McKenzie and Matheson 1966, Dygdeman, Svensjo and Tollerz 1970, Ricketts 1973, Aberg, Bergentz and Hedner 1975). It has been shown to improve blood flow and to prevent post-operative venous thrombosis. Dextran has been used recently to lyse large thrombi with moderate success, (Aberg, Bergentz and Tollerz 1975).

Another property noted in experimental work is that dextran coats blood vessel walls and hence improves the blood flow, (Bennet, Dhall, McKenzie, /

McKenzie and Matheson 1966, Bloom, Harmer, Bryant and Brener 1966).

Unfortunately some disadvantages have been discovered, the most important one being that it acts as an anti-coagulant and may be contraindicated in the treatment of haemorrhage if the site of loss is unknown, (Bonnar and Walsh 1972, Ricketts 1973). It has been noted that low molecular weight dextran administered to patients with renal malfunction can result in deposition of the molecules in the tubules which leads to an osmotic nephropathy which can be permanent, (Feest 1976).

Most preparations of dextran are hypertonic ( 6 % and 10 % ) and when infused cause tissue dehydration, (Ricketts 1973). This can be overcome by the addition of a hydrating solution such as normal saline or Ringer lactate simultaneously.

The following recommendations are made,

- i) Never exceed the recommended dose
- ii) Check the urine output regularly
- iii) Use with care in animals where there is an elevated blood urea level.

7. Haemaccel is a plasma substitute derived from bovine gelatin and composed of polypeptides of varying molecular weight, from 5,000 to 50,000 , the average being 35,000. The molecules are globular in character and are similar to albumin. It is pharmacologically inert, non-antigenic, sterile and pyrogen free, consistent from batch to batch, and can be stored for 8 years withstanding wide temperature variations, (Hoechst 1977).

It was discovered in 1962 and the solution contains gelatin, water/

water, sodium, chloride, potassium, calcium, phosphate and sulphate, (Schmidt, Thame, Mayer and Schoner 1962). Its pH is approximately the same as plasma and it is relatively isotonic. Its effect lasts for two to eight hours, the larger molecules lasting longest. It does not appear to affect coagulation or renal function, and it has advantages over dextran as a volume expander because of its inert nature, (Lutz and Hallwachs 1969).

8. Plasma is difficult to obtain in veterinary practice and although freeze-dried plasma is used in some practices, the equipment and time necessary to freeze dry plasma are not readily available. Whole blood transfusion is perhaps of more direct benefit and is outlined briefly here.

The production of ready for use blood bags for the collection of whole blood from humans has made blood transfusion relatively straightforward. These packs contain Citrate phosphate dextrose (CPD) which prevents clotting as well as prolonging the life of the blood if stored at 4°C. Its use has been advocated for many years in veterinary practice, (Penny 1953, Armistead 1954, Hall 1967, Foster 1967, Foster 1970, Finco 1974). It is the natural replacement fluid for blood loss and it adequately maintains the circulation.

Blood groups exist in dogs, but a single transfusion can usually be performed without reaction. In 10 to 15 % of second transfusions there will be some degree of reaction, ranging from agglutination of the transfused red cells to cell lysis. The signs of reaction include jaundice, anaphyllaxis and the lack of improvement, (Foster 1967).

Blood typing is possible but time consuming and cross-matching is easier.

Cross-/

Cross-matching involves taking blood samples from the donor and recipient, and separating the plasma and red cells. The red cells are washed and added to the plasma of the other dog, and incubated at 38°C for one hour. After this period, 30 % bovine albumin is added and the incubation is repeated for a further hour. The test is then read, by observing haemolysis or agglutination, (Hall 1967).

Blood transfusion is recommended for the replacement of major blood loss , though it should be administered with care and close observation.

There are some commercially produced composite fluids which contain amino acids, sugars, electrolytes and vitamins intended as infusions providing parenteral nutrition as well as fluid imbalance correction. These solutions are expensive and for complete utilisation of the amino acids, large quantities of sugar are required to provide the necessary calories. These calories can be derived from fats which are infused intravenously. This is a gross subject and is not considered here.



ADJUNCTS TO PARENTERAL FLUIDS

The use of fluids alone is generally satisfactory but certain other substances can be added to the infusions, for example,

- a) Extra electrolytes
- b) Antibiotics
- c) Vitamins
- d) Diuretics
- e) Analgesics
- f) Tranquillisers

a) Extra electrolytes These include potassium chloride and sodium bicarbonate. Dogs produce large quantities of acidic metabolic end products which constantly are buffered by the body systems. In cases of pH regulation upset where there is a metabolic acidosis, it has been recommended that sodium bicarbonate be infused at the rate of 2 to 6 mmol. per kilogram body weight, (Soma, Burrows and Marshall 1974).

Extra potassium can be added to infusion fluids or given orally which is safer if there is an indication to do so, for example a low serum potassium level. When there is cellular destruction potassium is lost from the body, and this must be replaced to permit the growth of new cells, (McMurray and Law 1961). Before the addition of potassium to the body, a diuresis should be present which can control the serum level of potassium by excreting excesses which may prove toxic to the heart, although in animals high serum potassium levels have been recorded without/

without evidence of cardiac malfunction.

Oral potassium can be administered in the form of potassium citrate or chloride, both of which have diuretic properties.

b) Antibiotics                      These should be administered when indicated and a broad spectrum antibiotic is best unless specific organism isolation and drug sensitivity have been performed. All patients in a state of circulatory failure should receive an antibiotic prophylactically to prevent infection and septicaemia from bowel stasis, (Soma, Burrows and Marshall 1974). The correct dosages, routes of administration and timing are given in Appendix 4.

c) Vitamins                      Vitamin supplements can be added to maintain body levels in patients who have been anorexic for prolonged periods or who are not permitted oral intake. Common supplements contain vitamins B and C which may be given intravenously with relative safety.

d) Diuretics                      Diuretics may be administered to patients who are oliguric even after fluid infusion, one drug being frusemide at 2 mg, per kilogram initially every six hours until a diuresis is established. The osmotic diuretic Mannitol can be given, although there is little information regarding its use in veterinary patients. It has been used experimentally at the rate of 0.5 to 2 grams per kilogram body weight per 24 hours, and adequate fluid should be infused to permit the correct diuresis. It is effective in reducing tissue oedema, (Soma, Burrows and Marshall 1974). It has been suggested that the use of mannitol is/

is to be preferred to frusemide in the treatment of oliguria, (Lee 1977).

e) Analgesics                    These have a place in veterinary cases where pain is evident or suspected. Suitable drugs are pethidine and pentazocine.

f) Tranquillisers                Tranquillisers should only be used in cases where the circulating blood volume has been re-established. Most sedative drugs produce a degree of hypotension which in a patient with a compensated circulation might prove fatal by inhibiting the normal body compensatory mechanism. In cases of severe circulatory collapse which appear refractory to fluid therapy alone, acepromazine maleate may be administered in an attempt to dilate the post-capillary sphincters and permit better body perfusion. This should only be done once parenteral fluid therapy is well underway, (Lillehei and MacLean 1958, Lillehei, Longerbeam, Bloch and Manor 1964).

## TREATMENT

There are three objectives of treatment,

- i) To ensure survival by maintenance of an adequate circulatory volume, thus giving renal control.
- ii) Stopping or treating the loss or imbalancing factor in fluid equilibrium.
- iii) Administering fluid in a corrective manner,

Success depends on treating each case as an individual, assessing the needs, and initiating suitable and correct therapy. Treatment does not cease once the calculated deficit has been administered or when the clinical signs resolve, but when the animal, preferably alive, leaves with the owner, (Hall 1967, Soma, Burrows and Marshall 1974). There is little doubt that there is a great improvement in the state of the fluid depleted patient following infusion even when minor error may be a feature of that application, (Foster 1970).

Treatment can be considered as two entities. Replacement therapy is the rehydration, correction of electrolyte imbalance and the re-establishment of blood volume. Maintenance therapy is needed to provide the daily requirement of water and electrolyte when a patient is incapable of self support,

There are two methods of determining the amount of fluid required to restore a body to its normal or previous state, (Hall 1967). An accurate history obtained from the owner can be correlated with the clinical/

clinical findings from which it is possible to determine a quantity of fluid relating to the patient's deficit. This is achieved by calculating the number of days of anorexia or reduced intake and using the known daily requirement of 40 ml. per kilogram per day. For the first day of illness, the animal will be deficient by 40 ml. per kg. body weight of water. For each succeeding day only 20 ml. per kg. is calculated since the output of urine will be reduced, (Hall 1967). Hence,

<u>Days of zero intake</u>	<u>Fluid loss</u> ml./kg.
1	40
2	20
3	20
4	20

Any additional losses are then considered such as vomiting, diarrhoea, pyrexia, tachypnoea and hyperpnoea, wound or organ discharge or effusion and other abnormal loss. It is difficult to calculate these losses, but an attempt should be made. This then gives a figure which is added to the normal loss to determine the fluid required for infusion. This method is relatively satisfactory but does not take into account a depleted circulatory volume and has inherent errors due to the additional losses.

The ancillary aids of biochemistry and haematology can be used prior to treatment and the total serum protein and haematocrit levels are correlated to assess the fluid deficit. The first error possible in this method is in the fact that the base line for each animal is not known, and therefore average values must be used. This could prove misleading in an/

an anaemic patient whose haematocrit might appear normal, but is an indication of haemoconcentration. From the results one determines the percentage dehydration and calculates the fluid requirement, (Hall 1967). This is not a satisfactory method and should not be used alone. It is better to treat the patient than to treat its blood chemistry, haematology or weight, (Moyer 1950).

Perhaps a third method should be considered. After assessment of the clinical appearance and the presenting signs, it is advisable to treat these symptoms and to establish a satisfactory renal output. This method requires monitoring and recording but is effective in the treatment of fluid imbalance cases. The first method of assessment can be used in conjunction with this, and the ancillary aids can be used to determine the success or failure of therapy.

The stages of therapy are now considered following calculation of the losses.

Replacement Therapy                      It is important to establish a circulating blood volume, (Penny 1953, Armistead 1954, Hall 1967, Finco 1974). A decrease in the blood volume constitutes a degree of hypovolaemic shock and it is important to prevent this becoming refractory to treatment. The initial signs of decreased blood volume are tachycardia and reduced pulse volume. This state progresses until the mucous membranes become pale and clammy and have a poor C.R.T.. In these cases the blood volume requires reinforcing with fluid administered intravenously.

This can be achieved initially with an infusion of Ringer lactate and a plasma volume expander or substitute, preferably dextran or/

or Haemaccel, in cases where blood loss is minimal or moderate. If the haematocrit is less than 20 %, an infusion of whole blood is indicated to replace losses. The amount infused should be sufficient to reverse the symptoms, and commonly 10 to 20 ml. of dextran 40 or 30 ml. of Haemaccel per kilogram body weight are required. The simultaneous infusion of Ringer lactate commences the rehydration process and prevents tissue dehydration.

Once the heart rate has decreased, the C.R.T. has improved and the pulse volume has increased, rehydration can continue until a satisfactory and steady output of urine is present assessed by bladder catheterisation and urine collection. Treatment of water and electrolyte deficits can be continued with Ringer lactate.

Patients who were not initially exhibiting signs of circulatory failure need only receive the rehydration form of therapy. In animals which have been vomiting, diarrhoeic or have wound or organ discharge or effusion, Ringer lactate is the replacement solution of choice, except in those cases where vomiting is from the stomach alone and the animal is alkalotic. It is perhaps advisable to use 0.9 % sodium chloride in these patients to avoid exacerbation of the pH imbalance. In animals with a pure water loss, for example due to hyperpnoea or tachypnoea, electrolytes are not as important and replacement may be achieved with 0.18 % Na Cl and 4.3 % dextrose.

The replacement of water and electrolyte can be undertaken in a short period of four to eight hours until a constant output of urine has been established. The rate of output should approximate to the normal 1 ml. per kilogram per hour, measured by means of a bladder catheter and graduated/

graduated container. The infusion of fluids at very high rates results in increased renal outputs and it is doubtful if total body fluid equilibrium is satisfactorily achieved.

Once a satisfactory renal output has been established, the specific gravity of the urine being produced is checked to assess the concentrating capabilities of the kidneys. If dilute urine is produced in quantity, it may indicate overinfusion or overhydration and the rate of infusion should be decreased. The normal output of urine in the dog is 20 ml. per kilogram per 24 hours and an output in excess of this is an indication of overinfusion. The normal specific gravity of dog urine is between 1.020 and 1.035, and if this increases it implies haemoconcentration or should it decrease it implies haemodilution or renal failure to concentrate.

If there is doubt regarding the output of urine, there are several possible reasons. Rehydration may not have been achieved and then it is important to check the other clinical signs such as the mucous membranes colour and consistency and the pulse rate and volume. If the mucous membranes are wet and the colour is normal, the pulse rate and volume are satisfactory, then overinfusion is probably due to renal impairment. A diuretic may be administered, preferably frusemide since this can be given without further increasing the circulatory volume. The infusion should be stopped and a urinary catheter passed to determine the output of urine. If the administration of frusemide fails after repeat doses, it is difficult to assist these patients other than by the slow infusion of fluid and possibly the addition of small quantities of mannitol. Peritoneal dialysis may be attempted but often in these cases would/



would be a necessary daily occurrence to prevent the accumulation of toxic agents, for example urea, in the body.

Anuria due to renal failure is difficult to treat and can be caused by chronic interstitial nephritis, glomerulonephritis or nephrosis. In cases of chronic nephritis it may be that the kidneys are so pathologically abnormal that any diuresis is impossible and treatment of these cases is hopeless. The use of diuretics is limited since most drugs depend on the kidney being capable of some normal function. Mannitol is a useful drug but it can cause circulatory overload in overinfused patients. It is advisable in suspected nephritic patients to administer a test dose of 20 to 30 ml. per kilogram over two hours, closely monitoring the output of urine. If the signs of overinfusion ( wet mucous membranes, increased respiratory rate and generalised oedema ) arise, then the infusion should cease and a diuretic be administered.

It is advisable in all dehydrated and shocked patients to insert a bladder catheter connected to a graduated bag to permit monitoring of the renal output.

Drug therapy should be instituted at this time with antibiotics, anti-emetics, analgesics and sedatives where indicated. If surgery is indicated, it can usually be performed once an output of urine is established since the body fluid homeostasis is approaching normality.

Maintenance Therapy                      Following the initial treatment to replace the body fluid loss, the animal requires to be maintained until it is capable of self support by oral intake. Routine maintenance therapy can be satisfactorily achieved with 0.18 % Na Cl + 4.3 % dextrose solution infused/

infused at 40 ml. per kilogram per day, the total daily fluid requirement. Additional losses due to pyrexia, vomiting, diarrhoea or discharges should be estimated and replaced with Ringer lactate. If animals have been anorexic for a prolonged period, extra potassium can be added at the rate of 2 mmol. per kilogram per day with caution and in the presence of a diuresis, and vitamins may be given as necessary.

An important part of maintenance therapy is to encourage the patient to consume fluid and food orally since the natural uptake of water and electrolyte is of more benefit than parenteral maintenance, (Moyer 1950, Hall 1967). The maintenance of veterinary patients using amino acid and vitamin infusions, parenteral nutrition, is not at present commercially viable. If cheaper, more satisfactory solutions can be developed, there will undoubtedly be a place for parenteral nutrition.

One possible contraindication for replacement therapy is in cases of intestinal foreign body where there may be occlusion of blood vessels at the site of obstruction. This results in the sequestration of fluid, oedema and congestion, and eventually in a few cases perforation of the intestinal wall, (Wilkinson 1955). Surgery is indicated in these animals and should be performed immediately following re-establishment of the circulating blood volume, further replacement of fluid to compensate dehydration taking place during and after the time of operation.

In other cases of body fluid deficiency where surgery is indicated, this should be performed as soon as possible following satisfactory parenteral fluid therapy. It is useful to administer fluids intravenously to patients undergoing prolonged or complex surgical procedures/

procedures to compensate for losses prior to, during and after operation, Ringer lactate is the fluid of choice and may be infused at a rate of 30 ml per kilogram body weight during the period of surgery. Any moderate to severe blood loss during surgery should be replaced either with whole blood or a plasma substitute such as "Haemaccel". The prophylactic use of parenteral fluid therapy during the period of surgery has been shown to be advantageous, (Shires 1961, Moore and Shires 1967, Roth, Lax and Maloney 1967).

The use of antibiotics pre-operatively in cases where surgery is expected to involve infected tissue is recommended to reduce the likelihood of post-operative infection and septicaemia, (Keen 1975, Rodgers 1976, Stone, Hooper, Kolb, Geheber and Dawkins 1976).

One form of fluid therapy which requires emphasis is the treatment of the acutely ill fluid deficient patient. An adequate airway is ensured, an intravenous cannula is inserted and an urinary catheter is passed to monitor the output of urine which is a sign of treatment success in animals with circulatory failure. An infusion of Ringer lactate and a plasma substitute is commenced without delay and a full clinical examination is performed once therapy is instituted. If the emergency therapy is successful, the previous forms of treatment can be instituted until the animal is capable of self support or corrective surgery is performed, (Soma, Burrows and Marshall 1974).

A use of fluid therapy which has not been discussed is the treatment of animals with an elevated blood urea level. The high blood urea level can be reduced by forced diuresis using a hydrating solution and diuretics. This may be performed once a satisfactory renal output has been achieved, and an urinary catheter is passed to avoid the discomfort of a continually full bladder. The solution of choice is 0.18 % Na Cl + 4.3 % dextrose with the addition of mannitol ( 1.5 grams every 24 hours ) . This procedure is satisfactory if the kidneys are normal or were relatively normal before the cause of the impairment occurred. It is essential to monitor the output of urine to ensure that overinfusion does not occur,

### SECTION 3

#### STUDY OF CLINICAL CASES

## STUDY OF CLINICAL CASES

### INTRODUCTION

During one year of this study, many cases requiring supportive parenteral fluid therapy were examined and treated. The most common cases are outlined below and the number of each type seen in the year is recorded.

1. Pyometritis	28	cases
2. Intestinal foreign body	20	"
3. Oesophageal obstruction	5	"
4. Diaphragmatic hernia	10	"
5. Road traffic accidents	14	"
6. Other trauma	4	"
7. Ruptured bladder	2	"
8. Intussusception	6	"
9. Miscellaneous	11	"
10. Intra-operative support	93	"
11. Post-operative support	26	"
	<u>219</u>	cases

All cases were referred by local practitioners and were presented most times by the owner from whom an accurate history could be obtained. The two groups of cases which were of most interest were,

Intestinal foreign body

Pyometritis

The dogs suffering from these conditions eventually underwent corrective surgery and were selected for investigation of parenteral fluid therapy, particularly during surgery. After selection of these two groups, various parameters were noted and measured regularly as outlined later. The two groups were chosen because these two conditions are relatively common in general practice and could be handled adequately by practitioners using fluid therapy.

An investigation was carried out to determine the advantages or disadvantages of the administration of fluid intravenously during surgery. To permit comparison, an untreated group was chosen and these dogs did not receive fluid supportive therapy. The other group received fluids during the period of surgery, and some received fluids pre-operatively and post-operatively as was necessary. Arguably all patients should have received parenteral fluid support, but with careful monitoring of set parameters and a strict method of assessment, it appeared that the study was justified. The initial selection of patients was based on the clinical examination and assessment, for example pulse rate and quality and C.R.T., at the time of admission with some correction of selection after consideration of the biochemical and haematological results, though in most cases the two forms of examination reached similar conclusions. It is perhaps interesting to note the adequacy of clinical examination and assessment when compared with the blood analyses. Other ancillary aids were used, commonly radiography, and this is recorded.

## MATERIALS AND METHODS

On presentation, all animals underwent a full clinical examination and assessment and history was obtained from the owner. It was often possible to evaluate the fluid requirements of most patients at this point. The questioning of the owner was at first general regarding age, sex, breed, past history, recent history, eating habits and normal appearance, and then went on to the present condition, attempting to determine the exact duration and the circumstances surrounding the condition. The history regarding eating habits, vomiting, (frequency and amount), defaecation, drinking (frequency and amount), urination (frequency and amount), discharges, coughing, movement and appearance was considered. The reproductive history of bitches suspected of having pyometritis was sought regarding irregular oestrus, previous parturition, pseudopregnancies and the date of the last season. Weight loss or gain was also assessed by the owner and this fact was used in the assessment.

The clinical examination involved all systems of the body with particular reference to appearance, weight, core and peripheral temperature, pulse rate and volume, heart rate and sounds, respiratory rate and nature, oral mucosa colour and consistency, capillary refill time, tissue turgor, mouth odour and tongue appearance. Tissue turgor was assessed at the elbow in all cases. Abdominal palpation was performed gently and many intestinal foreign bodies could be felt without discomfort to the animal. Often the dogs with pyometritis were not palpated in case uterine rupture should occur. Examination for discharge at the vulva and perineum was performed in bitches suspected of pyometritis.

The/



The clinical examination was recorded by the Termatrex system,  
( Glasgow University Veterinary School ).

The ancillary aids to diagnosis were investigated. Radiography  
was first to confirm the diagnosis and in one of the cases of intestinal  
foreign body, contrast radiography was performed.

10 ml. of blood was collected from the jugular vein and added  
to bottles containing E.D.T.A. or heparin for haematological or biochemical  
examination respectively. The following parameters were recorded,

Haematology	Packed cell volume
	Haemoglobin
	White blood cell count
Biochemistry	Blood urea
	Serum sodium
	" potassium
	" chloride
	Total serum protein

After confirmation of the diagnosis, surgery was arranged  
usually within 24 hours of admission. Some cases required pre-operative  
parenteral fluid therapy for shock and this was administered. All cases  
were given antibiotics on admission, (Penicillin 20,000 units/kg.,  
Streptomycin 10mg./kg. intramuscularly). This therapy was repeated every  
12 hours for a minimum of 72 hours.

The selection of cases was commonly made at this time. The main  
considerations were the degree of tachycardia, the pulse volume and the  
capillary/

capillary refill time, since any animal in a state of hypovolaemia was deemed in need of supportive fluid therapy. The final choice of the groups to receive or not to receive intra-operative fluid therapy was based on the blood urea and the haematocrit. Most animals with a haematocrit in excess of 50 ml. per 100 ml. or a blood urea in excess of 10 mmol. per litre were thought to require fluid therapy. It is seen in the results that some dogs did stray from one group to the other, but the comparison is still the same.

The dogs to receive fluid intravenously were give compound sodium lactate, ( Ringer lactate ), the rate being one-third of blood volume during the period of surgery. The calculation of blood volume was performed using the dog's weight at the time of surgery and the fact that the average blood volume is 90 ml. per kg., (Ashworth and Tiegertt 1940, Krieger, Storaasli, Friedell and Holden 1948, Penny 1953, Hall 1967, Finco 1977 ). This regime was strictly adhered to in order that some uniformity of case assessment would be possible.

Prior to surgery, premedication was with acepromazine maleate, ( 0.1 mg./kg. ), 30 minutes before induction by the intravenous administration of sodium thiopentone, the required dose being recorded. Acepromazine maleate was given to reduce the required dose of sodium thiopentone. The induction dose of barbiturate was administered slowly till the swallow reflex disappeared. An intravenous cannula was inserted for the administration of the Ringer lactate. Maintenance of anaesthesia was via a cuffed endotracheal tube and semi-closed circuit using halothane in oxygen. The duration of anaesthesia for surgery was recorded, being the time from induction to the cessation of administration of anaesthetic agent. The recovery time from the cessation of administration to extubation and/

and conscious movement was noted.

Surgery was always a midline laparotomy followed by enterotomy for the removal of a foreign body, or a total ovariectomy for the treatment of pyometritis. The degree of surgical trauma was assessed and recorded according to the system described on P. 32.

Fluid administration was started after the induction of anaesthesia and continued to the end of surgery.

Post-operatively, there was strict observation of the patients and recording of various parameters. This post-operative attention lasted for three to five days. The parameters monitored are listed later. The time of discharge was noted.

# RECORDING OF RESULTS

The actual recording of results presented little problem, but the day by day tabulation for this study did. It is my hope that the presentation, although apparently cumbersome, will provide the necessary information and conclusions.

The two groups were divided according to treatment status, the intestinal foreign body cases being considered first and the pyometritis cases second. The actual measurements made are outlined below.

<u>On Admission</u>	<u>General</u>	Number
		Breed
		Age
		Sex
		Weight
	<u>History</u>	Duration of present illness
		Eating habits since onset of illness
		Drinking " " " "
		Urination " " " "
		Defaecation " " " "
		Vomiting
	(Pyo. only)	Last oestrus date
		No. of litters
		Pseudopregnancies
		Irregular oestrus cycles
		Vaginal discharge
		Treatment
	<u>Clinical/</u>	

Arrangements for corrective surgery were generally made within 24 hours of admission and the following factors were noted.

Anaesthesia

Premedication

Induction thiopentone dose

Maintenance

Duration

Recovery time

Surgery

Operation

Trauma factor

Duration

Position of foreign body

Size of uterus

Clinical Examination

Appearance

Rectal temperature

Pulse rate

" volume

Respiratory rate

" nature

Mucous membrane colour

" " consistency

Capillary refill time

Tissue turgor

Abdominal palpation

Abdominal pain

Ancillary Aids

Radiology Visible signs

Size of lesion

Number

Other

Biochemistry

Blood urea

Serum sodium

" potassium

" chloride

Total serum protein

Haematology Packed Cell Volume

Haemoglobin

White blood cell count

The following measurements were made at 12, 24, 48, 72 and 96 hours post-operatively or until the time of discharge.

History - input/output

1. Eating
2. Drinking
3. Urination
4. Defaecation
5. Vomiting
6. Therapy

Clinical

7. Appearance
8. Rectal temperature
9. Pulse rate
10. Pulse volume
11. Respiratory rate
12. Respiratory nature
13. Mucous membrane colour
14. Mucous membrane consistency
15. Capillary refill time
16. Tissue turgor
17. Abdominal pain

Ancillary

18. Blood urea
19. Plasma sodium
20. Plasma potassium
21. Plasma chloride
22. Total serum protein
23. Packed cell volume
24. Haemoglobin
25. White blood cell count

The general information at admission was essential for further identification. The history from the owner regarding the present illness was collected including facts about duration, input and output, diagnostic aids such as the past history and information of treatment from the referring veterinary practitioner.

The clinical examination and assessment was thorough in each case to determine the various parameters of interest in this study. Appearance was based on the animal's normal appearance as assessed by the owner and also the expected as seen in dogs of that breed and age. Body temperature was recorded per rectum, the normal level being taken as 100.5 to 102.0 °F ( 38-39 °C).

The pulse was recorded at the femoral artery, the rate being assessed for breed, age, weight and temperament for the purposes of comparison. The pulse volume was similarly assessed.

The respiratory rate and nature were recorded. The mucous membrane colour and consistency were recorded commonly at the oral mucosae or the conjunctiva. The capillary refill time was recorded at the gum edge, the normal value being less than two seconds. Tissue turgor was assessed at the elbow, normal being instant return of the skin on "tenting".

The presence of abdominal pain was noted.

Blood samples were taken at regular intervals for analysis.

Any fluids administered post-operatively were recorded.

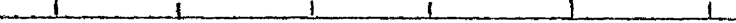


# HECAScore

To permit a direct comparison of cases, untreated and treated patients, a scoring system was established called the HECAscore, ( H - history, E - examination, C - clinical assessment, A - ancillary aids). The normal score for each of the 25 parameters numbered earlier is 10, giving a maximum score of 250 per recording interval. Each of the parameters was then assessed and given a score according to the nature of the results. Each parameter is considered on a + or - scale depending on the parameter and accorded a score of -15 through 0 to + 10 which is the maximum score being considered normal. The exact HECAscore system is explained in tabular form below.

## A. History - input/output ( H )

Degree of function	-	+	..++	+++	++++	+++++
1. Eating	0	5	10	5	0	-5
2. Drinking	0	5	10	5	0	-5
3. Urination	0	5	10	5	0	-5
4. Defaecation	0	5	10	5	0	-5
5. Vomiting	10	0	0	-5	-10	-15
6. Therapy	10	0	-5			

score 

Maximum 60

B. Clinical - examination and assessment ( EC )

7. Appearance	Collapsed	-----	-10
	Very dull	---	-5
	Dull	--	0
	Bright	-	5
	Normal	N	10

8. Rectal temperature

less than 97 <sup>0</sup> F	-----	-10
97 - 98	---	-5
98 - 99	--	0
99 - 100	-	5
100 - 102	N	10
102 - 103	+	5
103 - 104	++	0
104 - 105	+++	-5
more than 105	++++	-10

9. Pulse rate

N	10
+	5
++	0
+++	-5
++++	-10

10. Pulse volume

-----	-10
---	-5
--	0
-	5
N	10

11. Respiratory rate	N	10
	+	5
	++	0
	+++	-5
12. Respiratory nature Dyspnoea	--	0
Hyperpnoea	-	5
Normal	N	10
13. Mucous membranes colour		
Congested	-	0
Normal	N	10
Pale	+	5
14. Mucous membranes consistency		
Dry	---	0
Moist	-	5
Normal	N	10
15. Capillary refill time		
less than 2 secs.	N	10
2 - 4 seconds	+	5
4 - 6 "	++	0
6 - 8 "	+++	-5
more than 8 secs.	++++	-10
16. Tissue turgor	-----	-10
	----	-5
	---	0
	-	5
	N	10

17./

17.	Abdominal pain	Absent	N	10
		Present	+	0
			++	-5
			+++	-10

Maximum 110

C. Ancillary - ancillary aids ( A )

18.	Blood urea	0 - 6 mmol/l	N	10
		6 - 10 "	+	5
		10 - 20 "	++	0
		20 - 30 "	+++	-5
		30 - 40 "	++++	-10
		more than 40	+++++	-15
19.	Plasma sodium	115 - 125 mmol/l	---	-5
		125 - 135 "	--	0
		135 - 145 "	-	5
		145 - 160 "	N	10
20.	Plasma potassium	1.5 - 2.5 mmol/l	--	0
		2.5 - 3.5 "	-	5
		3.5 - 5.5 "	N	10
21.	Plasma chloride	less than 60	-----	-15
		60 - 70 mmol/l	-----	-10
		70 - 80 "	----	-5
		80 - 90 "	--	0
		90 - 100 "	-	5
		100- 120 "	N	10

22. Total serum protein

50 - 70 g/l	N	10
70 - 80 "	+	5
80 - 90 "	++	0
90 - 100 "	+++	-5

23. Packed cell volume

less than 32	--	0
32 - 38 ml/100ml	-	5
38 - 42 "	N	10
42 - 47 "	+	5
47 - 52 "	++	0
52 - 57 "	+++	-5
more than 57	++++	-10

24. Haemoglobin

less than 10	-	0
10 - 14 g/100ml.	N	10
14 - 16 "	+	5
16 - 18 "	++	0
18 - 20 "	+++	-5
more than 20	++++	-10

25. White Blood Cell count

5 - 10 thsnd.	N	10
10 - 20 "	+	5
20 - 30 "	++	0
30 - 40 "	+++	-5
40 - 50 "	++++	-10
more than 50	+++++	-15

Maximum 80

The HECA scores are recorded for each group for each time interval, and then compared for each group.

The maximum score for any individual dog at any one time is

250

TABLES OF RESULTS

The tables of results of the measurements taken at the set intervals of on admission and post-operatively at 12, 24, 48, 72 and 96 hours are given in Appendix 5 for the cases of intestinal foreign body. The tables indicate the HECAscores calculated for each of the three groups of measurments and the total score for each dog on each day.

## RESULTS

The cases of intestinal foreign body are considered first.

General information regarding each case was obtained.

Table 1.

<u>Number</u>	<u>Breed</u>	<u>Age</u> (years)	<u>Sex</u>	<u>Weight</u> (kg)
1	Labrador	3	M	34
2	Westie	6	M	9.75
3	Collie	8/12	M	10
4	Collie	4	M	16
5	Jack Russell Terrier	2	F	6
6	Labrador	10	M	44
7	C.Spaniel	7	F	13
8	Collie	1	M	18
9	Collie	5	F	11.75
10	Collie	7/12	M	10.25
11	Alsatian	1	M	11
12	Alsatian	1.5	M	25
13	Sheltie	5	F	14.25
14	Labrador	4.5	F	33
15	Dobermann	3	F	24.25
16	Jack Russell Terrier	4	M	6.25
17	Collie	1	M	16.25
18	Collie	7	M	27.5
19	Labrador	1	M	17
20	Dalmatian	8/12	M	14



The selection of cases for the two groups,

Untreated

Treated

was based on the clinical signs and then confirmed or otherwise by the blood analysis results. The clinical signs which determined that a dog should receive treatment were increased tissue turgor, dry, tacky, congested mucous membranes, increased pulse rate but decreased volume and general dullness. Dogs with a blood urea level in excess of 10 mmol. per litre and a P.C.V. of 50 % or over were deemed in need of parenteral fluid therapy. In the following sections any numbers areas of histograms or lines on tables or graphs which are in red indicate the untreated dogs. These animals received no fluid supportive therapy during the period of surgery.

There were 7 dogs in the untreated group and 13 dogs in the treated group. Of the 13 treated dogs, 4 required fluid therapy pre-operatively, one on admission as emergency therapy to prevent the advance of shock, and one dog required fluid therapy post-operatively. All 13 treated dogs received 30 ml. per kg. body weight of Ringer lactate during the period of surgery. None of the untreated patients received pre-operative parenteral fluid therapy.

The breed, sex and age incidence is now considered. There were 14 male dogs and 6 female dogs in the survey, but since the number involved is small, little can be concluded from this. The breeds involved varied, though more collie type dogs and labradors had intestinal foreign bodies than other breeds. The numbers are,

Table 2.

Collie	7
Labrador	4
Alsatian	2
Jack Russell Terrier	2
Westie	1
Dobermann	1
Sheltie	1
Dalmatian	1
C. Spaniel	1

There is perhaps a breed disposition, but it was my impression that the largest number of dogs seen in the hospital were of the collie cross type.

The age incidence was variable from 7 months to 10 years of age.

The ages of dogs have been divided into 4 groups as follows,

Table 3.

i)	Less than 1 year	3 dogs
ii)	1 - 5 years	10 "
iii)	6 - 10 years	7 "
iv)	more than 10 years	0 "

It therefore appears that there is a higher incidence in dogs under 5 years of age, although it seems that most ages of dogs may be affected, and/

and that it is not only a condition of young inquisitive animals. The average age was 3.4 years.

The weight of dog concerned was as variable as the breed involvement and weights varied from 6.25 kg. to 44 kg.

The historical background to all the cases was then considered and the following list of details are discussed. The duration of illness was variable.

Table 4.

<u>Days</u>	<u>Dogs</u>
2	1
3	3
4	5
5	2
6	1
14	3
21	3
28	1
42	1

On further analysis it was determined that there was little correlation between the duration of illness, the clinical signs, the biochemical and haematological data and the position of the foreign body in the intestine. 13 dogs had only been ill for less than one week, whereas the other 7 had been ill for some time before veterinary advice was sought. In fact 3 of the dogs had been treated by the referring veterinary surgeons without success, the diagnosis in all three cases being incorrect. These three/

three dogs were referred to the Glasgow University Veterinary Hospital Medicine Department as possible cases of gastro-enteritis.

The most common complaint of the owner was of the dog vomiting and it was found most suitable to assess cases on the number of times they vomited each day of the illness. This information was quite readily obtained from the owners, and is given below.

Table 5.

i)	1 - 2 times per day (+)	0	dogs
ii)	2 - 4 " " " (++)	2	"
iii)	5 - 10 " " " (+++)	16	"
iv)	10 - 20 " " " (++++)	1	dog
v)	continuously (+++++)	1	"

16 dogs vomited five to ten times per day and only one dog vomited continuously. When these figures were analysed in relation to duration of illness, position of object in the bowel and the biochemical data, there was no obvious correlation. It would appear, however, that characteristically dogs with intestinal foreign bodies do vomit, and most commonly quite often each day. It was not possible to relate the vomiting to feeding or drinking water, but some owners reported that vomiting was spontaneous and unrelated to other factors.

The other symptoms reported by the owner were anorexia and commonly oligodipsia or normodipsia. Only three dogs were reported to be polydipsic. Most animals were still passing urine and faeces. Despite the loss/

loss of electrolytes in these animals by vomiting, they were not drinking excessively to compensate for this loss which is contrary to the theoretical situation.

The clinical examination and assessment was the next stage of investigation, although one case received emergency treatment for shock before this was performed, ( dog number 20 ). This dog died post-operatively.

The general appearance of most dogs was either dull or collapsed and this was assessed on a minus scale for purposes of comparison. The incidence of each is shown below.

Table 6.

Normal appearance	{N}	0	dogs
Bright	{-}	0	"
Dull	{--}	13	"
Very dull	{---}	6	"
Collapsed	{----}	1	dog

The one collapsed dog was number 20. The untreated dogs were all considered to be dull.

The rectal temperature was recorded in all cases on admission and was found to vary from dog to dog as is seen below.

Table 7/

Table 7.

Less than 100.5 °F	1	dog
100.5 - 102 "	5	dogs
102 - 103 "	12	"
103 - 104 "	6	"
more than 104 "	0	"

The average temperature was 102.2 °F ( 38.5 °C ) and in almost all dogs (15) was elevated. This is most likely due to inflammation at the site of obstruction and endogenous pyrogen release. Infection did not appear to play an important part, since at laparotomy there was little evidence of infective peritonitis or enteritis. There was no relationship between temperature and the selection of the treated and untreated groups.

The cardiovascular system was evaluated in all cases, the pulse rate and volume being recorded and assessed for the breeds involved. The results are given below on the basis of the assessment, since marked differences existed in the rates of large and small breeds. A scoring system of + for every 10 pulses per minute increase in rate above that normally expected was adopted.

Table 8.

Normal	5	dogs
+	5	"
..++.	5	"
+++	3	"
++++	2	"

The volume of the femoral pulse was assessed by a similar technique dependant on the breed of dog. The results were,

Table 9.

Volume		
-	11	dogs
--	8	"
---	1	dog

It is apparent that all the dogs had some decrease in pulse volume and 15 showed an increase in the heart rate, presumably due to the dogs being in a state of circulatory compensation as a result of the body fluid deficiency. The untreated dogs were least affected. The one dog with the markedly decreased pulse volume was number 20.

The respiratory rates and nature were observed and recorded, and it was noted that although all the dogs had acceptable rates, 4 were hyperpnoeic. There was no apparent reason for this respiratory characteristic other than the presence of abdominal pain in these dogs.

The mucous membranes were examined and were found to be congested in 19 dogs and pale in one ( number 20 ). The membranes were dry in 16 dogs and moist but not normal in 4 dogs. The untreated dogs were divided between the two. The membrane appearance was confirmed haematologically, since all dogs had increased haematocrits, except number 20.

The capillary refill time was estimated in all dogs. The times are recorded below.

Table 10/

Table 10.

Normal	(0 - 2 seconds)	10	dogs
+	(3 - 4 " )	9	"
+++	(7 - 8 " )	1	dog

The untreated dogs all had relatively normal refill times, and in all but one case the C.R.T. was less than 4 seconds. The other dog was number 20.

The tissue turgor was assessed in the dogs at the point of the elbow and a scoring system was adopted, based on the time taken for the return of the tented piece of skin. The results are given below.

Table 11.

Normal	2	dogs
-	6	"
--	7	"
---	5	"

The untreated dogs had better tissue turgor than the treated cases. All but two dogs showed signs of dehydration.

Abdominal pain was assessed at the time of abdominal palpation and was recorded using a scale + to +++ to indicate intensity. The results were,

Table 12/



Table 12.

Pain		
-	8	dogs
+	6	"
++	4	"
+++	2	"

Abdominal pain was present in 12 cases, but there was little correlation between the other clinical signs and this finding. Pain was not related to the size of the foreign body or the duration of the illness.

An abdominal mass was palpable in all but one dog, and this dog ( number 2 ) was rotund and obese as well as being fractious.

The ancillary aids to diagnosis are considered.

#### Biochemistry

The blood urea level was assayed and the results were,

Table 13.

Normal (0 - 6 mmol./l.)	9	dogs
+ (6 - 10 " )	3	"
++ (10 - 20 " )	5	"
+++ (20 - 30 " )	1	dog
++++ ( over 40 " )	2	dogs

The/

The majority of dogs had blood urea estimations below 12.5 mmol. per litre and the untreated dogs were all within this number. The other three dogs had raised blood urea levels, two over 40 mmol. per litre. The exact reason for these high levels is not fully understood, but this is not due to renal disease and is referred to as pre-renal uraemia.

The serum electrolytes are considered. Sodium was assayed in all dogs and the results are given below. The normal level and range was divided into a high and low normal range as is seen in the following table.

Table 14.

Normal	{160 - 148 mmol./l}	6	dogs
"	{148 - 136 " }	10	"
-	{135 - 126 " }	3	"
--	{125 - 116 " }	1	dog

6 dogs had normal sodium levels in the upper range and 10 dogs had sodium levels within the lower normal range. All the dogs in this study had been vomiting and low sodium levels were expected. Most of the dogs were clinically dehydrated and therefore the amount of plasma water would be reduced, hence there would be a relative increase in the electrolyte values. Although the serum sodium levels are apparently normal, in some of the dogs they may be reduced. The untreated dogs were all but one within the normal range.

Potassium/

Potassium in the serum was measured and the results were,

Table 15.

Normal	{5.5 - 4.5 mmol./l.}	3	dogs
"	{4.5 - 3.5 " }	9	"
-	{3.5 - 2.5 " }	7	"
--	{2.5 - 1.5 " }	1	dog

The potassium level was lower than normal in all but 3 dogs, and all the untreated dogs had acceptable levels. Once again the degree of relative increase due to dehydration must be considered. It is shown, therefore, that the potassium level does fall with vomiting since the kidneys excrete potassium to conserve sodium.

The chloride level was estimated and the results were,

Table 16.

Normal	{115 - 100 mmol./l.}	5	dogs
-	{100 - 90 " }	5	"
--	{90 - 80 " }	4	"
---	{80 - 70 " }	3	"
----	{70 - 60 " }	2	"
-----	less than 60 "	1	dog

The/

The chloride levels showed the greatest change and the values ranged from 121 to 45 mmol. per litre. The lower chloride level cases were all treated. This result is expected since vomit is rich in hydrochloric acid. It was of interest to note that there was no correlation between the duration of illness, the persistence of vomiting or the position of the object in the bowel and the chloride value in the serum. There was some relationship between the electrolyte levels in that dogs with low chloride values also had low sodium and potassium levels.

The total serum protein level was assayed to allow an assessment of the degree of dehydration and the results were,

Table 17.

Normal	(50 - 70 g./l.)	12	dogs
+	(70 - 80 " )	6	"
++	(80 - 90 " )	2	"

The plasma protein levels appear normal in 12 dogs and elevated in 8 dogs, but it should be noted that the pre-illness levels are not known. The value of this estimation in the determination of dehydration is discussed later.

#### Haematology

The packed cell volume was estimated and the results were assessed and recorded below.

Table 18/

Table 18.

Normal	{38 - 42 %}	2	dogs
+	{43 - 47 %}	4	"
++	{48 - 52 %}	4	"
+++	{53 - 57 %}	4	"
++++	{58 - 62 %}	2	"
+++++	{more than 62%}	3	"

One dog had a P.C.V. on admission of 27 % and it is not included in the above table. Most animals appear to have an elevated haematocrit and 6 of the untreated dogs had levels below 50 %. These increases are due to the reduction in the extracellular water content which directly affects the intravascular water, and therefore the P.C.V. increase is relative.

If the haematocrit and total serum protein levels are considered in millilitres per 100 ml. and grams per litre and are added together, the figure is of the order of 100 in the normal animal. Acceptable limits are between 88 and 112. If the previous results for these two estimations are considered, the following is seen,

Table 19.

Table 19.

i)	80 - 90	1	dog
ii)	90 - 100	2	dogs
iii)	100 - 110	2	"
iv)	110 - 120	5	"
v)	120 - 130	4	"
vi)	130 - 140	3	"
vii)	140 - 150	2	"
viii)	150 - 160	2	"

It is later seen that the level decreases as the patients improve. The treated cases all had relatively higher values. This method of dehydration assessment requires further investigation.

The haemoglobin level is considered in all dogs and as expected the result parallels the change in the haematocrit. The results were,

Table 20.

Normal	(10 - 14 g/l.)	0	dogs
+	(15 - 16 " )	6	"
++	(17 - 18 " )	6	"
+++	(19 - 20 " )	4	"
++++	(more than 20 g/l.)	3	"

The haemoglobin level in all but one of the dogs was elevated and/

and the rise was relative to the reduction in plasma water.

The total white blood cell count was calculated to determine the degree of inflammation or infection that was present. The increase in most dogs was due to an increase in neutrophils. The results are given below.

Table 21.

Normal	{ 0 - 10 th./c.mm)	2	dogs
+	{ 10 - 20 " }	9	"
++	{ 20 - 30 " }	6	"
+++	{ 30 - 40 " }	2	"
++++	{ 40 - 50 " }	1	dog

Most dogs had an elevated white blood cell count but there was little correlation between this and other findings. The changes seen post-operatively are of interest as described later.

The other commonly used ancillary aid was radiography and the results are given below. Lateral and dorso-ventral plain films were taken of each dog on admission. In one instance contrast radiography was necessary to outline the foreign body. In another dog the radiographic examination was negative, but a foreign body could be palpated within the abdomen. The size and number of foreign objects seen on the lateral radiographs are recorded below.

Table 22/

Table 22.

<u>Dog</u>	<u>Radiographic Diagnosis</u>	<u>Number of objects</u>	<u>Size of object(s)</u> (cm.)
1	+	1	6 x 4
2	+	1	3 x 2
3	-	1	10 x 1
4	+ with Ba	1	6 x 6
5	+	2	3 x 2 5 x 1
6	+	1	5 x 6
7	+	1	4 x 1
8	+	1	4 x 3
9	+	1	4 x 3
10	+	1	2 x 5
11	-		
12	+	1	4 x 3
13	+	1	3 x 3
14	+	1	4 x 3
15	+	1	3 x 2
16	+	2	2 x 2 2 x 2
17	+	1	3 x 3
18	+	16	Av. 3 x 2
19	+	1	4 x 4
20	+	1	4 x 4



Examples of the radiographs are given, ( Figs.9 - 14 ).

Most foreign objects were visible on the plain X-ray film, and it was possible in these dogs to estimate the size of the object. It was found that in most instances lateral films of the abdomen would suffice. In dog number 3 a longitudinal mass was palpable, though there was some doubt as to its form. The barium contrast examination quite adequately outlined a linear non-radioopaque foreign body.

It was of interest to compare the objects removed from the bowel at laparotomy with those seen at radiographic examination. Stones and similar objects were quite clearly seen whereas rubber and cork were more difficult to determine.

The presenting symptoms of these dogs are now discussed. The owner's complaint in most cases was of anorexia, oligodipsia and vomiting. The duration of illness varied from a few days to several weeks, but the severity of the symptoms was not related to the duration, except that those dogs which had been ill for some time were thin and often weak. All the dogs were dull in appearance and had a tachycardia with a decrease in the pulse volume. The mucous membranes were dry and congested but the capillary refill time was relatively normal. The rectal temperature was  $102.2^{\circ}\text{F}$  on average. A loss of tissue turgor was noted in all dogs and abdominal pain was only present in a few of the animals. In all but one dog the foreign object was palpable per abdomen.

The blood analyses revealed changes in the electrolyte levels, especially a decrease in the chloride, and increases in the haematocrit and the total serum protein indicative of dehydration. All the dogs had an/

Fig. 9 .      Dog 8      Foreign body with gas proximally

Fig. 10 .      Dog 18      Foreign bodies in proximal and mid-  
jejunum.

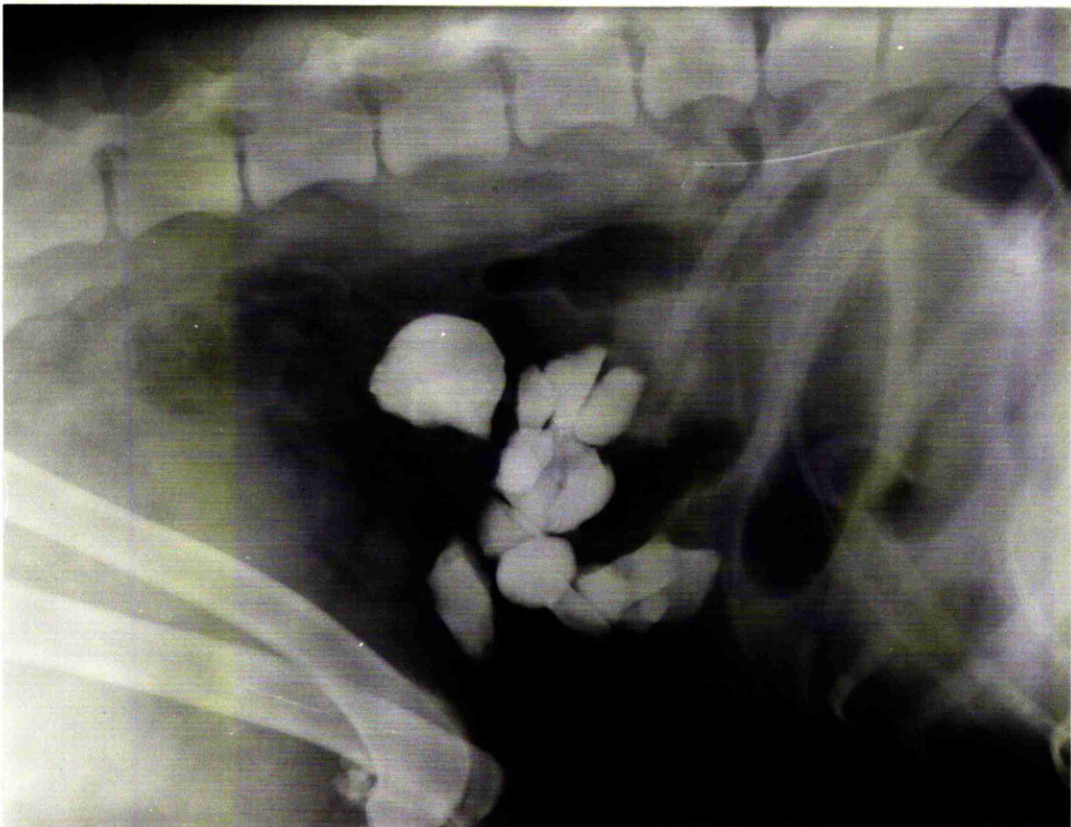
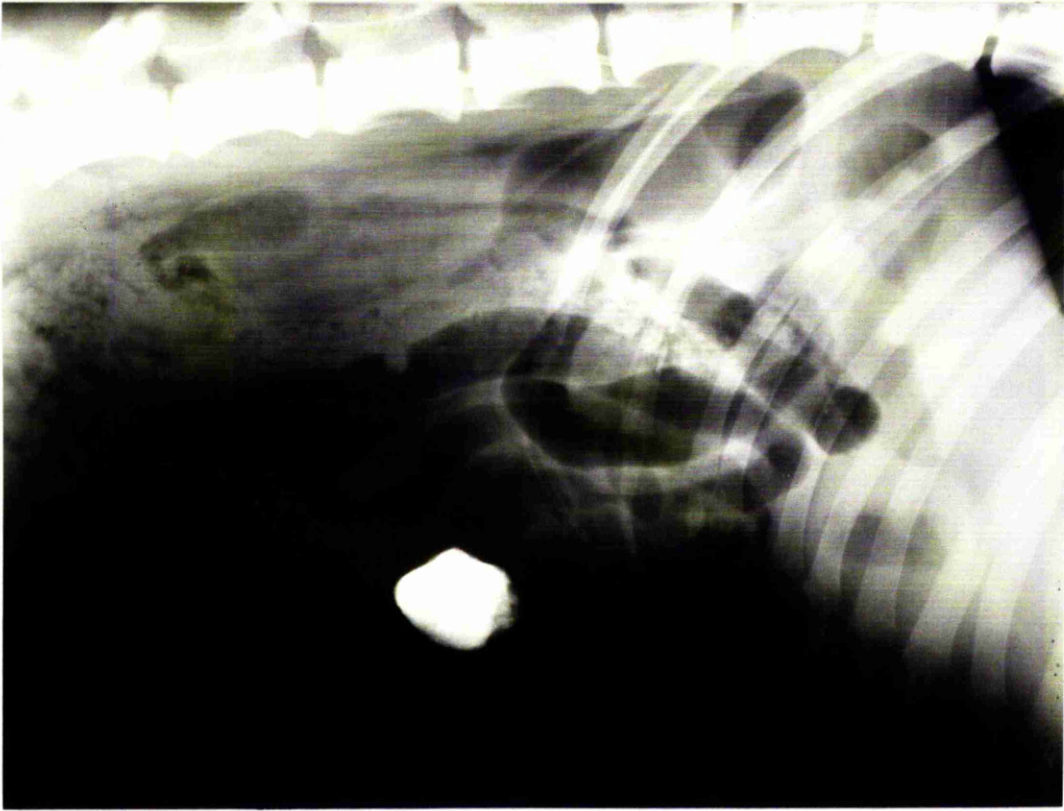


Fig. 11. Dog 3 Non-radioopaque foreign body.

Fig. 12. Dog 3 Foreign body outlined with barium.

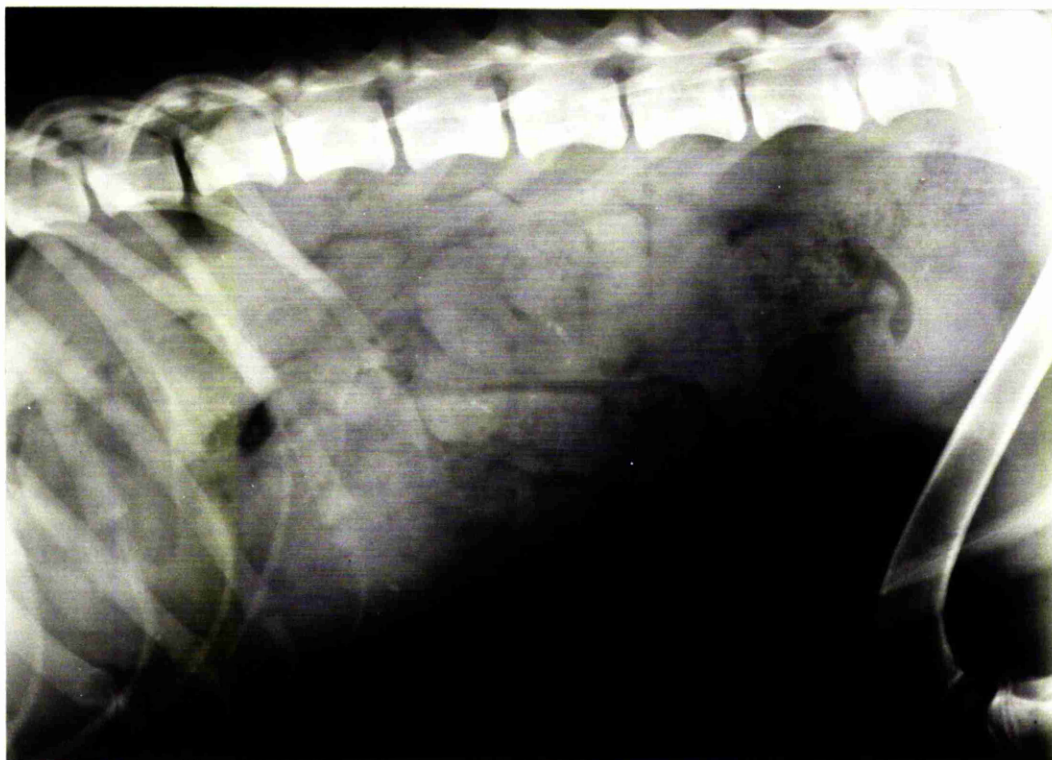
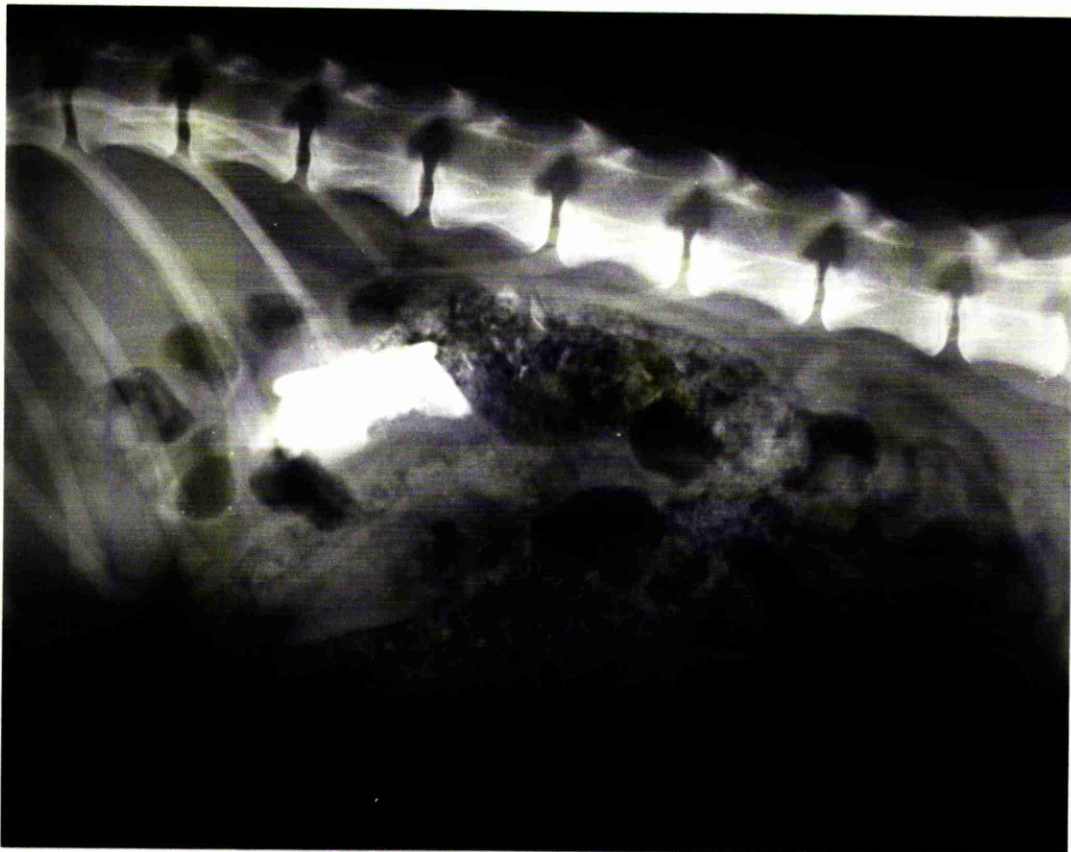


Fig. 13. Dog 1 Corn cob foreign body - mid-jejunum.

Fig. 14 . Dog 10 Piece of shower unit - distal jejunum.





an increased white blood cell count.

Radiography of the abdomen confirmed all but one case.

Surgery was arranged in all cases within 24 hours of admission.

Four cases required parenteral fluid therapy pre-operatively, and one dog required fluid supportive therapy post-operatively. The selection of dogs for the two groups, treated and untreated, was performed after the clinical examination and assessment and confirmed by the blood analyses results. It is worthwhile to note the selection of cases and to see how adequate the clinical examination was in the determination of the physical status. The following table outlines the choice of group on admission, the correct group biochemically and haematologically, and finally the group to which dog was assigned. The dogs receiving fluid pre-operatively are also indicated.

Table 23/

Legend:        U - Untreated group  
                 T - Treated group  
                 PT - Pre-operative fluid and treated group



Table 23.

<u>Dog</u>	<u>Clinical selection</u>	<u>Blood analysis selection</u>	<u>Actual group</u>
1.	U	U	U
2.	U	U	U
3.	U	U	U
4.	U	U	U
5.	U	U	U
6.	U	U	U
7.	U	T	U
8.	U	U	T
9.	U	T	T
10.	T	U	T
11.	T	T	T
12.	T	T	T
13.	T	T	T
14.	T	T	T
15.	T	T	T
16.	T	T	PT
17.	T	T	T
18.	T	T	PT
19.	T	T	PT
20.	T	T	PT

It is seen that one dog selected for the untreated group should have received treatment according to the results of the blood analysis. Two dogs which were treated were clinically selected as untreated dogs but were reassigned following the blood analyses results. One dog allocated to the treated group clinically had blood results suitable for the untreated group but remained within the treated group. It appears that there is much to be gained by assessing the cases clinically since in only one dog in this study was the selection wrong, and the blood results for this dog were only marginally over the selection level, (Blood urea 11.9 mmol./l., Packed cell volume 52 %).

Anaesthesia was routine in all cases and the information regarding the induction dose of sodium thiopentone given intravenously by titration and observation, the duration of anaesthesia and the recovery time were recorded. The dose of Ringer lactate was noted, and the infusion rate calculated. The results are given below.

Table 24./

Table 24.

<u>Dog</u>	<u>Thiopentone (mg./kg.)</u>	<u>Duration (hours)</u>	<u>Recovery (mins.)</u>	<u>Fluid dose (ml.)</u>	<u>Infusion rate (ml./kg./hr.)</u>
1.	8	1.25	10		
2.	7.5	2	15		
3.	12.5	1.5	20		
4.	12	1.5	10		
5.	12	1	20		
6.	9	1.25	30		
7.	11.5	1	15		
8.	11	2	5	540	15
9.	7	1.25	5	360	24.5
10.	12	1.75	5	310	17
11.	9.5	1.5	10	330	20
12.	5	1.75	20	750	17
13.	10.5	1.5	5	430	20
14.	8.5	1.5	5	990	20
15.	10	1.75	5	750	17
16.	8	1.25	10	185	24
17.	10	1.5	5	490	20
18.	12	2	10	850	15
19.	8	1.5	5	400	20
20.	2	1	60	420	30

The averages for some of these facts were calculated and are given in the following table, considering the difference between the treated and the untreated groups. One dog was discounted from the treated group since it died later, ( dog number 20 ).

Table 25.

<u>Measurement</u>	<u>Overall average</u>	<u>Untreated average</u>	<u>Treated average</u>
Thiopentone dose (mg./kg.)	9.3	10.3	8.7
Duration (hours)	1.5	1.5	1.5
Recovery (minutes)	13.5	17.1	7.5
Fluid infusion rate (ml./kg./hour)			20

The induction dose of sodium thiopentone was marginally higher in the untreated group, but the difference is not of significance. The dose was administered by titration until the swallow reflex abated.

The duration of anaesthesia in all cases was between one and two hours.

The recovery time for the untreated group was noticeably longer than that of the treated group by about 10 minutes. This result is of possible significance and may be attributable to the intravenous fluid support during the period of surgery. No more after-care was given to the treated group above that given to the untreated group and the recovery was in similar circumstances on a theatre trolley and in a kennel.

The/

The surgery in all the dogs involved a midline laparotomy and then single or multiple enterotomy. The degree of surgical trauma was assessed and recorded. The number and type of the foreign objects were recorded, and these results are given below.

Table 26/

Table 26.

<u>Dog</u>	<u>Surgical Trauma</u>	<u>Enterotomies</u>	<u>Site of F.B.</u>	<u>Number of F.B.'s</u>	<u>Type of F.B.</u>
1.	4	1	M.J.	1	Corn cob
2.	3	1	P.J.	1	Rubber bung
3.	4	1	I.	1	Wood stick
4.	3	1	P.J.	1	Stone
5.	3	2	P.J.	3	Stone Leather Rubber ball
6.	4	1	M.J.	1	Stone
7.	3	1	M.J.	1	Bone
8.	3	1	M.J.	1	Stone
9.	2	1	M.J.	1	Stone
10.	3	1	D.J.	1	Shower unit
11.	3	1	D.J.	1	Plastic teat
12.	4	1	I.	1	Stone
13.	3	1	M.J.	1	Stone
14.	3	1	I.	1	Stone
15.	3	1	M.J.	1	Stone
16.	3	2	D.J.	3	Corks
17.	2	1	P.J.	1	Rubber ball
18.	4	3	P.M.J.	17	Stones
19.	2	1	P.J.	1	Rubber ball
20.	4	1	I.	1	Stone

P.J. proximal jejunum

M.J. mid "

D.J. distal "

I. ileum

The average trauma factor was 3 and commonly only one enterotomy was required. ( multiple enterotomies in 3 dogs , 5, 18, 18 ) The most common sites were jejunum and ileum, there being 6 objects in the proximal jejunum, 8 in the mid-jejunum, 3 in the distal jejunum and 4 in the ileum.

17 dogs had only one foreign object, 2 had three and one dog had seventeen stones in its intestine. The most common object was the stone.

One point of interest was that at the time of surgery it was noted that 4 dogs ( 16, 18, 19 and 20 ) had oedema of the bowel wall proximal and adjacent to the foreign object. These dogs had received pre-operative fluid therapy and the bowel oedema has been attributed to this, (Wilkinson 1955). The infused fluid appears to sequester at the site of obstruction and may make closure of the enterotomy site more difficult. The treatment given to these dogs was as follows,

Dog 16.	200 ml. Ringer lactate
Dog 18.	250 ml. dextran 40, 750 ml. Ringer lactate
Dog 19.	200 ml. dextran 40, 500 ml. Ringer lactate
Dog 20.	150 ml. dextran 40, 450 ml. Ringer lactate

The treatment in these dogs was necessary to prevent shock progressing further and to make anaesthesia safer. The question of bowel cedema in these cases was secondary to life itself, but it is of interest to note what difference pre-operative fluid therapy can make to the degree of surgical difficulty.

### HECAscores

The HECAscores for the dogs are now considered. The origin=1 scores on admission are used as a reference level, and the recording of parameters was made at 12, 24, 48, 72 and 96 hours post-operatively.

The scores for each group of parameters is discussed as follows,

A.	Input/output	Maximum score	60
B.	Clinical signs	" "	110
C.	Ancillary aids	" "	80
H.	HECAscore	"	250

The untreated group is always outlined in red. For the purpose of comparison of the two groups, untreated and treated, the scores have been averaged and graphed. Dog number 20 is only included in the original scores and was discounted thereafter since it died, and would unnecessarily lower the values at 72 and 96 hours.

The scores for the above three groups are tabulated below and graphed.

Table 27.

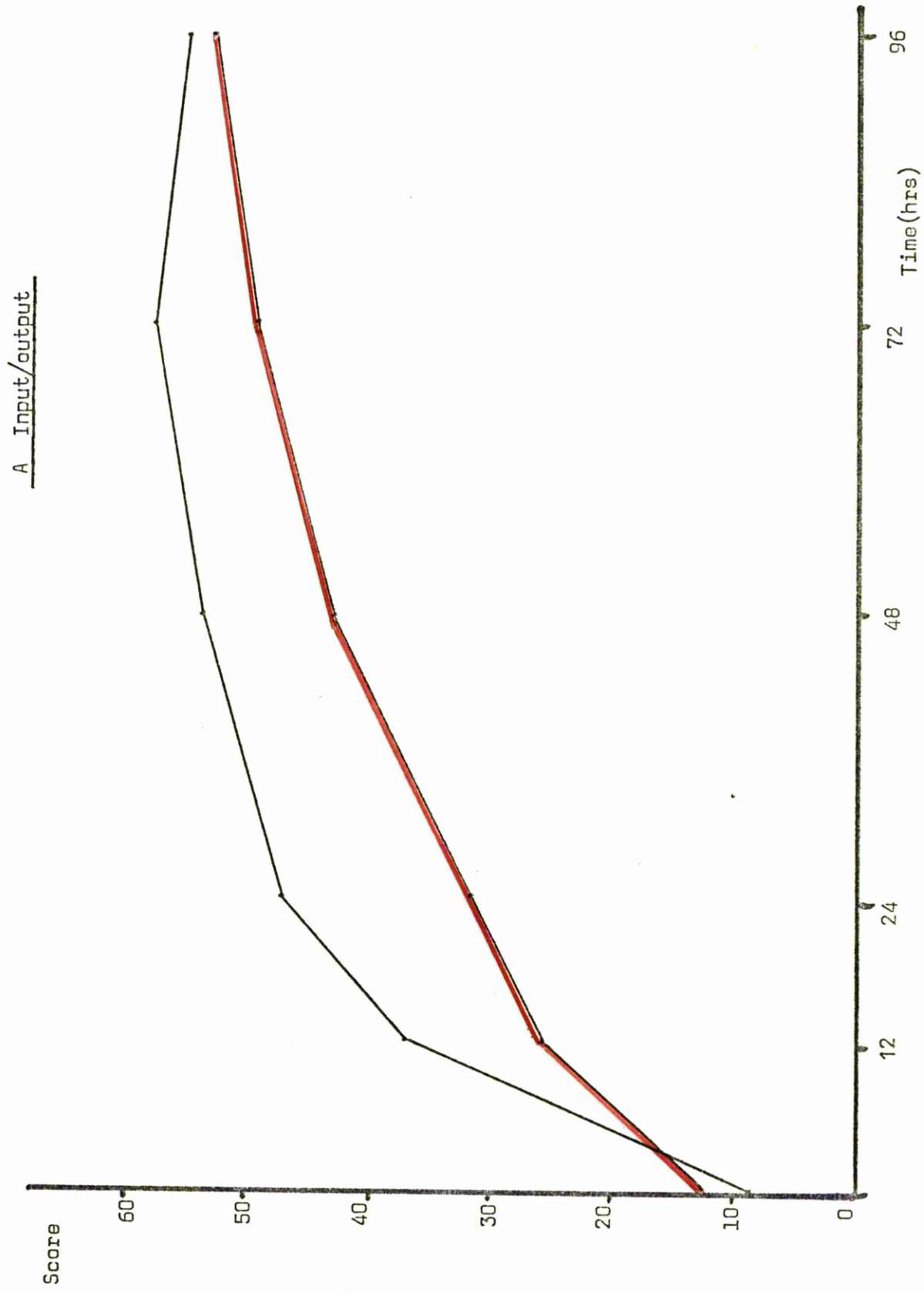
Graphs 1 - 3.



Table 27.

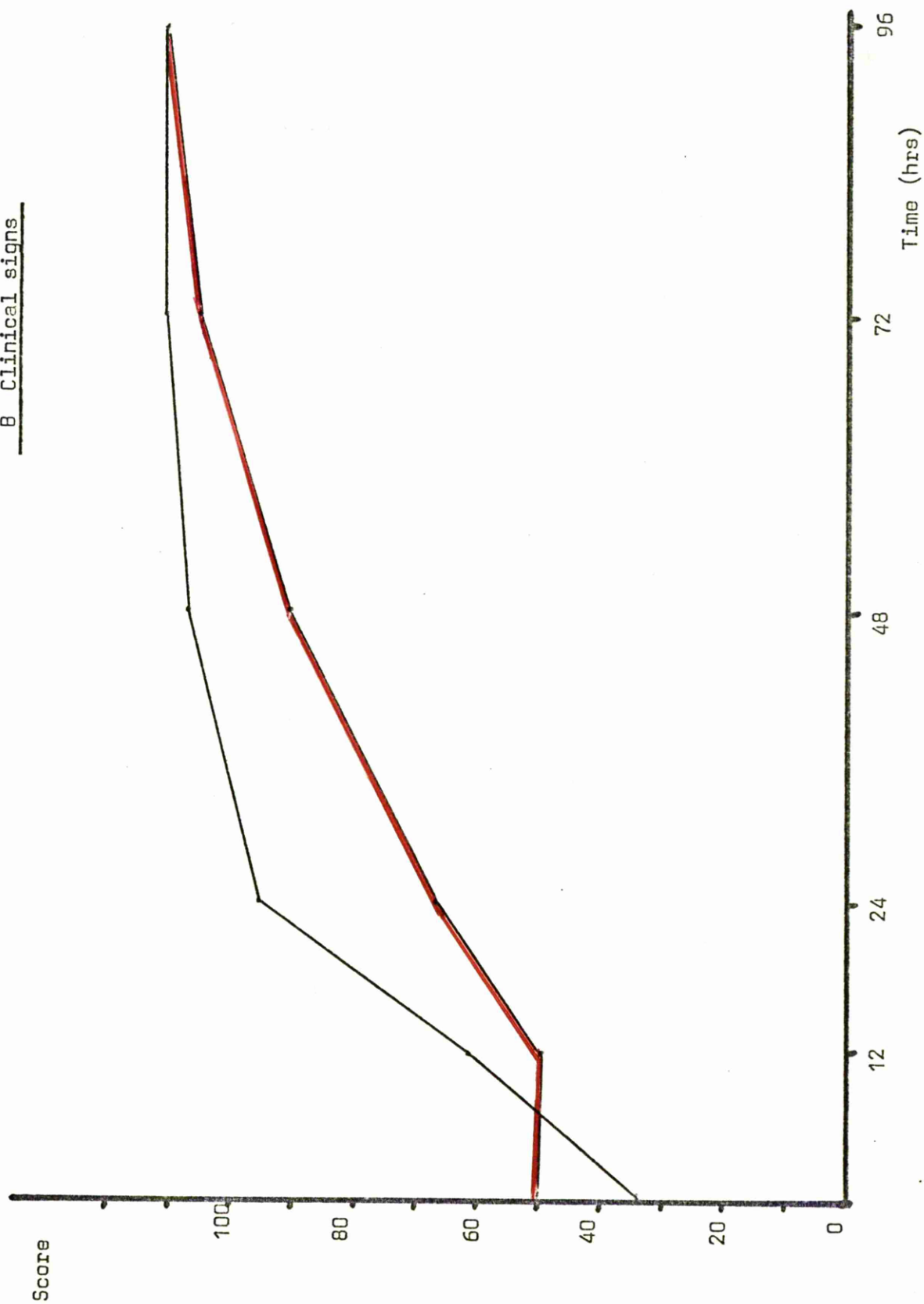
ble 27.		Scores and $\frac{24}{4}$ HECA scores																								Hours	
		0				12				48				72				96									
Dog	A	B	C	H	A	B	C	H	A	B	C	H	A	B	C	H	A	B	C	H	A	B	C	H			
1	15	70	60	145	35	65	55	155	40	95	65	200	50	105	70	225	55	110	70	235							
2	20	50	55	125	25	40	50	115	40	60	55	155	40	100	60	200	55	105	60	220	55	110	70	235			
3	15	45	65	125	15	50	35	100	35	70	55	160	45	100	75	220	50	105	75	230	55	110	75	240			
4	15	45	60	120	30	45	60	135	30	55	35	120	50	85	50	185	60	110	70	240							
5	5	50	50	105	35	60	45	140	35	70	30	135	40	100	65	205	45	105	75	225	55	110	75	240			
6	10	45	45	100	20	45	35	100	15	70	45	130	40	70	10	120	55	85	35	185	50	110	65	225			
7	0	50	30	80	20	45	30	95	30	50	15	95	35	75	35	145	40	100	55	195	50	110	65	225			
8	25	85	50	160	45	85	45	175	55	100	70	225	55	110	70	235											
9	10	60	40	110	40	80	55	175	45	100	70	215	60	110	65	235											
10	10	35	60	105	40	85	60	185	50	110	70	230	60	110	80	250											
11	20	45	20	95	45	65	60	170	55	105	70	230	60	110	75	245											
12	10	25	25	60	35	75	55	165	55	105	35	195	55	110	50	215	55	110	60	225							
13	10	45	5	60	35	70	40	145	30	105	60	195	40	110	70	220	60	110	75	245							
14	5	30	15	50	35	60	60	155	40	110	65	215	50	110	65	225											
15	15	15	20	50	40	35	50	125	55	85	45	185	50	110	65	225	55	110	80	245							
16	15	45	-35	25	25	65	15	105	45	75	55	175	45	85	50	170	60	110	65	235	50	110	75	235			
17	10	40	-25	25	35	80	25	140	50	110	55	215	55	110	60	225											
18	10	0	10	20	40	10	35	85	35	85	50	170	60	105	65	230	60	110	70	240							
19	-5	25	-15	5	30	30	15	75	45	65	30	140	50	105	50	205	55	110	70	235							
20	-10	-10	15	-5	5	40	-10	55	10	60	20	90	35	75	25	135	45	65	25	135	5	50	25	80	Died		

Graph 1.

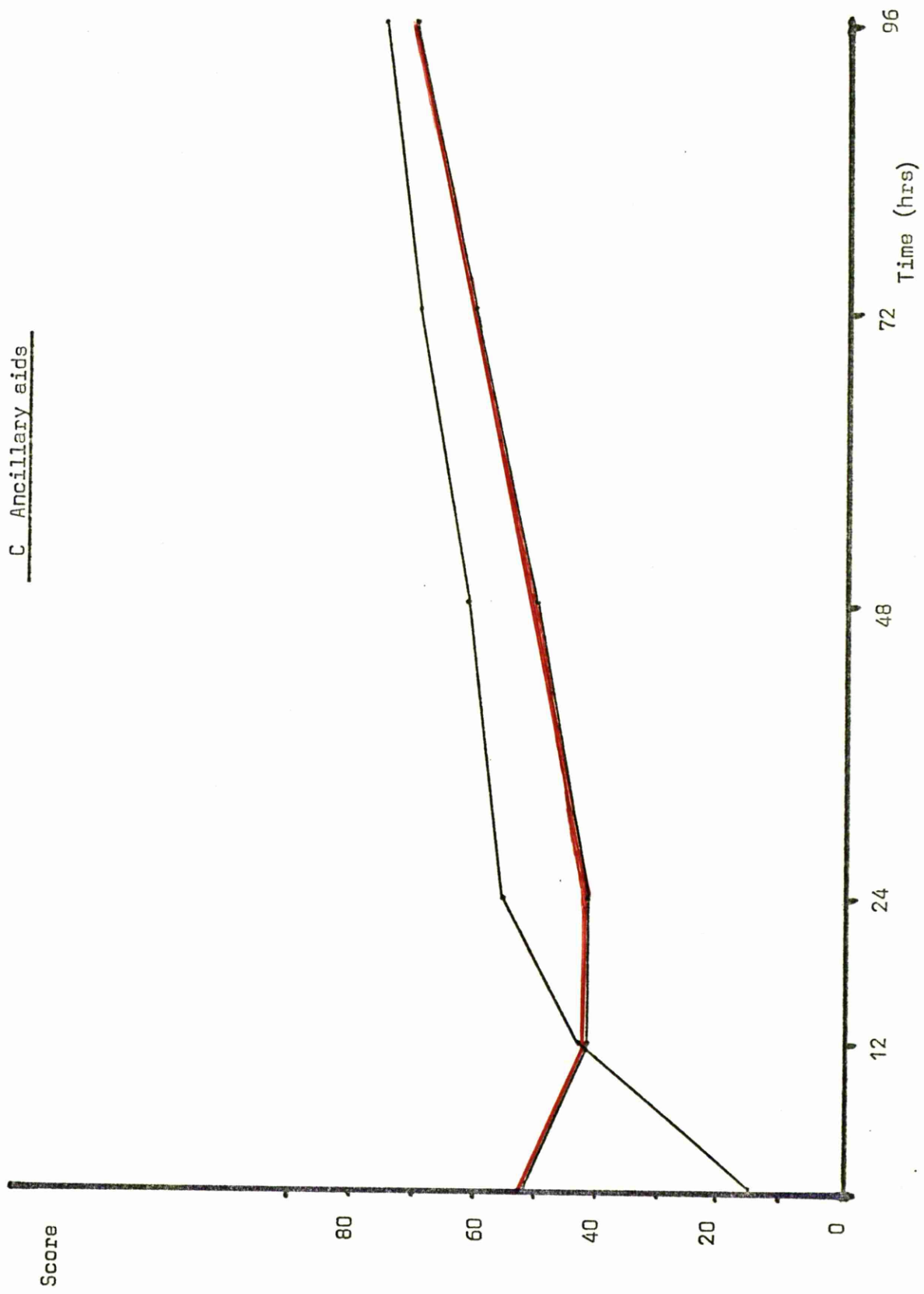


Graph 2.

B Clinical signs



Graph 3.



It is now possible to analyse the effect of administering fluids to animals undergoing surgery for the removal of an intestinal foreign body. The scoring system appears quite satisfactory in the overall assessment of the various parameters and gives a good indication of the progress of cases. The scores are considered separately and then overall as the HECA score.

#### A. Input/output

The initial scores in the two groups were similar since all the dogs had been anorexic, oligodipsic, vomiting and had decreased urine and faeces outputs.

Within 12 hours of surgery, the treated group showed more improvement than the untreated group, and most of the treated dogs drank water and urinated satisfactorily. The untreated dogs did improve but not as rapidly as those receiving fluids parenterally during surgery.

At 24 hours post-operatively, the treated dogs were approaching normality, but the untreated group were still behind and it took these latter dogs a further 48 hours to reach the same level as the treated group at the 24 hour time.

By 96 hours and discharge, the two groups had similar scores which were normal.

#### B. Clinical signs

The clinical appearance of the untreated group was marginally better than the treated group since this was one of the methods of group selection/

selection.

12 hours post-operatively, the untreated dogs showed no improvement, but the treated group demonstrated a marked improvement which continued to the 24 hour time when the scores were approaching normality. It was evident that the untreated dogs took an extra 48 hours to reach a similar state of improvement. At 96 hours and discharge, all the dogs had similar scores except number 20 which died.

C. Ancillary aids

The aids considered were the blood analyses.

Initially the untreated group were markedly better, but within 12 hours of surgery, the scores for the two groups were similar. At 24 hours post-operatively, the treated group showed a marked improvement, greater than the untreated group and it was not until the 96 hour time that the two groups had similar scores. It appeared that the treated group improved 48 hours earlier than the untreated dogs.

The blood analyses are now considered individually to determine the exact changes taking place in the body fluid composition. This is achieved by histograms which illustrate the day by day progression of the blood analysis results. Some dogs were discharged at 72 hours since they were considered normal and hence the histograms are not complete. The histograms are to be found in Appendix 5.

a)/

a) Blood urea

Histogram 1.

The blood urea decreased in all the treated dogs and by 24 hours the urea levels were less than 20 mmol. per litre. In some of the untreated dogs, however, the blood urea level increased post-operatively and took 24 to 48 hours to return to normal. It is seen in the histogram that the untreated dogs remained relatively static during the first 48 hours and only improved after this point.

b) Plasma sodium

Histogram 2.

The sodium levels in the blood return to within normal limits in 24 hours in all the dogs and it is of interest to note that the untreated dogs change little if at all. After 24 hours all the sodium levels were within normal limits.

c) Plasma potassium

Histogram 3.

The potassium levels take longer to return to normal than the sodium and the progress is slower in the untreated dogs. By 72 hours most of the animals had normal potassium levels in the plasma.

d) Plasma chloride

Histogram 4.

The decrease in the chloride level in the plasma in these dogs was greater than for any other electrolyte. Similarly the increase post-operatively took longer and the length of time was the same in the untreated/

untreated and the treated groups. The levels of chloride in the untreated dogs was initially higher and it may be evident that the treated dogs did improve more rapidly, especially within the first 24 hours. By 96 hours all the dogs had relatively normal chloride levels.

e) Total serum protein/Haematocrit

Histogram 5.

These two assays were considered together enabling an assessment of the degree of dehydration. The untreated dogs do not appear to rehydrate as rapidly as the treated group, and the untreated animals took 48 hours longer than the treated dogs to improve.

f) Haemoglobin

Histogram 6.

The haemoglobin level was shown to be increased in all the dogs on admission, and it appears that the untreated group took longer to return to normal than the treated animals. By 72 hours, all the dogs were considered to be normovolaemic, except dog number 20 which was anaemic.

g) White blood cell count

Histogram 7.

The W.B.C. count was moderately increased in all the dogs on admission and within 24 hours of surgery, the levels had increased. This increase was transient and the levels returned to normal within 72 hours. There were no differences between the two groups.



HECAscores.

Graph 4.

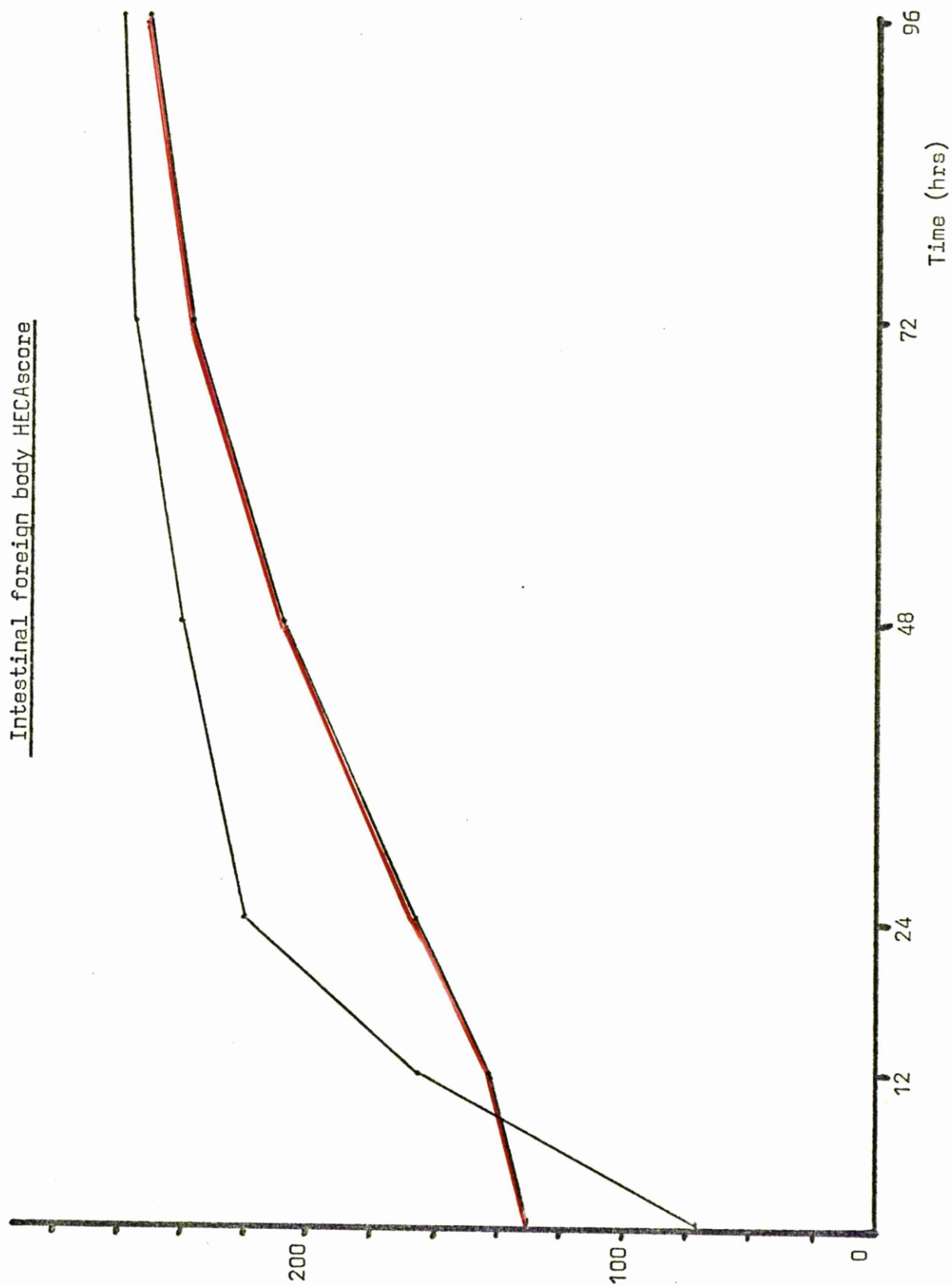
The three scores, A - input/output, B - clinical signs and C - ancillary aids were added together to give the HECAscore.

Initially the HECAscore is higher in the untreated group, but within 12 hours of surgery, the treated group score is higher than the untreated group, which remains relatively static during this period. Within 48 hours, the score of the treated group of dogs was within normal limits and only improved slowly after this time. The untreated group took 48 hours to reach the same level of improvement.

It would appear that there is an advantage to be gained from the administration of fluids intravenously during the period of surgery in cases of intestinal foreign body. The amount of fluid given was small, ( 30 ml. per kg. ), but it seemed to advance the overall recovery by at least 48 hours. The untreated group were initially clinically, biochemically and haematologically better than the treated group, and hence the improvement in the treated animals is better than is demonstrated since they were in a poorer state at the time of surgery.

The use of Ringer lactate can be recommended during the period of surgery in dogs undergoing the removal of an intestinal foreign body. The infusion improves the recovery rate and therefore the time of discharge. It appears from the results that the infusion may aid in the restoration of the body fluid balance in these dogs, firstly by supplying some electrolyte and water, and secondly by ensuring that the dogs drank and ate post-operatively/

Graph 4.



operatively which would allow correction of any further fluid imbalance.

One observation made during this study was that the treated dogs all appeared to be thirsty post-operatively, moreso than the untreated group who showed little interest in food or water during the first 24 hour period after surgery.

Tables of Results

The tables of the results of the measurements taken at the set intervals of on admission and post-operatively at 12, 24, 48, 72 and 96 hours are given in Appendix 5 for the cases of pyometritis. The tables indicate the HECA score calculated for each of the three groups and the total score for each dog on each day.

The cases of pyometritis are now considered. General information regarding each case was recorded.

Table 28.

<u>Number</u>	<u>Breed</u>	<u>Age</u> (yrs)	<u>Weight</u> (kg.)
1.	Staff. Bull Terrier	8	20
2.	Cairn	14	6.75
3.	Airedale	9	25.25
4.	Poodle	14	4
5.	Collie	12	20.5
6.	Westie	5	9.5
7.	Labrador	4	22.5
8.	Collie	10	14.5
9.	Great Dane	6	62
10.	Cairn	6	7.75
11.	Alsatian	8	16.75
12.	Alsatian	8	33
13.	Poodle	5	11.75
14.	Keeshound	9	15.5
15.	C.Spaniel	6	17.75
16.	Sheltie	9	19
17.	Labrador	8	16.5
18.	Collie	11	22
19.	Labrador	9	30
20.	Whippet	7.5	10
21.	Sheltie	10	15
22.	Collie	11	22.5
23.	Alsatian	9	40.5

The selection of cases for the groups,

Untreated

Treated

was based initially on the clinical examination and assessment and then confirmed/

confirmed by the blood analyses. In the following sections, any number, area of histogram or line on a graph/<sup>in red</sup> indicates the untreated dogs. These animals received no supportive fluid therapy during the period of surgery and there were 8 bitches in this group. The other group of treated cases contained 15 bitches, and of these two ( 21 and 22 ) received fluid therapy care pre-operatively on admission as emergency treatment, and four dogs ( 18, 20, 22 and 23 ) required post-operative parenteral fluid therapy. All the treated dogs received 30 ml. per kilogram body weight of Ringer lactate during the period of surgery.

The untreated dogs were selected on their clinical signs and if there was any doubt as to the eventual outcome, the dog was treated. The blood analysis gave the added facility of determining if the clinical assessment was correct. Dogs with a P.C.V. in excess of 50 % and a blood urea in excess of 10 mmol. per litre were deemed in need of fluid therapy.

The breed and age incidence of pyometritis are now considered. The breeds involved varied, although there were again more collie type dogs than any other breed. There was little breed predilection.

The age incidence was of more interest, since classically pyometritis is a condition of the older female dog. The ages have been divided into four groups for analysis and are recorded in the following table.

Table 29./

Table 29.

i)	0 - 4 years	1	dog
ii)	5 - 8 years	10	dogs
iii)	9 - 12 years	10	"
iv)	more than 12 years	2	"

The highest incidence does appear to be in the older dog between the age of 5 and 12 years. The average age was 8.7 years.

The weight of the dogs was as variable as the breed involvement and weights ranged from 4 to 62 kilograms.

The historical background to all the cases was investigated with particular reference to the duration of illness, the date of the last oestrus, history of pseudopregnancies and of irregular inter-oestrus periods. The results are recorded below.

Table 30/

Table 30.

Dog	Duration of illness (days)	Last oestrus (weeks)	Litters	Pseudop.	Irr. oestrus
1	42	10	0	-	+
2	21	52	0	-	-
3	3	4	1	-	-
4	4	3	0	++	++
5	6	4	0	-	-
6	14	6	0	++	++
7	5	4	0	-	-
8	28	9	3	-	-
9	56	16	0	++	-
10	2	52	1	-	+
11	7	16	0	-	-
12	5	10	3	+	+
13	7	12	0	++	++
14	4	6	2	-	-
15	4	3	0	+	-
16	4	6	0	+	-
17	4	6	0	-	-
18	5	5	0	++	++
19	6	6	0	-	-
20	4	6	3	-	-
21	14	6	0	+	+
22	7	8	0	-	-
23	7	6	0	-	++

The interoestrus periods and the incidence of pseudopregnancies were assessed on a plus scale depending on their occurrence.

The duration of illness is considered first, and it seems that most of the dogs were only ill for less than one week (17 dogs), but that there/



there was little correlation between the duration and the symptoms.

Some of the untreated dogs ( 5 ) had been ill for some time, but were not as apparently ill as some of the others.

The reproductive history is of interest. Most of the dogs ( 17 ) were nulliparous. The date of the last oestrus was analysed and the following table compiled.

Table 31.

<u>Weeks since oestrus</u>	<u>Dogs</u>
0 - 2	0
3 - 4	5
5 - 6	9
7 - 8	1
more than 8	8

It would appear that the dogs had either been in season within the last 6 weeks or had been in season many weeks ago. There was no apparent relationship between the duration of illness and the last oestrus date.

The other two factors, pseudopregnancies and irregular inter-oestrus periods were considered and the following table compiled.

Table 32.

	-	+	++	
Pseudopregnancies	14	4	5	dogs
Irregular interoestrus	14	4	5	"

There was no direct relationship between these conditions and a predisposition to pyometritis in this study.

The owner's complaint is now considered. Most reported anorexia, polydipsia, dullness and some reported vomiting and a vaginal discharge. Vaginal discharge is often a characteristic of pyometritis cases, and the incidence is recorded in this study.

Table 33.

<u>Discharge</u>	<u>Dogs</u>
-	7
+	9
++	6
+++	1

16 dogs had a vaginal discharge as reported by the owner and 7 showed no sign of discharge. Some bitches were reported as having been ill for a few days before the discharge was noted. In other bitches, the discharge was the first sign accompanied by polydipsia.

Vomiting was a presenting sign in 16 dogs, the incidence being once or twice each day. The untreated dogs vomited least, and 4 of them did not vomit at all. The exact cause of this vomiting is not known and on further analysis it did not appear to be related to any other change, either biochemical or clinical.

Anorexia was noted in all the dogs, being total in 17 of them. 6 of the untreated dogs were eating a little food each day. Polydipsia was/

was reported in many dogs and was assessed for the purposes of comparison, using ++ as normal.

Table 34.

+	2	dogs
++ (normal)	2	"
+++	14	"
++++	4	"
+++++	1	dog

The majority of dogs were polydipsic (19), but there was no apparent relationship between the untreated and the treated groups. The thirst is presumably stimulated by the excess fluid loss due to pyrexia and the vaginal discharge. However, on analysis, there was little correlation between the discharge, pyrexia and the degree of polydipsia.

Most of the dogs were reported as being dull.

In conclusion therefore, it would appear that most of the bitches with pyometritis are between 5 and 12 years of age, are polydipsic, anorexic, dull, may vomit and may have a vaginal discharge.

The clinical signs are now considered. Two dogs were given emergency supportive fluid therapy on admission. The general appearance of most dogs was dull, and this was assessed for the purposes of comparison, the results being recorded below.

Table 35/

Table 35.

Normal	(N)	0	dogs
Bright	(-)	4	"
Dull	(--)	12	"
Very dull	(---)	6	"
Collapsed	(----)	1	dog

The one collapsed dog was treated on admission with parenteral fluid therapy.

The rectal temperature was recorded and assessed on a rising scale. The results are given below.

Table 36.

Less than 100 °F	(-)	2	dogs
100 - 102 °F	(N)	10	"
102 - 103 "	(+)	8	"
103 - 104	(++)	2	"
more than 104 °F	(+++)	1	dog

The majority of dogs had a rectal temperature between 100 and 103 °F, and the average was 102.2 °F.. The most likely reason for the pyrexia is the inflammation and infection within the uterus, causing endogenous pyrogen release. This is confirmed by the increase in the white blood cell count.

The/

The cardiovascular system was evaluated. The results are based on the expected for each breed and age of dog. The results are given below for the pulse rate and the pulse volume.

Table 37.

<u>Pulse rate</u>		
Normal (N)	4	dogs
(+)	5	"
(++)	9	"
(+++)	4	"
(++++)	1	dog

Table 38.

<u>Pulse volume</u>		
Normal (N)	5	dogs
(-)	9	"
(--)	7	"
(---)	2	"

Most of the dogs had a tachycardia and a decrease in the pulse volume, and all but two of the treated dogs showed these signs. The untreated dogs were less severely affected. This result is expected due to the body fluid deficiency present in these dogs as demonstrated by other clinical signs and the blood analyses results.

The respiratory rate and nature were recorded and assessed,  
and/

and it was noted that there were changes in both. The results were,

Table 39.

Normal rate	(N)	16	dogs
	(+)	4	"
	(++)	3	"

Table 40.

Normal character	17	dogs
Hyperpnoeic	6	"

Most dogs had a normal respiratory function, but the common changes were tachypnoea and hyperpnoea, often related to pyrexia.

The mucous membranes were examined in all the dogs and assessed for colour and consistency. The results were,

Table 41.

Pale	membranes	3	dogs
Normal	"	5	"
Congested	"	15	"

Table 42./

Table 42.

Normal consistency		5	dogs
Moist	"	3	"
Dry	"	15	"

The membranes were commonly congested and dry in both the untreated and treated groups. The membrane appearance was confirmed haematologically, there being an increased haematocrit.

The capillary refill time was noted in each dog and recorded according to the time.

Table 43.

Normal	(N)	14	dogs
3 - 4 seconds	(+)	6	"
4 - 5	" (++)	0	"
5 - 6	" (+++)	2	"
more than 6	(++++)	1	dog

Most dogs had a normal or near normal capillary refill time and one dog with a time in excess of 6 seconds was given emergency therapy on admission. It would appear that a state of circulatory failure did not exist in these dogs, or at least in the majority of them.

The tissue turgor was assessed at the elbow and recorded.

Table 44./

Table 44.

Normal	(N)	6	dogs
	(-)	4	"
	(--)	10	"
	(---)	3	"

17 of the dogs showed some decrease in tissue turgor indicative of dehydration. The untreated dogs were almost as equally affected as the treated group.

Abdominal pain was assessed in all the dogs though abdominal palpation was not practised to avoid uterine rupture. Pain was recorded as being present or absent. It was absent in 17 dogs and present in 6 dogs, and it might appear that abdominal pain is not a typical sign of pyometritis.

The ancillary aids to diagnosis are now considered.

#### Biochemistry

The blood urea level was assayed in all the dogs and the results recorded below.

Table 45.

Normal	(0 - 6 mmol./l.)	13	dogs
(+)	(7 - 10 " )	4	"
(++)	(11 - 20 " )	4	"
(+++)	(21 - 30 " )	0	"
(++++)	(31 - 40 " )	0	"
(+++++)	(more than 40 )	2	"



21 dogs had a blood urea less than 20 mmol, per litre and 13 dogs were within normal limits. The untreated dogs were all normal except one whose blood urea level was 10.9 mmol, per litre. One of the dogs with a blood urea in excess of 40 mmol, per litre was given emergency therapy on admission. The cause of the raised blood urea may be renal or pre-renal in origin. It has been suggested that the renal lesion is a glomerulonephritis caused by the deposition of immune complexes most likely comprising antibody and antigen. The antigen in most cases of pyometritis is of E. coli, this being produced in excess in the uterus and absorbed through the distended uterine wall. Antibody attached to this antigen is deposited in the glomeruli and causes renal impairment, (Asheim 1965). Fortunately in most cases this lesion is reversible on removal of the source of antigen, and this is achieved by ovariectomy.

The plasma sodium level was measured and recorded in all the dogs and the results were,

Table 46.

Normal	(N)	22	dogs
(125 - 136 mmol./l.)	(-)	1	dog

The sodium levels appear to be relatively normal, but one should note the degree of dehydration present in some of these dogs and therefore the sodium levels may be increased relatively. The untreated dogs were all within normal limits.

The/

The plasma potassium was assayed and recorded. The normal level was divided into a high and low group.

Table 47.

{5.5 - 4.5 mmol./l.}	{N}	3	dogs
{4.4 - 3.5 " }	{N-}	14	"
{3.4 - 2.5 " }	{-}	6	"

The potassium levels in the plasma were generally lowered and this could be due to the loss in the vaginal discharge, although there was little correlation between the potassium level and the amount of discharge. Anorexia may contribute to the lowered plasma potassium result.

The plasma chloride was considered and recorded. Some of the dogs had been vomiting and one might expect altered chloride levels.

Table 48.

{115 - 110 mmol./l.}	{N}	19	dogs
{99 - 90 " }	{-}	2	"
{89 - 80 " }	{--}	2	"

The chloride levels were normal in 19 dogs, 12 of whom had been vomiting. The 4 dogs with lowered chloride levels had all been vomiting/

vomiting. The degree of dehydration should be noted when considering these results.

The total serum protein was assayed to assess the degree of dehydration. The results were,

Table 49.

(50 - 70 g./l.)	(N)	6	dogs
(71 - 80 " )	(+)	10	"
(81 - 90 " )	(++)	5	"
(91 - 100 " )	(+++)	2	"

The protein level is elevated in 17 dogs, 7 being untreated cases and this is due to the dehydrated state of these bitches.

#### Haematology

The haematocrit was recorded in the dogs and the results were,

Table 50.

(23 - 27 % )	(---)	1	dog
(28 - 32 " )	(--)	2	dogs
(33 - 37 " )	(-)	8	"
(38 - 42 " )	(N)	3	"
(43 - 47 " )	(+)	5	"
(48 - 52 " )	(++)	4	"

The/

The haematocrit was highly variable and a sole measurement it did not mean much. Most animals were dehydrated clinically and it is seen that some of the dogs must have been mildly anaemic prior to the onset of illness, or had lost whole blood in the vaginal discharge. It is perhaps better to consider the sum of the total serum protein and the packed cell volume estimation. The normal acceptable range is 88 to 112. The results were,

Table 51.

i)	80 - 90	0	dogs
ii)	91 - 100	1	dog
iii)	101 - 110	9	dogs
iv)	111 - 120	7	"
v)	121 - 130	3	"
vi)	131 - 140	2	"
vii)	141 - 150	1	dog

It is seen that the dogs had levels in excess of 100 and 13 dogs were above 110 which would be indicative of dehydration which was diagnosed clinically.

The haemoglobin level in the blood was assayed and recorded, and it is seen that the results are similar to the haematocrit results.

Table 52/

Table 52.

(8 - 10	g./100ml.)	(-)	2	dogs
(10 - 14	" )	(N)	14	"
(15 - 16	" )	(+)	5	"
(17 - 18	" )	(++)	0	"
(19 - 20	" )	(+++)	1	dog
more than 20	"	(++++)	1	"

This indicates that the dogs were marginally dehydrated in some cases or anaemic in others. The untreated dogs were less affected than the treated dogs.

The white blood cell count was performed and the results were,

Table 53.

(0 - 10 thousand per cmm.)	(N)	0	dogs
(10 - 20	" )	(+)	4 "
(21 - 30	" )	(++)	5 "
(31 - 40	" )	(+++)	5 "
(41 - 50	" )	(++++)	1 dog
more than 50,000 per cmm.	(+++++)	8	dogs

The white blood cell count was elevated in all the dogs as expected. The range was from 14,100 to 110,000 cells per cmm..

The/

The other commonly used ancillary aid was radiography and the results of lateral plain films are given below. Lateral films of the abdomen were taken of all the bitches, and it has been possible to estimate the size and diameter of the uterus on the X-ray films. The results compare favourably with the actual uterine size at laparotomy. A scale of assessment was used to evaluate the uterine size.

Table 54./

Table 54.

Dog	Breed	X-ray diagnosis	Uterine size	Uterine diameter (mm)
1.	Staff. B.T.*	+	++	14
2	Cairn	-		
3	Airedale	+	+++	32
4	Poodle	-		
5	Collie	+	+++	32
6	Westie	+	++	10
7	Labrador	+	++	12
8	Collie	+	+++++	45
9	Great Dane	+	++	30
10	Cairn	+	+	7
11	Alsatian	+	+++++	42
12	Alsatian	+	++++	40
13	Poodle	+	++	12
14	Keeshound	-		
15	C. Spaniel**	-		
16	Sheltie	+	++	11
17	Labrador	+	+++	22
18	Collie	+	+++++	48
19	Labrador	+	++++	32
20	Whippet	+	+++++	31
21	Sheltie	+	+++++	31
22	Collie	+	++	12
23	Alsatian	+	+++++	55

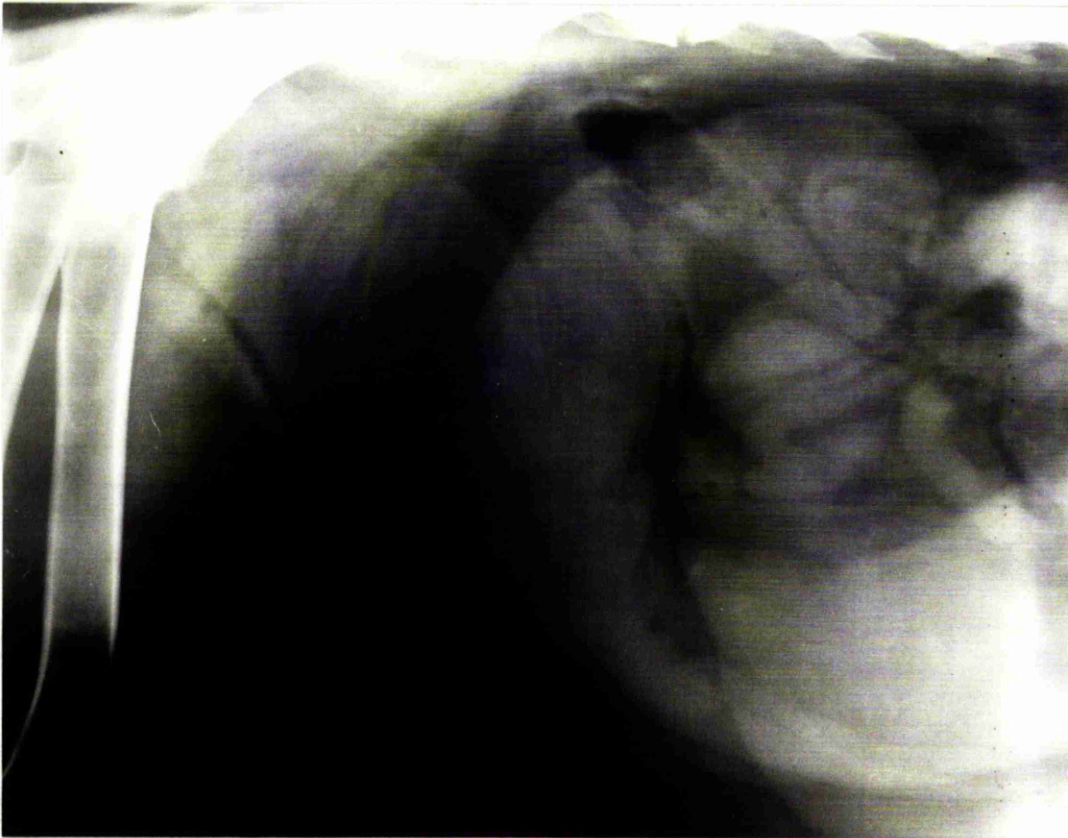
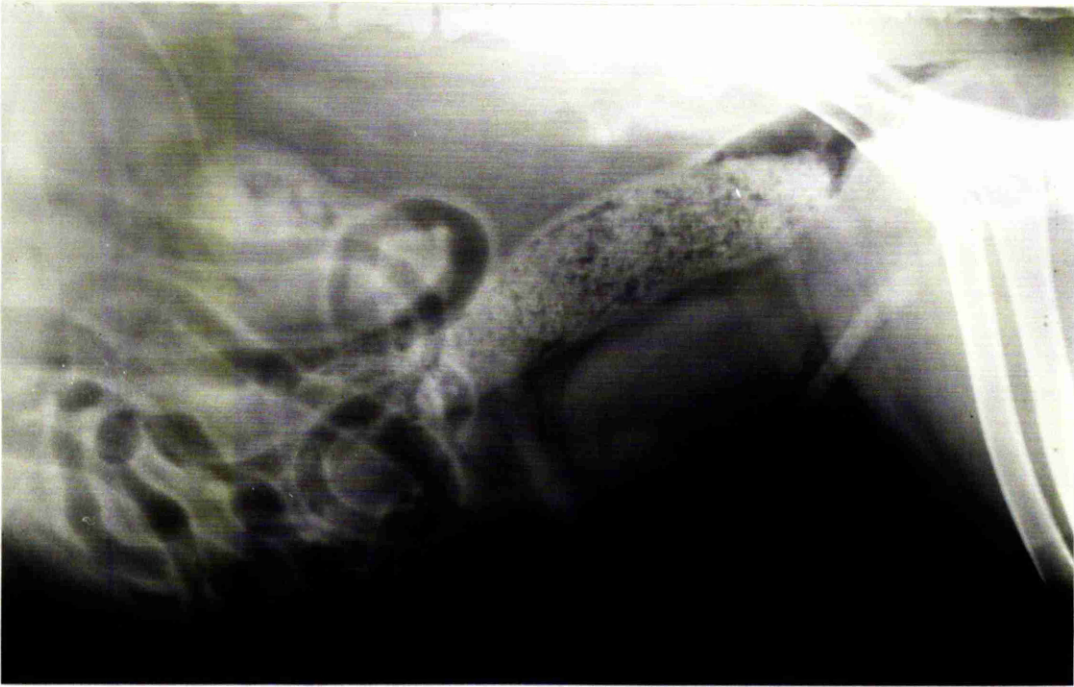
\* Staffordshire Bull Terrier

\*\* Cocker Spaniel

Some examples of radiographs are given, (Figs. 15,16.)

19 enlarged uteri were visible on lateral radiograph of the abdomen. There was some correlation between the size of the uterus and the historical and clinical signs, though some of the very large uteri were discharging freely. Most of the dogs with very large uteri were clinically worse than those with small uteri. Only one large pyometra was found in an untreated dog. The uterus was not visible in 4 dogs, but at surgery, although there was change in these uteri, the diameter of these uteri was relatively normal.





The presenting symptoms of the pyometritis case appear to be polydipsia, anorexia and perhaps vomiting. The dogs had been in oestrus within the last 4 to 8 weeks, though some were outwith this period. They are characteristically dull in appearance and the majority are dehydrated with changes in the cardiovascular system including tachycardia and a decreased volume pulse. The mucous membranes are dry and congested although the capillary refill time is relatively normal. Abdominal pain was evident in only a few cases. Tissue turgor is commonly decreased.

On analysis of the blood, there were some changes in the electrolyte levels and the state of dehydration was confirmed. The white blood cell count was elevated in all cases.

Radiography confirmed 83 % of the cases.

Surgery was arranged within 24 hours of admission, and two cases, (21 and 22) required pre-operative supportive fluid therapy. 4 dogs, (18, 20, 22 and 23) required some form of supportive therapy post-operatively.

The 2 dogs requiring pre-operative fluid therapy received the following.

Dog 21. 450 ml. Ringer lactate

Dog 22. 300 ml. dextran 40, 600 ml. Ringer lactate

The 4 dogs requiring post-operative fluid therapy received,

Dog 18. Day 1 post-op. 450 ml. 0.18%NaCl+4.3%dextrose

Dog 20. Day 1 " 200 ml. "

Dog 22. Day 1 " 500 ml. "

Dog 23. Day 1 " 1000 ml. "

The/

The selection of cases for the two groups, untreated and treated, has already been discussed. The selection is outlined in a table to illustrate the adequacy of the clinical examination in the assessment of these cases. Any dog in which there was any doubt as to the eventual outcome post-operatively was given fluid intra-operatively.

Table 55/

Legend :

U - Untreated group

T - Treated group

PT - Pre-operative fluid and treated group

TP - Post-operative fluid and treated group

Table 55.

Dog	Clinical Selection	Blood analysis selection	Actual Group
1.	U	U	U
2.	U	U	U
3.	U	T	U
4.	U	U	U
5.	U	T	U
6.	U	U	U
7.	U	U	U
8.	U	U	U
9.	U	U	U
10.	U	T	T
11.	U	U	T
12.	U	U	T
13.	U	T	T
14.	U	U	T
15.	T	U	T
16.	T	T	T
17.	T	T	T
18.	T	T	TP
19.	T	T	T
20.	T	T	TP
21.	T	T	PT
22.	T	T	PTP
23.	T	T	TP

Two bitches selected for untreated cases should have been treated according to the blood results. Four of the treated dogs could have been in the untreated group, but it was thought worthwhile to leave them in the treated group to assess their progress. One dog thought clinically to be in need of fluid therapy did not demonstrate this need on blood analysis, but it should be noted that the haematocrit was not a good indicator of the state of hydration in cases of pyometritis since some of the dogs were anaemic. There appears therefore to be much value in the clinical assessment of these cases.

Anaesthesia was routine in all cases and the information regarding the induction dose of sodium thiopentone, the duration of anaesthesia and surgery and the recovery time were recorded. The dose of Ringer lactate was recorded with the infusion rate.

Table 56/

Table 56.

Dog	Thiopentone dose (mg/kg)	Duration (hrs)	Recovery (mins)	Fluid dose (ml)	Infusion rate (ml/kg/hr)
1	7.5	1.25	10		
2	12	0.75	20		
3	7	2	30		
4	8.5	1	10		
5	10	1.5	20		
6	15	2.5	15		
7	12	1.75	15		
8	7	1	15		
9	11	1.5	5	1860	20
10	7	1.5	10	230	20
11	10	1.25	15	500	24
12	6	1	5	990	30
13	5	1	5	350	30
14	9	0.75	5	465	40
15	10	1.5	10	530	20
16	9	1	10	570	30
17	8	0.75	10	495	40
18	10	0.75	5	660	40
19	7.5	2.25	10	300	13
20	12	0.75	5	300	40
21	5	1	15	450	30
22	6	1.5	30	675	20
23	6	1.75	10	1215	17

Some average figures were calculated from these results and a comparison was possible for the two groups of dogs.

Table 57.

<u>Measurement</u>	<u>Overall average</u>	<u>Untreated average</u>	<u>Treated average</u>
Thiopentone dose (mg./kg.)	8.7	9.8	8.1
Duration (hours)	1.3	1.5	1.2
Recovery (minutes)	12.4	16.8	10.1
Fluid inf. rate (ml./kg./hr.)			28

The induction dose of sodium thiopentone was marginally higher in the untreated group, but the difference is not of significance.

The duration of anaesthesia and surgery in all dogs was relatively similar, the average being 1.3 hours. The recovery time for the untreated group was noticeably longer than that of the treated group. The average rate of infusion was related to the duration of surgery and was 28 ml. per kilogram per hour.

The surgery in all dogs was a midline laparotomy and total ovariectomy. The degree of surgical trauma was assessed and recorded in each case. The size of the uterus was noted for comparative studies with the radiographic appearance. The results are recorded.

Table 58/

Table 58.

<u>Dog</u>	<u>Surgical trauma</u>	<u>Uterine size (mm.diameter)</u>
1	3	15
2	3	5
3	4	30
4	3	5
5	4	30
6	3	10
7	3	10
8	4	50
9	3	30
10	3	10
11	3	40
12	4	40
13	3	10
14	3	15
15	4	10
16	3	10
17	3	25
18	4	50
19	4	40
20	3	25
21	4	30
22	4	10
23	4	60

The average trauma factor was 3.4.

There/



There was an obvious correlation between the size of the uterus visualised on the lateral abdomen radiograph and the size of the uterus removed at laparotomy.

The surgery and anaesthesia in all the dogs was satisfactory and the dogs recovered well, the untreated dogs taking slightly longer than the treated group.

HECAscores

The HECAscores for the pyometritis cases are now considered. The original scores on admission are used as the base reference level and the recording of parameters was performed at 12, 24, 48, 72 and 96 hours post-operatively. The scores for each group of parameters is discussed as follows,

A.	Input/output	Maximum score	60
B.	Clinical signs	" "	110
C.	Ancillary aids	" "	80
D.	HECAscore	" "	250

The untreated group is always outlined in red. For the purposes of comparison the scores for the two groups, untreated and treated, have been averaged and graphed.

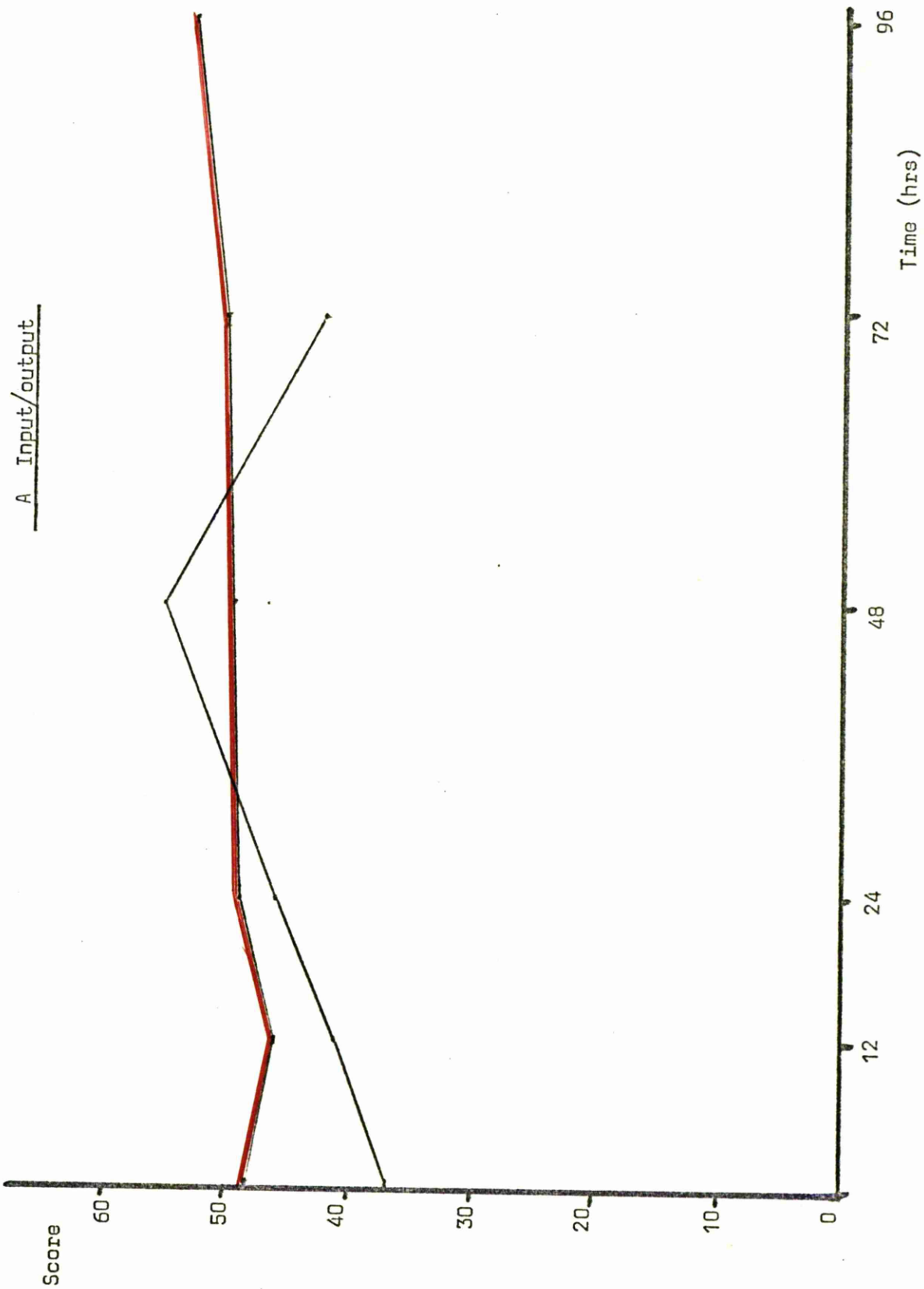
The scores for the above three groups and the HECAscore are tabulated and described below.

Table 59.

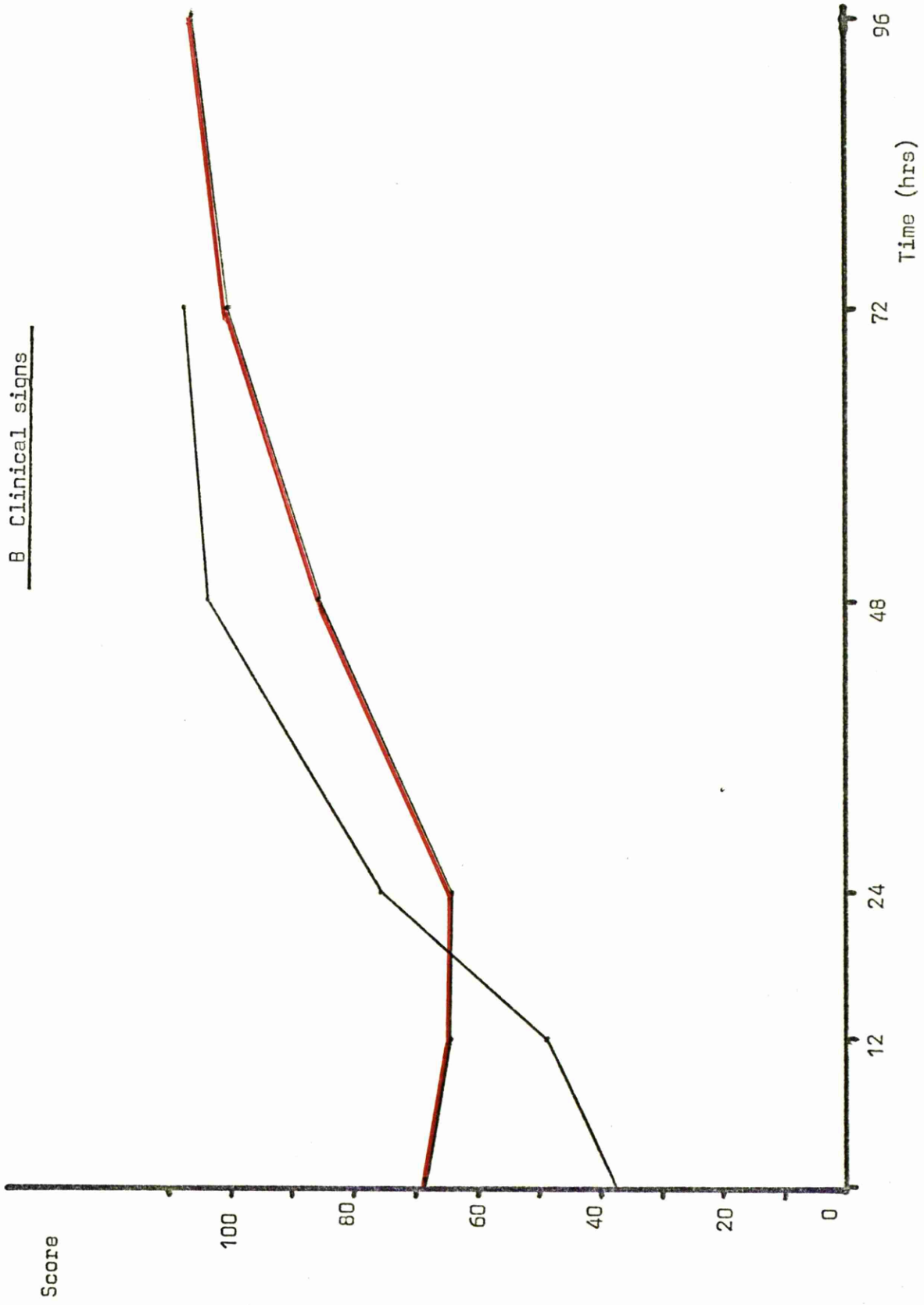
Graphs 5 - 7. /

Table 59.		Scores and												Hecascors												48												72												96												Hours																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																	
		0				12				24				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours				0				12				24				48				72				96				Hours	

Graph 5.

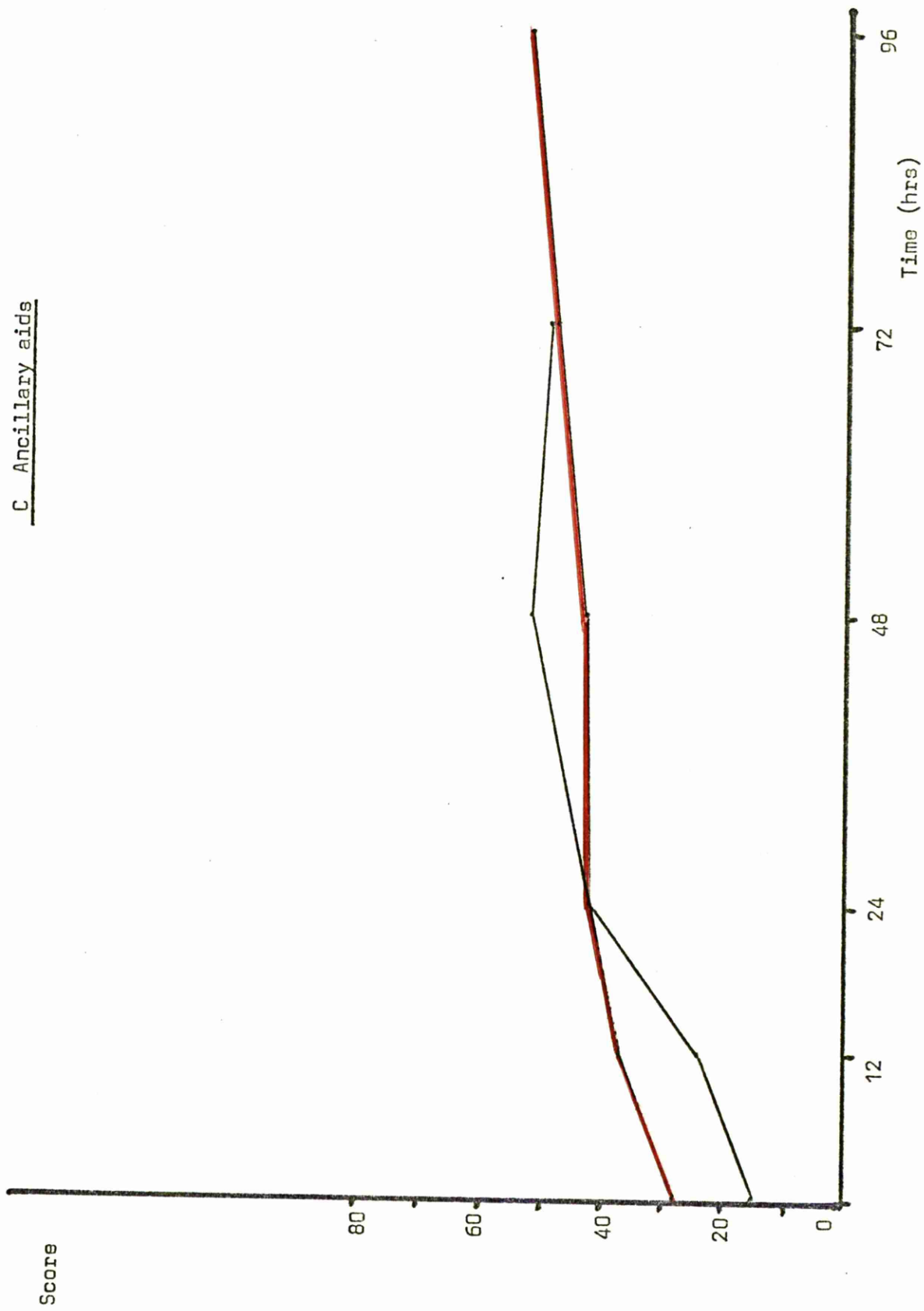


Graph 6.



Graph 7.

C Ancillary aids



It is now possible to analyse the effects of administering fluids intravenously during the period of surgery to cases with a pyometra. The graphs for the treated group show a decline in graphs 5 to 7 due to dogs improving and being discharged, leaving less improved dogs with lower scores. The normal score should perhaps have been added to the remaining dog's scores to show the full extent of recovery. The HECA score system appears quite adequate in the comparison of the two groups and of the progress of cases. The scores are considered separately and then overall.

A. Input/output

The initial scores for the two groups were relatively similar, the untreated group score being higher than the treated group. Most of the dogs were anorexic and polydipsic and had increased urine outputs.

Within 24 hours of surgery both groups had improved, the treated group making the most marked recovery. The untreated group did improve but not over the first 12 hour period.

The treated group approached normality within 48 to 72 hours, and by 96 hours only one dog had not been discharged, compared with 5 untreated dogs.

B. Clinical signs

The clinical appearance of the untreated group was markedly better/

better than the treated group as expected since this was one of the methods of selection.

12 hours post-operatively the untreated group had deteriorated slightly and did not improve till 48 hours after surgery. The treated group showed a marked improvement within 48 hours of surgery and were normal within 72 hours. It appears that the untreated dogs took approximately 24 hours longer to improve than the treated dogs post-operatively.

#### C. Ancillary aids

The aids considered were the blood analyses.

Initially the untreated group were 12 points better than the treated group, but these former dogs deteriorated during the first 12 hours post-operatively. The treated group made a marked recovery within 48 hours and were relatively normal by 72 hours. The untreated group, meanwhile, did not improve until after 72 hours. The parameters are considered separately, and the histograms are to be found in Appendix 5.

##### a) Blood urea

Histogram 8.

The blood urea levels took 24 to 48 hours to return to normal in these dogs, the treated group showing a marginally better improvement than the untreated dogs. By 96 hours all but one dog had a normal blood urea, and its level had decreased from 51.4 mmol./l. to 7.4 mmol./l..

b/



b) Plasma sodium

The sodium levels were relatively normal initially, and by 24 hours post-operatively, all the dogs had an acceptable plasma sodium level.

c) Plasma potassium

Histogram 9.

The plasma potassium levels stayed around the initial level for at least 48 hours before showing any sign of a return to normal. At the time of discharge some of the dogs still had a subnormal potassium level in the plasma. The untreated group appeared to take longer to improve than the treated group.

d) Plasma chloride

Histogram 10.

The chloride levels improved within 24 hours and by 72 hours all but two dogs had acceptable plasma chloride levels. The untreated group took longer than the treated group to improve.

e) Total serum protein/haematocrit

Histogram 11.

These two assays were considered together to assess the rehydration of the dogs. It is seen that the treated group improved more rapidly than the untreated group and that by 96 hours all the dogs were relatively normovolaemic.

f) Haemoglobin

Histogram 12.

The haemoglobin levels which were elevated prior to surgery returned to normal indicating rehydration of the dogs. Some dogs were anaemic/

anaemic and remained so post-operatively. The treated group responded well to fluids and the state of dehydration was resolved quicker than in the untreated dogs.

g) White blood cell count

Histogram 13.

The counts were elevated throughout the study and most began to resolve by 48 hours post-operatively. By 96 hours after surgery, most of the dogs had a W.B.C. count below 40,000 per cmm..

HECAscores.

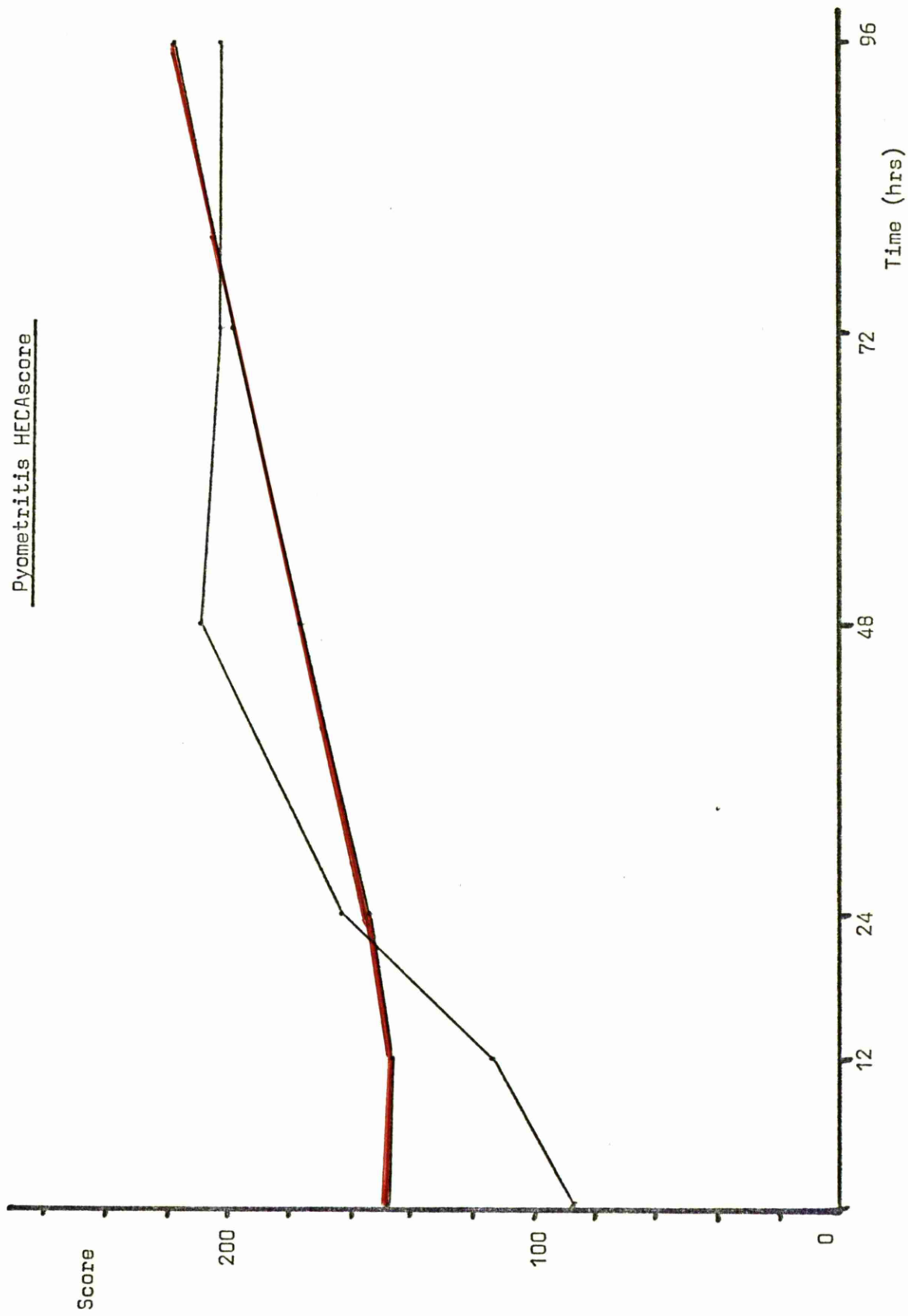
Graph 8.

The three scores, A - input/output, B - clinical signs and C - ancillary aids were added together to give the HECAscore.

Initially the HECAscore in the untreated group was higher than the treated group, but within 12 hours of surgery the difference between them was reduced due to an improvement in the treated group and a slight deterioration in the untreated dogs. The untreated group took a further 48 hours to reach the same degree of improvement. It must be noted that initially the untreated group were in better physical and biochemical condition, and it would suggest from the results that the administration of fluid intravenously during the period of surgery is advantageous.

Therefore, the use of Ringer lactate administered during the period of surgery is to be recommended to improve the recovery and to ensure a more rapid return to normal. The oral consumption of food and water was more readily acceptable to the treated group of dogs post-operatively.

Graph 8.



## DISCUSSION

The untreated group of dogs in both studies took 48 hours longer to reach a relative state of normality, both clinically and biochemically. The treated groups made dramatic improvements within the first 24 hours post-operatively and steadily responded to treatment over the next 48 hours. Most of the treated dogs were outwardly normal by 48 to 72 hours.

The recovery time post-operatively and post-anaesthesia was improved by the administration of parenteral fluids.

The animals given Ringer lactate were more willing to eat and drink post-operatively than the untreated dogs.

The amount of fluid infused did not correct the total body fluid imbalance, but it did seem to promote recovery. The use of intravenous fluids intra-operatively in the fluid deficient dog would seem to be of benefit both to the dog, and to the owner and the veterinary surgeon, since it is always advisable and desirable to present the owner with a dog which can stand, eat and drink after any form of surgery. Any procedure which reduces the period of hospitalisation is desirable from the economic and social viewpoint, the owner paying less and having the dog home sooner.

It may be recommended, therefore, that Ringer lactate be infused to dogs undergoing surgery for the removal of either an intestinal foreign body or a pyometra.

PART 3 - GENERAL DISCUSSION

## GENERAL DISCUSSION

Many aspects of parenteral fluid therapy in the dog have been reviewed, investigated, analysed and discussed. This study has dealt in depth with the problems of detecting and preventing overinfusion, the physiology and pathophysiology of fluid therapy and acute illness due to various causes and the methods of case analysis and treatment. A section of the appendices researches in detail problems associated with the parenteral administration of fluids to dogs and the recording and monitoring of such treatment. It was hoped that a complete study of parenteral fluid therapy could be made, but this was found impossible and many areas of pathophysiology, treatment and monitoring still require fuller investigation.

Overinfusion is one of the principal hazards of parenteral fluid therapy and central venous pressure has been advocated as one of the best methods of monitoring infusions since it was reputed to increase with excessive fluid input. It has been stated that should the central venous pressure exceed a certain level, overinfusion might be diagnosed. There are many difficulties associated with the measurement of central venous pressure in dogs and the experimental work comparing the peripheral venous pressure and the central venous pressure showed that changes in both were parallel. Peripheral venous pressure was monitored from a cephalic vein being greater than central venous pressure but by a variable amount. The readings obtained were only of value in the dogs if trends were observed since single measurements were relatively meaningless. The position of the dog and the location of the intravenous cannula/

cannula were important factors in deciding the initial peripheral venous pressure reading, and it was possible to establish that ranges of pressure existed for different postural positions relative to the zero point.

It was concluded from this experimental study that peripheral venous pressure was perhaps more beneficial to the veterinary profession than central venous pressure since it required little expenditure on equipment and avoided some of the problems associated with central venous pressure monitoring. These problems included location of the jugular vein, sequelae to venipuncture such as air embolism and haematoma formation, and positioning of the cannula in the vena cava. The long cannula inserted into the vena cava often kinked, occluding the lumen and thus preventing the monitoring of venous pressure. It is usually advisable to check the position of the central venous line by contrast radiography since it is not uncommon for the cannula to enter a peripheral vein instead of the vena cava.

Since doubt existed regarding the effectiveness of central venous pressure monitoring for the prevention of overinfusion, some other parameters which are commonly used in human hospitals to monitor acutely ill cases were compared with venous pressure in an experimental study. The other parameters were the output of urine, the partial pressure of oxygen in the plasma and the appearance of the lungs on radiographic examination of the chest. A group of dogs were infused at increasing rates of administration with four different crystalloid solutions in an attempt to produce overinfusion and pulmonary oedema. The colloid dextran was infused to determine the changes caused by increasing the blood volume. Following the administration of the colloid, Ringer lactate was infused. The/



The results of this experiment showed that some parameters were useful, whilst other monitoring aids were misleading.

The output of urine was perhaps the best indication of parenteral fluid therapy success or failure and it would increase above the normal one millilitre per kilogram per hour if the patient was overinfused. The body will not produce a constant flow of urine at or above this rate until some major degree of rehydration has taken place and there is surplus fluid in the tissues and circulatory system. Rates of 2 to 5 millilitres per kilogram per hour are acceptable and although indicative of overinfusion, did not appear to cause pulmonary oedema or embarrassment to the animal in any way. Outputs exceeding five millilitres per kilogram per hour could result in symptoms of overinfusion such as pulmonary oedema, wet oedematous mucous membranes, oedema of the feet and flanks and occasionally watery discharges from the nose and eyes. The clinical signs described were most useful in animals suffering from a nephropathy which caused oliguria or anuria, and which were undergoing parenteral fluid infusion.

The other parameters were considered but all had disadvantages associated either with equipment availability and cost or reliability. The central venous pressure was found to be of little use in the assessment of fluid infusion since it did not change consistently when overinfusion was present or imminent. The measurements of central venous pressure in all the dogs were of variable level initially and the changes which took place with the intravenous infusion of fluid did not relate to the input. It was decided that central and peripheral venous pressure were misleading haemodynamic values and should not be recommended for routine fluid/

fluid infusion monitoring in the dog.

The presence or absence of pulmonary oedema can be demonstrated radiographically, but the use of X - rays to monitor fluid infusion is both uneconomical and undesirable due to the excessive exposure to radiation. It was useful in this study to prove that overinfusion pulmonary oedema was produced by excessive fluid infusion and that it could be detected radiographically. It was also of interest to observe how rapidly the pulmonary oedema cleared once the infusion of fluid ceased, this being adequately demonstrated on the X - ray plates.

The arterial blood pressure was shown to remain relatively stable during the infusion of crystalloids, but did as expected increase with the infusion of colloid. This increased level of pressure was maintained during the period of the experiment proving that dextran was indeed capable of replacing some blood or plasma loss for a short time provided some form of crystalloid was infused simultaneously to avoid tissue dehydration and permit an output of urine.

The arterial partial pressure of oxygen altered when overinfusion pulmonary oedema was present, but because of the expense of the equipment necessary for such an assay, it is not recommended for routine use in the prevention of overinfusion. It is a parameter which unfortunately changes once overinfusion is present and present to the degree of causing interference with pulmonary function. Normally this stage of overinfusion is to be avoided.

The conclusion from the experimental study was that fluid could be infused to dogs if set levels of infusion were not surpassed. The recommended rates of infusion were 30 millilitres per kilogram per hour for/

for a maximum of four hours for crystalloids and 20 millilitres per kilogram per 24 hours for colloid. When a colloid and crystalloid are to be administered, they should be infused simultaneously at a maximum rate of 20 millilitres per kilogram of colloid and 20 millilitres per kilogram of crystalloid. Further crystalloid may be infused until a satisfactory renal output is established.

The infusion of fluid may be monitored successfully by the regular recording of the output of urine by means of a bladder catheter connected to a collecting unit, and observation of the clinical signs. The one apparent indication for the use of venous pressure monitoring is in the patient with a confirmed or suspected cardiac abnormality where the cardiovascular system may be unable to accommodate large quantities of intravenously infused fluid. The venous pressure in these patients will rise with the infusion of fluid by the intravenous route. It may be useful in cases of renal failure to monitor venous pressure to prevent overloading of the circulatory system. The treatment and management of these cases was described in the text of the clinical study and patients suffering from renal and cardiovascular disease are perhaps the only major areas of concern for the practitioner who undertakes parenteral fluid therapy.

It is apparent from the historical background of parenteral fluid therapy that the veterinary profession has been somewhat reluctant to implement some of the knowledge discovered regarding this subject, both from practical experience and from experimental studies performed in animals. The correct use of supportive fluid therapy has been recognised as of immense benefit to human patients with body fluid imbalances/

imbalances. Animals suffer from a variety of similar body fluid disturbances and should receive therapy of a suitable nature to correct both the imbalance and possibly the underlying cause. A survey of general practitioners regarding the use made of fluid therapy in small animal practice indicated that although most veterinary surgeons claimed to use fluids parenterally in animals, few cases were seen which were considered in need of such therapy and fewer still actually received treatment. It is hoped that publication of some of the information contained in this study will encourage much more willing use of such treatment.

The physiology of body fluids was outlined briefly to permit discussion of the various forms of fluid imbalance which can occur, and what treatment was necessary to correct these disturbances. The examination and assessment of body fluid imbalance cases has been described and with a little experience it is relatively straightforward to identify and select cases in need of fluid replacement therapy. After the selection of cases, the correct treatment must be sought and although many different parenteral fluids exist, only a few are genuinely required in veterinary practice. Most cases of blood or plasma loss can be treated with a colloid such as dextran and the simultaneous infusion of a crystalloid such as Ringer lactate. Animals suffering body fluid loss from the circulation and tissues can be treated successfully with Ringer lactate or normal saline solution.

From the analysis of the survey it appeared that a percentage of practitioners used one specific fluid, - " Duphalyte ". This fluid is not totally satisfactory for body fluid replacement or maintenance and its widespread use is not the fault of the practitioners, but is due to the/  
the/

the lack of information in veterinary publications and misleading advertising by pharmaceutical companies. Many composite fluids have been produced and retailed to practitioners without much consideration of the correct methods of use. The infusion of " Duphalyte " to provide the nutritional and fluid needs of any animal is of doubtful value and parenteral nutrition is a complex subject which is ill-understood by the veterinary profession. Although it may be a necessary form of therapy in human patients, similar treatment may not be applicable to animals, and parenteral nutrition in animals is a part of fluid therapy which requires much fuller investigation.

It was demonstrated that the intravenous administration of fluid during the period of surgery improves the recovery period and the rate of recovery and discharge from the veterinary clinic. These improvements are of benefit not only to the patients, but also to the owners and the veterinary surgeons since a patient which appears bright and capable of self support is desirable. This may decrease the period of hospitalisation and diminish owner concern. This was shown in cases of both intestinal foreign body and pyometritis which were presented to the University of Glasgow Veterinary School hospital. These dogs were admitted for surgery to remove the foreign body from the intestine or to perform a total ovarohysterectomy, and certain cases required pre-operative fluid therapy to restore the circulating blood volume as well as commencing rehydration. Most patients underwent surgery within 24 hours of admission and the majority of cases were discharged by 72 hours post-operatively. It was shown that in two groups of dogs, one treated with parenteral fluids at the time of surgery and the other left untreated, that/

that the former group recovered some 48 hours sooner than the latter group of dogs. Recovery was assessed by the degree of normal function demonstrated by each dog and it was seen that the treated dogs drank and ate much sooner than the untreated dogs. The dogs for this study were carefully selected, and the untreated animals were those which were least ill. The use of parenteral fluids intra-operatively is therefore more impressive since the treated group consisted of dogs which were more seriously affected by dehydration and might have been expected to have taken longer to recover.

It was of interest to note that in the cases of intestinal foreign body treated with fluid pre-operatively to prevent shock that sequestration of fluid occurred at the site of obstruction as described in the literature. This accumulation of fluid around the foreign body made surgery difficult and it may be one instance in which fluids should be administered during and after surgery rather than pre-operatively.

The information derived from the study of these clinical cases applies not only to dogs with intestinal foreign bodies or pyometritis, but equally well to dogs which have any form of body fluid imbalance whether present pre-operatively or unfortunately attained during the period of surgery. Any single factor which can improve the overall performance of animals undergoing surgery must be beneficial.

Many ancillary aids to diagnosis were used to confirm and assess the condition of the dogs presented to the University of Glasgow Veterinary School hospital with body fluid imbalance. Radiography and haematological and biochemical analysis of the blood and urine were performed in most cases to aid diagnosis. Blood analysis was used to assess/

assess the degree of dehydration or shock present and the deficiency of electrolyte or the change in plasma protein content. It was noted during analysis of the haematocrit and total serum protein results that if the two were added together, the result was invariably around 100 in normal dogs. This figure rose as expected with increasing dehydration and although not initially considered as a method of assessing cases, it was later shown in some dogs, which were presumably anaemic prior to the onset of dehydration, where the haematocrit was low the total serum protein was high, hence the figure of 100 had some relevance. This method was used to assess the state of dehydration in the clinical cases described in this thesis suffering with an intestinal foreign body or pyometritis.

In general practice there is an increasing need for ancillary aids to diagnosis and since much of the necessary equipment is relatively inexpensive, there is no longer reason for abstension. The results of the survey of general practitioners indicated that a small number of veterinary surgeons used ancillary aids such as the haematocrit and the blood urea estimation. There were no other aids in use at the time of the survey in the general practices considered and it is hoped that the need for some minor degree of sophistication will be progressively apparent. Small blood analysis units are available which can be of direct benefit in assessing patients prior to treatment as well as determining the course of therapy.

The route of administration of fluids in cases of body fluid deficiency is important. In this study the intravenous route is recommended/

recommended since it provides access to the body fluid compartments and permits accurate control of the infusion. Supportive therapy is only administered until the patient is capable of self support by the oral intake of food and water to satisfy the daily requirement. Neither the subcutaneous nor the intraperitoneal routes are as effective as the intravenous route in the restoration of the blood volume and the extracellular and intracellular fluid volumes. Certain fluids must be administered intravenously and it would appear sensible to infuse fluids by this route in most cases since it causes less irritation and inconvenience to the recipient. It is recommended that the intraperitoneal route should not be used at all and the subcutaneous route only used to maintain an animal which has been previously rehydrated intravenously. The subcutaneous route may be used where intravenous administration is not possible.

Many different forms of intravenous cannulae and needles are available which permit the infusion of fluids and are readily inserted into all sizes of animal. Most common designs were used and evaluated in a study which is illustrated and located in the appendices. It was obvious when undertaking this study that some designs were more useful than others and although cost was important, it was necessary to spend some money to save time and damage to body tissues when inserting cannulae or connecting and controlling the infusion of fluid. Plastic cannulae cause little damage to veins and are better maintained in situ than metal units which often irritate the walls of the vein and occasionally repenetrate the vein allowing the leakage of fluid perivenously. The type of administration unit can determine the likelihood of overinfusion and the/



the correct design of set should be chosen for each respective patient. A graduated burette chamber may be used attached to the administration set, and will aid in the prevention of overinfusion by not permitting more than the desired volume of fluid to be infused. Some forms of flow regulator are more effective than others and the best type should always be chosen since this will avoid accidents involving increased or decreased infusion rate.

Monitoring equipment is described and discussed at length in this study although central venous pressure monitoring is not recommended, it has been considered. Most manometer units are easily attached to the administration sets and operate using the infusion fluid. Other means of monitoring often require recording and this has been illustrated and discussed in the appendices. Recording of every fact and measurement which are found at the clinical examination, the results of the ancillary tests and the day by day, minute by minute account of the progress or otherwise of the patient is essential. It is important to have these records available to other members of staff for consideration at anytime, and the information regarding each case should be stored for future reference.

In conclusion therefore, parenteral fluid therapy is a necessary and rewarding means of supporting animals prior to, during and after surgery. It reduces the period of recovery and ensures more rapid self support. The monitoring of patients undergoing supportive fluid therapy may be adequately performed using the rate of output of urine and the clinical signs without the measurement of venous pressure. The monitoring of central venous pressure and peripheral venous pressure is of doubtful use in the care of dogs with body fluid deficiencies.

The/

The time for fluid therapy is on the presentation of an animal which is showing signs of circulatory collapse or body fluid deficit and which would benefit directly from some form of parenteral support. Sophistication is not required, but care and attention to detail will ensure a satisfactory conclusion.

## BIBLIOGRAPHY

BIBLIOGRAPHY

1. ABERG, M., BERGENTZ, S.E., HEDNER, U., (1975), " The effect of Dextran on the lysability of ex-vivo thrombi ", Annals of Surgery, 181, 342.
2. ADACHI, H., STRAUSS, H.W., OCHI, H., WAGNER, H.N., (1976), " The effect of hypoxia on the regional distribution of cardiac output in the dog ", Circulation Research, 39, 314.
3. ADAR, R., MOZES, M., (1971), " Fatal complications of central venous catheters ", British Medical Journal, 3, 746.
4. ADAR, R., (1976), " Potentially fatal complications of central venous catheters ", Surgery, 79, 726.
5. ADOLPH, E.F., (1949), " Quantitative studies in the physiological constitution of mammals ", Science, 109, 579.
6. ALEXANDER, R.S., (1954), " The participation of the venomotor system in pressor reflexes ", Circulation Research, 2, 405.
7. ALTSCHULE, M.D., GILLIGAN, D.R., (1938), " The effect on the cardiovascular system of fluids administered intravenously in man ", Journal of Clinical Investigation, 17, 401.
8. AMES, A.C., (1977), " Lactic acidosis ", Health Care World Wide, Abbot, 31.
9. ANDERSON, R.W., SIMMONS, R.L., COLLINS, J.A., BREDENBERG, C.E., JAMES, P.M., LEVITSKY, S., (1969), " Plasma volume and sulphate spaces in acute combat casualties ", Surgery, Gynaecology and Obstetrics, 128, 719.
10. ANNIS, J.R., (1961), " Metabolic response of the surgical patient to trauma ", Veterinary Medicine, 56, 467.

11. ARCHIBALD, J., (1976), " Abdominal trauma ", Symposium, Edinburgh.
12. ARCHIBALD, J., (1976), " Thoracic trauma ", Symposium, Edinburgh.
13. ARCHIBALD, J., (1974), Canine Surgery, California, American  
Veterinary Publications Incorporated, 74 - 106.
14. ARMISTEAD, W., (1954), " Some surgical considerations in pyometra ",  
Veterinary Medicine, 49, 533.
15. ARTZ, C.P., (1966), " Volume replacement in shock ", Surgery,  
Gynaecology and Obstetrics, 122, 112.
16. ASHEIM, A., (1965), " Pathogenesis of renal damage and polydipsia  
in dogs with pyometra ", Journal of the American Veterinary  
Medical Association, 147, 736.
17. ASHWORTH, C.T., TIGERTT, W.D., (1940), " A single rapid method of  
determining relative blood volume changes by specific  
gravity studies ", Journal of Laboratory and Clinical  
Medicine, 26, 1545.
18. ASHWORTH, C.T., HUTHESON, Z.W., PAYNE, W.T., JESTER, A.W., (1944),  
" The effect of crystalloid and protein containing solutions  
on the body fluids and circulating plasma proteins ",  
American Journal of Physiology, 140, 589.
19. ASTRUP, P., (1963), " Acid, Base disorders ", New England Journal of  
Medicine, 269, 817.
20. AUSTAD, R., BJERKAS, E., (1976), " Eclampsia in the bitch ", Journal  
of Small Animal Practice, 17, 793.
21. BARRAS, J., (1969), " Apparent viscosity of red blood cell suspensions  
in different plasma substitutes ", Bibliotheca Haematologica  
33, 298.

22. BARTLETT, R.M., BINGHAM, D.D.L., PEDERSEN, S., (1938), " Salt balance in surgical patients ", Surgery, 4, 414.
23. BARTLETT, R.M., BINGHAM, D.D.L., PEDERSEN, S., (1938), " Salt balance in surgical patients ", Surgery, 4, 441.
24. BAUE, A.E., TRAGUS, E.T., WOLFSON, S.K., CARY, A.C., PARKINS, W.M., (1967), " Haemodynamic and metabolic effects of ringer lactate solution in haemorrhagic shock ", Annals of Surgery, 166, 29.
25. BAUE, A.E., TRAGUS, E.T., PARKINS, W.M., (1967), " A comparison of isotonic and hypertonic solutions and blood on blood flow and oxygen consumption in the initial treatment of haemorrhagic shock ", Journal of Trauma, 7, 743.
26. BEDFORD, D.E., WRIGHT, T.S., (1924), " The venous pressure in normal individuals ", Lancet, 2, 106.
27. BELL, G.H., DAVIDSON, J.N., SCARBOROUGH, H., (1961), Textbook of Physiology and Biochemistry, Edinburgh and London, E. & S. Livingstone.
28. BENNET, P.N., DHALL, D.P., MCKENZIE, F.N., MATHESON, N.A., (1966), " The effects of dextran infusion on the adhesiveness of human blood platelets ", Lancet, 2, 1001.
29. BERMAN, I.R., SPENCER, F.C., (1972), " The wet lung - diagnostic considerations ", Annals of Surgery, 175, 458.
30. BERRY, R.E.L., IOB, V., CAMPBELL, K.N., (1948), " Potassium metabolism in the post-operative period ", Archives of Surgery, 57, 470.
31. BEVAN, D.R., DUDLEY, H.A.F., HORSEY, P.J., (1973), " Renal function during and after anaesthesia and surgery - significance for water and electrolyte management ", British Journal of Anaesthesia, 45, 968.

32. BLACK, D.A.K., (1953), " Body fluid depletion ", Lancet, 1 305.
33. BLACK, D.A.K., (1953), " Body fluid depletion ", Lancet, 1 353.
34. BLACK, D.A.K., (1957), Essentials of Fluid Balance, Oxford, Blackwell Scientific Publications.
35. BLALOCK, A., BEARD, J.W., THUSS, C., (1932), " Intravenous injections ; a study of the effect on the composition of blood of the injection of various fluids into dogs of normal and low blood pressures ", Journal of Clinical Investigation, 11, 267.
36. BLALOCK, A., (1934), " Acute circulatory failure as exemplified by shock and haemorrhage ", Surgery, Gynaecology and Obstetrics, 58, 551.
37. BLOCH, J.H., PIERCE, L.H., MANAX, W.G., LYONS, G.W., LILLIHEI, R.C., (1965), " Experimental cardiogenic shock ", Archives of Surgery, 91, 77.
38. BLOCH, J.H., DIETZMAN, H., PIERCE, C.H., LILLIHEI, R.C., (1966), " Theories on the production of shock ", British Journal of Anaesthesia, 38, 234.
39. BLOOD, D.C., HENDERSON, J.A., (1963), Veterinary Medicine, London, Balliere, Tindall and Cox.
40. BLOOM, F., (1960), The Blood Chemistry of the Dog and Cat, New York, Gamma Publications Incorporated.
41. BLOOM, W.C., HARMER, D.S., BRYANT, M.F., BREWER, S.S., (1964), " Coating of vascular surfaces and cells - a new concept in the prevention of intravascular thrombi ", Proceedings of the Society for Experimental Biology and Medicine, 115, 384.
42. BOBA, A., LANDMESSER, C.M., (1961), " Renal complications after anaesthesia and operation ", Anaesthesiology, 22, 781.

43. BOBA, A., GAINOR, J., POWERS, S.R., (1962), " The influence of mannitol on water and electrolyte excretion following trauma ", Surgery, 52, 188.
44. BONNAR, J., WALSH, J., (1972), " Prevention of venous thrombosis after pelvic surgery by British Dextran 70 ", Lancet, 1, 614.
45. BOROW, M., AQUILIZAN, C., KRAUSZ, A., STEFANIDES, A., (1965), " The use of central venous pressure as an accurate guide for body fluid replacement ", Surgery, Gynaecology and Obstetrics, 120, 345.
46. BRIGGS, I.A., ( 1977), " Renal considerations in shock ", Lecture, Royal college of Physicians and Surgeons, Glasgow.
47. BRISCOE, C.E., (1973), " A comparison of jugular and central venous pressure measurements during anaesthesia ", British Journal of Anaesthesia, 45, 173.
48. BROGAN, T.D., (1976), " Antibiotic treatment of chest infections ", Antibiotics in General Practice, Symposium, Manchester, Abbott.
49. BROWN, E.B., (1966), " Acid base debate " , Anaesthesiology, 27, 333.
50. BULL, J.P., RICKETTS, C., SQUIRE, J.R., MAYCOCK, W.dA., SPOONER, S.J.L., MOLLINSON, P.D., PATERSON, J.C.S., (1949), " Dextran as a plasma substitute ", Lancet, 1, 614.
51. BULL, J.P., (1963), " Transfusion fluids ", Annals of the Royal College of Surgeons of England, 33, 175.
52. BURROWS, C., (1976), " Intensive Care ", B.S.A.V.A. Congress, London.
53. BURROWS, C.F., KOLATA, R.J., SOMA, C.R., (1977), " Shock - pathophysiology and management ", in Current Veterinary Therapy (ed. Kirk), VI, 26.



54. BUXTON, D.W., (1920), Anaesthetics, London, H.H.Lewis & Co..
55. CARTER, A.E., EBAN, R., (1973), " The prevention of deep venous thrombosis with dextran 70 ", British Journal of Surgery, 60, 681.
56. CAUGHEY, J.L., (1935), " Effect of rapid infusion on venous pressure", Proceedings of the Society for Experimental Biology, 32, 973.
57. CHAIT, A., (1972), " Interstitial pulmonary oedema ", Circulation, 45, 1323.
58. CLARKE, R., TOPLEY, E., FLEAR, C.T.G., (1955), " Assessment of blood loss in civilian trauma ", Lancet, 1, 629.
59. COHN, J.N., LURIA, M.H., (1964), " Studies in clinical shock and hypotension ", Journal of the American Medical Association, 190, 89.
60. COLLIER, F.A., MADDOCK, W.G., (1932), " Dehydration attendant on surgical operations ", Journal of the American Medical Association, 99, 875.
61. COLLIER, F.A., MADDOCK, W.G., (1933), " The water requirements of surgical patients ", Annals of Surgery, 98, 952.
62. COLLIER, F.A., MADDOCK, W.G., (1935), " A study of dehydration in humans ", Annals of Surgery, 102, 947.
63. COLLIER, F.A., DICK, V.S., MADDOCK, W.G., (1936), " Maintenance of normal water exchange with intravenous fluids ", Journal of the American Medical Association, 107, 1522.
64. COLLIER, F.A., BARTLETT, R.M., BINGHAM, D.L.C., MADDOCK, W.G., PEDERSEN, S., (1938), " The replacement of sodium chloride in surgical patients ", Annals of Surgery, 108, 769.

65. COLLIER, F.A., CAMPBELL, M.D., VAUGHN, H.H., IOB, V., MDYER, C.A.,  
(1944), " Post-operative salt intolerance ", Annals of  
Surgery, 119, 535.
66. COLLIER, F.A., CROOK, M.D., IOB, V., (1944), " Blood loss in surgical  
patients ", Journal of the American Medical Association, 126, 1.
67. COLLIER, F.A., IOB, V., VAUGHN, H.H., KALDER, N.D., MDYER, C.A., (1945)  
" Translocation of fluid produced by the intravenous  
administration of isotonic salt solution in man post-  
operatively ", Annals of Surgery, 122, 663.
68. COOKE, R.E., CROWLEY, L.G., (1952), " Replacement of gastric and  
intestinal fluid losses in surgery ", New England Journal  
of Medicine, 246, 637.
69. COOPER, D.R., IOB, V., COLLIER, F.A., (1949), " Response to parenteral  
glucose of normal kidneys and of kidneys of post-operative  
patients ", Annals of Surgery, 129, 1
70. CULLEN, S.C., (1961), " Estimation of blood loss in the operating  
room ", Anaesthesiology, 22, 318.
71. CUTTER, R.K., (1936), " The use of large volume intravenous injections "  
Journal of the American Medical Association, 106, 1250.
72. DACIE, J.V., Practical Haematology, (1956), 2nd. Ed., Churchill, London.
73. DANIEL, W.J., MOHAMMED, S.D., MATHESON, N.A., (1966), " Treatment of  
mesenteric embolism with dextran 40 ", Lancet, 1, 567.
74. DANOWSKI, T.S., WINKLER, A.W., ELKINTON, J.R., (1946), " The  
treatment of shock due to salt depletion : comparison of the  
haemodynamic effects of isotonic saline, of hypertonic  
saline and of isotonic glucose solutions ", Journal of  
Clinical Investigation, 25, 130.

75. DAVIES, J.W.L., (1966), " Methods of assessment of blood loss in the shocked and injured patient ", British Journal of Anaesthesia, 38, 250.
76. DAVIES, J.W.L., TOPLEY, E., (1959), " A critical evaluation of red cell and plasma volume techniques in patients with civilian injuries ", Journal of Clinical Pathology, 12, 289.
77. DILLON, J., LYNCH, L.J., MYERS, R., BUTCHER, H.R., MOYER, C.A., (1966), " A bioassay of treatment of haemorrhagic shock ", Archives of Surgery, 93, 537.
78. DODMAN, N.H., (1977), " Feline anaesthesia survey ", Journal of Small Animal Practice , 18, 653.
79. DOTY, D.B., HUFNAGEL, H.W., MOSELEY, R.W., (1970), " Distribution of body fluids following haemorrhage and reconstitution in combat casualties ", Surgery, Gynaecology and Obstetrics, 130, 453.
80. DOUGAN, L.R., FINLAY, W.E.I., (1973), " Fluid and electrolyte balance : an assessment of the patient ", British Journal of Anaesthesia, 45, 945.
81. DOUGLAS, T.A., (1977), " Standard international units ", Veterinary Record, 100, 28.
82. DUCOK, deW., C., COENEGRACHT, N.A.J.C., POLL, P.H.A., LINDE, V.D., (1967), " The practical importance of blood groups in dogs ", Journal of Small Animal Practice, 8, 285.
83. DUNCUM, B.M., (1947), The Development of Inhalation Anaesthesia, Oxford University Press.
84. DURNIN, J.V.G.A., RAHAMAN, M.H., (1967), " The assessment of the amount of fat in the human body from measurements of skin fold thickness " British Journal of Nutrition, 21, 681.

85. DYGDAMAN, S., SVENSJO, E., TOLLERZ, G., (1970), " Prevention of venous thrombosis ", Lancet, 2, 419.
86. EDELMAN, I.S., LEIBMAN, J., O'MEARA, M.P., BIRKENFIELD, L.W., (1958), " Interrelations between serum sodium concentrations serum osmolarity and total exchangeable sodium, total exchangeable potassium and total body water ", Journal of Clinical Investigation, 37, 1236.
87. EDITORIAL, (1875), " New and Recent Introductions and Improvements ", Medical Times and Gazette , 2, 508.
88. EDITORIAL, (1883), " Reports and analysis and descriptions of new inventions ", British Medical Journal, 2, 722.
89. EDITORIAL, (1941), " Fatality in blood donor ", British Medical Journal, 2, 311.
90. EDITORIAL, (1963), " New classification of physical status ", Anaesthesiology, 24, 111.
91. EDITORIAL, (1969), " Administration of crystalloids in shock and surgical trauma ", Lancet, 1, 1298.
92. EHRLY, A.M., (1969), " The effect of plasma substitutes on erythrocyte agglutination and blood viscosity ", Bibliotheca Haematologica, 33, 302.
93. EICHLER, J., STEPHAN, G., (1969), " The effect of plasma substitute on coagulation ", Bibliotheca Haematologica, 33, 452.
94. ELKINTON, J.R., GILMOUR, M.T., WOLFF, W.A., (1939), " The control of water and electrolyte balance in surgical patients ", Annals of Surgery, 110, 1050.
95. ELKINTON, J.R., TAFFEL, M., (1942), " Prolonged water deprivation in the dog ", Journal of Clinical Investigation, 21, 787.

96. ELKINTON, J.R., DANOWSKI, T.S., WINKLER, A.W., (1946), " Haemodynamic changes in salt depletion and in dehydration ", Journal of Clinical Investigation, 25, 120.
97. ELMAN, R.S.G., WEINEL, D.O., (1939), " Intravenous alimentation -- protein metabolism ", Journal of the American Medical Association, 112, 796.
98. ELMAN, R.S.G., LEMMER, R.A., WEICHSELBAUM, T.E., OWENS, J.G., YORE, R.W., (1949), "Minimum maintenance requirements for parenteral water, sodium, potassium, chloride and glucose ", Annals of Surgery, 130, 703.
99. FALCONER, M.A., LYALL, A., (1937), " The requirements of sodium chloride ", British Medical Journal, 2, 1116.
100. FANTUS, B., (1936), " Fluid post-operatively ", Journal of the American Medical Association, 107, 14.
101. FEEST, T.G., (1976), " Low molecular weight dextran - a continuing cause of acute renal failure ", British Medical Journal, 2, 1300.
102. FIEBER, W.W., JONES, J.R., (1966), " Intra-operative fluid therapy with 5% dextrose in lactated ringer's solution ", Anaesthesia and Analgesia Current Researches, 45, 366.
103. FINCO, D.R., (1974), " Fluid therapy ", in Current Veterinary Therapy, (ed. Kirk), Philadelphia, W.B.Saunders Company, V, 3.
104. FINCO, D.R., (1975), " Newer developments in fluid therapy ", Scientific Presentations of the American Animal Hospitals Association, 42, 233.
105. FINCO, D.R., (1977), " Fluid Therapy ", in Current Veterinary Therapy, (ed. Kirk) VI, 3.

106. FISCHER, M., (1969), " The effect of different plasma expanders, (Rheomacrodex, Haemaccel), on the fibrinolytic system and some platelet functions ", *Bibliotheca Haematologica*, 33, 457.
107. FISHER, E.W., (1969), " Hydrogen ion concentration ", in *Textbook of Clinical Pathology*, (ed. Medway), Baltimore, Prior & Wilkinson, 152.
108. FISHER, E.W., (1978), " Regulation of volume and tonicity of body fluids ", *Personal Communication*.
109. FISHER, W.D., HEIMBACH, D.M., McARDLE, C.S., MADDERN, M. HUTCHISON, M.M., LEDINGHAM, I. McA., (1973), " A circulating depressant effect following canine haemorrhagic shock ", *British Journal of Surgery*, 60, 392.
110. FOSTER, S.J., (1967), " Blood transfusion techniques in practice ", *Journal of Small Animal Practice*, 8, 587.
111. FOSTER, S.J., (1970), " Some aspects of fluid therapy in practice ", *Journal of Small Animal Practice*, 11, 337.
112. FOSTER, S.J., (1972), " Fluid therapy in the dog ", in the *Veterinary Annual*, Bristol, John Wright, 215.
113. FOX, C.L., WINFIELD, J.M., SLOBODY, L.B., SWINDLER, C.M., LATTIMER, J.K., (1952), " Electrolyte solutions approximating plasma concentrations ", *Journal of the American Medical Association*, 148, 827.
114. FRANK, E.D., (1969), " The hazard of interstitial pulmonary oedema ", *Annals of Surgery*, 169, 641.

115. FREEMAN, N.E., MORISON, R.S., MCKAY-SAWYER, M.E., (1933), " The effect of dehydration on adrenal secretion and its relation to shock ", American Journal of Physiology, 104, 628.
116. FREEMAN, N.E., (1935), " Haemorrhage in relation to shock ", Annals of Surgery, 101, 484.
117. FREEMAN, N.E., WALLACE, W.M., (1938), " The effect of concentrated serum on plasma volume and serum protein concentration ", American Journal of Physiology, 124, 791.
118. FRIEDMAN, B.A., JURGELEIT, H.C., (1968), " Perforations of the atrium by poly-ethylene central venous catheters ", Journal of the American Medical Association, 203, 1141.
119. GARDINER, A.J.S., DUDLEY, H.A.F., (1962), " The measurement of blood loss at operation ", British Journal of Anaesthesia, 34, 653.
120. GARRET, T.A., (1967), The fundamentals of body water and electrolytes, Travenol .
121. GARRET, T.A., (1970), Travenol guide to fluid therapy, Travenol.
122. GATCH, W.D., LITTLE, W.D., (1924), " The amount of blood lost during some common operations ", Journal of the American Medical Association, 83, 1077.
123. GAUER, O.H., SIEKER, H.O., (1956), " The continuous recording of central venous pressure changes from an arm vein ", Circulation Research, 4, 74.
124. GAUER, O.H., HENRY, J.P., SIEKER, H.O., (1956), " Changes in central venous pressure after moderate haemorrhage and transfusion in man ", Circulation Research, 4, 79.
125. GAUER, O.H., HENRY, J.P., (1963), " Circulatory basis of fluid volume control ", Physiological Reviews, 43, 424.

126. GEISTLICH NUTRITION, (1977), Geistlich Sons Ltd. Chester.
127. GILBERTSON, A.A., Personal Communication (1978).
128. GOLDENBERG, M., CRANE, M.D., POPPER, H., (1947), " The effects of intravenous administration of dextran on macromolecular carbohydrates in animals ", American Journal of Clinical Pathology, 17, 939.
129. GRAY, T.C., NUNN, J.F., (1971), General Anaesthesia, 3rd. Ed., Vol. 1, Basic Sciences, London, Butterworth.
130. GRONWALL, A., INGLEMAN, B., (1944), " Dextran as a plasma substitute ", Acta Physiologica Scandinavica, 7, 97.
131. GUTHRIE, O., (1945), A History of Medicine, London, Nelson.
132. GUYTON, A.C., (1961), Textbook of medical physiology, Philadelphia and London, W.B.Saunders Company.
133. GUYTON, A.C., (1971), Textbook of medical physiology, 4th. Ed., Philadelphia, London, Toronto, W.B.Saunders Company.
134. HADEN, R.L., ORR, T.G., (1923), " The effetc of sodium chloride on the chemical changes in the blood of the dog after pyloric and intestinal obstruction ", Journal of Experimental Medicine, 38, 55.
135. HADEN, R.L., ORR, T.G., (1924), " Use of sodium chloride in the treatment of intestinal obstruction ", Journal of the American Medical Association, 82, 1515.
136. HALL, L.W., (1967), Fluid balance in canine surgery, London, Balliere, Tindall and Cassell.
137. HALL, L.W., (1974), Wright's Veterinary Anaesthesia and Analgesia, London, Balliere Tindall.
138. HARDAWAY, R.M., BRUNE, W., GEEVER, E., BURNS, J.W., MOCK, H.P., (1962)/



- (1962), " Studies on the role of intravascular coagulation in irreversible haemorrhagic shock ", Annals of Surgery, 155, 241.
139. HARDAWAY, R.M., (1962), " The role of intravascular clotting in the aetiology of shock ", Annals of Surgery, 155, 325.
140. HARDAWAY, R.M., DIXON, R.S., FOSTER, E.F., KARABIN, B.L., SEIFERS, F.D., MOYERS, T., (1976), " The effect of haemorrhagic shock on disseminated intravascular coagulation ", Annals of Surgery, 184, 43.
141. HARDY, J.D., GODFREY, L., (1944), " Effect of intravenous fluids on dehydrated subjects and on normal subjects ", Journal of the American Medical Association, 126, 23.
142. HARDY, J.D., (1958), Pathophysiology in surgery, Baltimore, The Williams & Wilkins Company.
143. HARPSTER, N.K., (1974), " Pulmonary oedema ", in Current Veterinary Therapy, (ed. Kirk), V, 219.
144. HARRISON, J.B., SUSSMAN, H.M., PICKERING, D.E., (1960), " Fluid and electrolyte therapy in small animals ", Journal of the American Veterinary Medical Association, 137, 637.
145. HARTHOORN, A.M., (1951), The therapy of shock in the dog, Ph.D. Thesis.
146. HARTWELL, J.A., HOGUET, S.P., (1912), " Experimental intestinal obstruction in dogs with special reference to cause of death and treatment by large amounts of normal saline solution ", Journal of the American Medical Association, 59, 82.
147. HAYES, M.A., (1968), " Water and electrolyte therapy after operation ", New England Journal of Medicine, 278, 1054.

148. HEENE, D.L., LASCH, H., (1969), " Effect of gelatin plasma substitute on homeostatic mechanism ", Bibliotheca Haematologica, 33, 428.
149. HENRY, J.P., GAUER, O.H., SIEKER, H.O., (1956), " The effect of moderate changes in blood volume on left and right atrial pressures ", Circulation Research, 4, 91.
150. HEWITT, E.A., (1952), " Fluid therapy ", in Canine Surgery, (ed. Hopkins and Lacroix), American Veterinary Publications Incorporated Ltd..
151. HINTZE, T.H., KALEY, G., (1977), " Prostaglandins and the control of blood flow in the canine myocardium ", Circulation Research, 40, 313.
152. HOLLIS, R.H., (1952), " Fluids, blood and plasma therapy in veterinary medicine ", North American Veterinarian, 33, 317.
153. HORSEY, J.S., (1933), " The intravenous administration of dextrose in ringer's lactate ", Annals of Surgery, 100, 678.
154. HOWIE, J., (1976), " Reasons for failure of antibiotic treatment ", Antibiotics in General Practice, Symposium Manchester, Abbott.
155. HUGHES, R.E., MACGOVERN, G.J., (1959), " The relationship between right atrial pressure and blood volume ", Archives of Surgery, 79, 238.
156. HULME, B., LAWSON, L.J., (1956), " Renal failure and low molecular weight dextran ", British Medical Journal, 2, 1455.
157. JACKSON, W.F., (1976), " Heart disease in the dog ", Symposium, Edinburgh.
158. JACOBS, H.R.D., HOFMANN, W.S., (1931) Journal of Biological Chemistry, 93, 685.

159. JAMES, P.M., MYERS, R.T., (1972), " Central venous pressure monitoring ", Annals of Surgery, 175, 693.
160. JONES, C.M., EATON, F.B., (1933), " Post-operative nutritional oedema ", Archives of Surgery, 27, 159.
161. JONES, D.R.E., DENNY, H.R., MULLOWNEY, P.C., (1976), " Disseminated intravascular coagulation in the dog - a case report ", Journal of Small Animal Practice, 17, 387.
162. JONES, R.R., (1963), " venous pressure in general anaesthesia ", Anaesthesia and Analgesia Current Researches, 42, 470.
163. KALTREIDER, N.L., MENEELY, G.R., (1940), " The effect of exercise on the volume of the blood ", Journal of Clinical Investigation, 19, 627.
164. KEEN, P., (1975), " Some aspects of the pharmacology of antibiotics in the cat and dog ", Journal of Small Animal Practice, 16, 767.
165. KEITH, N.M., (1924), " Experimental dehydration - changes in blood composition and body temperature ", American Journal of Physiology, 68, 80.
166. KIEF, H., (1969), " Morphological findings following single or multiple administration of gelatin plasma substitutes ", Bibliotheca Haematologica, 33, 367.
167. KIRK, R.W., (1965), " Fluid therapy ", in Canine Surgery, ( ed. Archibald), American Veterinary Publications Incorporated.
168. KIRK, R.W., (1977), Current Veterinary Therapy, VI, Philadelphia, London, W.B.Saunders Company .
169. KLUTSCH, K., HEIDLAND, A., (1969), " The influence of various volume expanders on renal function during hypo- and normovolaemia ", Bibliotheca Haematologica, 33, 380.

170. KOLATA, R.J., (1977), " Management of the severely injured animal ", Modern Veterinary Practice, 58, 142.
171. KREEL, L., SANDIN, B., (1977), " Chest symposium - the radiology of pulmonary oedema ", X-Ray Focus, 16, 2, 30.
172. KRIEGER, H., STORAASLI, J.P., FRIEDEL, H.L., (1948), " A comparative study of blood volume in dogs ", Proceedings of the Society for Experimental Biology, 68, 511.
173. LANDIS, E.M., BROWN, E., FAUTEUX, M., WISE, C., (1946), " Central venous pressure in relation to cardiac competence, blood volume and exercise ", Journal of Clinical Investigation, 25, 237.
174. LANDIS, E.M., HORTENSTINE, J.C., (1950), " Functional significance of venous blood pressure "; Physiological Reviews, 30, 1.
175. LATTA, T.A., (1832-33), " Saline venous injection in cases of malignant cholera ", Lancet, 2, 173.
176. LATTA, T.A., (1832-33), " Saline venous injection in cases of malignant cholera ", Lancet, 2, 208.
177. LEDINGHAM, I.McA., McARDLE, C.S., FISHER, W.D., MADDERN, M., (1974), " The incidence of the shock syndrome in a general hospital ", Postgraduate Medicine Journal, 50, 420.
178. LEDINGHAM, I.McA., (1975), " Septic shock ", British Journal of Surgery, 62, 777.
179. LEDINGHAM, I.McA., (1976), Shock - clinical and experimental aspects, Amsterdam, Excerpta Medica.
180. LEDINGHAM, I.McA., (1977), " Shock- aetiology and mechanisms " Lecture, Glasgow, Royal College of Physicians and Surgeons.

181. LEDINGHAM, I.McA., (1977), Personal communication.
182. LEE, H.A., (1977), " The management of acute renal failure following trauma ", Britisn Journal of Anaesthesia, 49, 697.
183. LEFER, A.M., (1973), " Blood borne humoral factors in the patho-physiology of circulatory shock ", Circulation Research, 32, 129.
184. LILLEHEI, R.C., (1951), " Relationships of appearance of abnormal plasma hemin pigment to development of irreversible haemorrhagic shock in dogs ", Circulation Research, 6, 438.
185. LILLEHEI, R.C., WASHINGTON, D.C., (1957), " The intestinal factor in irreversible haemorrhagic shock ", Surgery, 42, 1043.
186. LILLEHEI, R.C., MacLEAN, L.D., (1958), " The intestinal factor in irreversible endotoxic shock ", Annals of Surgery, 148, 513.
187. LILLEHEI, R.C., LONGERBEAM, J.K., BLOCH, J.H., MANAX, W.G., (1964), " The nature of irreversible shock - experimental and clinical observations ", Annals of Surgery, 160, 682.
188. LOCKHART, C.E., ELMAN, R.S.G., (1949), " The effect of intravenous glucose and amino acid on glycosuria and urinary output in humans " Surgery, Gynaecology and Obstetrics, 88, 97.
189. LUTZ, H., (1969), " Animal experiments on the effect of colloidal plasma substitutes in haemorrhagic shock in the dog ", Bibliotheca Haematologica, 33, 232.
190. LUTZ, H., HALLWACHS, O., (1969), " Changes in renal function after shock and volume replacement with various plasma substitutes " Bibliotheca Haematologica, 33, 398.

192. LYONS, R.H., JACOBSON, S.D., AVERY, N.L., (1944), " Increase in the plasma volume following the administration of sodium salts ", American Journal of the Medical Sciences, 208, 148.
193. MACKINTOSH, R.R., PASK, E.A., (1940), " Apparatus for giving intravenous anaesthetics continuously ", Lancet, 2, 650.
194. MacCOY, D.M., (1977), " Peritonitis ", in Current Veterinary Therapy, (ed. Kirk), VI, 49.
195. MacDONALD, J.A.E., MILLIGAN, G.F., MELLON, A., LEDINGHAM, I.McA., (1975), " Ventricular function in haemorrhagic shock ", Surgery, Gynaecology and Obstetrics, 140, 572.
196. McDONELL, W., PRESNELL, H.R., SATTLER, F.P., (1974), in Canine Surgery, (ed. Archibald ), 53.
197. MacGOVERN, G.J.,(1976), " Complications of central venous pressure ", Surgery, 79, 726.
198. McGOWAN, G.K., WALTERS G., (1963), " The value of measuring central venous pressure in shock ", British Journal of Surgery, 50, 821.
199. McIVER, M.A., GAMBLE, J.L., (1928), " Body fluid changes due to upper intestinal obstruction ", Journal of the American Medical Association, 91, 1589.
200. McKENZIE, A.I., DONALD, J.R.,(1959), " Urine output and fluid therapy during anaesthesia and surgery ", British Medical Journal, 3, 619.
201. McLAIN, D.L., (1975), " Percutaneous and cut-down intravenous catheterisation, C.V.P. measurement and indwelling drainage of the bladder ", Scientific Presentations of the American Animal Hospital Association, 42, 248.

202. MacLEAN, L.D., WEIL, M.H., (1956), " Hypotension (shock) in dogs produced by E.coli endotoxin ", Circulation Research, 4, 546.
203. MacLEAN, L.D., (1964), " Blood volume versus central venous pressure in shock ", Surgery, Gynaecology and Obstetrics, 118, 594.
204. MacLEAN, L.D., DUFF, J.H., SCOTT, H.M., PERETZ, D.J., (1965), " Treatment of shock in man based on haemorrhagic diagnosis ", Surgery, Gynaecology and Obstetrics, 120, 1.
205. MacLEAN, L.D., (1966), " The clinical management of shock ", British Journal of Anaesthesia, 38, 255.
206. McMURRAY, J.D., LAW, S.W., (1961), " Post-operative changes in electrolyte balance ", Anaesthesiology, 22, 819.
207. MARSHALL, D.E., SOMA, L.R., HARP, J.R., NEUFELD, G.R., WURZEL, H.A., DODD, D.C., (1974), " Pulmonary function after exchange transfusion of stored blood in dogs ", Annals of Surgery, 179, 46.
208. MARSHALL, D.E., THORNTON, G.W., (1974), " Intensive care " in Current Veterinary Therapy, (ed. Kirk), V, 17.
209. MARRIOT, H.L., (1950), Water and Salt Depletion, Springfield, Illinois, C.C.Thomas.
210. MASSON, A.H.B., (1971), " Latta - pioneer in saline infusions ", British Journal of Anaesthesia, 43, 681.
211. MATAS, R., (1924), " The continued intravenous drip ", Annals of Surgery, 79, 643.
212. /

212. MILLER, J.R., POINDEXTER, C.A., (1932), " The effects observed following the intravenous and subcutaneous administration of fluid - experimental study on dogs ", Journal of Laboratory and Clinical Medicine, 18, 287.
213. MILLER, J.R., STOETLING, V.H., PARADISE, R.R., (1963), " A study of fluids and electrolytes during and after surgery ", Anaesthesia and Analgesia, 42, 640.
214. MILLER, M.E., CHRISTENSEN, G.C., EVANS, H.E., (1964), Anatomy of the Dog, Philadelphia, London, W.B.Saunders Company.
215. MILLER, W.C., WEST, G.P., (1970), Black's Veterinary Dictionary, London, Adam and Charles Black.
216. MOORE, F.D., BULL, M.R., (1952), The metabolic response to trauma , Springfield, Illinois, C.C.Thomas.
217. MOORE, F.D., SHIRES, G.T., (1967), "Moderation", Annals of Surgery, 166, 300.
218. MOORE, N.S., VAN SLYKE D.D., (1930), " The relationship between plasma specific gravity, plasma protein content and edema in nephritis " , Journal of Clinical Investigation, 8, 337.
219. MORGAN, T.O., LITTLE, J.M., EVANS, W.A., (1966), " Renal failure associated with low molecular weight dextran ", British Medical Journal, 2, 737.
220. MOYER, C.A., (1947), " Fluid and electrolyte balance ", Surgery, Gynaecology and Obstetrics, 84, 586.
221. MOYER, C.A., (1950), " Acute temporary changes in renal function associated with major surgical procedures ", Surgery, 27, 198.



222. MULLER, K.H., (1969), " Comparative studies on the influence of plasma substitutes on coagulation in vivo ", Bibliotheca Haematologica, 33, 434.
223. NADAL, J.W., PEDERSEN, J., MADDOCK, W.G., (1941), " A comparison between dehydration from salt loss and from water deprivation ", Journal of Clinical Investigation, 20, 691.
224. NATIONAL BLOOD TRANSFUSION SERVICE, (1973), Notes on Transfusion, Department of Health and Social Security ( Scottish Home and Health Department ).
225. NEMIR, P., HAWTHORNE, H.R., COHN, I., DRABKIN, D.L., (1949), " The cause of death in strangulation obstruction ", Annals of Surgery, 130, 857.
226. NIALL, J.F., DOYLE, J.C., (1966), " Treatment with dextran 40 ", Lancet, 1 817.
227. OMAMEGBE, J., (1976), M.V.M. Thesis, (Glasgow).
228. ORDWAY, C.B., (1974), " Air embolus via C.V.P. catheter without positive pressure ", Annals of Surgery, 179, 479.
229. ORR, T.G., HADEN, R.L., (1928), " Chemical factors in toxæmia of intestinal obstruction ", Journal of the American Medical Association, 91, 1529.
230. OSER, B.L., Hawks Physiological Chemistry, (1968), McGraw Hill Book Company, 14th, Ed., New York, Toronto, Sydney, London.
231. PAGE, I.H., KOHLSTAEDT, K.C., (1947), " Haemorrhagic hypotension and its treatment by intra-arterial and intravenous infusion of blood ", Archives of Surgery, 47, 178.

232. PAREIRA, M.D., SOMOGYI, M., (1948), " Rationale of parenteral glucose feeding in the post-operative state ", Annals of Surgery, 127, 417.
233. PAREIRA, M.D., (1959), " The effects of plasma and dextrose compared to saline for fluid replacement following traumatic shock ", Surgery, 45, 623.
234. PENNY, S.G., (1953), " Blood substitutes or plasma expanders in small animal practice ", Veterinary Medicine, 48, 151.
235. PERCIVAL, A., (1976), " Management of urinary tract infections ", Antibiotics in General Practice, Symposium, Manchester, Abbot, 17.
236. PETERS, G., BRUNNER, H., (1963), " Mannitol diuresis in haemorrhagic hypotension ", American Journal of Physiology, 204, 555.
237. PFEIFER, G.W., JENTSCH, D., KRUGER, J., DUDECK, E., DIETZ, M., (1969), " Influence of rapid infusion of colloidal plasma volume expander on platelet function and plasma clotting factors ", Bibliotheca Haematologica, 33, 481.
238. PFEIFER, G.W., (1969), " Special indications for gelatin plasma substitutes in gynaecology ", Bibliotheca Haematologica, 33, 623.
239. PIERCE, V.K., BOYAN, C.P., MASTERTON, J.G., (1953), " Studies on venous blood pressure in patients undergoing major surgical procedures ", Surgery, Gynaecology and Obstetrics, 96, 310.
240. POLIWODA, K., (1969), " The effect of plasma expanders on the formation of platelet rich agglutination thrombi ", Bibliotheca Haematologica, 33, 466.

241. POWERS, S.R., SHAH, D., RYON, D., NEVELL, J., RALPH, C., SCOVILL, W., DUTTON, R., (1977), " Hypertonic mannitol in the therapy of the acute respiratory distress syndrome ", Annals of Surgery, 185, 619.
242. PRENTICE, T.C., OLNEY, J.M., ARTZ, C.P., HOWARD, J.M., (1954), " Studies of blood volume and transfusion therapy in the Korean battle casualty," Surgery, Gynaecology and Obstetrics, 99, 542.
243. PRICE, H.L., DEUTSCH, S., DAVIDSON, I.A., CLEMENT, A.J., BEHAR, M.G., EPSTEIN, R.M., (1966)," Can general anaesthetics produce splanchnic visceral hypoxia by reducing regional blood flow ", Anaesthesiology, 27, 24.
244. RAWLINGS, C.A., WINGFIELD, W.E., BETTS, C.W., (1976), " Shock therapy and anaesthetic management in gastric dilatation volvulus ", Journal of the American Animal Hospital Association, 12, 158.
245. REMINGTON, J.W., HAMILTON, W.F., BOYD, G.H., HAMILTON, W.F. Jr., CADDELL, H.M., (1950), " Role of vasoconstriction in the response of the dog to haemorrhage ", American Journal of Physiology, 161, 116.
246. REYNOLDS, M., (1949), " Cardiovascular effects of large volumes of isotonic saline infused intravenously into dogs following severe haemorrhage ", American Journal of Physiology, 158, 418.
247. RICHARDSON, B.W., (1866), " Physiological formula of cholera and of the treatment," Medical Times and Gazette, 2, 111.
248. RICKETTS, C.R., (1973), " Blood substitutes ", British Journal of Anaesthesia, 95, 958.

249. RITTMEYER, P., (1969), " Microcirculatory impairment during experimental shock in dogs ", Bibliotheca Haematologica, 33, 342.
250. ROBERTSON, J.D., (1935), " The effect of haemorrhage of varying degree on blood and plasma volume, on blood sugar, and on arterial pressure ", Journal of Physiology, 84, 393.
251. ROBERTSON, J.D., (1938), " Blood volume changes in health and haemorrhage and after various infusions ", Lancet, 2, 634.
252. ROBINSON, J.R., (1962), Fundamentals of Acid Base Regulation, Blackwell Scientific Publications.
253. RODGERS, K.B., (1976), " Intestinal infections ", Antibiotics in General Practice, Symposium, Manchester, Abbot, 31.
254. ROE, C.F., GARDINER, A.J.S., DUDLEY, H.A.F., (1962), " A simple instrument for the continuous accurate measurement of operative blood loss ", Lancet, 1 672.
255. ROSS, J.K., (1957), " Vena caval infusions ", Postgraduate Medical Journal , 33, 623.
256. ROTH, E., LAX, L.L., MALONEY, J.V., (1969), " Ringer lactate solution and extracellular fluid volume in the surgical patient - a critical analysis ", Annals of Surgery, 169, 149.
257. SARNOFF, S.J., (1955), " Regulation of heart performance ", Physiological Reviews, 35, 107.
258. SCHAFER, H., (1969), " Results of treatment with gelatin plasma substitutes in accident surgery" , Bibliotheca Haematologica, 33, 518.
259. SCHALM, O.W., JAIN, N.C., CARROL, L.J., (1975), Veterinary Haematology 3rd. Ed., Lea and Febiger, Philadelphia.

260. SCOTT, H.G., (1938), " Intestinal obstruction - experimental evidence on the loss of blood in intestinal obstruction ", Archives of Surgery, 38, 816.
261. SCRIBNER, B.H., BURNELL, T.M., (1956), " Interpretation of the serum potassium concentration ", Metabolism, 5, 468.
262. SCURR, C., FELDMAN, S., (1974), Scientific Foundations of Anaesthesia 2nd. Ed., London, William Heinemann Medical Books Ltd., 392.
263. SHARPEY\_SCHAFER, E.P., (1963), " Venous tone - effects of reflex changes, humoral agents and exercise ", British Medical Bulletin, 19, 145.
264. SHENKIN, H.A., CHENEY, R.K., GOUARD, S.R., HARDY, J.D., (1944), " On the diagnosis of haemorrhage in man ", American Journal of the Medical Sciences, 208, 421.
265. SHIELDS, R., (1965), " The absorption and secretion of fluid and electrolytes by the obstructed bowel ", British Journal of Surgery, 52, 744.
266. SHIRES, T., JACKSON, D.E., (1962), " Post-operative salt tolerance " Archives of Surgery, 84, 703.
267. SHIRES, T., COLN, D., CARRICO, J., LIGHTFOOT, S., (1964), " Fluid therapy in haemorrhagic shock ", Archives of Surgery, 88, 688.
268. SIMPSON, K., (1942), " An accident during transfusion ", Lancet, 1, 697.
269. SKEGGS, A., (1957), " Diacetyl monoxime method for estimation of blood urea ", American Journal of Clinical Pathology, 28, 311.
270. SOMA, C.R., BURROWS, C.F., MARSHALL, D.E., (1974), " Shock - aetiology and management " in Current Veterinary Therapy, (ed. Kirk) V, 26.

271. SOMA, C.R., BURROWS, C.F., MARSHALL, D.E., (1974), " Non-penetrating blunt pulmonary trauma " in Current Veterinary Therapy, (ed. Kirk), V, 32.
272. SPURR, G.B., FARRAND, E.A., HORVATH, S.M., (1956), " The effect of chlorpromazine on survival from haemorrhagic shock ", American Journal of Physiology, 185, 499.
273. STAHL, W.M., (1967), " Intra-operative support by sodium infusion ; an approach to quantitation ", Surgical Forum, 18, 39.
274. STANBRIDGE, T.N., (1976), " Choosing an antibiotic ", Antibiotics in General Practice, Symposium, Manchester, Abbott, 5.
275. STARR, I., COLLINS, L.H., (1931), " Fainting and syncope ", Journal of Clinical Investigation, 9, 552.
276. STARR, I., (1943), " Incoordination of the circulation ", Journal of Clinical Investigation, 22, 813.
277. STAUB, N.C., (1974), " Pulmonary oedema ", Physiology Reviews, 54, 3.
278. STEWART, J.D., ROURKE, G.M., (1942), " The effects of large intravenous infusions on body fluids ", Journal of Clinical Investigation, 21, 197.
279. STEWART, J.D., (1947), " Use of blood and blood substitutes ", Surgery, Gynaecology and Obstetrics, 84, 601.
280. STONE, H.M., HOOPER, C.A., KOLB, L.D., GEHEBER, C.E., DAWKINS, E.J., (1976), " Antibiotic prophylaxis in gastric, biliary and colonic surgery ", Annals of Surgery, 184, 443.
281. SWISHER, G.N., YOUNG, L.E., (1961), " The blood grouping systems of dogs " Physiology Reviews, 14, 495.

282. SYKES, M.K., (1963), " Venous pressure as a clinical indication of the adequacy of transfusion " *Annals of the Royal College of Surgeons of England*, 32, 185.
283. SYKES, M.K., ADAMS, A.P., FINLAY, W.E.I., WIGHTMAN, A.E., MONROE, J.P., (1970), " The cardiorespiratory effects of haemorrhage and overtransfusion in dogs ", *British Journal of Anaesthesia*, 42, 573.
284. SYKES, W.S., (1960), *Essays on the First Hundred Years of Anaesthesia*, Vol. I and II, Edinburgh and London, E.&S. Livingstone.
285. TALBOT, N.D., CRAWFORD, J.D., BUTLER A.M., (1953), " Homeostatic limits to safe fluid therapy ", *New England Journal of Medicine*, 248, 1100.
286. THEOBALD, G.W., VERNEY, E.B., (1935), " The inhibition of water diuresis by efferent nerve stimuli after complete denervation of the kidney ", *Journal of Physiology*, 83, 341.
287. THEYE, R.A., MOFFIT, E.A., (1962), " Blood transfusion therapy during anaesthesia and operation ", *Anaesthesia and Analgesia Current Researches*, 41, 354.
288. THOMAS, D.J.B., ALBERTI, K.G.M.M., (1978), " Hyperglycaemic effects of Hartmann's solution during surgery in patients with maturity onset diabetes ", *British Journal of Anaesthesia*, 50, 185.
289. THOMPSON, W.L., (1959), " The use of dextrans as infusion fluids ", *Surgery, Gynaecology and Obstetrics*, 109, 43.

290. TRUDNOWSKI, R.J., GOEL, S.B., LAM, F.T., EVERS, J.L., (1967),  
" Effect of ringer lactate solution and sodium bicarbonate  
on surgical acidosis ", Surgery, Gynaecology and Obstetrics,  
125, 807.
291. VARLEY, H., (1969), Practical Clinical Biochemistry, 4th. ed.,  
William Heinemann Medical Books Ltd..
292. WARREN, J.V., STEAD, E.A., (1943), " The effect of the accumulation  
of blood in the extremities on the venous pressure of  
normal subjects ", American Journal of the Medical  
Sciences, 205, 501.
293. WARREN, J.V., MERRIL, A.J., STEAD, E.A., (1943), " Role of the  
extracellular fluid in the maintenance of a normal  
plasma volume ", Journal of Clinical Investigation, 22,  
635.
294. WARREN, J.V., BRANNON, E.S., WEENS, H.S., STEAD, E.A., (1948),  
" Effect of increasing the blood volume and right atrial  
pressure on the circulation of normal subjects by  
intravenous infusions, " American Journal of Medicine,  
4, 193.
295. WARTHEN, H.J., (1935), " Massive intravenous infusion - experi-  
mental study ", Archives of Surgery, 30, 199.
296. WATSON, A.D.J., (1977), " Antimicrobial therapy ", in Current  
Veterinary Therapy, (ed. Kirk), VI, 15.
297. WEIL, M.H., SHUBIN, H., ROSOFF, L., (1965), " Fluid repletion in  
circulatory shock ", Journal of the American Medical  
Association, 192, 668.
298. WEST, J.B., (1965), Ventilation/blood flow and gas exchange,  
Blackwell Scientific Publications Ltd..



299. WHEATCROFT, J., (1858), " Cases of uterine haemorrhage treated successfully by transfusion ", British Medical Journal, 1, 290.
300. WIGGERS, C.J., (1945), " The failure of transfusion in irreversible haemorrhagic shock ", American Journal of Physiology, 144, 91.
301. WILEY, F.H., NEWBURGH, L.H., (1931), " The relationships between the environment and the basal insensible loss of weight ", Journal of Clinical Investigation, 10, 689.
302. WILKINSON, A.W., (1955), Body fluids in surgery, Edinburgh, Livingstone.
303. WILLIAMS, J.A., FINE, J., (1961), " Measurement of blood volume with a new apparatus ", New England Journal of Medicine, 264, 842.
304. WILSON, R.F., THAL, A.P., KINDLING, P.H., GRIFKA, T., ACKERMAN, E., (1965), " Haemodynamic measurements in septic shock ", Archives of Surgery, 91, 121.
305. WILSON, R.F., BASSET, J.S., WALT, A.J., (1965), " Five years of experience with massive blood transfusions ", Journal of the American Medical Association, 194, 851.
306. WILSON, R.F., SARVER, E., BIRKS, R., (1971), " Central venous pressure determination in clinical shock ", Surgery, Gynaecology and Obstetrics, 132, 631.
307. WILSON, J.N., GROW, J.B., DEMONG, C.V., PREVEDEL, A.E., OWENS, J.C., (1962), " Central venous pressure in optimal blood volume determination ", Archives of Surgery, 85, 563.

308. WILSON, J.N., (1965), " Rational approach to management of  
clinical shock ", Archives of Surgery, 91, 92.
309. WOLFSON, L.J., (1963), " Blood loss in trauma " Annals of the  
Royal College of Surgeons of England, 33, 158.
310. WRIGHT, C.J., (1976), " The effects of severe progressive  
haemodilution on regional blood flow and oxygen  
consumption ", Surgery, 79, 299.
311. ZEKORN, D., (1969), " Intravascular retention, dispersal, excretion  
and breakdown of gelatin plasma substitute ", Bibliotheca  
Haematologica, 33, 131.
312. ZIMMERMAN, B., WANGENSTEEN, O.H., (1952), " Observations on water  
intoxication in surgical patients ", Surgery, 31, 654.

## APPENDICES

LIST OF APPENDICES

	Page
APPENDIX 1. Venous pressure study results	326
APPENDIX 2. Experimental study results	327
APPENDIX 3. Survey of fluid therapy use by practitioners - results and graphs	328
APPENDIX 4. Methods of administration	343
Methods of monitoring and recording	363
Antibiotic administration	
APPENDIX 5. Study of clinical cases - results and analysis	364

## APPENDIX 1

APPENDIX 1.

VENOUS PRESSURE STUDY - tables of results

Tables 1 - 7.

No	Breed	Age	Sex	Weight	Dur.	ST	Time (Hrs.)												Fluid		
							0	.25	.5	.75	1	1.25	1.5	1.75	2	2.25	2.5	2.75	3	Av.	Post op.
1	J.R.T.	11/12	M	6	3	04	18	18	18	17	16	16	16	16	18	19	19	19	18	17.5	
							2	2	2	2	1.5	1.5	1.5	1.5	1.5	2	2	2	2	1.75	
2	Collie	1.5	M	20.75	1.25	03	20	20	18	16	17.5	17.5							18		
							4	4	3.5	3	3	3.5							3.5		
3	Poodle	10	M	7.5	1.25	N2	19	19	18.5	17	17	16							17.5		
							1.5	1.5	2	1	1	1							1.5		
4	Poodle	3	M	7.5	1.5	03	17.5	20	20	18.5	18	17.5	17.5						18.75		
							-1	0	0	0	-0.5	-0.5	-0.5						-0.5		
5	Greyhound	3	M	30.75	1.5	04	22.5	22	19	21	22	21	20.5						20.75		
							4	4	3.5	3	4.5	4.5	4						3.75		
6	Labrador	8	M	27	1.75	03	20	20	22	18	19	19.5	19						20		
							6	6	6	5	5	5	5						5.5		
7	Chinese Crested	4/12	M	3.5	1.25	03	14	13.5	12	12.5	13	13							13		
							1	1	0.5	0.5	0.5	0.5							0.75		
8	Mongrel	4/12	F	10.75	1.5	03	17.5	17	16	14.5	14	14	14						16.25		
							3.5	3	2.5	2.5	2	2	2.5						3		
9	Cairn	2	F	7.5	1.25	03	12	12	15	15	15	15							13.5		
							0	0	0	1	1	1							0.5		
10	Alsatian	6/12	M	13.25	1.25	04	11	11.5	11	11	11	10							10.5		
							-3	-2	-2	-2	-2	-2							-2.5		
11	Labrador	9	F	27	0.75	X1	14	14	15.5	16									15		
							4.5	4.5	4.5	4									4.25		
12	I. Setter	6	F	22	1.75	03	10.5	10.5	11	11	11	11.5	10						10.5		
							1.5	1.5	1.5	1.5	1.5	1.5	1.5						1.5		
13	Greyhound	3	M	37	1.25	03	22	22	19	18.5	19	19							19.75		
							6	6	5.5	5.5	5	5							5.5		
14	Alsatian	6/12	M	22.75	1.5	05	17	17	17	17	15	15	14.5						15.75		
							3	3	3.5	3	3	2.5	3						3		

TABLE 7

Continued.....

No	Breed	Age	Sex	Weight	Dur.	ST	Time (Hrs.)												Av.	Fluid Post op.
							0	.25	.5	.75	1	1.25	1.5	1.75	2	2.25	2.5	2.75	3	
15	WHWT	1	M	8.5	2	05	18	18	18	18	17.5	17.5	18	18	18				17.75	
							3.5	3.5	3	3	3	3	3	3	3				3.75	
16	Labrador	6	M	31	1.5	05	20	19	17	17	17	17	17	17.5					18.5	*
							2	2	1.5	1	1	1	1	1					1.5	
17	Visla	2	F	19	2	04	19	19	18	18.5	18	17.5	16	18.5	17				17.5	
							-1	-0.5	0	0	0	-1	-1	-1	-1				-0.5	
18	Collie	9	F	14.25	1	02	15	14	14	15	15								14.5	
							4	4	4	4	3.5								3.75	
19	Collie	7	M	10.25	1.75	04	11.5	12	12.5	15	13	14	14	14	14				12.75	
							1.	1.5	1	1.5	2	2	2	2	2				1.5	
20	Sheltie	13	F	7.5	1	X2	12	12	12	11	11								11.5	
							2.5	2.5	2	1.5	1								1.75	
21	Collie	<sup>B</sup> /12	M	12.5	1.75	03	15.5	15.5	15.5	15.5	16	16	16	16	16				15.75	
							3	3	3.5	3	4	4	4	4	4				3.5	
22	Poodle	2	F	9	1.5	X4	18	18	16	15	15	15	16						16.5	
							5.5	5.5	5	4.5	2	2	2						3.25	
23	Labrador	2.5	M	33.5	1.25	X4	16	16	14	15.5	16	15.5							15	
							0	0	-0.5	-0.5	-0.5	-0.5	-0.5						-0.25	
24	Alsatian	7	M	46.5	0.75	X2	17	17.5	17	17									17.25	
							4	4	4	4									4	
25	WHWT	8	F	6.25	1.25	03	14.5	14	15	15	15	15	15						14.5	
							2	2	2	1	0.5	1							1.25	

TABLE 7



APPENDIX 2

APPENDIX 2.

EXPERIMENTAL STUDY - Pre-anaesthetic clinical check of dogs  
Pre-anaesthetic blood sample results of dogs  
Tables of results of experimental work  
Graphs 1 - 10  
Tables 1 - 2

# APPENDIX 2

Pre-anaesthesia clinical check experimental dogs.

Dog	Breed	Age	Sex	Weight kg.	Appearance	C.V. system	Resp. system	Abd. palp.	Temp.
1	Labrador	A	M	29	Good	-	-	-	N
2	Foxhound	Y	F	19	"	-	-	-	N
3	Foxhound	Y	M	18.5	"	-	-	-	N
4	Foxhound	Y	M	20	"	-	-	-	N
5	Foxhound	Y	F	22	"	-	-	-	N
6	Lurcher	A	M	30	"	-	-	-	N
7	Dobermann	Y	M	25	"	-	-	-	N
8	Mongrel	A	M	20	"	-	-	-	N
9	Labrador	Y	M	28	"	-	-	-	N
10	Labrador	Y	M	20	"	-	-	-	N
11	Labrador	Y	M	24	"	-	-	-	N
12	Labrador	Y	M	31	"	-	-	-	N
13	Labrador	Y	F	23	"	-	-	-	N
14	Labrador	A	M	28	"	-	-	-	N
15	Labrador	Y	M	28	"	-	-	-	N
16	Mongrel	A	M	28	"	-	-	-	N

# APPENDIX 2

## Pre-anaesthesia blood sample results experimental dogs.

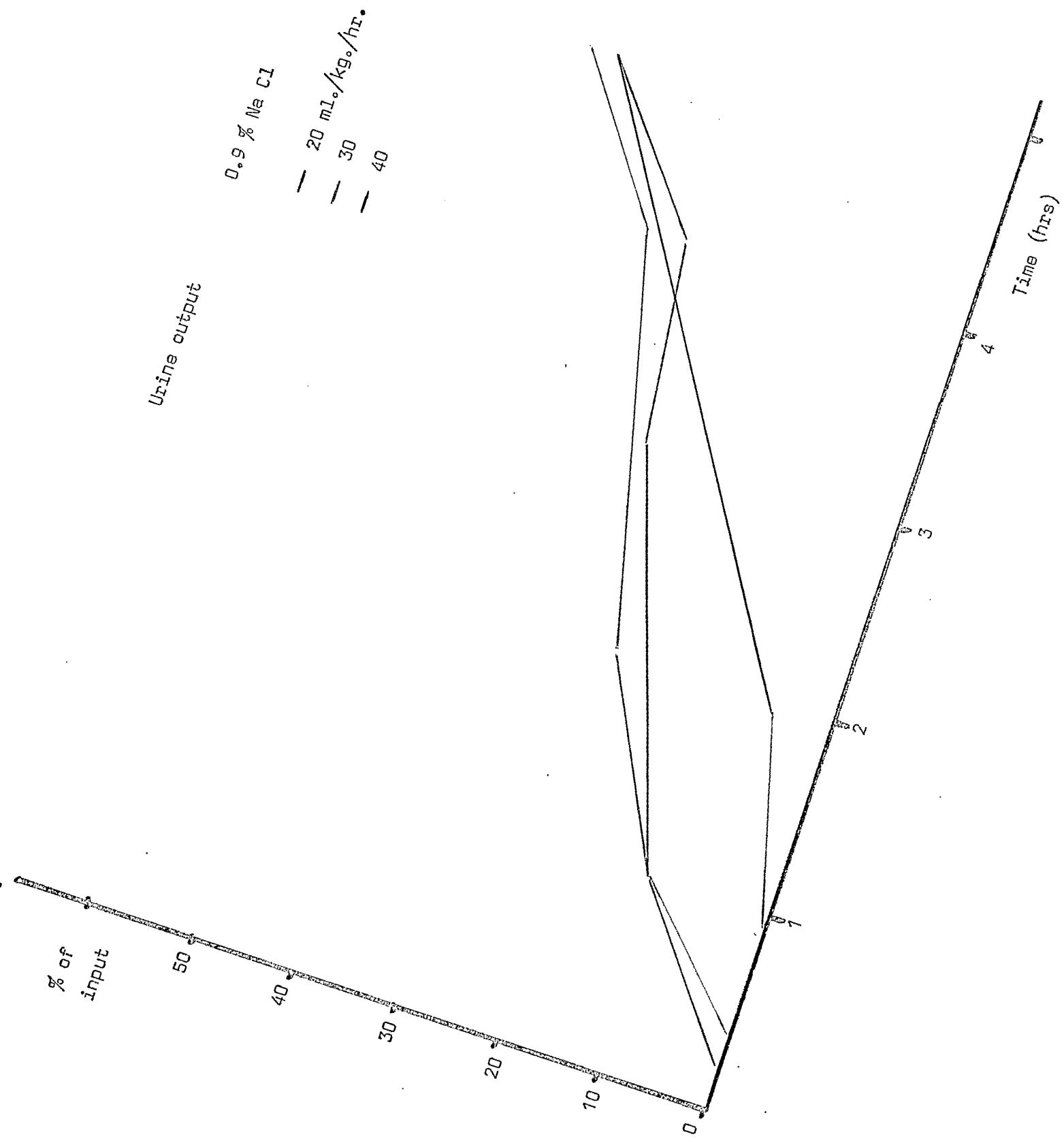
Dog	PCV	Hb	R.B.C.	W.B.C.	L.	N.	Urea	Na	K	Cl	Ca	Mg	BiI.	Alk.Ph.	AsT	ALT	TSP
	ml%	g/100ml	M/cmm	per cmm	%	%	mmol/l						umol/l	I.U.			g/l
1	41	13.5	5.46	5800	27	73	4.8	143	4.3	112	2.5	0.84	4	18	32	64	63
2	41	13.5	5.23	5300	30	70	4.3	150	4.5	115	2.3	0.80	1	41	30	30	53
3	37	12.9	5.23	3100	30	70	5.1	158	3.3	113	2.5	0.86	4	30	30	35	55
4	39	11.6	5.47	4300	25	75	3.2	152	4.2	115	2.4	0.75	4	61	57	55	65
5	36	10.7	5.10	3400	40	60	3.5	152	3.9	114	2.3	0.75	9	42	14	31	62
6	45	16.6	10.7	8600	26	74	6.7	154	4.3	101	2.5	0.88	1	35	25	42	54
7	43	15.0	5.72	5200	21	79	5.6	147	4.2	106	2.3	0.86	1	48	41	47	53
8	44	13.2	6.38	6600	22	78	4.6	141	4.1	104	2.3	0.65	4	21	26	48	60
9	43	11.9	5.99	3100	40	60	6.3	148	4.4	110	2.6	0.72	1	60	34	42	62
10	43	13.2	6.25	4500	40	60	6.3	157	5.2	111	2.5	0.80	6	21	18	18	62
11	42	13.8	5.77	4000	26	74	4.8	154	4.2	110	2.6	0.71	5	22	46	73	53
12	38	12.6	5.21	5200	40	60	41	153	4.2	111	2.9	0.71	5	38	28	16	53
13	38	11.4	5.26	3000	41	59	4.5	152	4.4	117	2.4	0.72	1	15	30	42	52
14	35	10.4	5.64	9000	28	72	4.3	142	5.7	112	2.3	0.74	1	28	22	34	60
15	40	12.2	5.62	7900	30	70	5.4	159	4.1	118	2.5	0.83	1	44	36	48	54
16	38	10.6	5.62	4200	42	58	5.1	151	3.8	114	2.3	0.90	7	31	64	58	59
Mean	40	12.6	5.83	5400	32	68	4.9	151	4.2	112	2.4	0.76	3	35	30	42	57
S.D.	3	1.6	1.21	2070	7.3	7.3	0.9	5.4	0.3	4.4	0.1	0.07	2.5	13.6	10.5	15.9	4.3

[illegible]

[illegible]

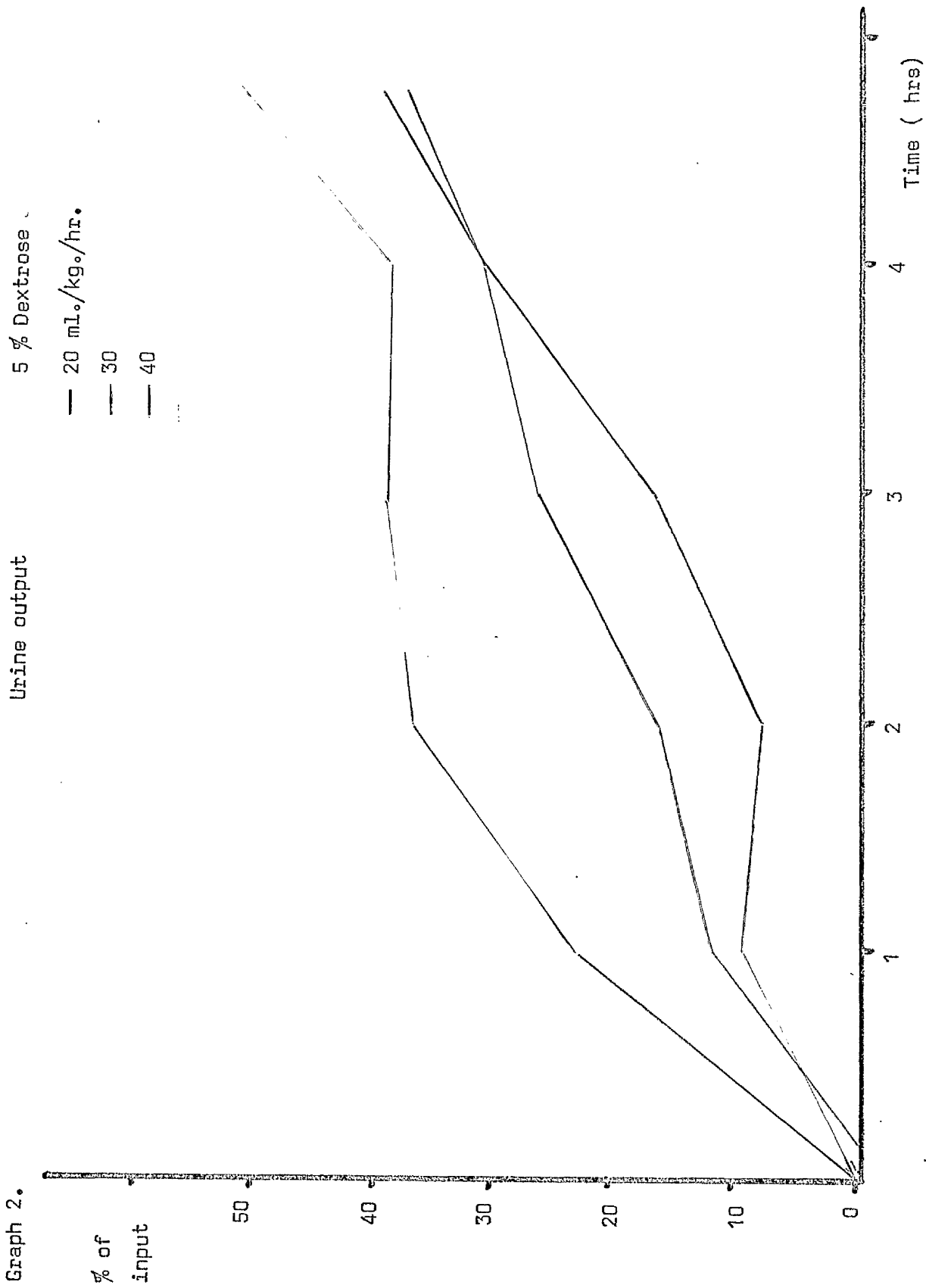
Temp. °C	30	30	30	30	37.5	37.5	37.5	37.5	37.5	37.5	37.5	37.5	37.5	37.5	37.5
Pulse/min.	120	115	120	130	150	160	170	175	180	180	190	190	200	160	160
Resp./min.	12	12	12	12	15	22	24	30	40	50	80	90	100	100	100
B.P. Syst.	90	90	95	95	140	150	160	165	160	160	160	160	160	160	160
B.P. Diast.	70	70	70	75	90	110	120	125	120	120	120	120	120	120	120
B.P. Mean	80	80	82	85	115	130	140	145	140	148	140	140	140	140	140
PVP mm Hg	5	5	5	5	11.5	14.5	20	20	20	17	15	15	17	16	14
CVP mm Hg	0	0	0	0	2.5	7	9	15	15	15	11	11	13	12	8
PaCO <sub>2</sub>	64		51.7					48.4				49.5	56.6	49.9	
PaO <sub>2</sub>	414		391					361				187	108	115	
X-ray signs			-					+				+++	++++	++++	
Urine output	20		0	20	20	20	20	20	100	170	240	410	580	730	910
Fluid input			0	280	560	560	560	1120	0	140	280	420	560	1120	
P.C.V.	30		29					18				21	18	23	
Na	151		152					150				151	152	152	
K	3.8		3.9					4.0				4.1	4.2	4.5	
Cl	114		115					119				118	117	117	
T.S.P.	58		56					26				22	18	24	
Oral M.M.	M		M					D				D	+	+	
Eyes M.M.	M		M					D				M	M	+	
Nose M.M.	M		M					D				D	D	D	
Feet edema	+		+					+				+	-	-	
Flank edema	+		+					+				+	+	+	
Auscultation	+		+					+				+	++	++	++

Graph 1.





Graph 2.



Graph 3.

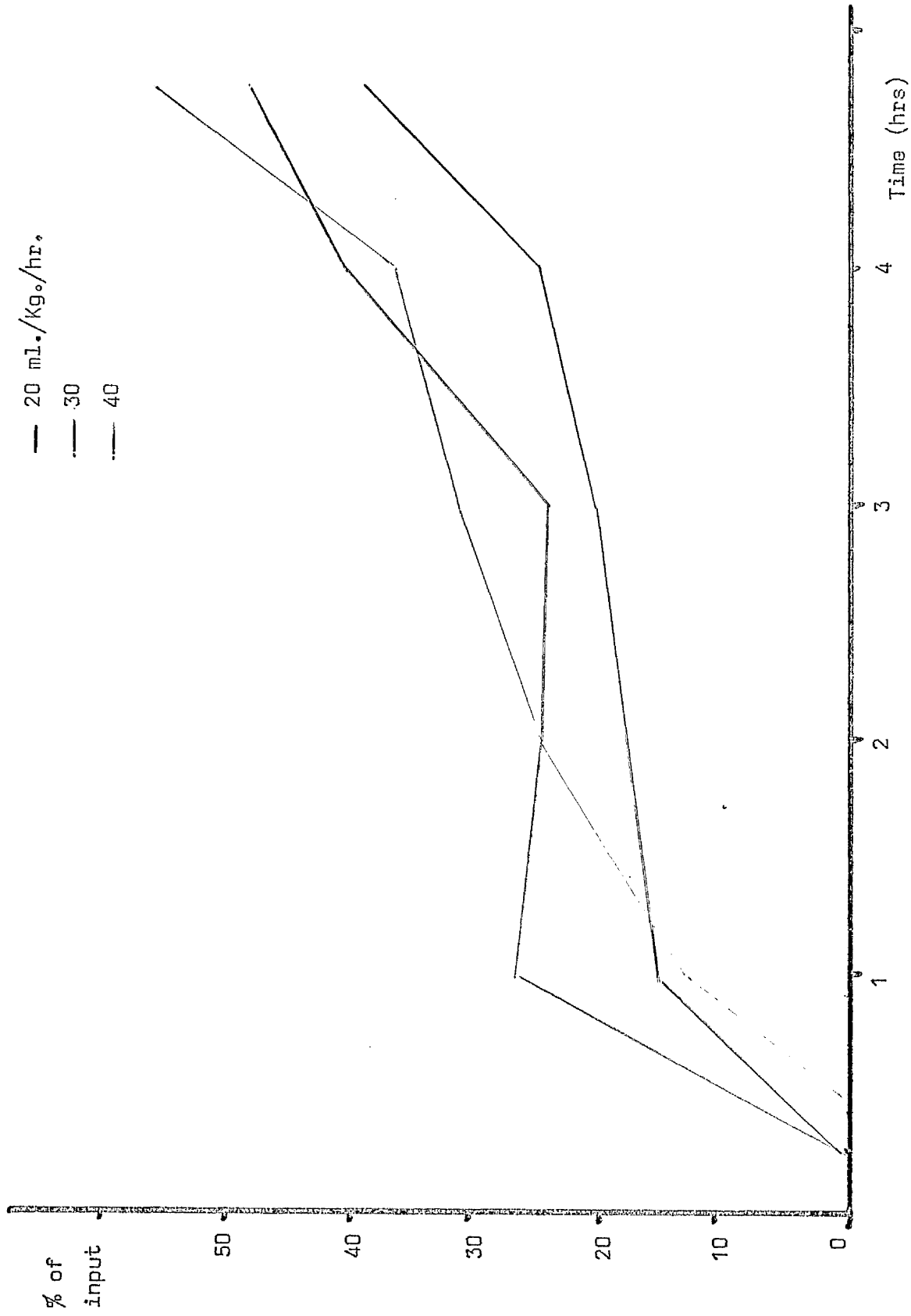
Urine output

0.18% NaCl 4.3% Dextrose

— 20 ml./kg./hr.

— 30

— 40



Graph 4

Urine output

Ringer Lactate

— 20 ml./kg./hr.

— 30

— 40

% of  
input

50

40

30

20

10

0

1

2

3

4

Time (hrs.)

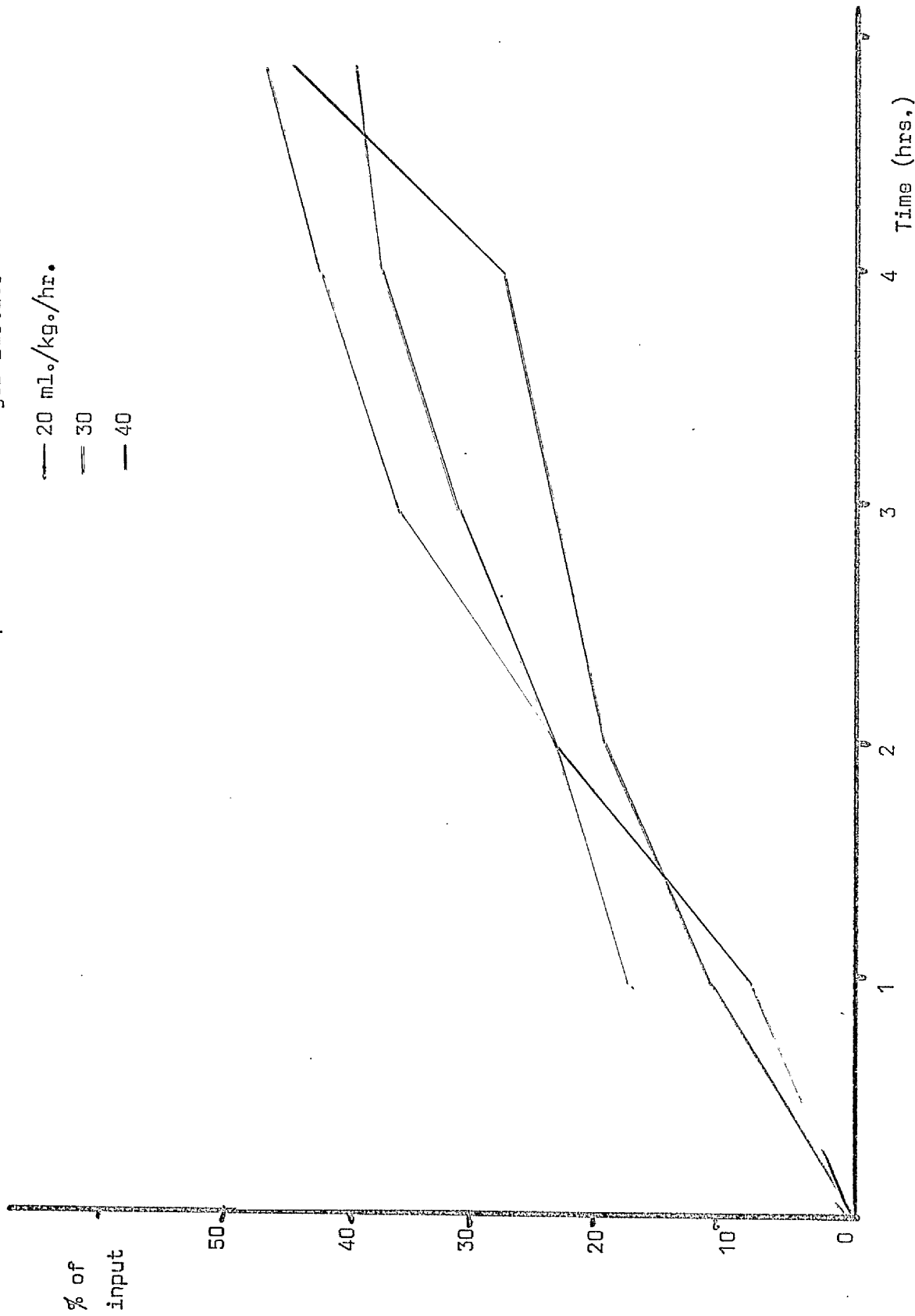


Table 1.

Dogs 1 - 12 urine output rate over 4 hour infusion

<u>Fluid</u>	<u>ml/kg</u>	<u>0</u>	<u>1</u>	<u>2</u>	<u>3</u>	<u>4</u> hours
Na Cl	20	2.7	3.4	4.4	6.8	21.6
(0.9%)	30	3.6	9.4	10.5	16.8	32.9
	40	4.8	10.8	13.5	16.2	43.2
Dextrose	20	2.5	4	9.5	8.5	16.6
(5%)	30	7.7	14	11.8	15.9	48.4
	40	4	1.6	15	23.6	55.5
Dext, saline	20	3.2	4	5.2	8	25
(0.18+4.3%)	30	4	11	14	14.5	45.3
	40	10.7	8.9	7.5	32.1	53.8
Ring. lactate	20	25	6	7.5	7.5	35.3
	30	5.8	10.4	16.6	20.8	35
	40	3.5	17.4	18.7	24.1	35.6

Graph 5

Urine output

Dextran 40

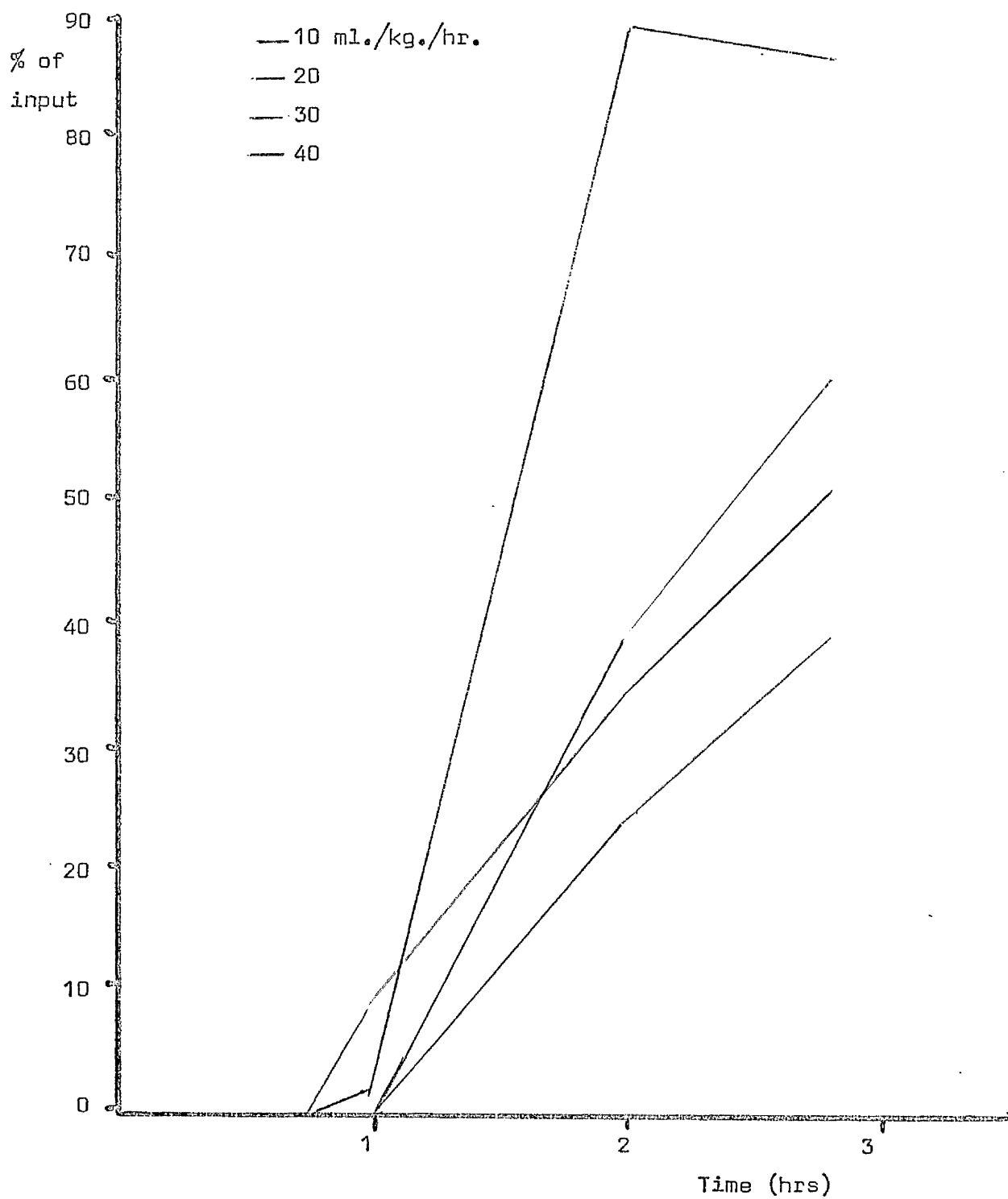


Table 2.

Dogs 13 - 16 urine output rate during colloid infusion

<u>ml / kg</u>	<u>0</u>	<u>1</u>	<u>2</u>
10	0.8	27.3	26
20	2.1	12.5	21.4
30	0	20	30.4
40	0.7	13.9	23.8

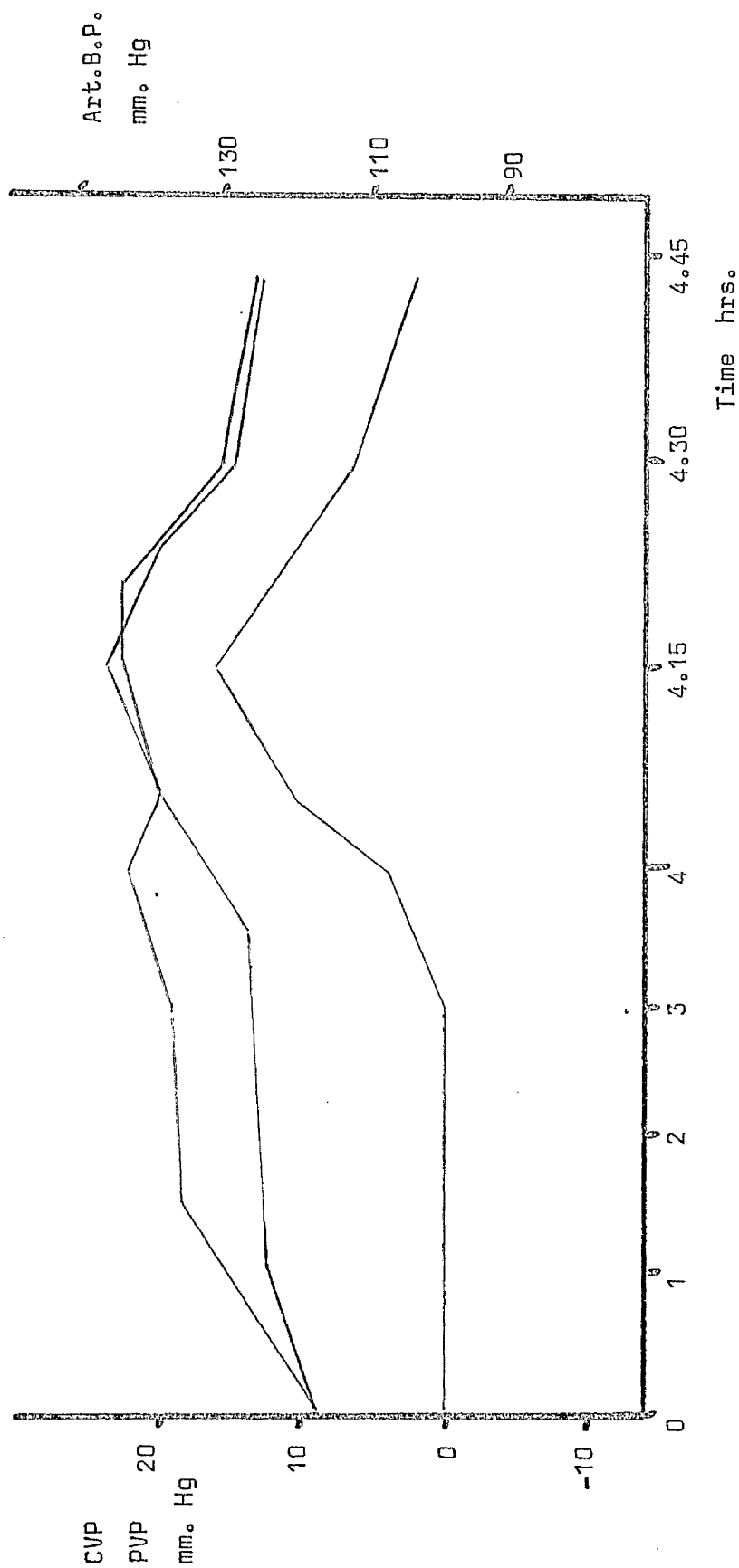
Graph 6 a.

Blood pressures

0.9% Na Cl

20 ml./kg./hr.

CVP —  
PVP —  
Art.B.P. —



Graph 6 b.

Blood pressures

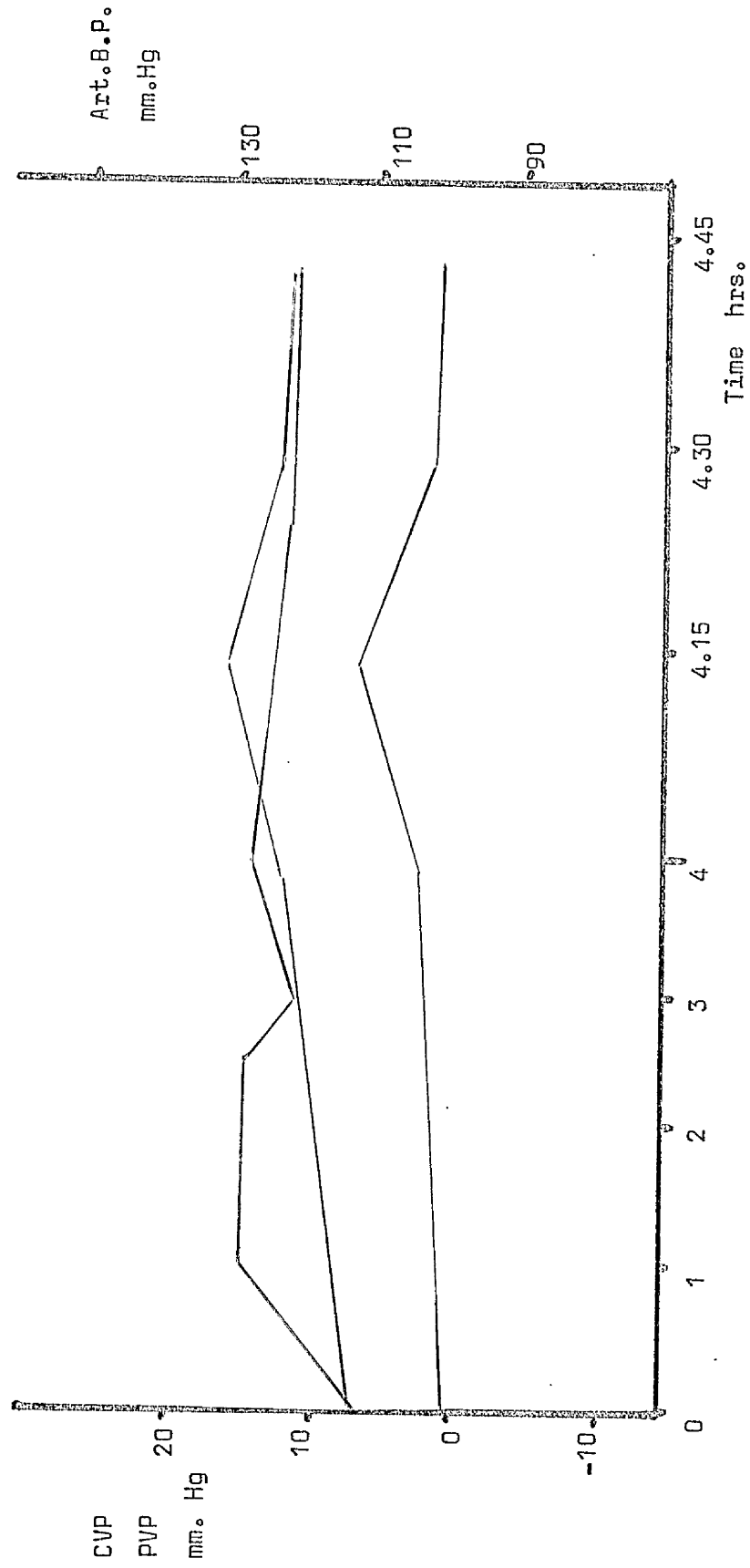
0.9% Na Cl

30 ml./kg./hr.

CVP

PVP

Art.B.P.





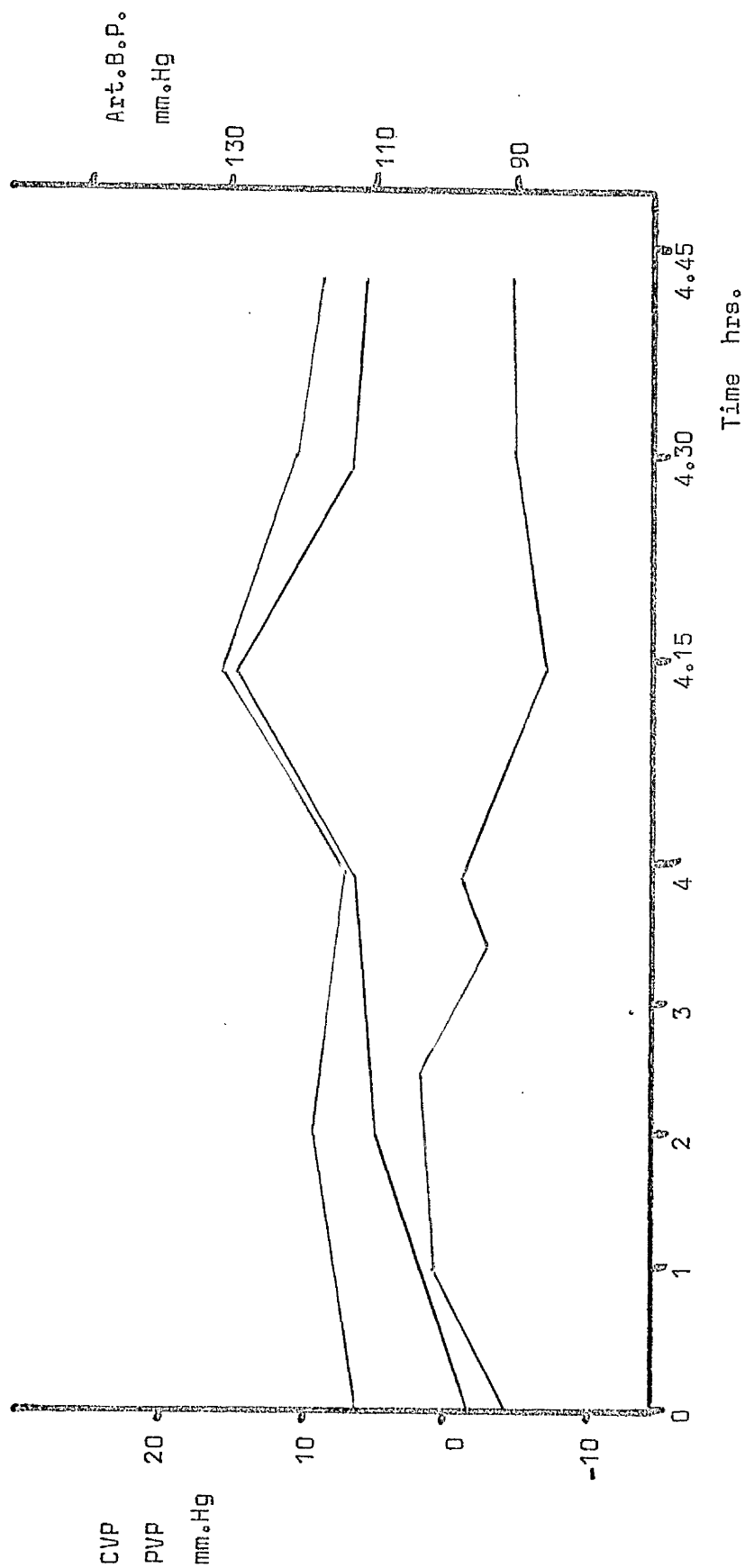
Graph 6 c.

Blood pressures

0.9% Na Cl

40 ml./kg./hr.

CVP —  
PVP —  
Art.B.P. —



Graph 7 a.

Blood pressures

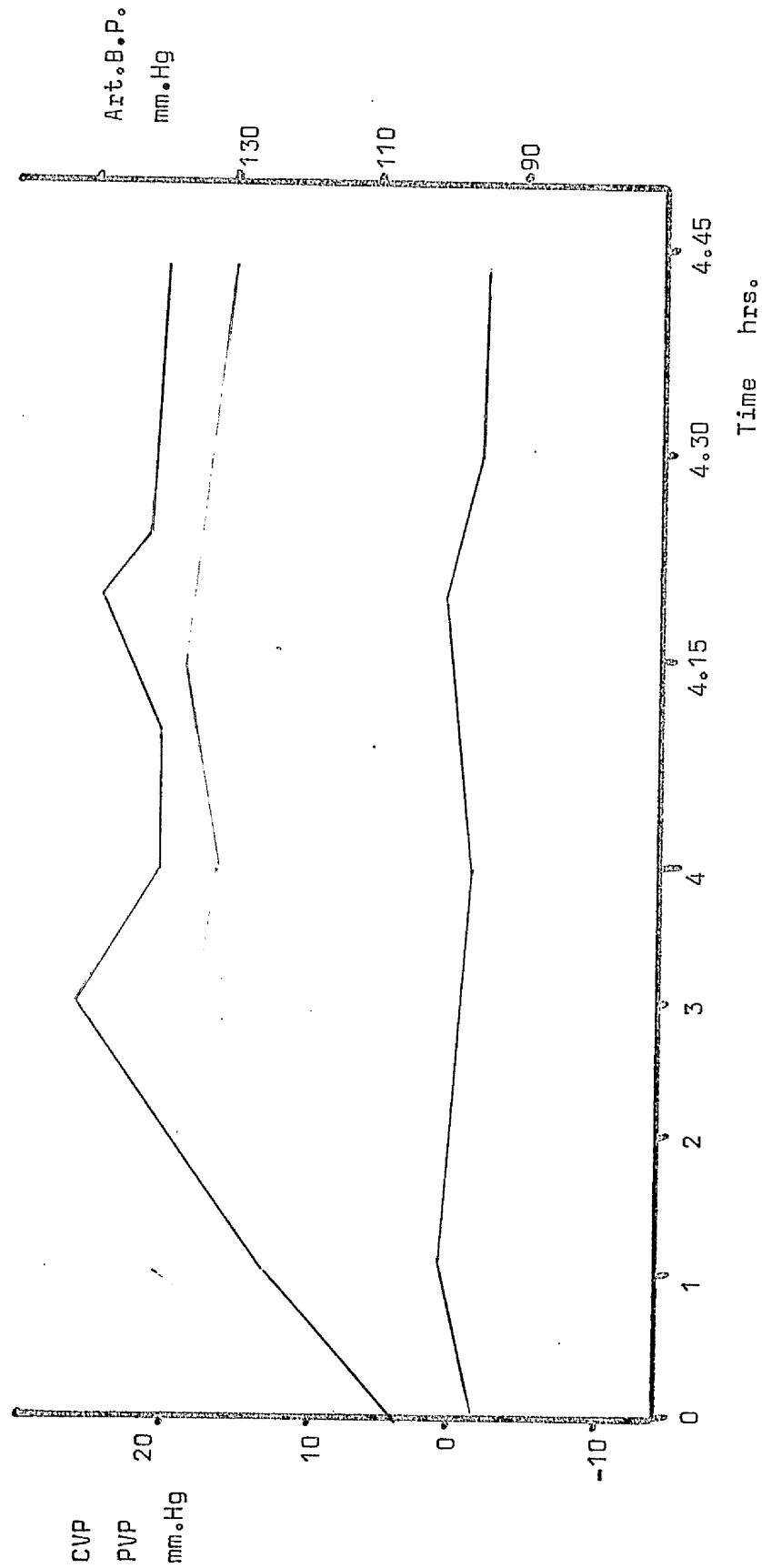
5% Dextrose

20 mL/kg./hr.

CVP

PVP

Art.B.P.



Graph 7 b.

Blood pressures

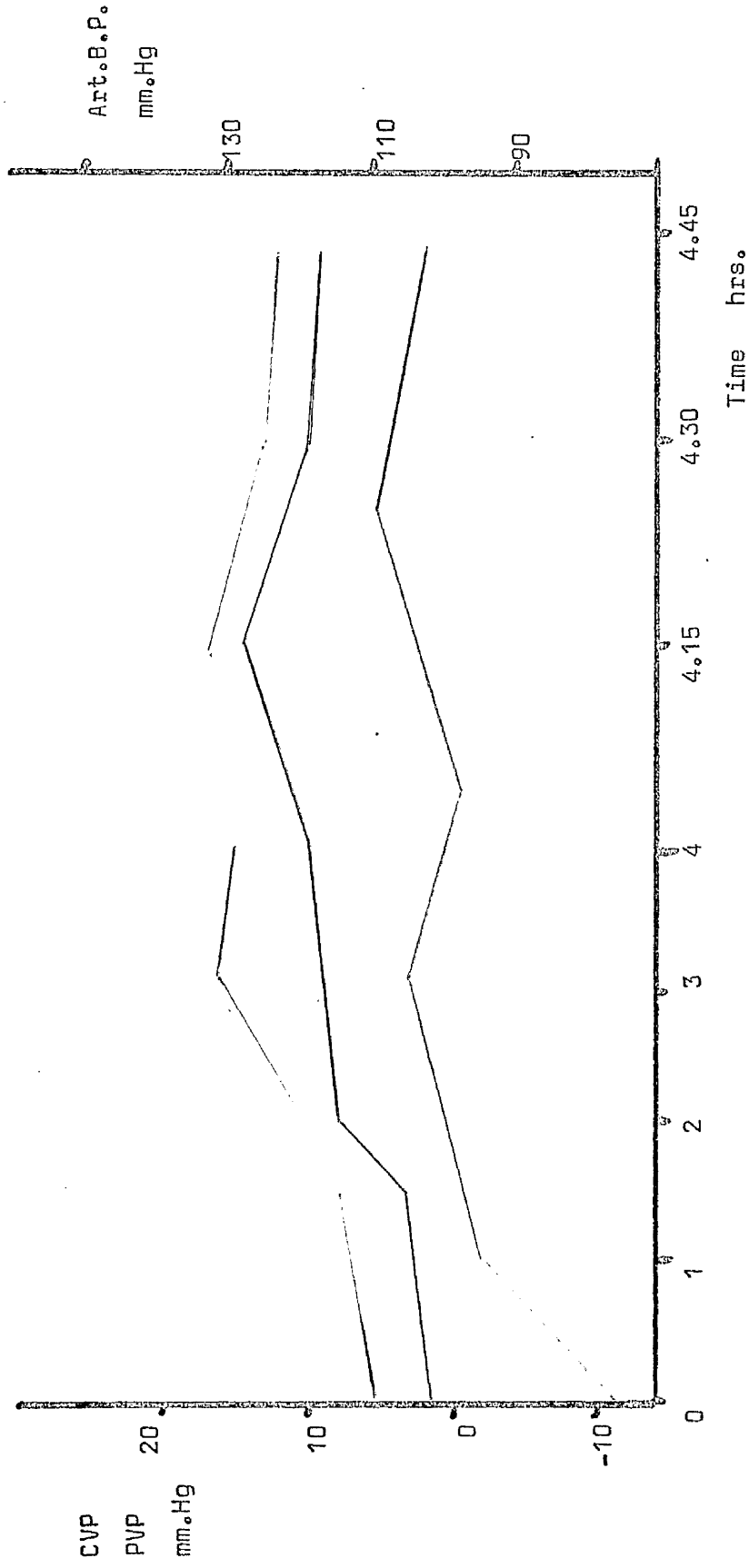
5% Dextrose

30 ml./kg./hr.

CVP

PVP

Art.B.P.



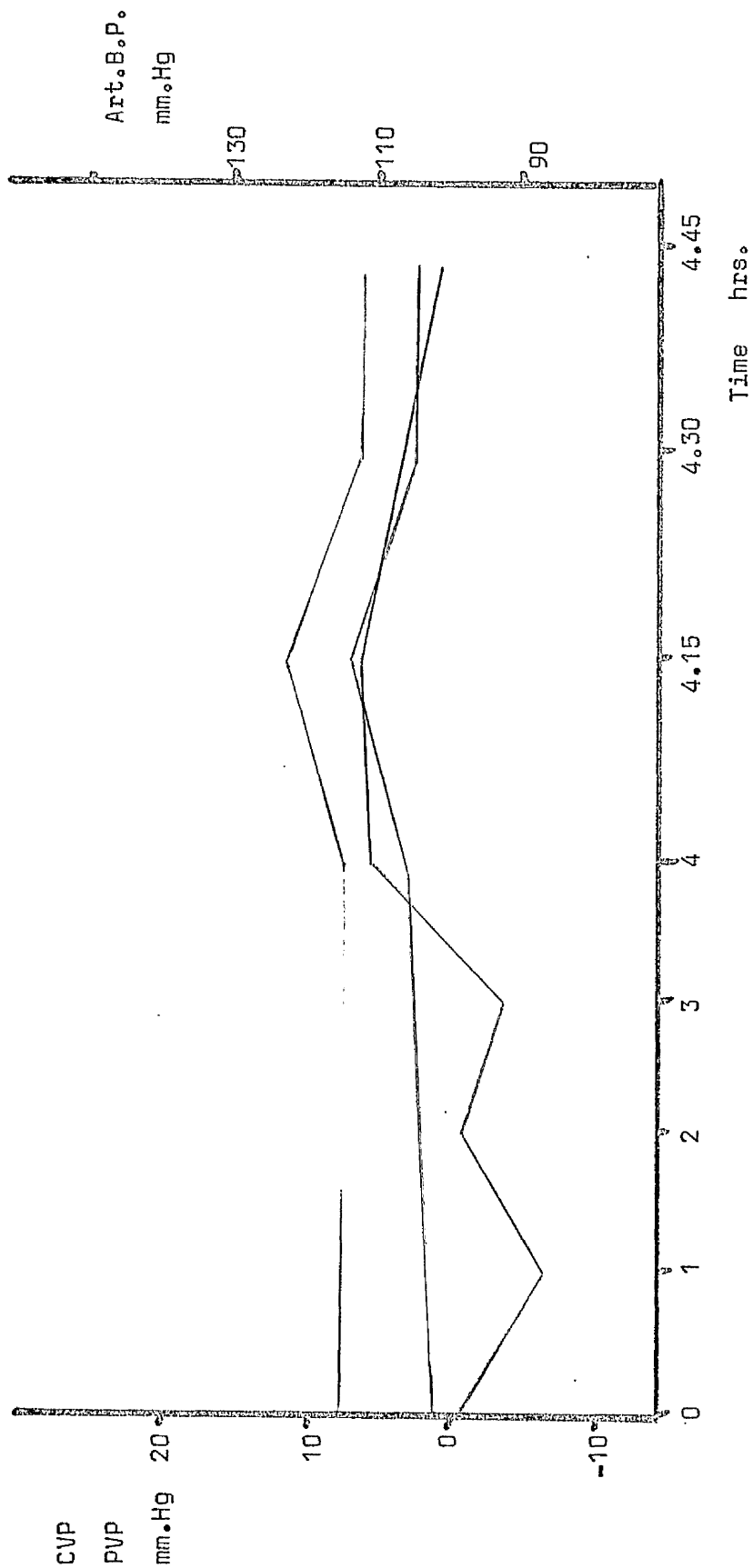
Graph 7 c.

Blood pressures

5% Dextrose

40 ml./kg./hr.

CVP —  
PVP —  
Art.B.P. —



Graph 8 a.

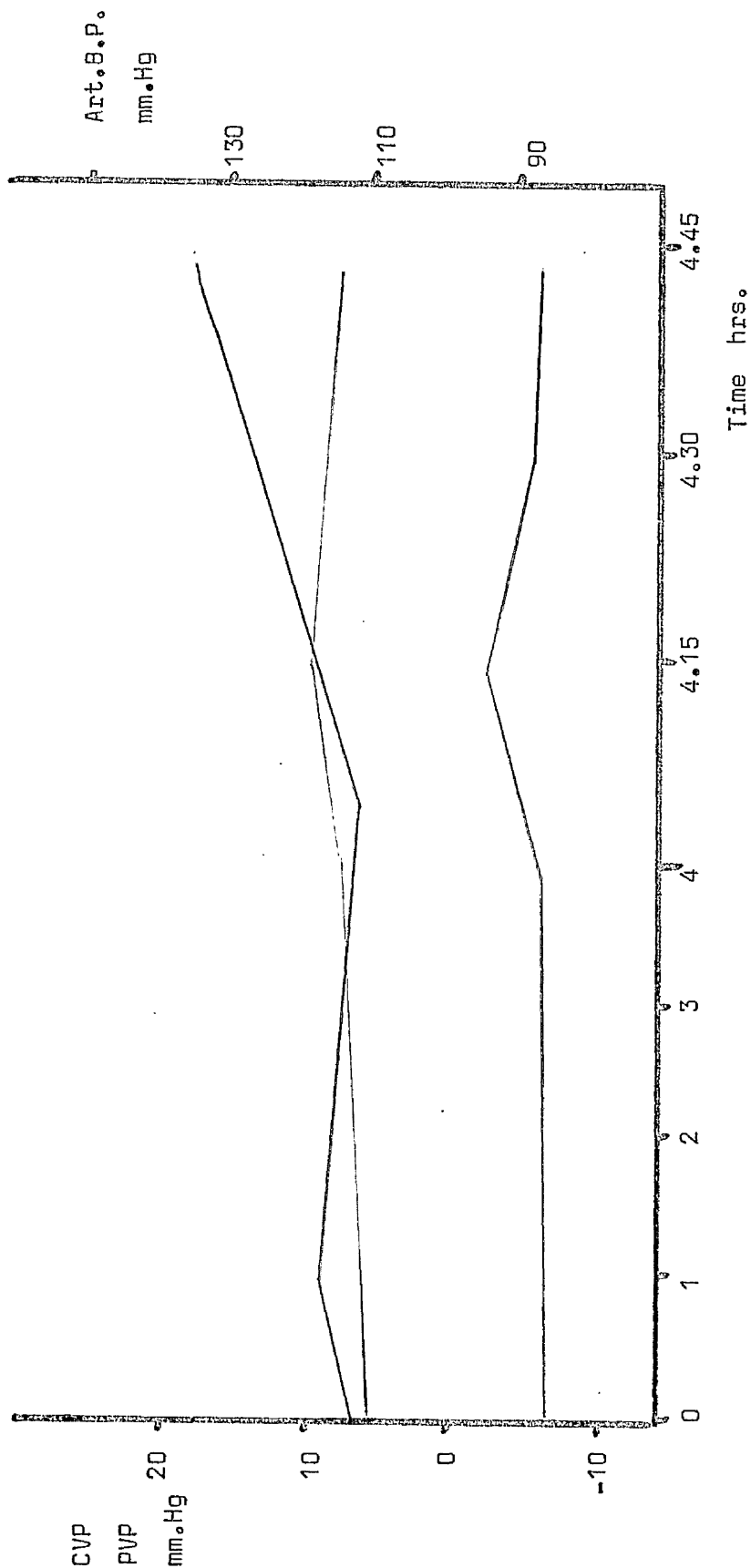
Blood pressures

0.18% Na Cl 4.3% Dextrose 20 ml./kg./hr.

CVP

PVP

Art.B.P.



Graph 8 b.

Blood pressures

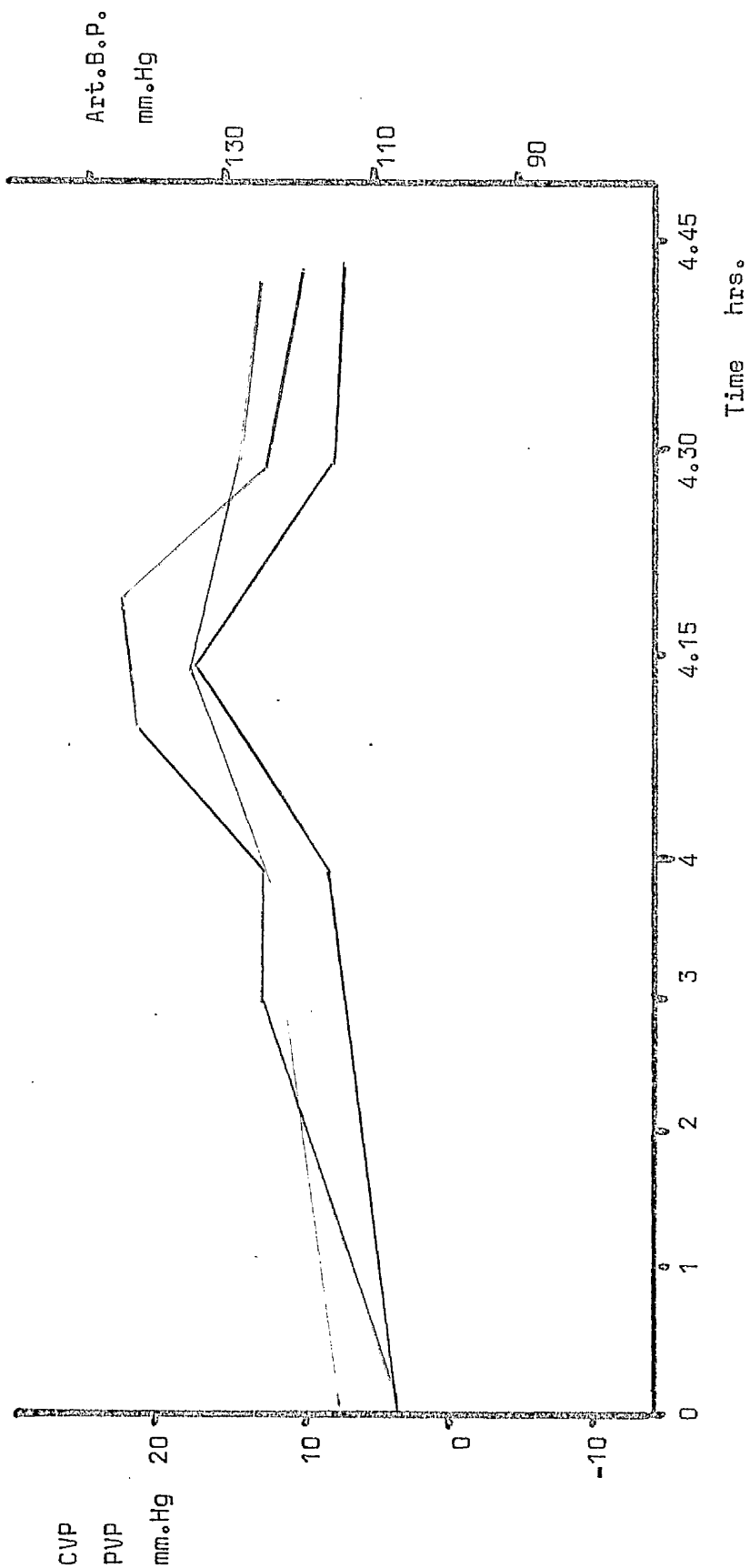
0.18% Na Cl 4.3% Dextrose

30 ml./kg./hr.

CVP

PVP

Art.B.P.



Graph 8 c.

Blood pressures

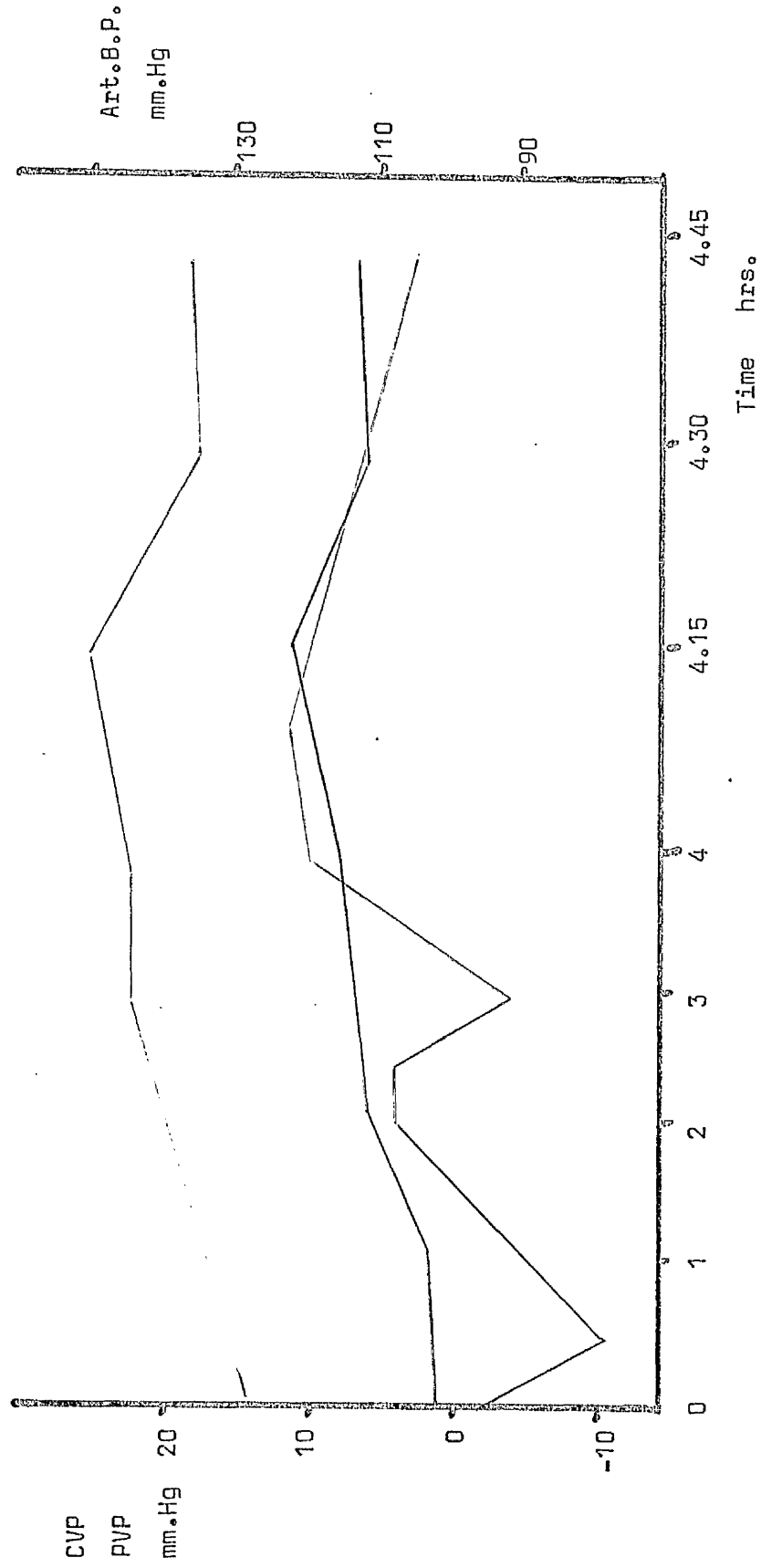
0.18% Na Cl 4.3% Dextrose

40 ml./kg./hr.

CVP

PVP

Art.B.P.



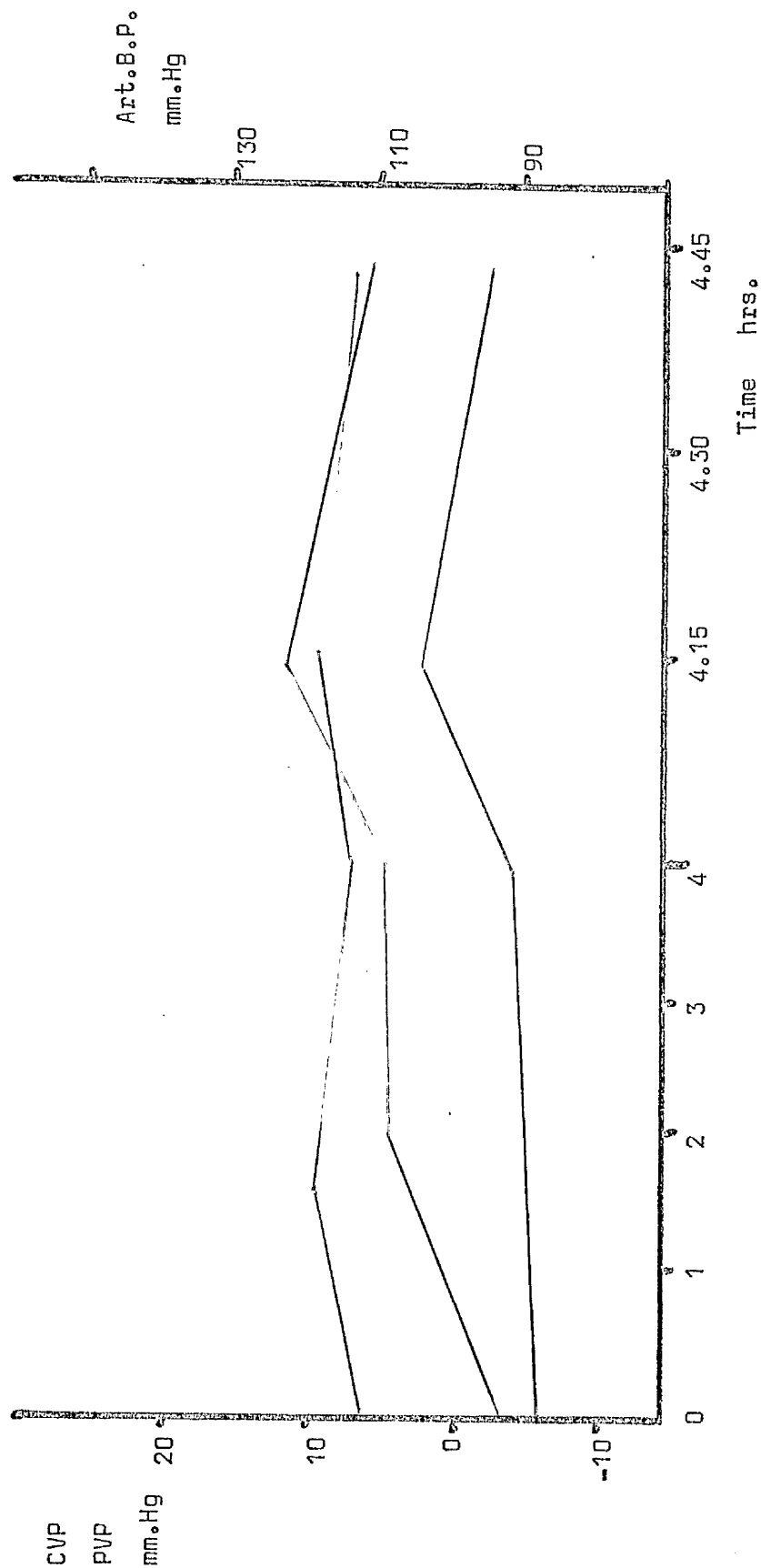
Graph 9 a.

Blood pressures

Ringer Lactate

20 ml./kg./hr.

CVP  
PVP  
Art.B.P.





Graph 9 b.

Blood pressures

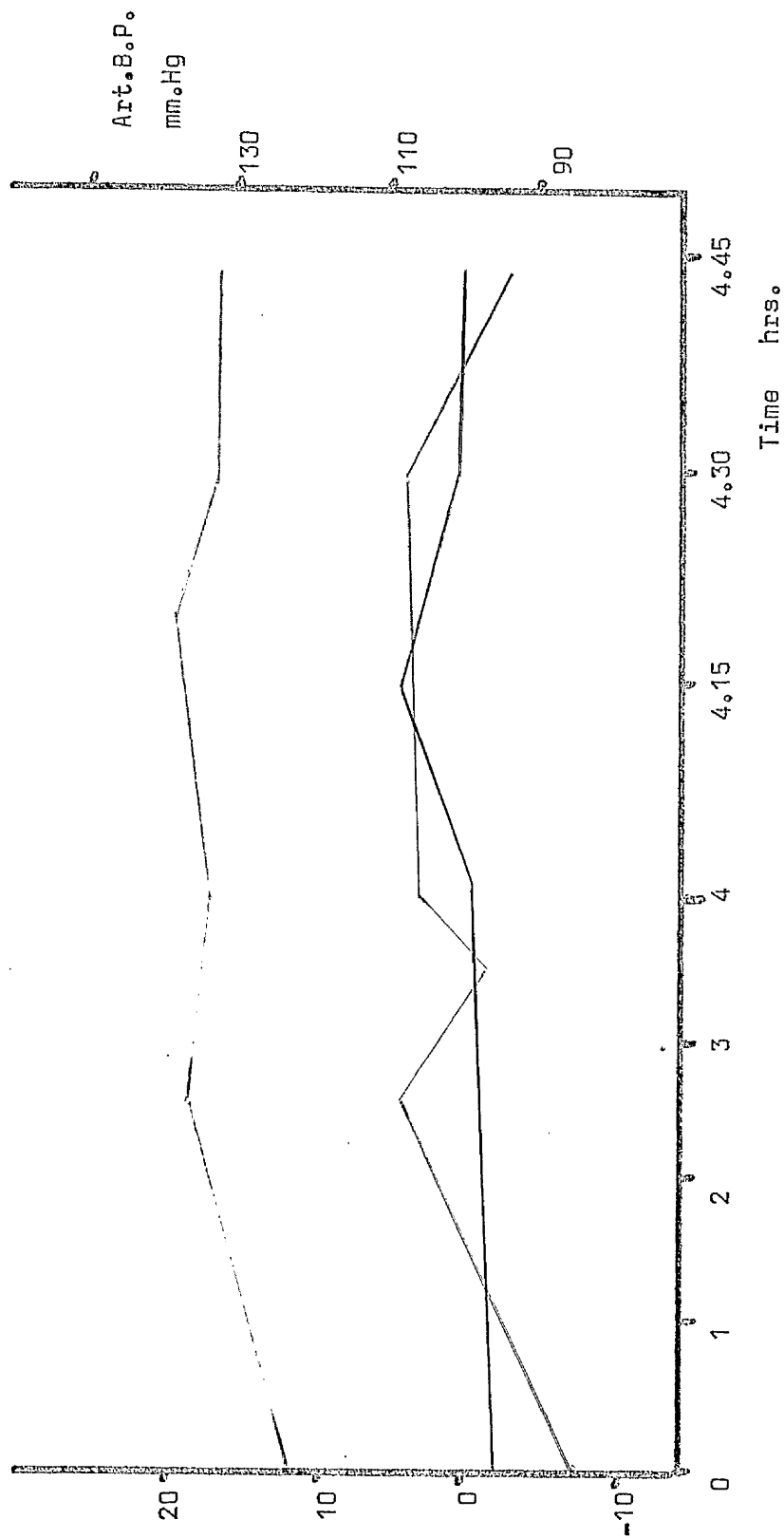
Ringer Lactate

30 ml./kg./hr.

CVP

PVP

Art.B.P.



Graph 9 c.

Blood pressures

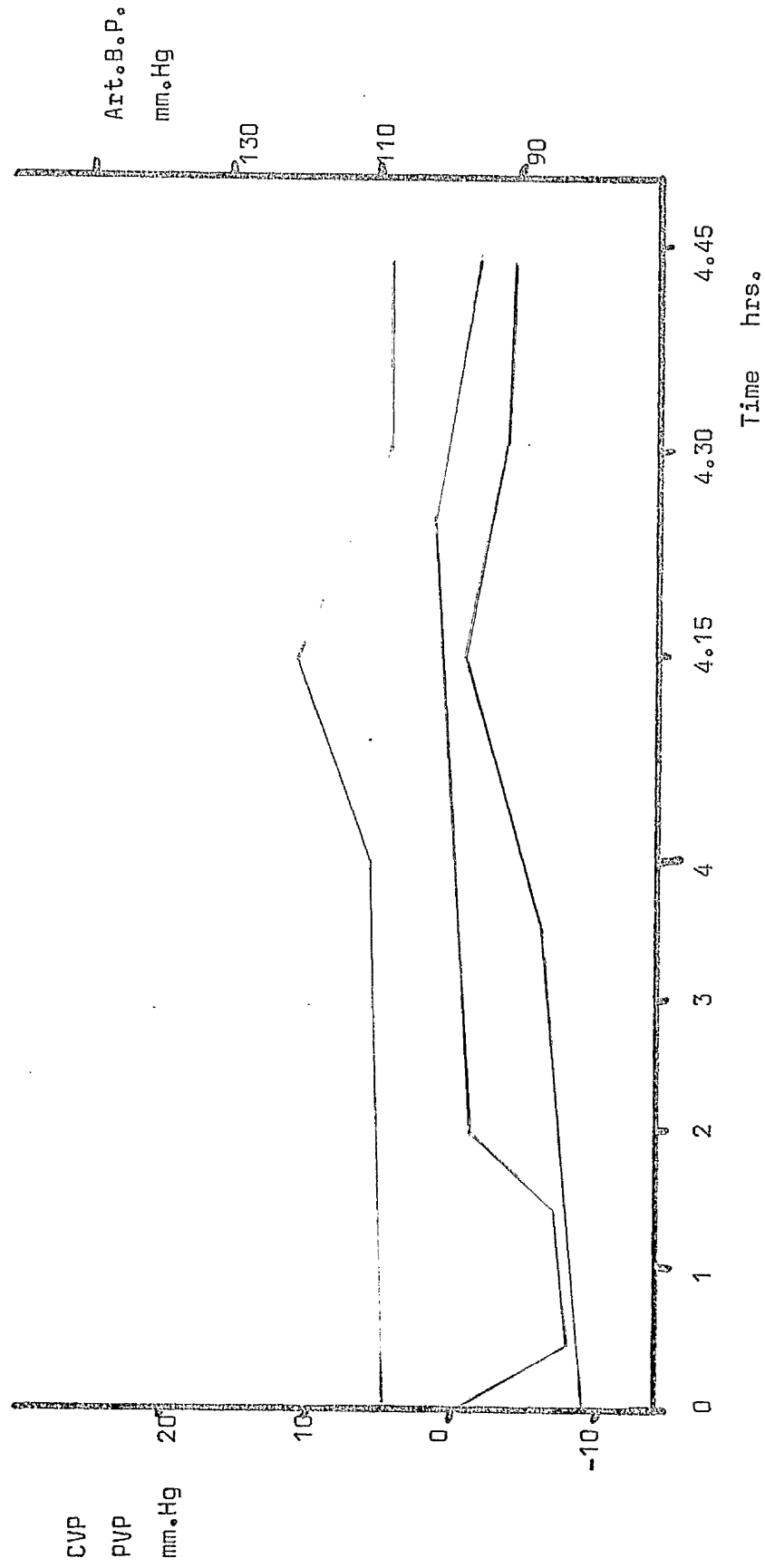
Ringer Lactate

40 ml./kg./hr.

CVP

PVP

Art.B.P.



10 mL/kg./hr.

Dextran 40

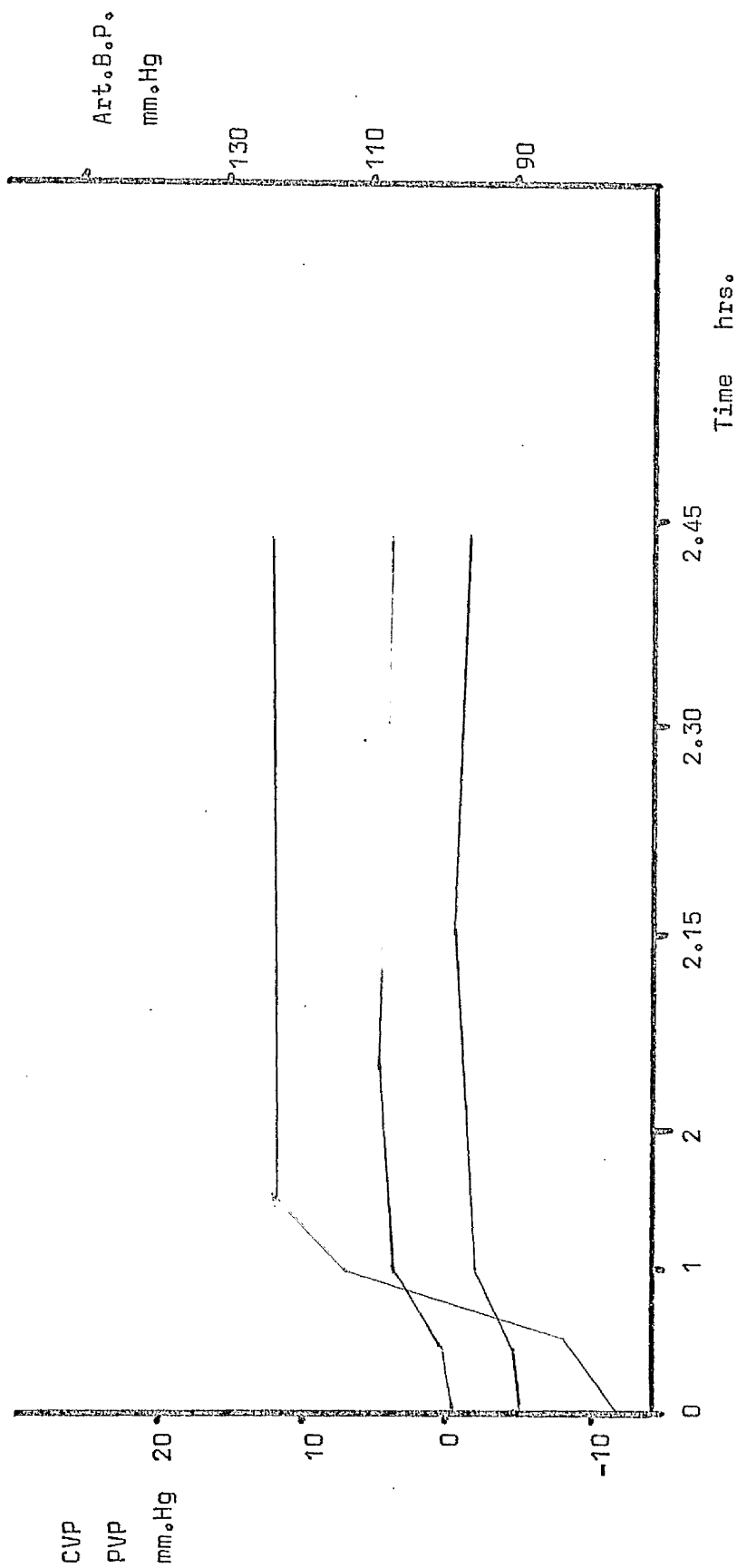
Blood pressures

Graph 10 a.

CVP —

PVP —

Art.B.P. —



20 ml./kg./hr.

Dextran 40

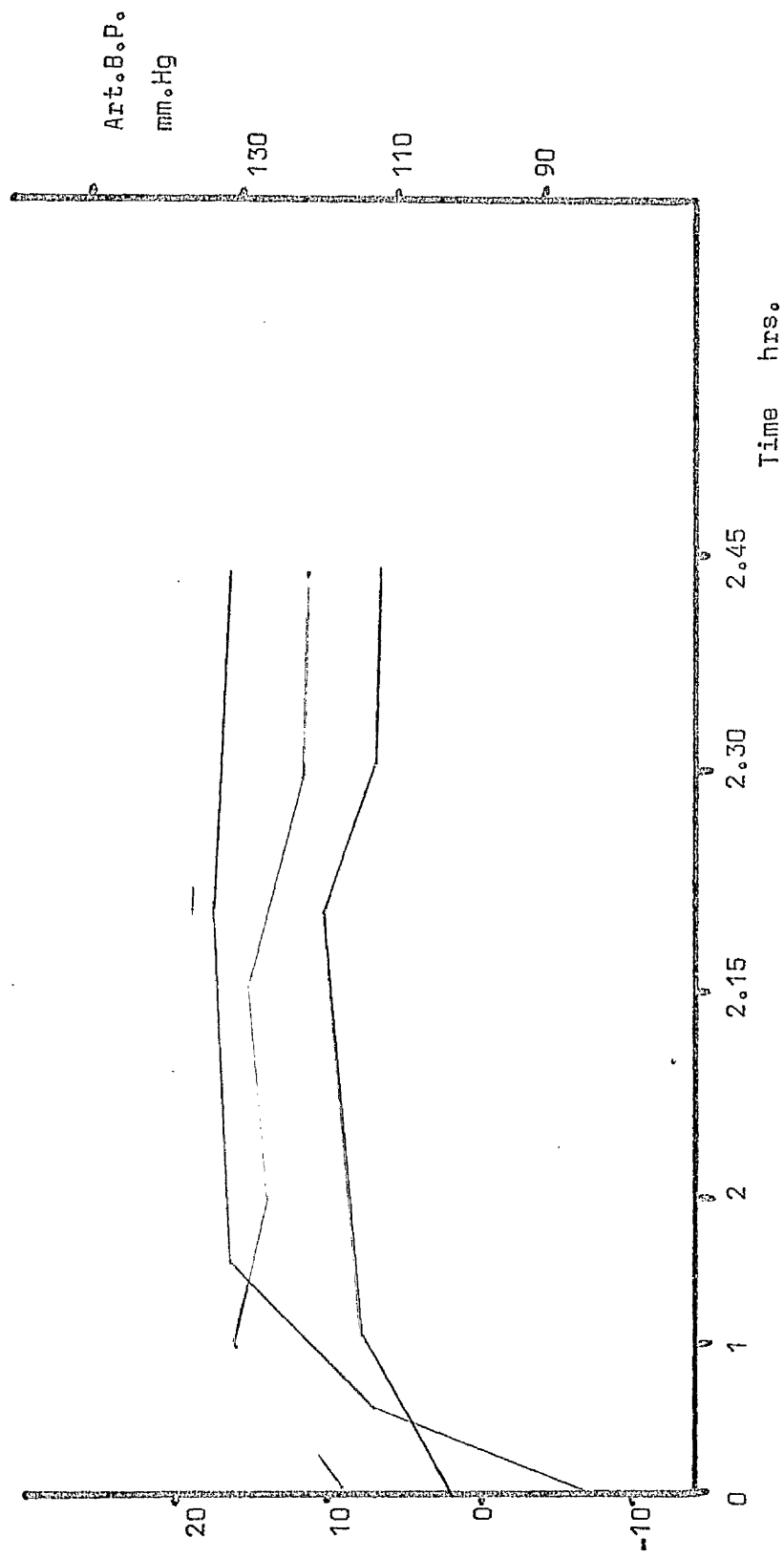
Blood pressures

Graph 10 b.

CVP

PVP

Art.B.P.



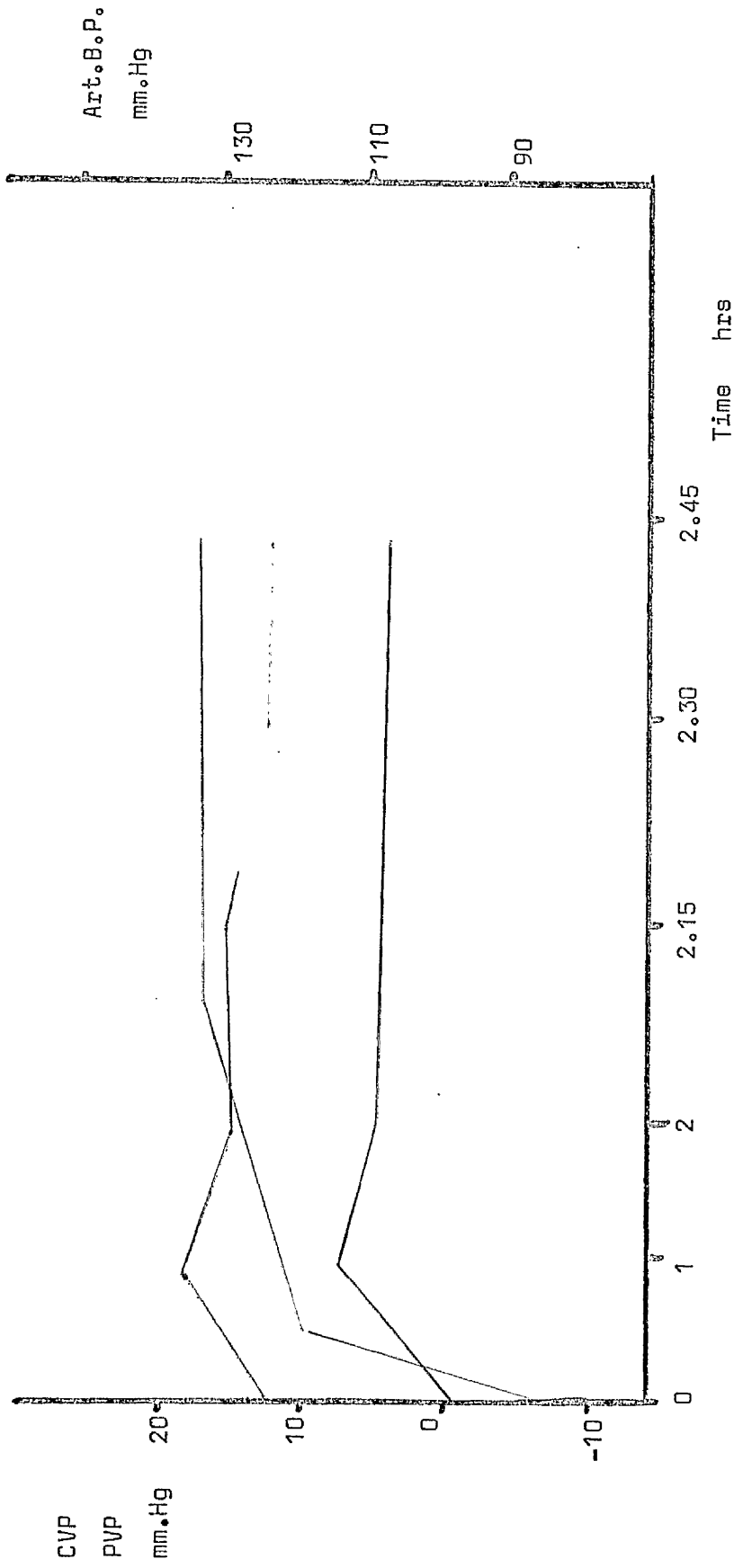
Graph 10 c.

Blood pressures

Dextran 40

30 ml./kg./hr.

CVP —  
PVP —  
Art.B.P. —



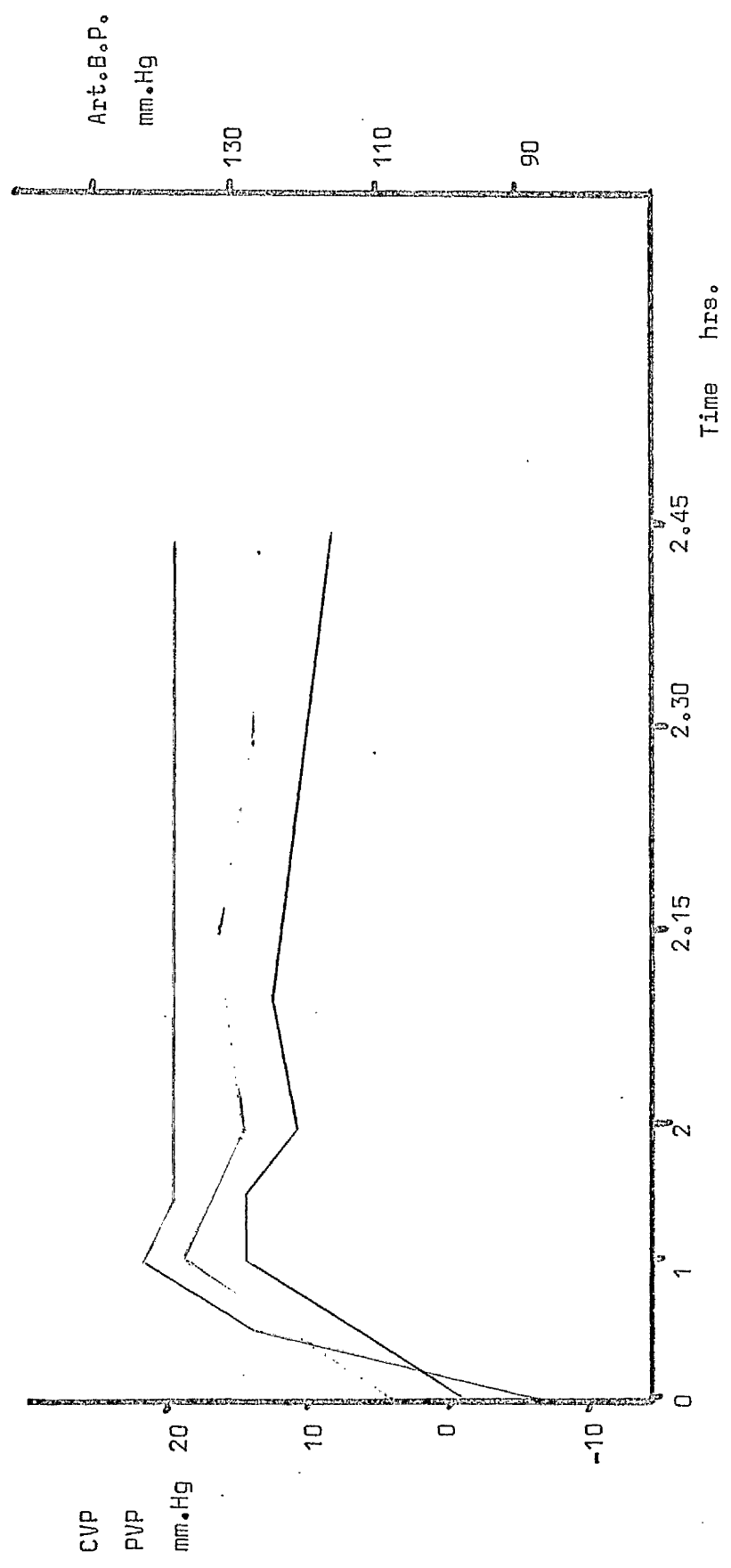
40 mL/kg/hr.

Dextran 40

Blood pressures

Graph 10 d.

CVP  
PVP  
Art.B.P.



### APPENDIX 3

APPENDIX 3.

SURVEY OF FLUID THERAPY USE BY GENERAL PRACTITIONERS - results and graphs.

Results - questions 1 - 7.



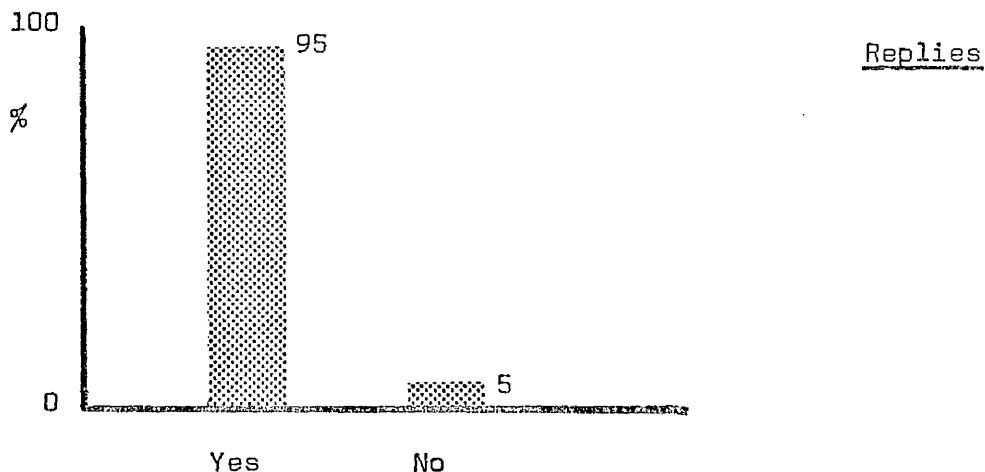
## RESULTS

Of the 125 questionnaires posted in January 1976, 92 replies had been received by the 1st. of March, the closing date for consideration and recording of the results. This gives a percentage return of 73.6 % which was well above the figure anticipated. Over 75 % of the replies were received within the first fourteen days of dispatch. and this may indicate some general interest in this subject.

Since two of the 92 replies came from English practices, it was presumed that mail had been forwarded to practitioners who had moved south of the border. These two results were discounted from the final analysis to maintain a purely Scottish assessment.

Question 1. Do you treat dehydrated or shocked patients by the parenteral administration of fluids ?

Of the 90 general practitioners in Scotland who replied to the survey, 86 answered in the affirmative, the other four being replies in the negative. This answer is expressed in a percentage histogram.



Most practitioners do administer fluids parenterally, but the following results indicate a wide variation in the number of cases treated by individual practices each week, and also a variation in the types of fluid used.

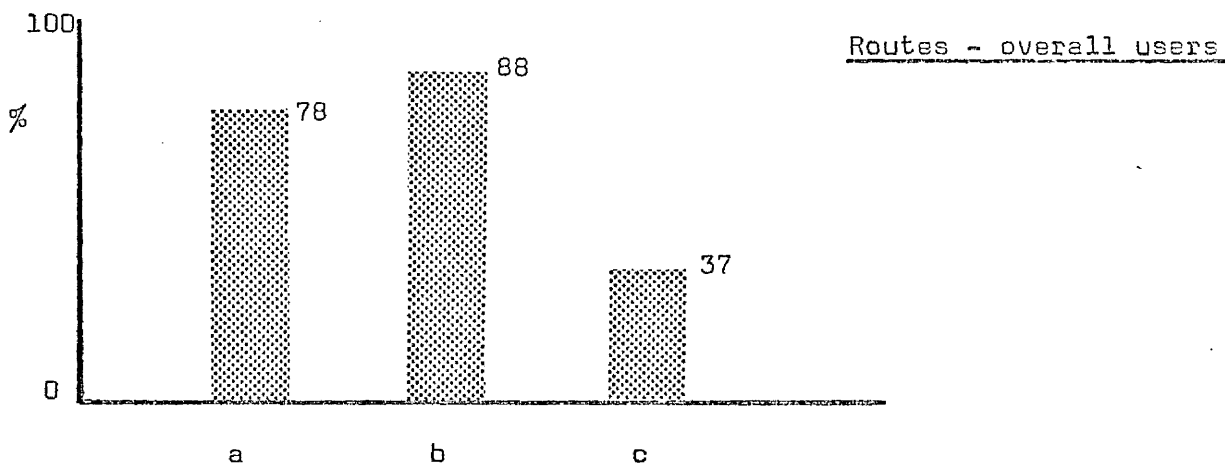
All further percentages are calculated from the 86 affirmative answers to the first question, except where specifically indicated in the text.

Question 2.      Indicate which routes are used.

Answers were straightforward and no practitioner used " other " routes.

- |    |                 |    |               |
|----|-----------------|----|---------------|
| a) | Intravenous     | 67 | practitioners |
| b) | Subcutaneous    | 76 | "             |
| c) | Intraperitoneal | 32 | "             |

This is represented as a percentage histogram.

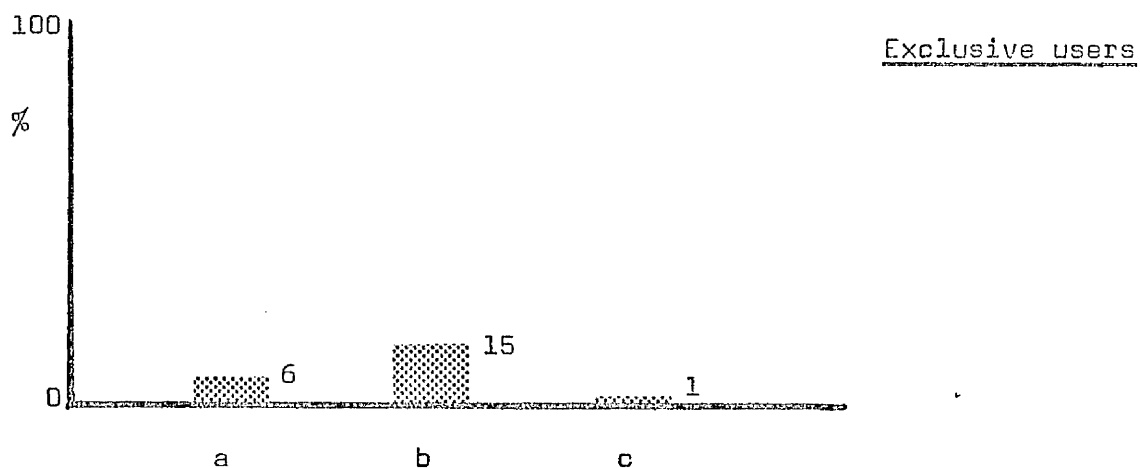


From/

From the answers to this question it was intended to discover whether practitioners consistently used one route or varied their methods of fluid administration. Firstly there were considered those practitioners who used one route exclusively.

a)	Intravenous	5 practitioners
b)	Subcutaneous	13 "
c)	Intraperitoneal	1 practitioner

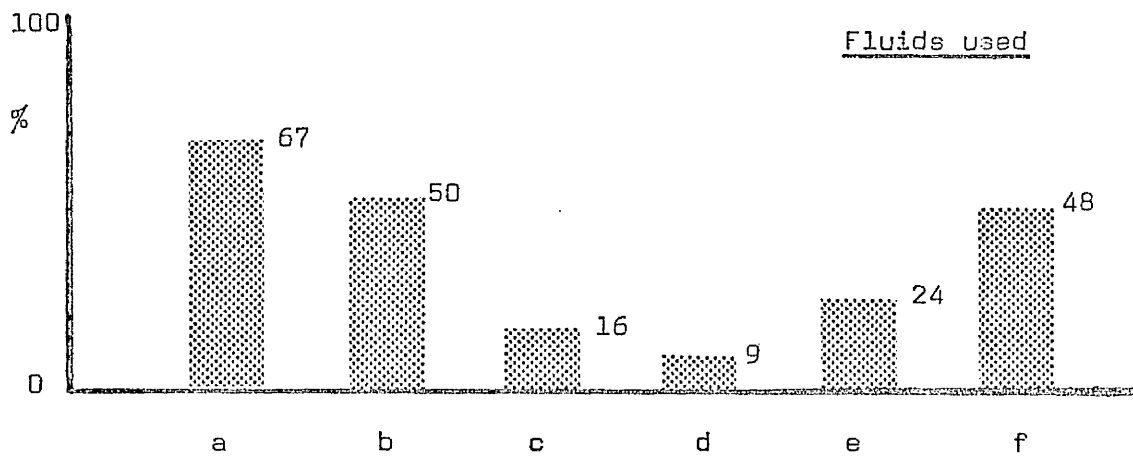
This is represented in a percentage histogram.



The number of practitioners using two or three routes were then considered, and 34 practitioners use the intravenous and subcutaneous routes, 5 practitioners use the subcutaneous and intraperitoneal routes, 4 practitioners use the intravenous and intraperitoneal routes and 22 practitioners use all three routes.

a)	NaCl (0.9%)	58	practitioners
b)	Dextrose (5%)	43	"
c)	Ringer Lactate	14	"
d)	Dextran	8	"
e)	Whole Blood	21	"
f)	Other	41	"

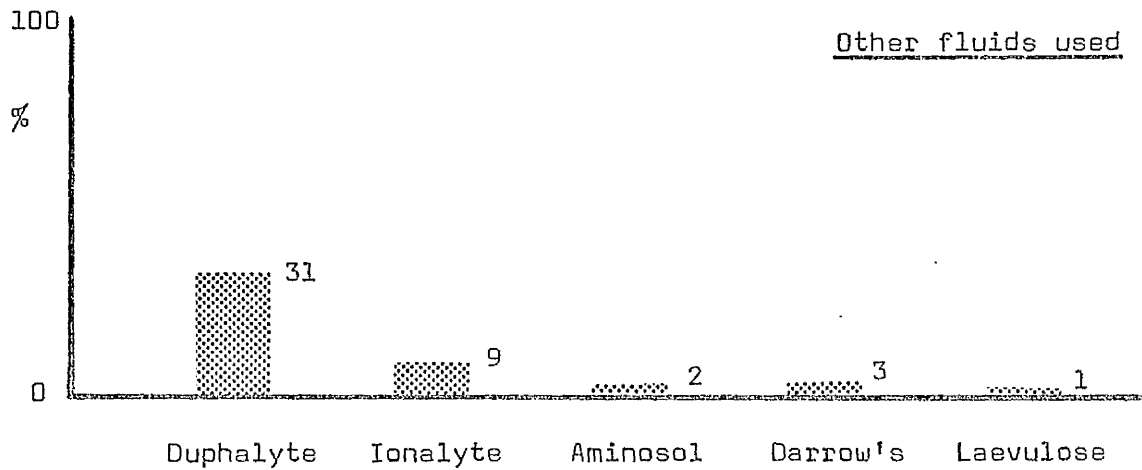
This is represented in a percentage histogram.



The answers to part f) of the question were then analysed to determine what other fluids were being used in general practice. The result is given in the following list.

"Duphalyte"	27	practitioners
"Ionalyte	8	"
Darrow's Solution	3	"
"Aminosol"	2	"
Laevulose	1	practitioner

This is illustrated in a percentage histogram.

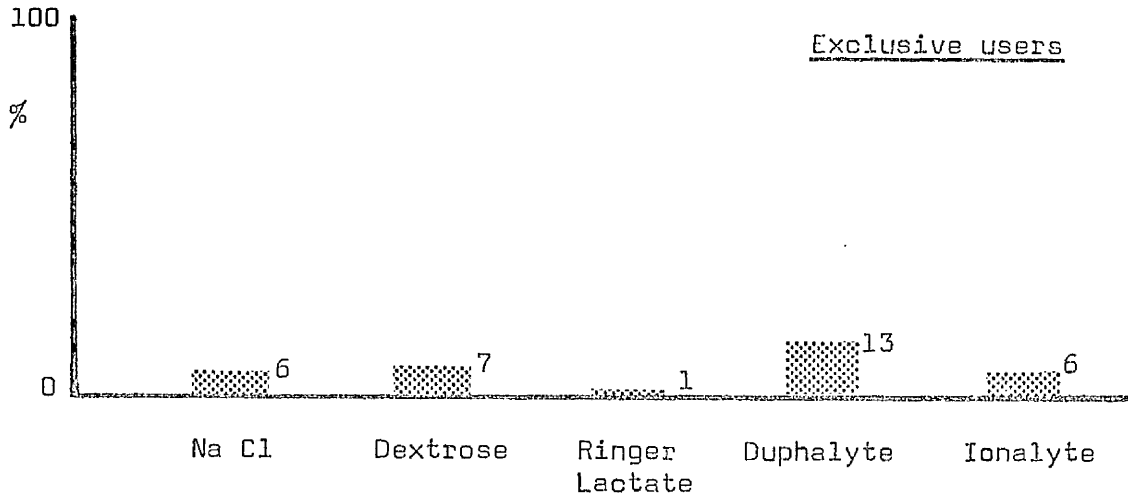


The above information enables an evaluation of the awareness of general practitioners of the available fluids and the use made of some of the newer composite infusions. The fluids listed were reckoned to be those most commonly used parenterally, and all except whole blood are readily available from veterinary suppliers. Some practitioners use a wide range of fluids, whereas others use only a few, and this is further analysed.

Those practitioners using only one fluid exclusively were considered.

a)	NaCl (0.9%)	6 practitioners
b)	Dextrose (5%)	7 "
c)	Ringer Lactate	1 practitioner
f)	Duphalyte	11 practitioners
	Ionalyte	6 "

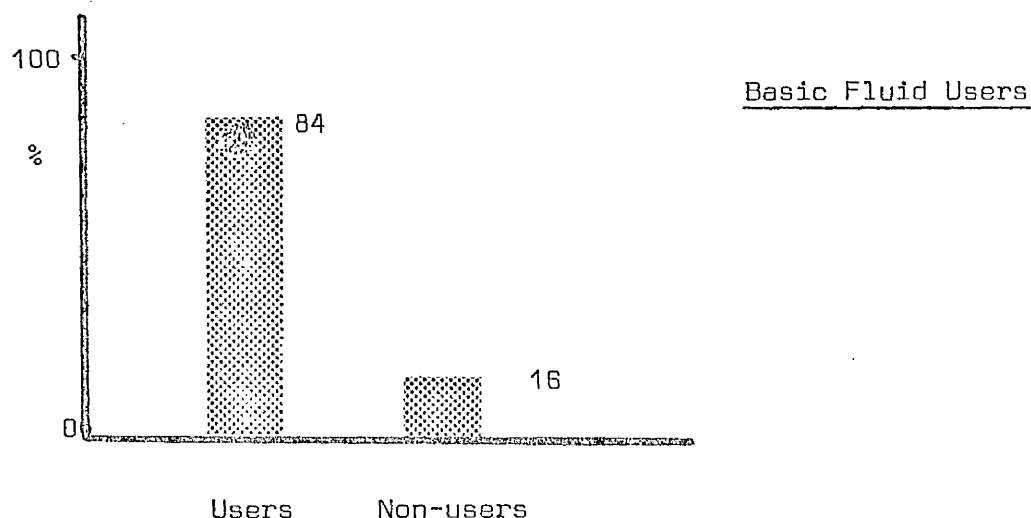
The other fluids attracted no exclusive users. The results are illustrated in a percentage histogram.



Only 3 of the 86 practitioners used all five listed fluids.

It is of interest to note the number of practitioners who do not use basic crystalloid solutions in the treatment of patients. These are basic fluids which can be administered with ease and safety to all suitable cases. They provide the foundation for fluid infusion and most other fluids are adjuncts which make possible fuller supportive parenteral therapy.

72 practitioners use these basic fluids and 14 do not, and this is illustrated in a percentage histogram.



In addition there are the users of "Duphalyte" and "Ionalyte" which contain electrolytes as well as other substances, and both these preparations have exclusive users. When administered in the recommended dosage, these solutions may not provide the recipient's total fluid requirements. Darrow's solution contains extra potassium as well as other electrolytes, but has no exclusive users in this survey.

Only 8 practitioners use the plasma substitute and expander Dextran and 21 practitioners use whole blood transfusion. Both these fluids have use in cases of massive haemorrhage where the circulating volume requires restoration.

Two practitioners use "Aminosol" which is presented in various forms, but all should be administered intravenously along with a source of energy such as lipid or dextrose to provide the necessary calories to fully utilise the amino acids. "Duphalyte" also contains amino acids and needs calories to be of complete use to the recipient, /

recipient.

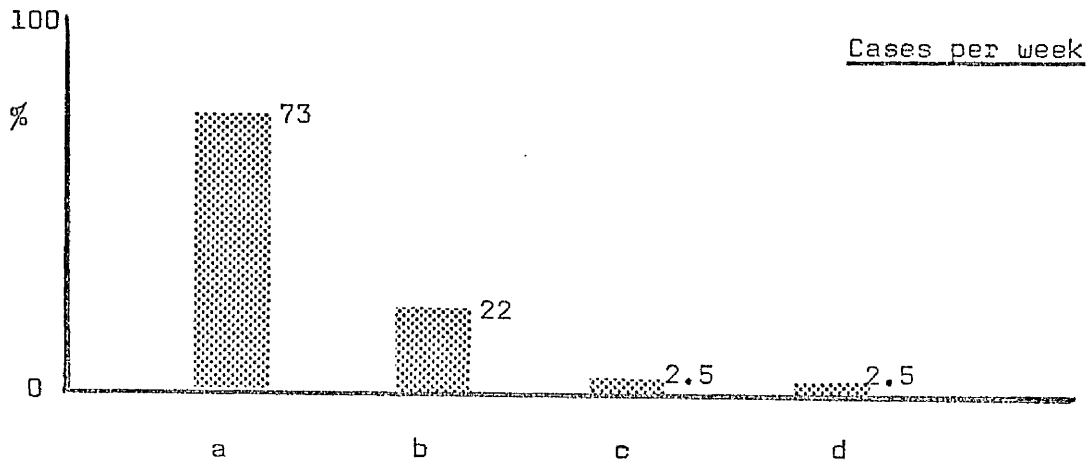
Question 4.      Approximately how many dehydrated or shocked patients  
do you see ?

A weekly number was chosen rather than a monthly or yearly figure because human memory tends to more accurate in the short term. An additional answer - less than one case per week - would have been useful, but such information as was forwarded by the practitioners was taken into account in the subsequent analysis.

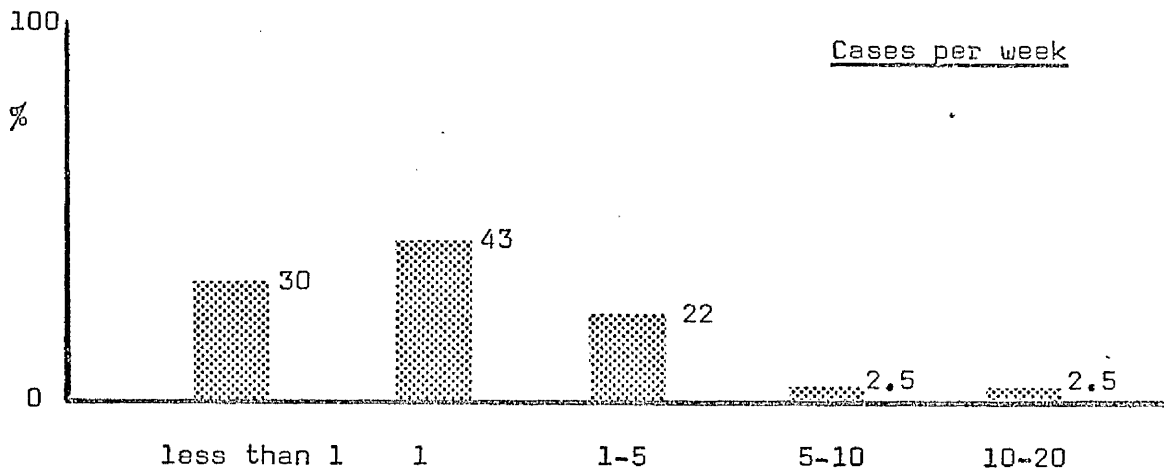
a)	1 case per week	73 practitioners
b)	1-5 cases per week	19      "
c)	5-10 cases per week	2      "
d)	10-20 cases per week	2      "

This is represented in a percentage histogram.





Taking into account the large number of practitioners seeing less than one case per week, the histogram should appear more like this.



Thus 95 % of practitioners see less than 5 cases per week of dehydration or shock and 73 % see one or less case per week.

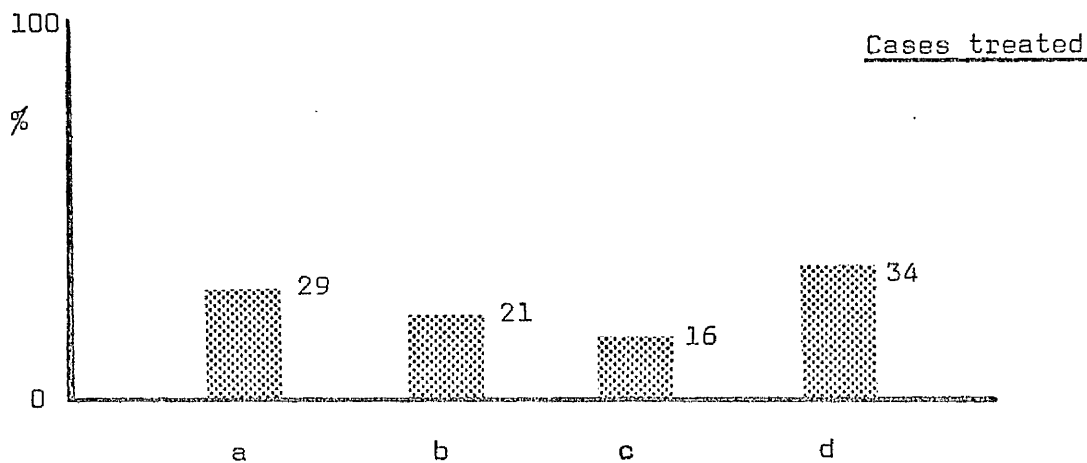
In the following question it is interesting to note how many of the cases of dehydration and shock are treated by practitioners in general practice. Most accident cases are quite rightly left until they have they have recovered from the "shock" themselves. Even though, there may be a case for treatment in some of these patients especially when blood has been lost or the animals are anorexic along with a reduced water intake, Treatment can improve the recovery or at least increase the speed of recovery in many instances.

Question 5.      What proportion of these receive fluids parenterally ?

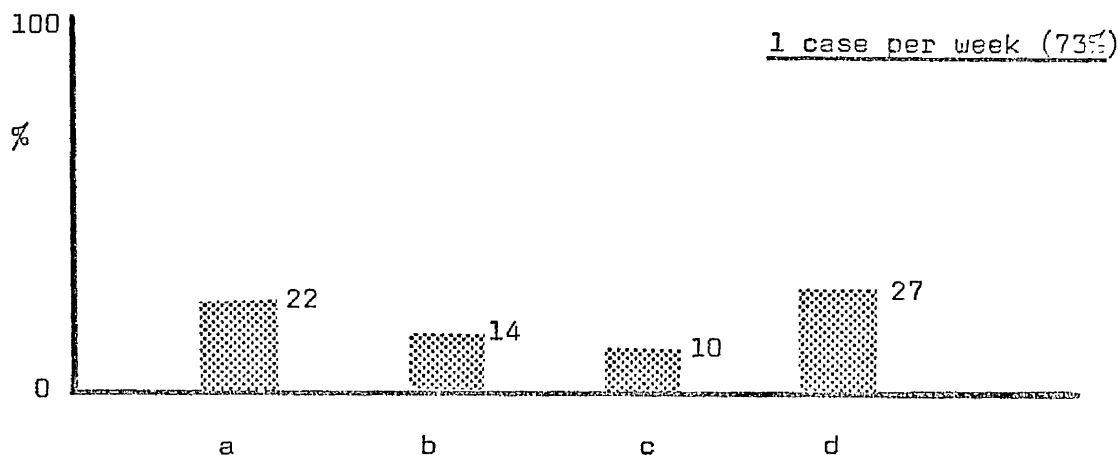
This question was intended to elicit the emphasis placed by practitioners upon intensive therapy. In general practice some cases need fluid therapy, but a large proportion of these are denied treatment and despite the initial deficit, may attain their normal body fluid balance by oral consumption. What happens to others is pure speculation.

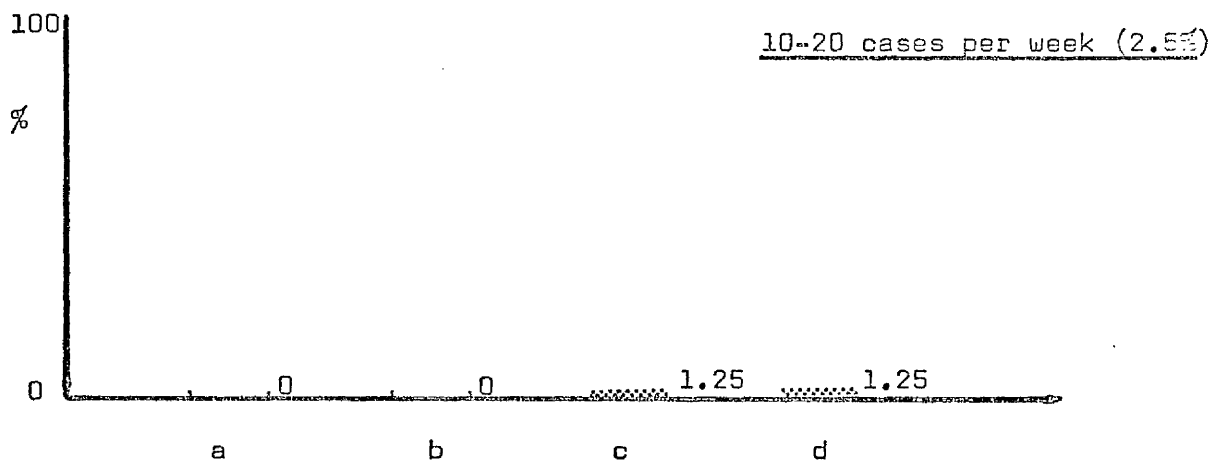
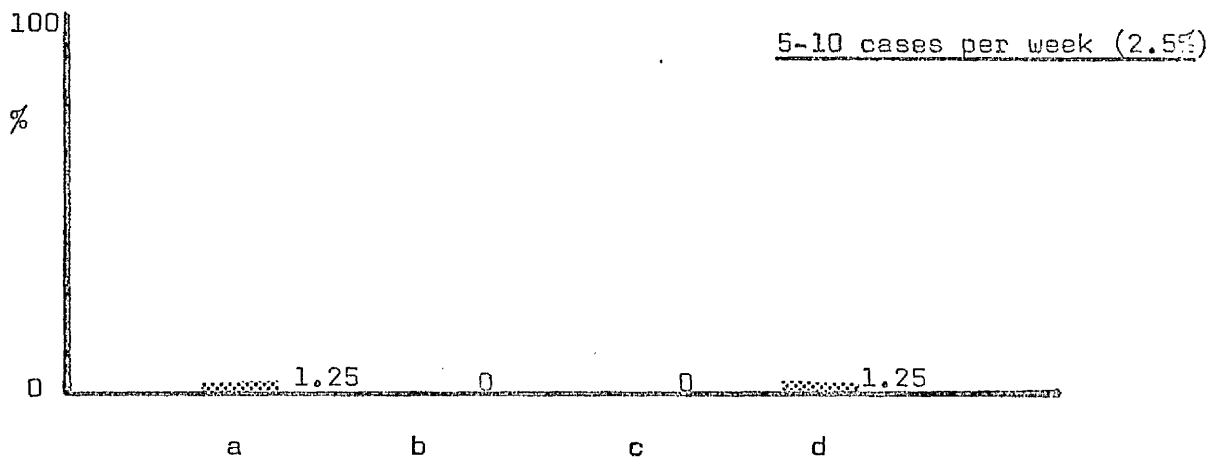
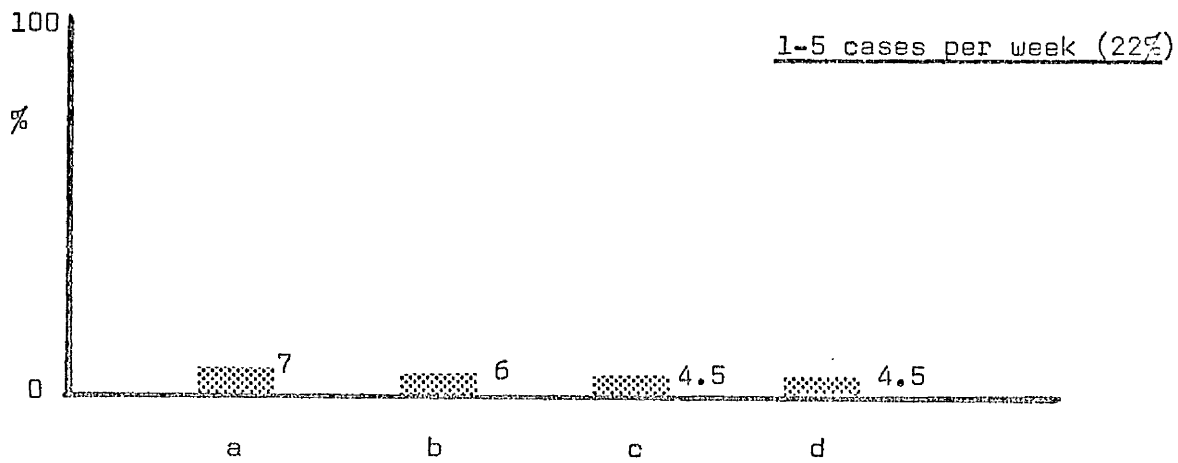
a)	0-25 %	26 practitioners
b)	25-50 %	17            "
c)	50-75 %	14            "
d)	75-100 %	29            "

This is represented in a percentage histogram.



It is interesting to note that 50 % of practitioners treat less than half of their cases and 50 % treat more than half of their cases. Further analysis of these two groups showed that there was little difference between practices seeing a large number of cases and those seeing less than one case per week. Of the group of practitioners treating less than 25 % of cases, 19 out of 26 (73%) saw one case or less per week, and of the 29 practitioners treating over 75 % of cases, only two saw more than five cases per week. The following histograms show the figures for the four groups according to the number of cases seen, as a percentage of practitioners using fluids.





No single group of practitioners treats either all or none of their cases, and in fact the spread is fairly even over all practitioners.

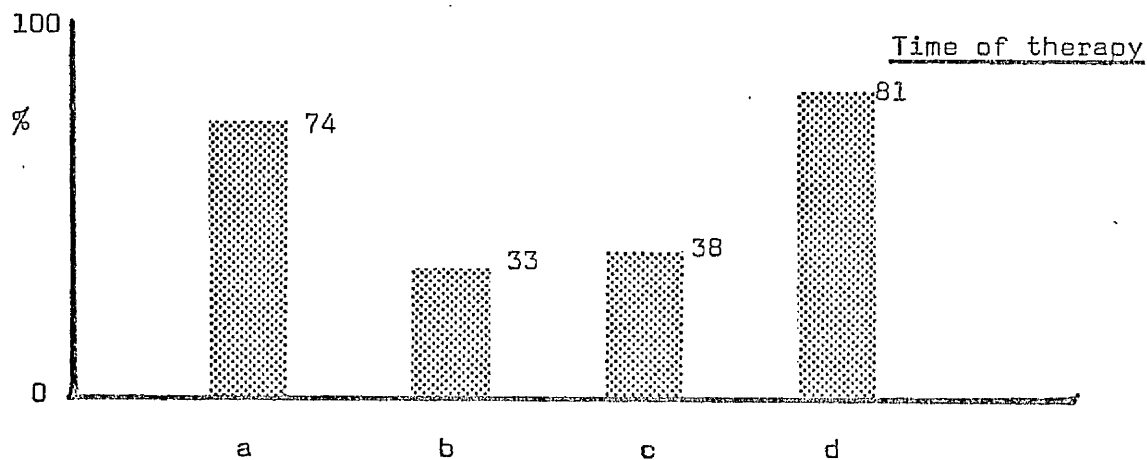
Question 6.      When do you administer fluids ?

This question covers the four most likely times of fluid administration. The answers revealed that most practitioners treat patients on admission and post-operatively.

The results were,

a)	On admission	64	practitioners
b)	Pre-operatively	28	"
c)	During surgery	33	"
d)	Post-operatively	70	"

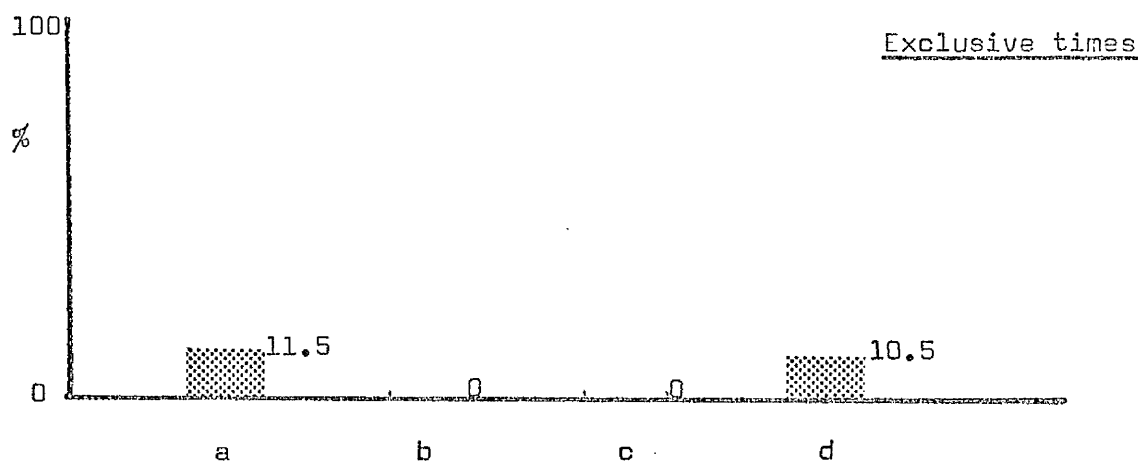
This is illustrated in a percentage histogram.



Most practitioners treat cases during several phases. It was thought relevant to establish if some practitioners gave fluids at only one stage during treatment, and two groups were noted.

- |    |                  |                  |
|----|------------------|------------------|
| a) | On admission     | 10 practitioners |
| b) | Post-operatively | 9 "              |

This is seen in a histogram.



Most practitioners, however, indicated that they administer fluids during more than one phase and 12 of these administered fluid during all four phases. The combinations of other phases are numerous and from a brief analysis it was found that 21 practitioners use fluids on admission and postoperatively only, and 6 practitioners use fluids only at the time of surgery and post-operatively. The other combinations each involve no more than two practitioners.

Question 7./

Question 7. How do you assess the degree of dehydration or shock ?

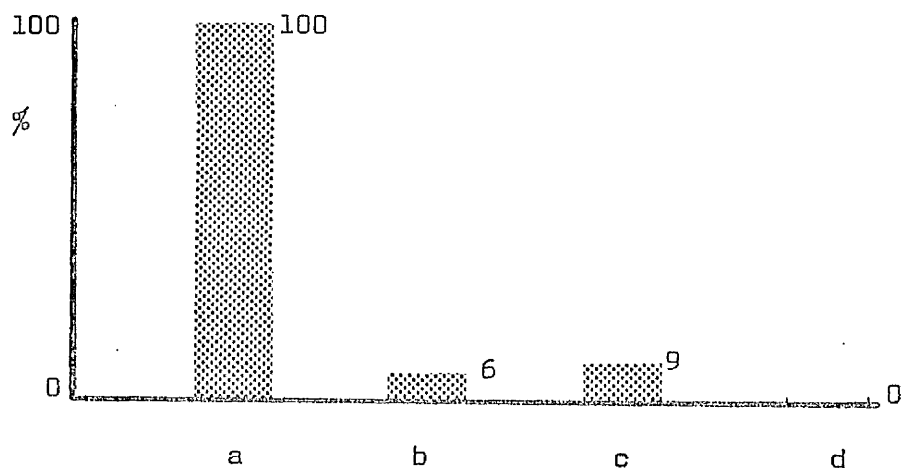
This question was designed to determine how many practices had facilities for carrying out the two basic tests listed and to enquire what other tests or methods of assessment are in use. It was expected that all practitioners would assess cases clinically considering pulse rate and quality, mucous membrane colour and consistency, capillary refill time, tissue turgor and skin temperature, and that a large number would make use of the stick blood urea test Azostix. The accuracy of this test is doubtful but may indicate if an animal's blood urea level is elevated. It was not expected that many practitioners would have the facilities for the estimation of P,C,V. and this was borne out in the results.

All 86 practitioners assessed cases clinically and none used tests other than those listed, except for one who stated that he hoped to use C,V,P. monitoring soon.

a) Clinically	86	practitioners
b) Packed Cell Volume	5	"
c) Blood Urea	8	"
d) Other	0	"

This is illustrated in a percentage histogram.

Assessment





#### APPENDIX 4

APPENDIX 4.

METHODS OF ADMINISTRATION - introduction

intravenous units

administration sets

flow regulation

fluid presentation

monitoring equipment

METHODS OF MONITORING AND RECORDING

ANTIBIOTIC ADMINISTRATION - routes and dosages

## METHODS OF ADMINISTRATION - LIST OF FIGURES

### Figure

1. Butterfly Cannula
2. Butterfly Cannulae
3. Plastic Cannula
4. Through-the-needle Cannula
5. Through-the-needle Cannula
6. Over-the-needle Cannula
7. Over-the-needle Cannula
8. Intravenous Unit
9. Administration Set
10. Blood Administration Set
11. Paediatric Administration Set
12. Roller Clamp
13. Dial-a-flo Flow Regulator Unit
14. Plastic Fluid Containers
15. Plastic Fluid Containers
16. Water Manometer Unit

## METHODS OF ADMINISTRATION

Within the past twenty years, the recognition that body fluid deficiencies might be restored parenterally has led to the development and employment of various techniques of administration. Not surprisingly, many different methods have been designed, used, abandoned, remodelled until some have been accepted for general use. Prior to this, such methods have included the use of feather quills connected to pig bladders, enema pumps with gold or ivory tubes, iron needles attached to funnel chambers, and more recently, highly sophisticated, expensive plastic designs, computer controlled, semi-reliable, but quite uneconomic. Clearly there must be a happy medium involving equipment which is economic, safe, practical, easily utilised and easily maintained. In the University of Glasgow Veterinary School, Surgery Department and a general practice numerous methods and pieces of equipment were used over four years to assess their relative advantages, merits or disadvantages. Acceptance for use was based on the ease of insertion, fixation and control of equipment, patient immobilisation or mobilisation, monitoring and expense. Availability of equipment was a problem and though discussed, applies only to that area of Scotland concerned in this work.

The areas of discussion are,

- A, Intravenous units
- B, Administration sets
- C, Flow regulation
- D, Fluid presentation
- E, Monitoring equipment

Each/

Each subject is considered on the basis of relative ease of use, patient satisfaction and economy. Patient satisfaction was assessed on the lack of equipment destruction and ease of attachment.

In this discussion it will be noted that only intravenous administration is mentioned and other routes are omitted since they were regarded as inadequate methods of parenteral fluid therapy.

Since veins are used, available sites are required which can be easily located, relatively easily punctured and allow fixation of the device in use. The dog provides six accessible sites which have their advantages and disadvantages, but are consistent in most animals. There are other possible veins, but they are relatively difficult to locate and pose problems with fixation and maintenance of a patent route. The selected veins are,

1. Jugular ( external ) veins
2. Cephalic veins
3. Saphenous ( lateral ) veins

1. External Jugular Vein                      The external jugular vein is easily located by palpating the trachea in the midline and the brachio-cephalic muscle laterally over which it runs latero-medially, and can be made prominent by applying pressure lateral to the trachea. This vein does not readily collapse even in shocked animals since it is one of the main vessels for venous return from the head. It is a large vessel relative to animal size and is ideal for venipuncture especially when withdrawing blood for analysis, even in very small animals. To permit venipuncture/

venipuncture, the animal is best restrained in a sitting position with the forefeet on the ground and the head extended upwards.

The infusion of fluid via a needle in the conscious or semi-conscious animal is virtually impossible because with movement of the subject and thereby the vein, the needle may repenetrate the vein or may become occluded due to apposition with the vessel wall. These difficulties have been overcome to a degree by the introduction of flexible plastic cannulae which do not repenetrate, but on occasion may kink and become occluded if the animal moves, especially laterally. The fixation of such a device poses another problem and usually requires stitching to the skin which incurs a little more work and inconvenience to the animal. An alternative is to perform a surgical cut-down operation and to tie the cannula into the jugular vein, thus preventing removal. If this can be performed near to the thoracic inlet there is less chance of the cannula becoming kinked, or of its removal by the patient. This technique is occasionally necessary in severely shocked patients where the peripheral veins are collapsed or impossible to locate and parenteral fluid is urgently required. This procedure can be completed in five to ten minutes, allowing parenteral fluid administration to commence rapidly.

2. Cephalic Vein                      That part of the cephalic vein used for venipuncture is the antebrachial segment as it runs proximocranially from the carpus until it reaches the cranial surface of the extensor carpi radialis muscle which it then follows to the flexor angle of the elbow joint, lying under the skin in loose connective tissue. In the proximal two-thirds of the antebrachium, the cephalic vein is ideal for venipuncture either/

either for injection or cannulation. Due to its smaller size, blood sampling from the cephalic vein is possibly worthwhile only in the larger breeds.

The cephalic vein is the vein of choice for the infusion of fluid due to its ease of location, relative ease of puncture which is easier due to the lack of surrounding superficial tissue which maintains the vein in a more stable position and its ideal position for the fixation of a device on a semi-permanent basis. The success of venipuncture can be demonstrated by the withdrawal of blood and by slowly injecting fluid observing the flow or otherwise into the vein. It is advisable to make the venipuncture distally in this vein, and should difficulty arise, it is possible to make further attempts proximally without the inconvenience of perivenous haematoma formation, "blowing the vein".

Both metal and plastic devices can be used successfully, although in short legged breeds re-penetration of the vein can occur with metal implants, especially on flexion of the limb. Similarly, it is best to insert a plastic cannula distally to prevent occlusion on flexion of the limb. To avoid both these problems, the leg may be supported in a splint with the limb in extension, thus preventing flexion of the elbow, but this causes inconvenience to the animal and extra work for the staff. Normally cannulae and needles are fixed in position with adhesive tape, and it is advisable to place gauze over the actual site of skin penetration to prevent the introduction of infection. Most devices fixed in this way cause little inconvenience to the patient, and many have been left in situ for several days for parenteral therapy with fluids and/or antibiotics. The commonest cause of failure is destruction of the device/

device by the patient. To prevent this, collars can be fitted - of the Elizabethan design - or the limb can be bandaged with the infusion unit delivery tube appearing at the shoulder where patient access is restricted. The cephalic vein appears to recover remarkably well on removal of implants and obstruction to blood flow is rarely recorded.

3. Lateral Saphenous Vein                      The lateral saphenous vein is found in the pelvic limb adjacent to the calcanean tendon and tibia. It is at this site that venipuncture is possible although perhaps difficult as it is a small diameter vessel very loosely held in the superficial connective tissue. It is difficult to maintain in use for any length of time, particularly when metal implants are inserted. Its mobility is its major drawback since fixation and maintenance are quite easy.

As a minor vessel, it collapses easily and is not suitable for long term intravenous infusions or treatment. Two minor disadvantages are its proximity to the hindquarters where the risk of infection is increased, and there is the constant soiling of dressings in incontinent animals. The implant and infusion unit in the conscious animal are compressed when it lies down, and this may prevent flow. It is ideal for short term infusions in an emergency when the thoracic veins are not patent, or when, for example, there are fractures of both forelimbs.

95 % of infusions in this study were given via the cephalic veins, as this was considered to be the most satisfactory route. The other routes were tried and evaluated, but, except in special circumstances were not used.

Venipuncture/



Venipuncture is performed under aseptic conditions along with antiseptics of the skin ensuring sterile insertion of any device, thus preventing the introduction of infection into the subcutaneous tissues or the vessel itself. Faulty technique may result in cellulitis, abscesses, phlebitis and finally septicaemia, both direct and indirect. ( Direct septicaemia is the introduction of infective pathogens from the external environment directly into the vascular system, whereas indirect septicaemia arises from an intermediate septic focus such as an abscess. )

Initially the hair should be close clipped over the area for venipuncture and prepared as for surgery with disinfecting agents to remove skin debris and pathogens, and to degrease the skin surface. A final application of disinfecting agent is made before actual puncture. This procedure is preferably carried out in clean conditions with scrub-up facilities for the staff performing the vessel puncture. All needles and cannulae provided by manufacturers are in sterile packs and care should be taken when handling them to ensure the maintenance of sterility.

The following sections are concerned with free and continuous administration of fluid from the supply containers without impedence from equipment, staff or patient.

- A. Intravenous units
- B. Administration sets
- C. Flow regulation
- D. Fluid presentation
- E. Monitoring equipment

The/

The implanted devices will receive detailed and constructive criticism with recommendations for use. It should be understood that all the equipment used in this study is manufactured for use in human patients and some designs require adaptation to veterinary needs.

A. Intravenous Units                      These devices are either needles or cannulae, ( the word cannula is used in preference to catheter, both being tubes which allow fluid or gas to pass into or out of the body ). The intravenous units are considered as two groups.

Needles come in all shapes and sizes from the basic steel tube with fitting for the syringe, to needles with plastic multiport fittings, rubber plugs, plastic caps and lengths of plastic tubing attached. Nowadays most needles are of a disposable type made of steel coated in silicone and with a plastic female fitting to allow insertion of other equipment. The steel to plastic bond is generally well made and will withstand sterilisation by most methods, but the sharp, machined point becomes blunt after use on one or two occasions, making further injections painful and difficult.

For venipuncture, needles with a transparent butt have the advantage of allowing visible " flashback " when the vein is entered. This " flashback " phenomenon is characterisic of venous puncture and is a certain sign that entrance has been achieved, it being demonstrated/

demonstrated by the sudden presence of blood in the butt chamber.

Needles are made by various manufacturers and for the purpose of intravenous infusion they are adequate only for short term treatment in those cases where the needle can be securely fixed without fear of venous repenetration. Therefore in general they are inadequate for medium or long term infusions due to the lack of fixation point, fear of repenetration, ease of removal by the patient and staff and the inconvenience to the animals. Needle implants seem to cause more irritation than plastic cannulae.

To permit the use of needles for fluid infusion, designs of a plastic butt with support appendages are manufactured. These are often referred to as "scalp" or "butterfly" cannulae and are primarily for use in children and adults for short term infusions. The basic design is a steel needle moulded to a flat plastic butt, not unlike a butterfly in shape, with a short length of plastic tubing attached and at the distal end of which is a plug arrangement. ( Fig. 1. ) One design tried in this study did not employ the tubing arrangement which in itself has the advantage of separating the needle from the female fitting of the unit and allows syringes and infusion units to be attached without placing tension on the venous implant. The type of tubing used is of minimal internal diameter or bore and contains very little fluid - between 0.2 millilitre and 1.0 millilitre. These designs allow fixation to the limb using the butterfly wings which also make insertion easier due to better finger control of the unit.

The/

The length of plastic tubing can be coiled and strapped down leaving only the distal female port visible,

These units are supplied by many manufacturers, are relatively inexpensive being available in a wide range of needle lengths and gauges suitable for most animals. ( Fig. 2. )

The advantages of needle and plastic butterfly designs are low cost, relative ease of insertion, maintenance, fixation and minimal patient inconvenience. More of these units were interfered with by patients, but overall only some 5 % were destroyed, the major incident being severance of the plastic tubing.

The disadvantages are vein repenetration - especially in short legged breeds, and then usually on limb flexion - phlebitis during long term infusions and relative inconvenience to patients.

Having regard for the above, it is recommended that butterfly type needles be used mainly for emergency and short term infusions or treatment. The design incorporating a length of plastic tubing attached to the needle end is preferred, since it can be easily fixed in position.

Cannulae are made of polyethylene or " Teflon " and are basically of three types, determined by their appearance and use in fluid deficient patients. The types are,

a/

- a. Plastic cannulae
- b. Through-the-needle cannulae
- c. Over-the-needle cannulae

The basic form is a plastic tube with a female fitting at one end to allow fixation of infusion units or syringes ; this butt or female fitting comes in different forms, from simple enlargement of the cannula to sophisticated hubs with numerous attachments. Most of the designs have simply a single female fitting, only a few have plugs and fewer still have the means for fixation to the patient.

a. Plastic cannulae Plastic cannulae are the simplest type consisting of a tube with a female fitting at one end. (Fig. 3.) Their use is limited to where a surgical cut-down operation is performed or where they are inserted through a needle of suitable bore. The last method raises an immediate problem which is perhaps more evident in the cannulae through-the-needle style. This problem arises if the cannula is withdrawn through the needle as there is a chance that the cutting edge of the needle will sever the cannula leaving the proximal end free within the vein. At worst it may travel proximally and come to rest in the right side of the heart. The consequences of such an accident are not fully apparent in the animal, but the concern regarding this problem in the human has led to the withdrawal of some makes and the inclusion with others of strict instructions related to use.

b. Through-the-needle cannulae The basic principle of this design is to insert a needle into the vein and through this is fed/

Fig. 1. Butterfly cannula

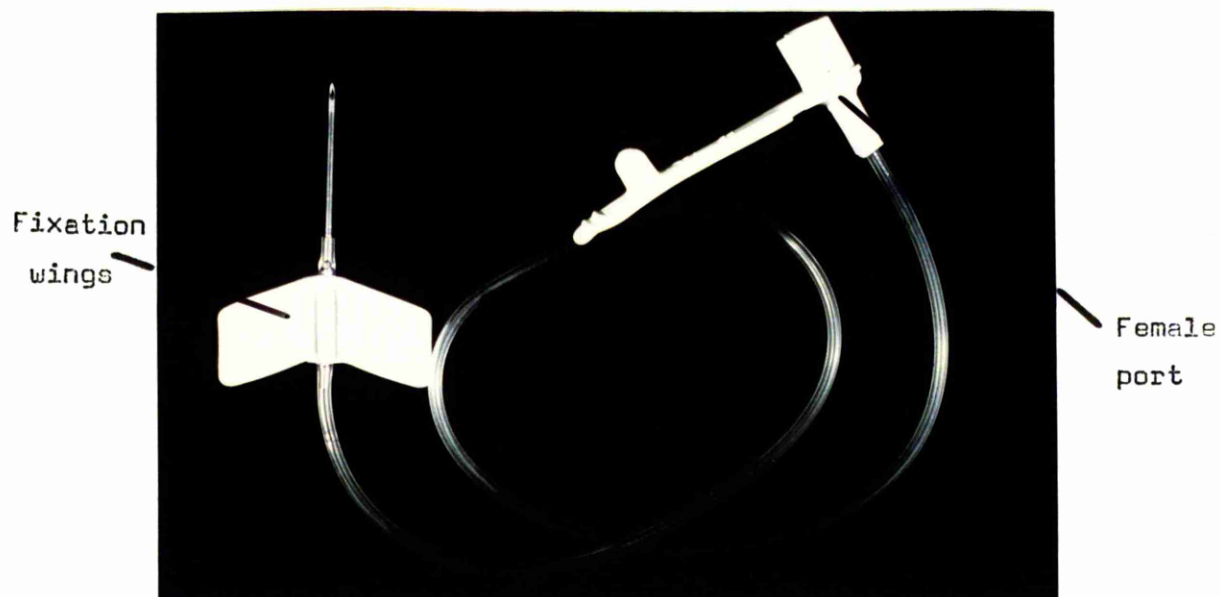


Fig. 2. Butterfly cannulae.

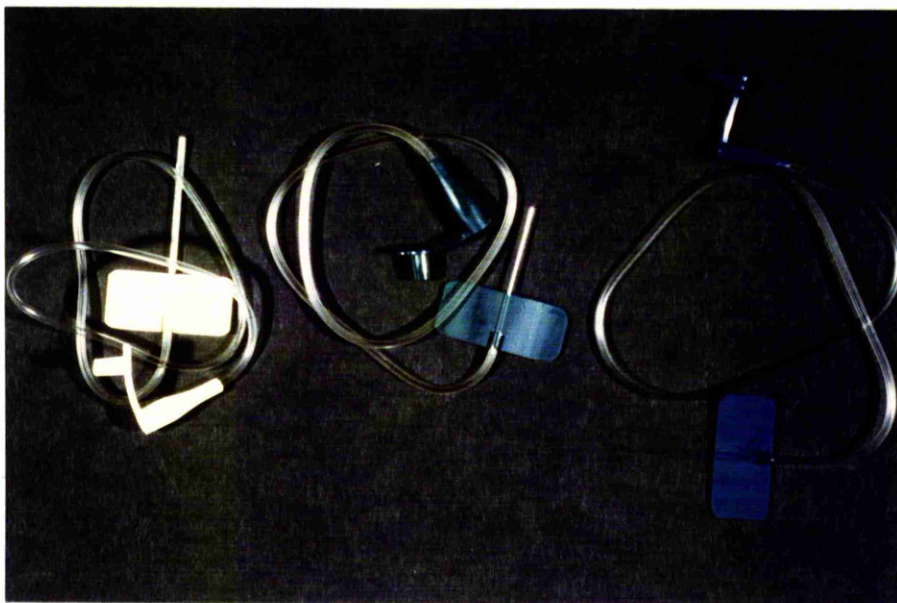
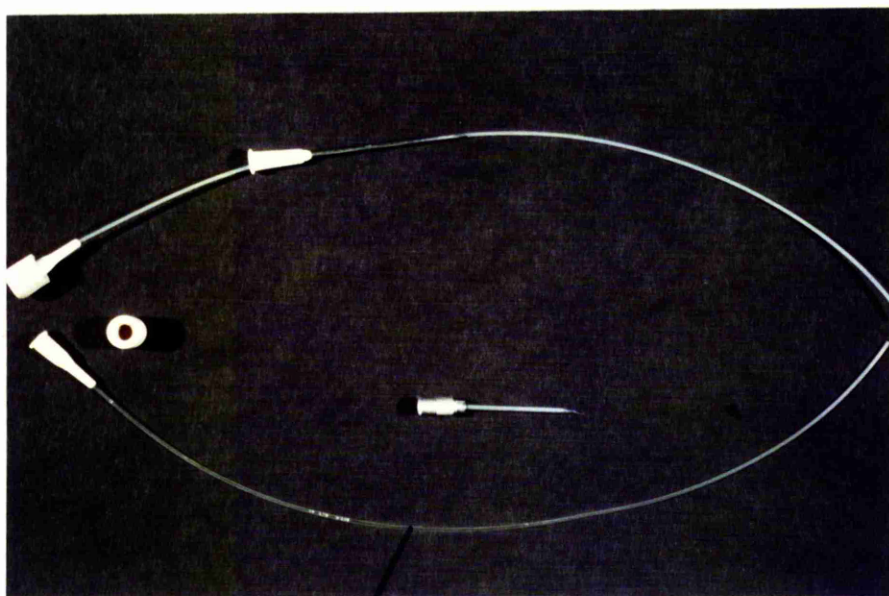
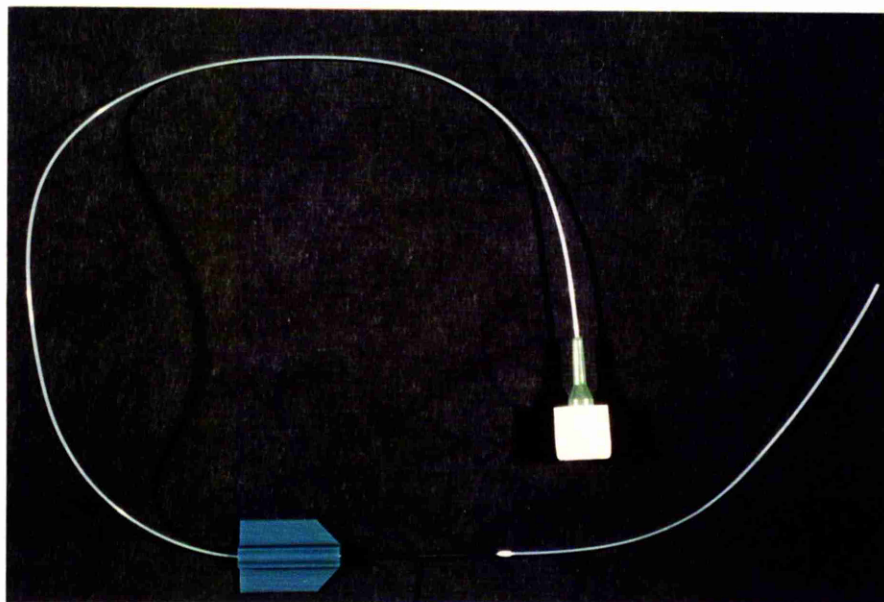


Fig. 3. Plastic cannula



Cannula

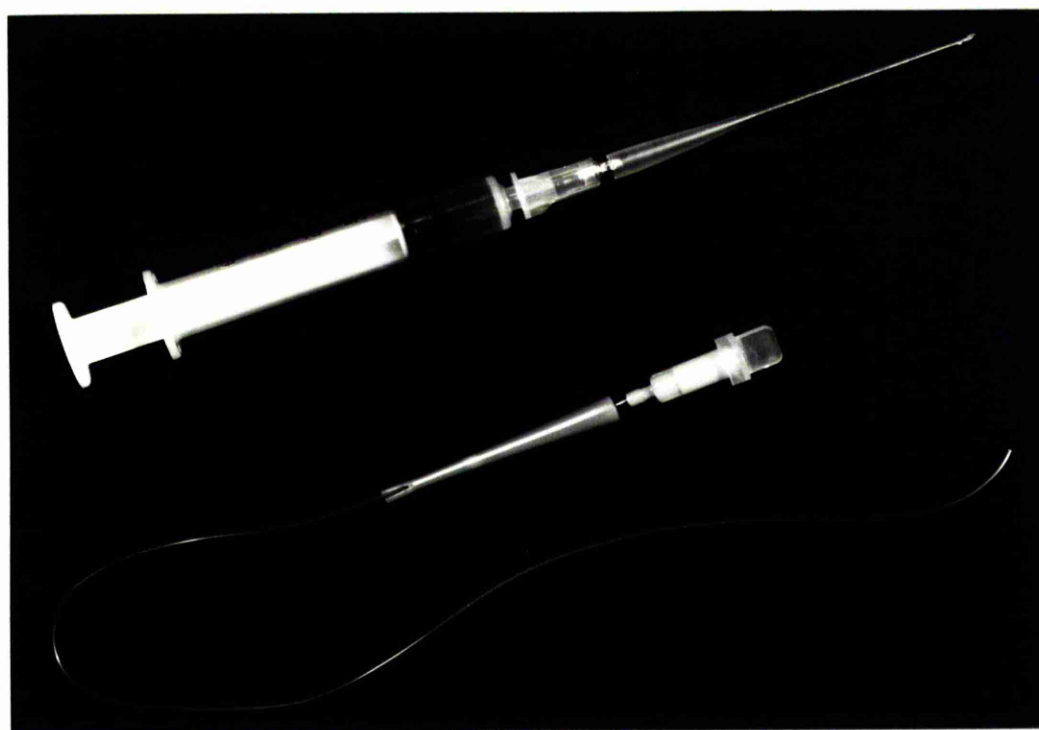
Fig. 4. Through-the-needle cannula



Cannula

Insertion needle

Fig. 5. Through-the-"needle" cannula





fed a plastic cannula of varying length which has a butt and female fitting at the external end. There are available combined units and separate units, each being self explanatory. (Figs. 3 and 4.) Most of the combined units have a needle with a locking device for the plastic cannula butt once inserted which prevents withdrawal of the cannula with the risk of severance. All of these designs are expensive and cumbersome to use and are not particularly useful in animals due to their length and gauge - most are between 14 and 18 gauge. The size is governed by the inserting needle which will allow a cannula of three sizes smaller in gauge to pass through.

One more complex design (Fig. 5.) differs in that it requires the insertion of an over-the-needle type of plastic cannula, the inserting needle of which is discarded prior to insertion of a longer through-the-needle type of plastic cannula. This latter cannula is used for infusion and has a locking device attached which determines the length inserted into the vein. A plastic screw plug is also provided to seal off this cannula when not in use. This type of unit does not cause cannula severance on withdrawal. It is expensive and is probably outwith normal veterinary use.

c. Over-the-needle cannulae This design involves a plastic cannula which is moulded around a needle which is withdrawn after venipuncture and discarded leaving the cannula in the vein. Most of these designs are basic and inexpensive relative to their advantages in small animals. The requirement in veterinary use is that skin penetration is possible without crinkling of the fine moulded end of the cannula which might cause injury to the vein. For this reason some types tried and/

and evaluated were not recommended for use. The closer the apposition of plastic to metal, particularly at the proximal or pointed end, the more successful the apparatus. The large number of available designs means that variables exist. Some have female butts of plastic, some of metal and some none at all ; a variety of flashback chambers is available; some have occluding plugs ; and lengths are quite variable as are gauges. (Figs. 6 and 7.) Only one design tried had butterfly wings to allow attachment to the animal, but this was more expensive and there tended to be crinkling in a large number of cases.

Insertion of these devices is not complicated, but requires care and attention to detail, and with acquired skill can be undertaken by most staff capable of intravenous injection. The whole unit is inserted through the skin and into the vein. Penetration of the vein is indicated by flashback, at which point the needle is gradually removed and the cannula advanced as far as the butt. The cannula is fixed in position with a sterile dressing over the puncture site by adhesive tape. In my experience, to have a three-way tap attached to the cannula makes fixation much more satisfactory and the unit easier to use. (Fig. 8.)

The major advantages of these over-the-needle cannulae are that once inserted there is little chance of venous repenetration, or other damage to the venous wall, and other points of note include less inconvenience to the patient and less work for the staff. In addition, these cannulae can be re-used provided the insertion needle is retained. When using the jugular or cephalic vein, long term infusion or therapy is possible without venous thrombosis occurring. Cannulae of this design have been used successfully over a period of three weeks in dogs/

Fig. 6. Over-the-needle cannula

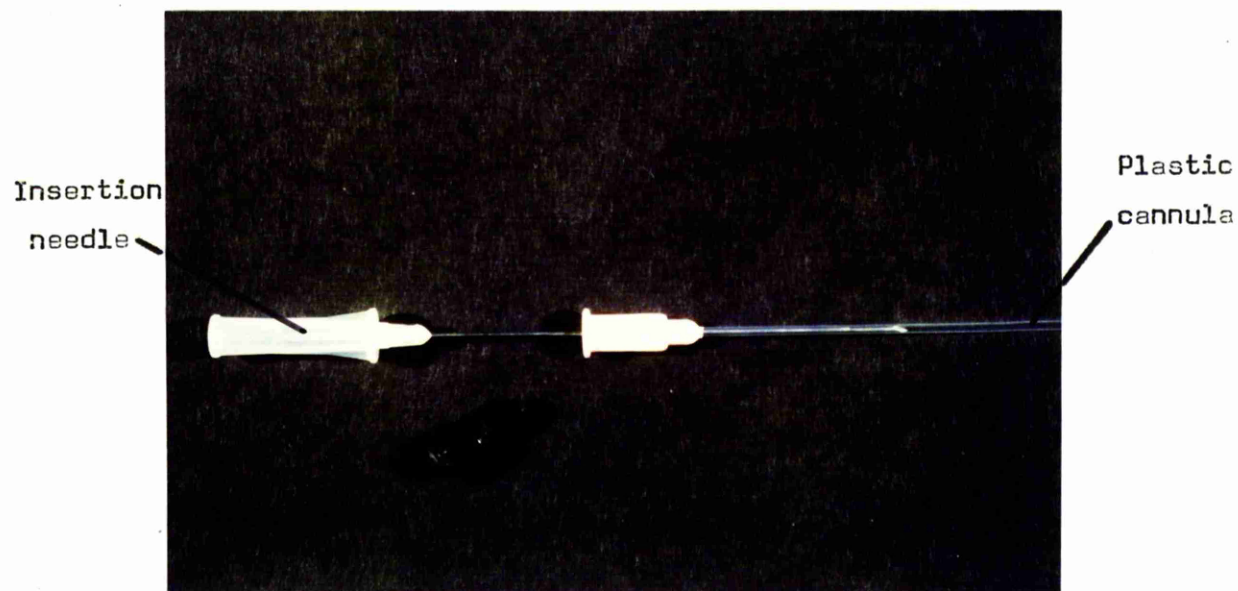


Fig. 7. Over-the-needle cannula

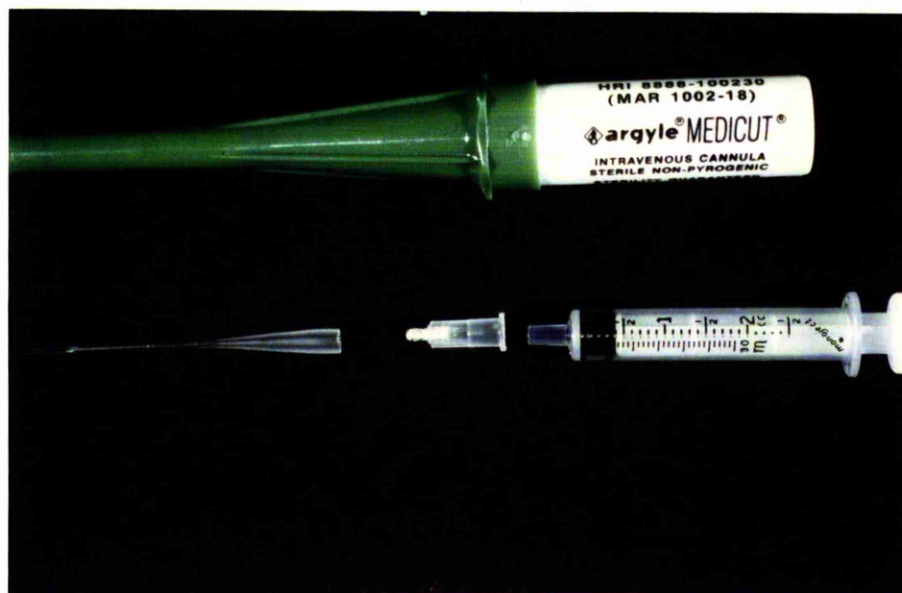
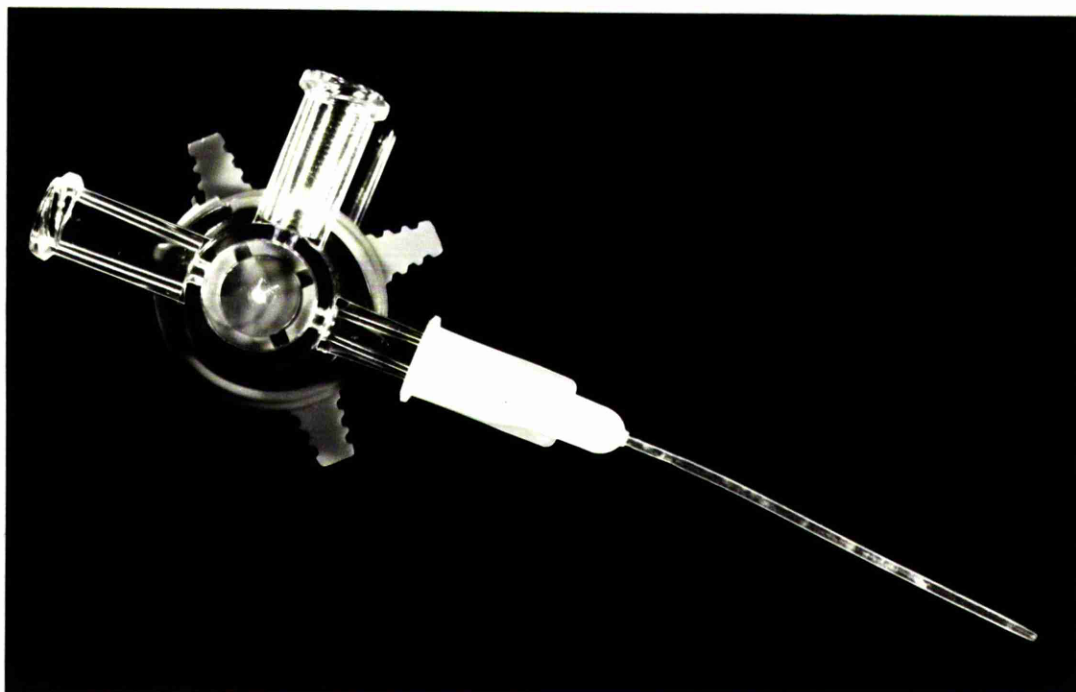


Fig. 8. Intravenous unit



dogs without problem and the same cannulae, after re-sterilisation were used again in other animals. This procedure of re-sterilisation and re-use was investigated and put into practice with a resultant reduction in treatment cost.

All systems of needles and cannulae require routine maintenance with particular reference to hygiene to allow successful, prolonged use. While not in use for infusion or injection, they should be flushed and left filled with heparinised saline (5,000 I.U. per litre). This helps to prevent clotting of blood in the needle or cannula, and allows re-use for injection or infusion. The site of insertion through the skin should be kept clean and can be protected with antibiotic cream. The female inlet port should be cleaned with disinfectant prior to use and kept covered when not in use to prevent the introduction of infection into the bloodstream.

In conclusion therefore, cannulae have the advantages of prolonged use in long term therapy, less inconvenience to staff and patient, the opportunity of re-use after sterilisation and relative ease of insertion. The disadvantages are the lack of fixation point in many designs, the lack of provision for temporary plugging of the cannula, the occasional crinkling of the tip and the relative expense compared with butterfly needles.

It is recommended that plastic cannulae, of the over-the-needle design, be used in conjunction with a three-way tap or extension set to allow fixation, for both short and long term therapy in small animals of all sizes. In addition, their use in large animals has been proved to be successful.

B./

B. Administration Sets

Consideration is now given to

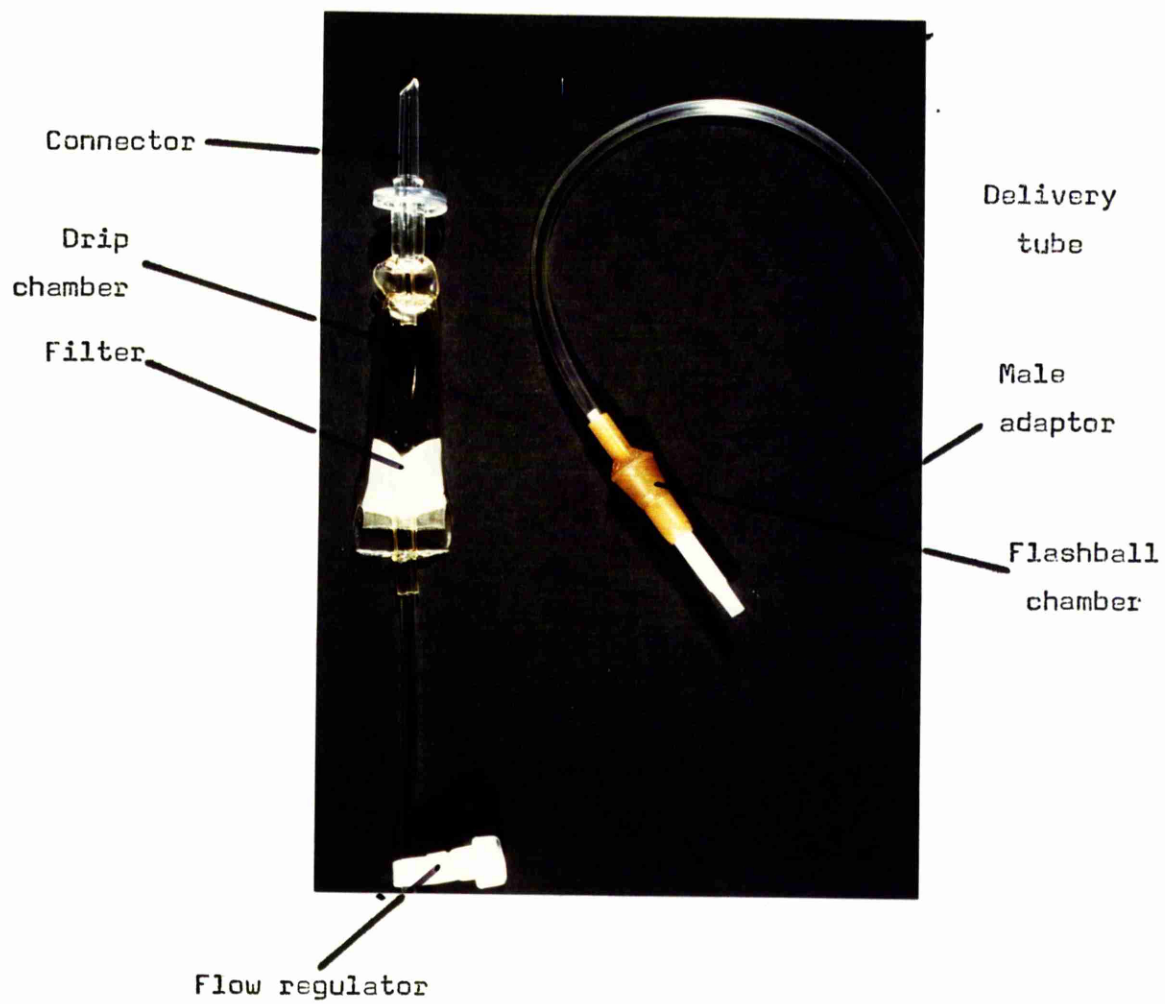
the apparatus required for fluid infusion. Most commercially available units are referred to as administration sets and are relatively standard. They are presented ready sterilised in packs with a variety of optional fittings and consist basically of a connector, drip chamber, filters, delivery tube, flow regulator, a rubber segment suitable for additive injections and a needle adaptor, typically a male fitting. Most sets are made of plastic throughout, though one design uses a metal needle moulded into the plastic connector for puncturing the rubber stopper in glass bottles. The rubber section is commonly found at the patient end of the administration set and takes on a variety of shapes, from a length of straight tubing to a chamber called a flashball or balloon chamber. This point is intended for the injection of additional substances into the infusion, and in my experience, it is best to use a fine gauge needle allowing easier resealing of the puncture hole in the rubber. Larger puncture holes may leak if fluid is being administered under pressure or when there is some obstruction to the flow.

The basic set is as described above ( Fig. 9. ) This design is ideal for fluid infusions of all kinds except whole blood or plasma. They are intended to be disposable, but can be cleaned, resterilised and used several times, thus reducing costs.

For whole blood and plasma infusions the basic set with the addition of a filter chamber placed above the drip chamber is required. ( Fig. 10. ) This marginally increases the cost but is necessary to remove cellular debris within the blood before it reaches the recipient.

The/

Fig. 9. Administration set



The final type to be discussed and illustrated is the paediatric unit which incorporates a burette chamber along with the filters of the administration set and is highly recommended for use in veterinary practice, particularly in the infusion of fluids in small animals where less than 150 millilitres is required. The design follows the simple infusion set but has a short upper chamber delivery tube and flow regulator attached to the connector. This delivery tube feeds the graduated burette chamber which allows an accuracy of 1 millilitre in infusions. Below this chamber is a filter unit and a drip chamber attached to the remainder of the set. (Fig. 11.) Most burette chambers, which are sealed, contain 150 ml. of fluid.

When using these units, the flow regulators are closed prior to insertion of the connector into the fluid container. The upper regulator is opened until 100ml. of fluid fill the burette. The filter and drip chamber chambers are gently squeezed until they contain some fluid, and then the lower regulator is opened slowly allowing fluid to flow into the long delivery tube. Once the delivery tube is filled, the male fitting can be attached to the patient. Between 15 and 30 ml. of liquid are needed to fill the unit. After the infusion is commenced, the burette chamber requires to be filled regularly, usually with 100 ml..

This is a worthy method of fluid infusion and is discussed in the section regarding the prevention of overinfusion.



Fig. 10. Blood administration set

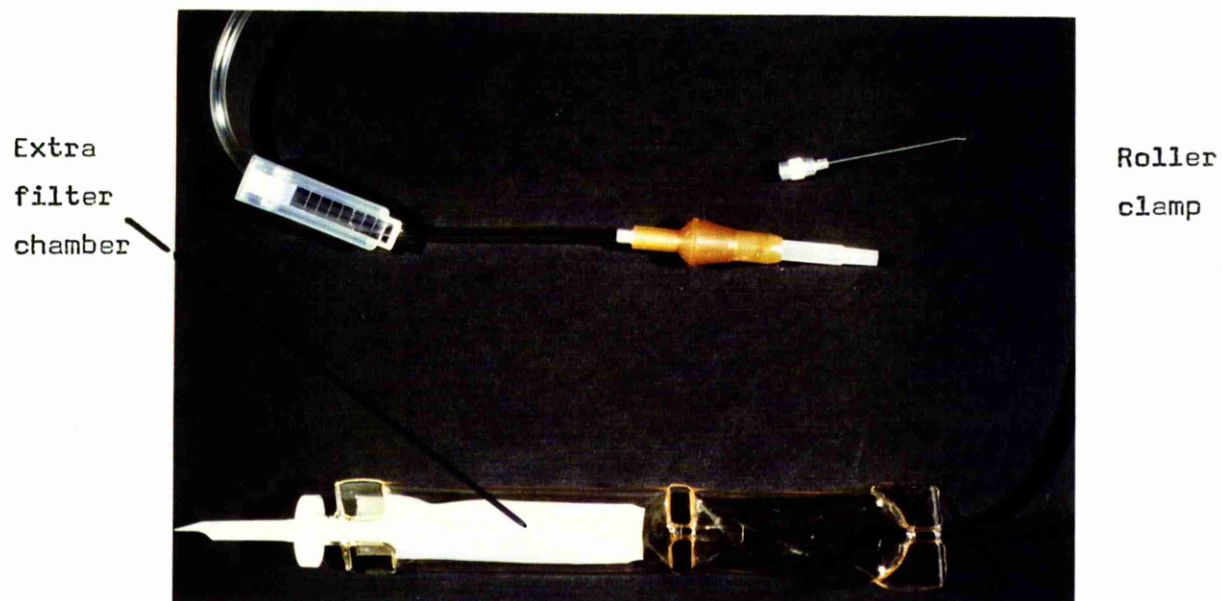
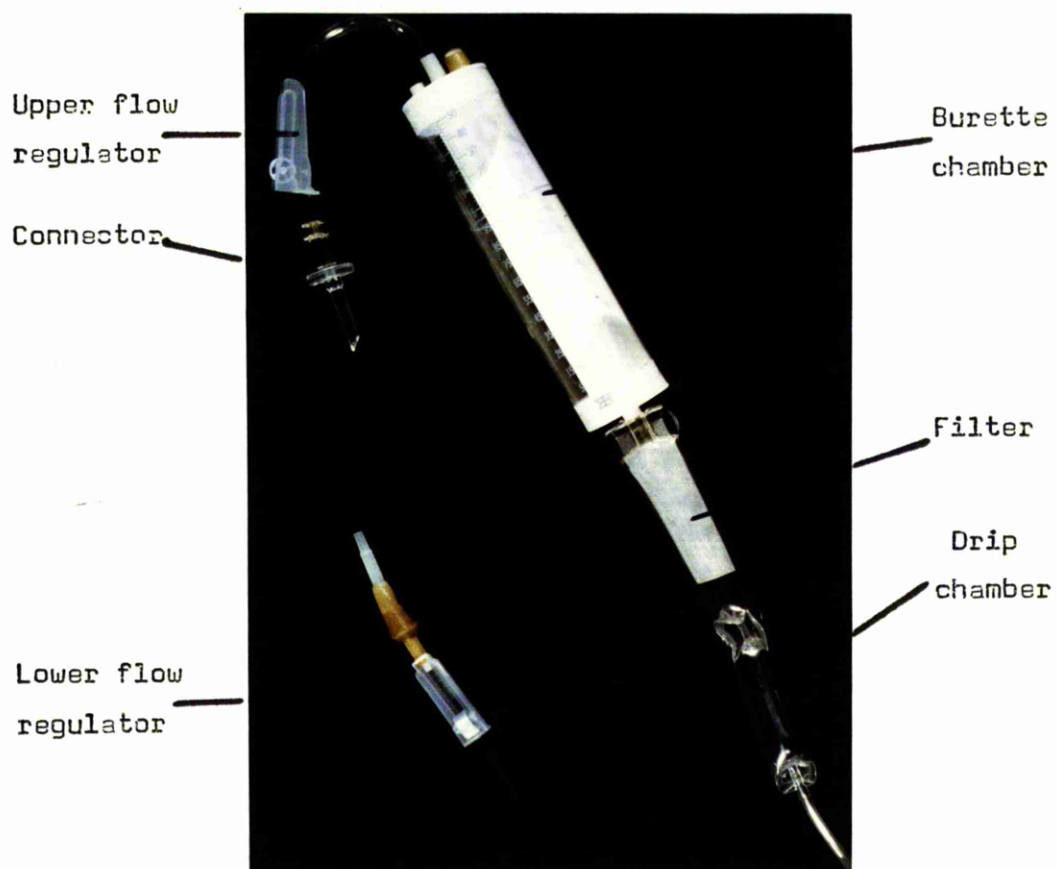


Fig. 11. Paediatric administration set



C. Flow Regulation

Flow control is one subject which has resulted in ingenuity in design and units vary from a thin metal disc folded in half to ultra-expensive and sophisticated pumps using all aspects of modern flow technology. Administration sets have a maximum flow rate and the guage of the needle or cannula along with the height of the drip chamber above the patient influence this flow. The commonly supplied drip chambers allow a flow of 15, 30 or 60 drops per millilitre, drip size being determined by the size of the aperture between the filter and the drip chamber. Therefore by counting the number of drips per minute and knowing the drip size, the infusion rate can be determined. To maintain this rate, a flow regulator is used on the delivery tube. The designs vary and have advantages and disadvantages. An even flow rate is essential for the correct administration of drugs and fluids, and to prevent the blockage of cannulae by blood clot.

In this study only simple devices which were economically available were considered. The simplest design is a soft metal disc which is folded around the delivery tube and when folded at right angles to the original fold narrows the tube lumen, ( see Fig. 15. ) Its disadvantage is that it cuts into the plastic delivery tube and renders the administration set useless.

One screw system supplied may give finer control, but has the disadvantage of cutting directly into the plastic tube when the infusion is stopped, ( Fig. 9. ) This type is not recommended where it is hoped to re-sterilise and re-use the administration set.

The common type is a roller clamp through which the delivery tube/

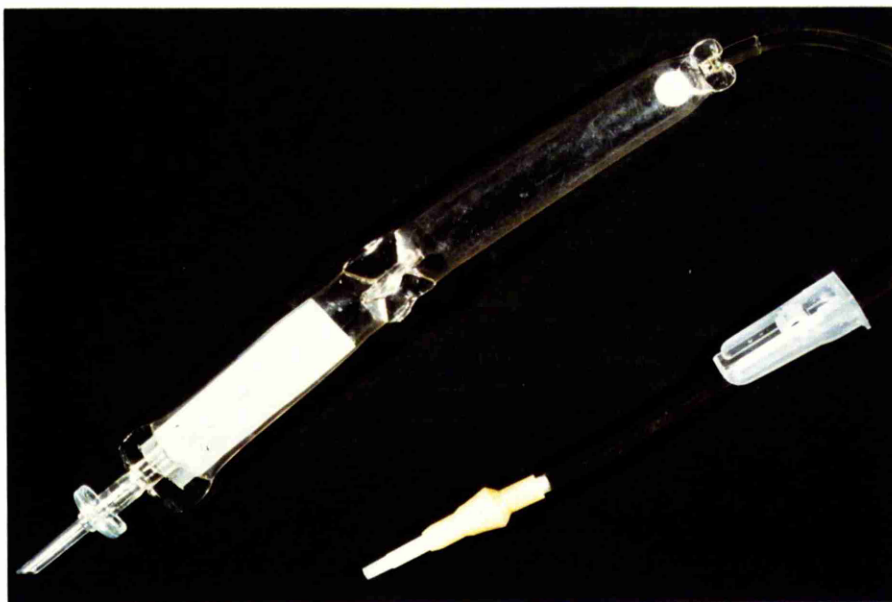
tube passes and which controls the flow by pressing directly on the tube. (Fig. 12.) This design is easy to use and provides adequate control of the flow rate. One disadvantage is that it tends to " creep " thus causing a spontaneous change in flow rate, more often an increase. This is not often immediately evident and the flow rate can be quite dramatically altered.

Recently a small barrel-shaped device has appeared which is sold as a separate entity, but it is expensive and can only be bought in bulk at present. This regulator is added to the administration set between the set and the patient. It is affected by the height of the drip chamber above the device and also the size of drip delivered, but once adjusted, gives an accurate and steady control of the flow rate. (Fig. 13.) In my experience, it can be used for long term infusions over 24 hours without varying the rate and is ideal for prolonged maintenance therapy.

The flow controller, therefore, must be efficient, easy to understand and use, of low cost and must not damage the administration set. The roller clamp design seems to satisfy the requirements successfully, although the newer type described would be of benefit.

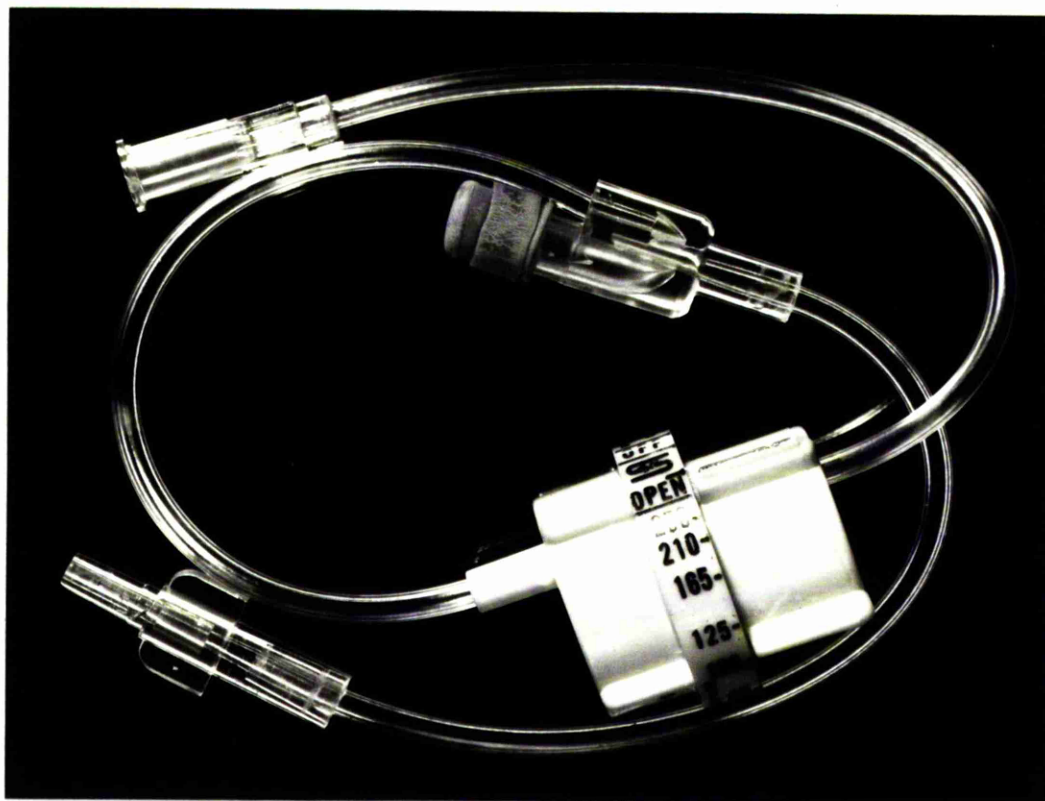
To give some indication of the advances made in technology, systems are now available which control flow to a 2% error and include audio-visual alarms which operate in the event of cannula or extraneous occlusions or emptying of the fluid container. They instantaneously respond to any desired change in flow rate, automatically adjusting to variations in venous pressure.

Fig. 12. Administration set with Roller clamp



Roller  
clamp

Fig.13. Dial-a-flo flow regulator unit



D. Fluid Presentation

Fluid containers vary from

manufacturer to manufacturer, but two basic designs exist - glass bottles and plastic bags. Most fluids are available in both and there are over one hundred different fluid compositions.

The glass container is gradually being replaced by the plastic bag which is simpler to use. Bottles require the addition of an air inlet which is usually a needle attached to a plastic tube with a filter at the other end. A hanging attachment for the drip stand is also required.

The plastic containers are soft or hard but all empty freely without an air inlet, thus reducing the risk of infection. Plastics have now been designed which do not react with the fluids and therefore storage times have been prolonged, but this is now disputed. These containers can also be used for rapid infusion as pressure can be exerted manually. Other advantages are low cost and less risk of breakage along with easier storage, and all varieties are provided with an eye or hook for suspension from drip stands. A major disadvantage is that once punctured they do not re-seal and the administration set must be left in place until the fluid is used or discarded. One point of interest is that most plastic bags have numbers on the side of the pack presumably to indicate the amount of fluid infused or fluid remaining, but unfortunately these figures are only very approximate and should not be used for accurate fluid infusion. (Fig. 14.)

Some plastic units have a simple administration set attached by moulding to the bag, but although adding to the cost, it does obviate the expense of a separate administration set. (Fig. 15.) All other fluid packs require an administration set before infusion can commence/

Fig. 14. Plastic fluid containers

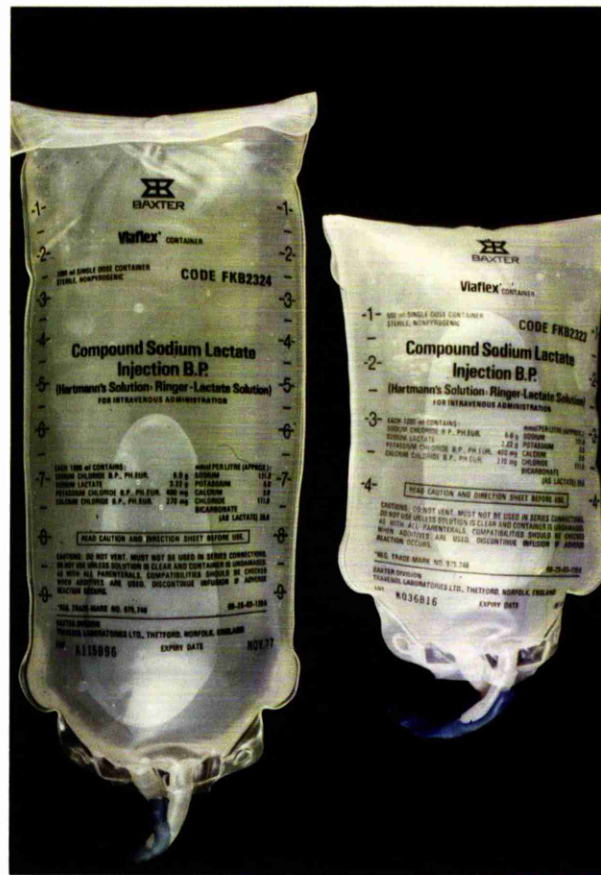
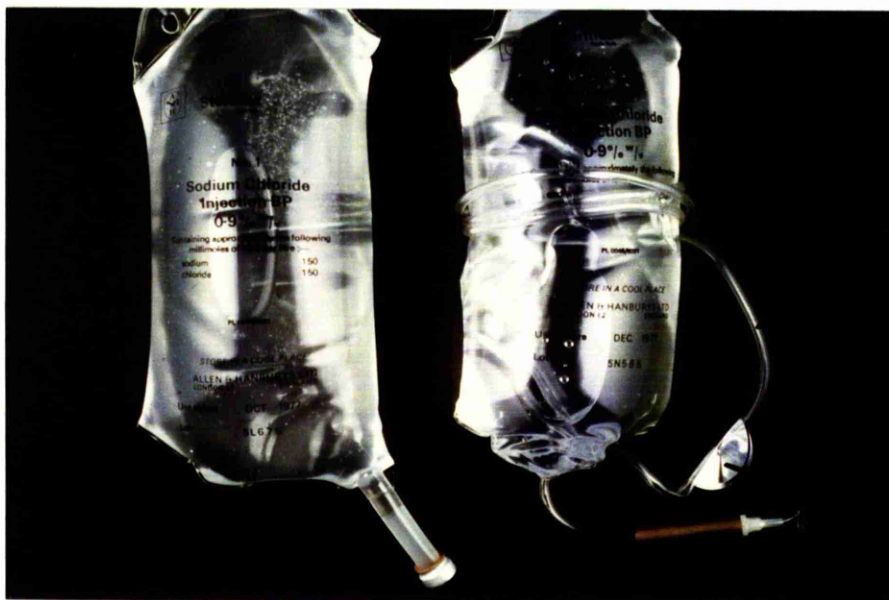


Fig. 15. Plastic fluid containers (with administration set)



Administration  
set

Flow  
regulator

commence.

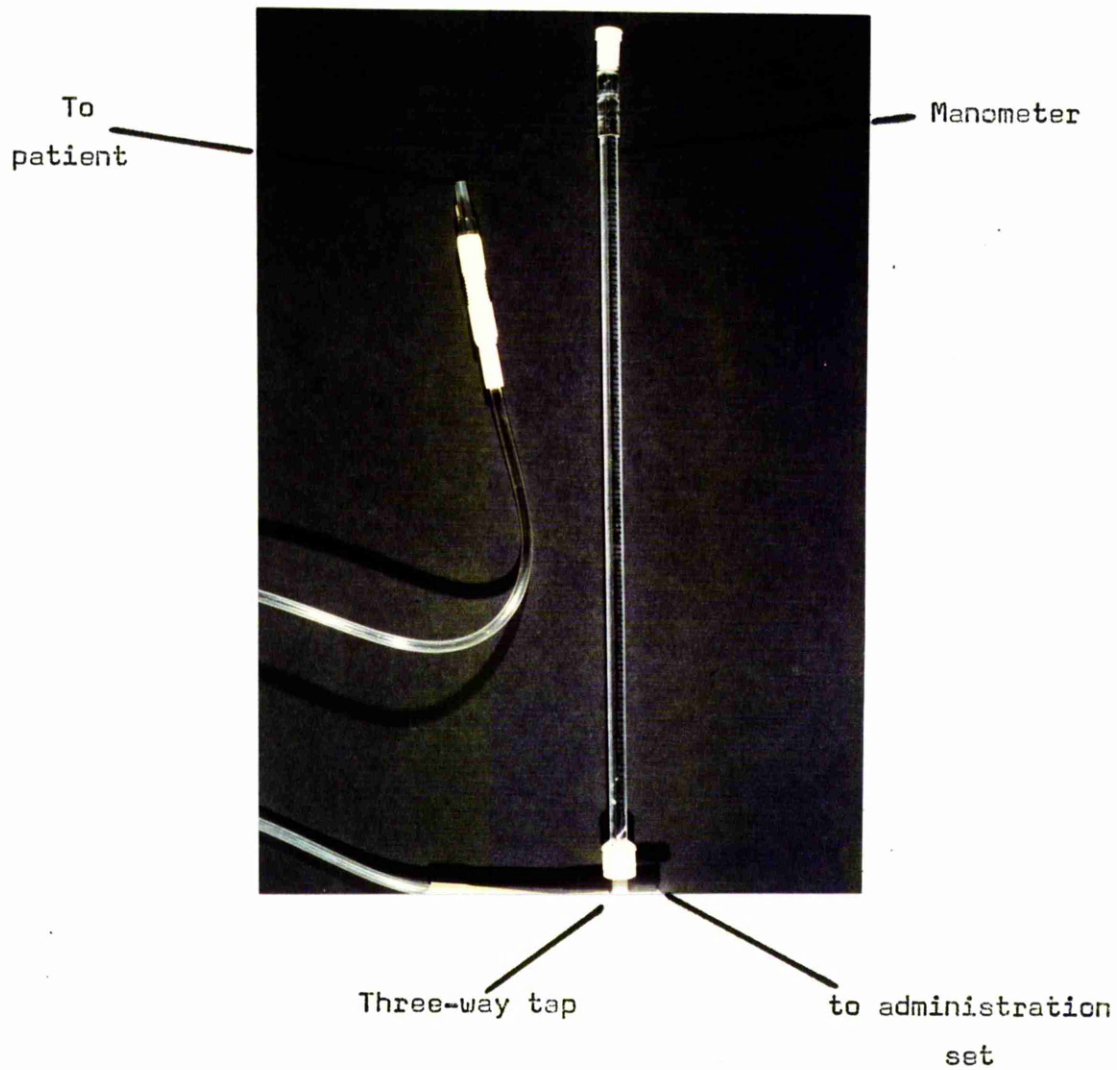
Therefore the advantages of plastic over glass are low cost, ease in storage and in use including suspension from the drip stand and disposal. The main disadvantage of plastic containers is that they do not re-seal after puncture. Having considered these points, my own experience indicates a preference for plastic containers.

#### E. Monitoring Equipment

The monitoring of venous pressure

is described in detail in another section, but it was thought relevant to describe the equipment used routinely. All designs incorporate a water manometer, graduated by various methods, and a three-way tap which permits the measurement of venous pressure from the patent line or the infusion of fluid. As with other pieces of equipment, the range and cost are highly variable, but all that is required is a three-way tap, a length of plastic tubing and a graduated scale which can be set at the respective zero point on the patient. (Fig. 16.) Normally the infusion flows through the unit, but when a venous pressure reading is being taken, the three-way tap blocks the infusion and the manometer is connected to the patient.

Fig. 16. Water manometer.





## MONITORING AND RECORDING

Soma, Burrows and Marshall (1974) state that, " It is not enough to place an animal in a cage with a bottle of fluid connected intravenously " and " only skilled technicians should be used in intensive therapy units and all information and times should be recorded ".

Monitoring and recording are two necessary components of intensive therapy. All the information regarding each case should be noted in a record file of some form. Since one person cannot totally care for a single patient, it is important that others have access to the information obtained by the initial clinician, and that this is readily available preferably beside the patient.

All the information derived from monitoring should be noted along with the time of the observation, and any system must be clear and easily understood by all personnel. Some forms of recording systems are illustrated below.

The monitoring of fluid replacement and patient status is a large field, and some possible methods are discussed elsewhere in this study. Clinical assessment is of use and other ancillary monitoring aids are available which are satisfactory in animals. Such aids as the output of urine, the venous blood pressure, the arterial blood pressure and the ancillary aids of blood analysis and radiography are discussed in the experimental section.

GLASGOW UNIVERSITY VETERINARY SCHOOL

CASE NUMBER.

OWNER. \_\_\_\_\_

BREED. \_\_\_\_\_ AGE. \_\_\_\_\_

INTENSIVE THERAPY UNIT

Date .....

Time of Admission .....

Diagnosis .....

.....

Day

Time

Pulse

B.P.

Rectal Temperature

Skin Temperature

Capillary Refill Time

C.V.P.

P.V.P.

Respiratory Rate

Ventilator ON

OFF

Suction

Urine Volume

Pupil Reactions

M.M. colour

Treatment 1.

2.

3.

4.

5.



# TEMPERATURE CHART

Time	A M		P M		A M		P M		A M		P M		A M		P M		A M		P M		
Temp's	2	6	0	2	6	0	2	6	0	2	6	0	2	6	0	2	6	0	2	6	0
Unit																					
106°																					
105°																					
104°																					
103°																					
102°																					
101°																					
100°																					
99°																					
98.4°																					
98°																					
97°																					
150																					
140																					
130																					
120																					
110																					
100																					
90																					
80																					
70																					
60																					
50																					
40																					
30																					
20																					
Day of Month																					
Date																					

C  
 41°  
 40.4°  
 39.8°  
 39.3°  
 38.7°  
 38.2°  
 37.6°  
 37.1°  
 36.8°  
 36.5°  
 36°  
 35°  
 34°  
 33°  
 32°  
 31°  
 30°  
 29°  
 28°  
 27°  
 26°  
 25°  
 24°  
 23°  
 21°  
 21°

NAME:

LABORATORY

CASE NO.

## DATA

DATE							NORMALS
HAEMATOLOGY							
P.C.V. (%)							35-40
Haemoglobin (g/100ml)							11-14
R.B.C. ( $10^3$ /cu mm)							3-5
W.B.C. ( $10^3$ /cu mm)							7-12
Neutrophils (%)							60-80
Eosinophils (%)							0-5
Lymphocytes (%)							20-30
Monocytes (%)							2-7
Platelet ( $10^3$ /cu mm)							100,000
Reticulocytes % r.b.c.							0.5
Clotting time (mins)							7-9

## BIOCHEMISTRY

Urea (mmol/l.)							0-7.5
Sodium "							134-152
Potassium "							3.4-5.9
Chloride "							95-115
Calcium "							2.2-2.6
Magnesium "							0.5-1.19
Phosphate "							1.3-2.3
Sugar "							2.5-5.02
Cholesterol "							1.1-7.6
Creatinine (mmol/l.)							44-132
Bilirubin							0-10
Alk. Phos (u/l)							0-105
Ast "							0-40
Alt "							0-40
Total Protein (g/l.)							59-79
Albumin							4-5
Globulin							2-1
T 4 (nmol/l.)							0-30
T 3 (g)							60-80
Progesterone (nmol/l.)							
Oestrogens (Pmol/l.)							
Androgens							
c.p.k. (u/l)							
Cortizole (Gms-nmol/l)							

## URINE ANALYSIS

Protein (mg/100ml)							Nil
Urea (mmol/l)							500
Sugar							Nil
Ketones							Nil
Bilirubin							Nil
Blood							Nil
pH							5-7
S.G.							1.02-1.04
Deposits							
Bence Jones Protein							
Creatinine (mmol/l)							
Calcium (mmol/l)							
Magnesium (mmol/l)							
Phosphate (mmol/l)							

## S.F.

Protein (mg/l)							<100
Cells							Nil

Y6C8b

## TREATMENT SHEET

[illegible]

# ANTIBIOTICS

<u>Trade Name</u>	<u>Drug</u>	<u>Form</u>	<u>Route</u>		<u>Dose</u>	<u>Hourly</u>	
			O.	IM.	IV.	per kg.	
Crystapen	Benzylpenicillin	Injection		x	x	20000 units	4 - 6
Mylipen	Procaine penicillin	Injection		x		20000 units	24
Penidural	Benzathine penicillin	Injection		x		20000 units	96
	Penicillin G	Tablets	x			20000 units	6
Dimycin	Dihydrostreptomycin	Injection		x		10 mg.	12
Terramycin	Oxytetracycline	Injection		x	x	6 mg.	6 - 12
	Oxytetracycline	Tablets	x			20 - 50 mg.	8
Penbritin	Ampicillin	Injection		x		20 mg.	8
Penbritin	Ampicillin	Tablets	x			10 mg.	12
Tribrissen	Trimethoprim + Sulphadiazine	Tablets	x			3 mg.	12
Trivetrin	Trimethoprim + Sulphadoxine	Injection		x	x	15 mg.	12
Septtrin	Trimethoprim + Sulphamethoxazole	Tablets	x			10 mg.	12

Trade Name	Drug	Form	O.	Route	Dose	Hourly
				IM.	IV.	
Septtrin	Trimethoprim + Sulpha methoxazole	Injection		x	x	12
Lincocin	Lincomycin	Injection		x	10 mg.	12
Lincocin	Lincomycin	Tablets	x		20 mg.	8
Intramycetin	Chloramphenicol	Injection		x	6 mg.	12
Chloromycetin	Chloramphenicol	Tablets	x		20 - 50 mg.	8
Flagyl	Metronidazole	Tablets	x		60 mg.	24
	Erythromycin	Tablets	x		10 mg.	8
Tylan	Tylosin	Injection		x	3 mg.	8
Tylan	Tylosin	Tablets	x		10 mg.	8



## APPENDIX 5

APPENDIX 5.

STUDY OF CLINICAL CASES - results and analysis

Tables of results

Histograms 1 - 13.

ABBREVIATIONS USED IN THE TABLES OF RESULTS

App.	Appearance
Temp.	Rectal Temperature °F
Pulse vol.	Pulse volume
Resp. rate	Respiratory rate
Resp. nat.	Respiratory nature
M.M.col.	Mucous Membrane colour
M.M.Cons	Mucous Membrane consistency
CRT.	Capillary Refill Time    - seconds
T.T.	Tissue Turgor
Abd. Pain	Abdominal Pain
Urea	Blood Urea            mmol/l
Na	Serum sodium           mmol/l
K	Serum potassium       mmol/l
Cl	Serum chloride        mmol/l
TSP	Total serum protein    g/l
PCV	Packed cell volume     ml/100ml
Hb	Haemoglobin            g/100ml
WBC	White Blood Cell count    per cmm

Results of Intestinal foreign body cases

no.	food	urine	faeces	vomit	Treat.	SCORE	App.	Temp.	Pulse rate	Pulse vol.	Resp. rate	Resp. nat.	M.M. col.	M.M. cone.	CRT.	T.T.	Abd. pain	SCORE
1	-	+	++	+++	-	15	--	103.0	90	-	32	N	Cong.	Moist	N	N	-	70
2	-	++	+	+++	-	20	--	102.8	120	-	30	Hyper	Cong.	Dry	N	-	+	50
3	+	+	+	+++	-	15	--	102.2	100	-	34	N	Cong.	Dry	3	-	+++	45
4	+	+	+	+++	-	15	--	102.4	152	-	26	N	Cong.	Moist	N	--	+	45
5	-	-	+	+++	-	5	--	103.0	130	-	35	Hyper	Cong.	Moist	N	N	++	50
6	-	+	+	+++	-	10	--	102.2	130	-	25	N	Cong.	Dry	N	-	+	45
7	-	-	-	+++	-	0	--	102.2	120	-	35	N	Cong.	Dry	3	-	+	50
8	+	++	+	+++	+	25	--	101.8	130	-	24	N	N	N	N	-	-	85
9	-	+++	+	+++	-	10	----	102.2	120	-	28	N	Cong.	Moist	N	-	-	60
10	-	++	-	+++	-	10	--	102.4	160	----	35	N	Cong.	Dry	4	----	-	35
11	-	+++	+	++	-	20	--	101.8	120	----	25	N	Cong.	Dry	N	----	-	45
12	-	+	-	++	-	10	----	101.5	150	----	40	Hyper	Pale	Dry	4	----	+	25
13	-	++	-	+++	+	10	----	102.2	135	----	34	N	Cong.	Dry	3	----	-	45
14	-	+	-	+++	-	5	----	101.2	140	----	30	N	Cong.	Dry	3	----	-	50
15	-	+	++	+++	-	15	--	102.2	180	----	40	N	Cong.	Dry	3	----	++	15
16	+	++	-	+++	-	15	----	102.2	110	-	30	N	Cong.	Dry	N	----	++	45
17	-	++	-	+++	-	10	--	100.2	140	-	40	N	Cong.	Dry	4	----	-	40
18	-	+++	++	++++	-	10	--	103.8	180	----	35	Hyper	Cong.	Dry	4	----	++	0
19	-	-	-	+++	-	-5	----	102.2	140	----	28	N	Cong.	Dry	N	----	+	25
20	-	-	-	++++	+	-10	----	103.8	160	----	28	N	Pale	Dry	8	----	+++	-10

INTESTINAL FOREIGN BODY CASES RESULTS

PRETESTINAL FOREIGN BODY CASES RESULTS										
No.	0 Hours (On Admission)					Ancillary Aids				
	Urea	Na	K	Cl	TSP	PCV	Hb	WBC	SCORE	HECAscore
1	6.9	152	4.9	120	69	46	14.4	16100	60	145
2	3.9	135	3.6	86	50	38	16.0	26200	55	125
3	5.0	147	4.0	111	58	44	15.4	8800	65	125
4	6.5	147	3.8	103	58	47	15.4	14300	60	120
5	5.8	151	4.4	107	76	48	16.6	11800	50	105
6	4.6	141	3.6	97	76	48	16.6	11000	45	100
7	11.9	141	3.4	96	81	52	15.9	32100	30	80
8	6.3	158	3.6	112	53	43	17.3	25000	50	160
9	4.5	151	4.5	91	68	52	16.6	28500	40	110
10	3.6	145	2.8	90	57	41	14.7	11700	60	105
11	5.6	135	2.8	80	73	53	18.5	11700	30	95
12	45.3	145	3.6	70	66	45	16.4	8400	25	60
13	12.1	134	2.7	64	67	52	19.4	24300	5	60
14	10.9	136	4.0	65	64	53	16.6	22000	15	50
15	5.9	149	2.8	92	69	65	21.4	13600	20	50
16	47.3	121	3.1	45	71	57	19.0	20000	-35	25
17	23.5	141	3.1	76	80	67	20.8	31000	-25	25
18	10.8	139	3.2	88	75	57	18.8	14700	10	20
19	12.2	136	2.5	73	80	69	20.7	17600	-15	5
20	5.9	151	5.0	83	49	27	7.9	41000	15	-5

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	
Temp.	103.2	103.2	102.4	102.2	102.8	102.2	102.4	102.4	101.8	102.0	102.4	101.8	102.6	100.4	102.4	102.4	101.8	102.0	103.6	102.4	102.8
Pulse rate	110	130	110	148	110	130	130	130	125	110	120	140	126	135	130	154	100	120	160	160	140
Pulse vol.	-	-	-	-	-	-	-	-	-	N	-	-	-	-	-	-	-	N	-	-	-
Resp. rate	30	34	32	28	28	30	32	29	24	34	30	32	24	40	38	30	36	45	30	32	
Resp. nat.	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Hyper	N	N
M. M. col.	Cong.	Cong.	Cong.	Cong.	Cong.	Cong.	Cong.	N	Cong.	Cong.	Cong.	Cong.	N	Cong.	Cong.	Cong.	Cong.	Cong.	Cong.	Pale	
M. M. cons.	Moist	Dry	Dry	Moist	Dry	Dry	Dry	N	Moist	N	Moist	N	N	Moist	Dry	Moist	Moist	Moist	Moist	Moist	
CRT.	N	3	3	N	N	N	3	N	N	N	N	N	N	N	3	N	N	6	N	3	
T. T.	N	-	-	-	N	-	-	-	N	N	N	-	-	-	-	-	-	-	-	-	-
Abd. pain	-	+	+	+	+	+	+	-	-	-	-	+	-	-	+	-	-	-	++	+	++
SCOR.	65	40	50	45	60	45	45	85	80	85	65	75	70	60	35	65	80	10	30	40	

INTESTINAL FOREIGN BODY CASES RESULTS

## 12 Hours post-operatively Ancillary Aids

No.	Urea	Na	K	Cl	TSP	PCV	Hb	WBC	SCORE	HECAsocore
1	6.2	140	4.1	103	71	47	15.4	18000	55	155
2	2.4	139	4.2	80	56	36	15.2	28400	50	115
3	5.8	155	5.3	99	60	58	16.9	26900	35	100
4	4.8	144	3.3	108	64	45	14.8	16700	60	135
5	6.4	148	4.1	101	79	49	17.0	14200	45	140
6	3.5	152	3.6	98	80	56	19.4	12800	35	100
7	14.2	141	3.2	91	80	50	15.2	26300	30	95
8	6.2	152	3.3	110	54	44	17.6	26300	45	175
9	4.2	150	4.2	98	64	47	14.2	26200	55	175
10	3.1	145	3.0	100	52	43	14.2	17700	60	185
11	5.6	136	3.5	91	53	46	14.1	16500	60	170
12	14.0	155	4.4	85	61	40	13.2	16200	55	165
13	13.7	131	3.1	83	41	35	13.2	14300	40	145
14	3.7	146	4.1	93	64	41	13.2	30900	60	155
15	8.8	144	2.5	105	56	47	15.7	18300	50	125
16	22.5	135	3.1	70	68	51	16.2	24200	15	105
17	8.7	146	3.2	90	72	54	16.3	42100	25	140
18	11.6	139	3.5	84	60	51	16.2	15000	35	85
19	11.9	147	2.5	75	58	53	16.9	18100	15	75
20	4.9	131	3.7	117	50	21	6.3	54300	10	55



No.	Sex	Age	Wt.	Hgt.	Temp.	Pulse rate	Pulse vol.	Resp. rate	Resp. nat.	M.M. col.	M.M. cons.	CRT.	T.T.	Abd. pain	Score
1	-	+++	++	+	-	40	--	102.8	100	N	28	N	N	-	95
2	-	++	++	-	-	40	--	102.6	110	-	24	N	Cong.	Dry	60
3	-	++	+	-	-	35	--	102.4	120	-	30	N	Cong.	Moist	70
4	-	++	+	+	-	30	--	102.4	140	-	30	N	Cong.	N	55
5	-	+	+	-	-	30	--	102.6	120	-	32	N	Cong.	N	70
6	-	+	-	+	-	15	--	101.4	130	-	24	N	Cong.	Moist	70
7	-	+	+	-	-	30	--	103.2	130	-	30	N	Cong.	Dry	50
8	++	+++	++	-	-	55	-	102.8	110	N	26	N	N	-	100.
9	+	+++	++	-	-	45	-	102.6	120	N	30	N	N	-	100
10	++	+++	++	+	-	50	N	100.4	108	N	30	N	N	-	110
11	+	++	++	-	-	55	-	100.4	92	N	20	N	N	-	105
12	++	++	++	+	-	55	N	102.2	85	N	26	N	N	-	105
13	+	++++	+++	-	-	30	-	100.4	115	N	28	N	N	-	105
14	+	+++	++	-	-	40	N	100.6	94	N	30	N	N	-	110
15	++	++	++	+	-	55	-	101.8	120	N	34	N	Cong.	Moist	85
16	+	++	++	-	-	45	--	102.2	110	N	32	N	Cong.	Moist	75
17	++	++	++	-	-	50	N	101.2	112	N	36	N	N	-	110
18	-	+++	+++	+	-	35	-	103.6	110	N	32	N	N	+	85
19	+	+++	++	+	-	45	--	103.2	120	N	30	N	Cong.	N	65
20	-	+	+	-	+	10	--	102.2	150	-	32	N	Pale	Moist	60

INTESTINAL FOREIGN BODY CASES RESULTS

## 24 Hours post-operatively

## Ancillary Aids

No.	Urea	Na	K	Cl	TSP	PCV	HB	WBC	SCORE	HECAscore
1	5.2	140	4.1	113	5.8	42	14.1	20100	65	200
2	3.4	153	3.9	100	61	46	13.6	47200	55	155
3	4.1	160	4.7	103	60	48	16.7	16900	55	160
4	11.3	157	4.7	107	68	48	16.4	30200	35	120
5	7.2	146	4.1	104	81	51	18.6	23100	30	135
6	7.7	155	3.8	102	73	48	16.8	14100	45	130
7	16.8	138	3.3	90	86	54	16.4	48200	15	95
8	4.3	154	4.1	111	51	40	14.2	14000	70	225
9	3.8	147	4.1	103	62	42	12.3	28000	70	215
10	2.2	151	4.3	112	49	40	13.2	15500	70	230
11	4.2	141	4.1	103	50	42	13.4	21900	70	230
12	12.4	168	4.4	84	62	45	15.3	33400	35	195
13	7.1	140	3.6	93	59	40	13.8	25700	60	195
14	2.3	150	4.1	96	40	31	9.7	21000	65	215
15	5.5	151	4.7	95	88	54	17.1	24000	45	185
16	18.3	141	2.6	89	65	47	12.6	22000	55	175
17	6.1	150	4.2	100	54	46	14.0	39000	55	215
18	8.0	141	3.0	102	61	54	16.2	26100	50	170
19	8.9	148	2.9	84	58	50	17.2	29100	30	140
20	3.2	157	3.6	113	41	23	6.6	31000	20	90

Case	Time	Temp.	Pulse	Pulse rate	Resp. rate	Resp. nat.	M.M. col.	M.M. cons.	CRT.	T.T.	Abd. pain	Score
1	+	++	++	90	N	22	N	N	N	N	-	105
2	-	+++	++	120	N	25	N	N	N	N	-	100
3	-	++	++	110	N	28	N	N	N	N	-	100
4	+	++	++	105	N	30	N	Cong.	N	N	-	85
5	-	+++	+	115	N	28	N	N	N	N	-	100
6	+	+	+	120	N	24	N	Cong.	Moist	N	-	70
7	-	++	+	120	-	26	N	Cong.	Moist	N	-	75
8	+++	++	++	90	N	26	N	N	N	N	-	110
9	++	++	++	110	N	24	N	N	N	N	+	110
10	++	++	++	104	N	30	N	N	N	N	-	110
11	++	++	++	96	N	20	N	N	N	N	-	110
12	+++	++	++	90	N	28	N	N	N	N	-	110
13	++	++++	+++	95	N	20	N	N	N	N	-	110
14	++	+++	++	100	N	32	N	N	N	N	-	110
15	+++	++	++	110	N	30	N	N	N	N	-	110
16	+	++	++	85	N	28	N	N	N	N	+	85
17	++	++	++	95	N	32	N	N	N	N	-	110
18	++	++	++	98	N	24	N	N	N	N	-	105
19	++	+++	++	80	N	22	N	N	N	N	-	105
20	-	++	++	125	N	30	N	Pale	N	N	+	75

INTESTINAL FOREIGN BODY CASES RESULTS

## 48 Hours post-operatively

## Ancillary Aids

No	Urea	Na	K	Cl	TSP	PCV	Hb	WBC	SCORE	HECAscore
1	4.5	149	4.3	111	54	41	13.9	23200	70	225
2	3.6	140	4.2	102	59	44	14.0	42900	60	195
3	4.6	151	5.0	105	55	41	13.6	14200	75	220
4	4.5	151	3.2	102	49	35	12.0	42000	50	185
5	7.3	150	4.1	115	59	35	12.2	11600	65	205
6	14.1	155	3.2	103	80	58	19.4	21000	10	120
7	9.2	141	3.1	93	76	45	14.2	33800	35	145
8	3.8	161	3.6	117	46	38	12.9	16100	70	235
9	2.2	154	4.8	110	49	41	12.6	23500	65	235
10	2.1	151	4.4	113	5.0	41	13.3	9200	80	250
11	3.6	151	4.1	101	50	41	13.1	11800	75	245
12	7.0	151	3.9	94	65	44	14.9	28900	50	215
13	3.8	149	4.1	103	60	41	13.1	28700	70	220
14	3.1	149	4.0	97	41	32	9.8	21000	65	225
15	5.2	149	4.1	101	63	48	15.8	9600	65	225
16	10.0	145	2.9	92	66	39	12.2	32400	50	180
17	6.4	141	4.4	98	49	44	12.8	32000	60	225
18	5.1	152	3.3	110	58	42	13.5	32300	65	230
19	7.2	151	3.1	96	56	46	15.0	24600	50	205
20	2.2	139	3.7	112	4.4	29	7.5	49200	25	135

No.	Food	Fluid	Urine	Faeces	Vomit	Treat.	SCORE	App.	Temp.	Pulse rate	Pulse vol.	Resp. rate	Resp. nat.	M.M. col.	M.M. cons.	CRT.	T.T.	Abd. pain	SCORE
1	++	++	++	+	-	-	55	N	100.4	70	N	18	N	N	N	N	N	-	110
2	++	++	++	+	-	-	55	N	101.5	130	N	28	N	N	N	N	N	-	110
3	+	++	++	+	-	-	50	-	101.4	110	N	28	N	N	N	N	N	-	105
4	++	++	++	++	-	-	60	N	101.5	85	N	30	N	N	N	N	N	-	110
5	+	+++	++	+	-	-	45	-	101.4	110	N	24	N	N	N	N	N	-	105
6	++	++	++	+	-	-	55	--	101.8	120	N	39	N	N	N	N	N	-	95
7	+	+++	++	-	-	-	40	--	102.0	110	N	24	N	N	N	N	N	-	100
8	Discharged																		
9	Discharged																		
10	Discharged																		
11	Discharged																		
12	+++	++	++	++	-	-	55	N	101.8	90	N	32	N	N	N	N	N	-	110
13	++	++	++	++	-	-	60	N	100.6	98	N	28	N	N	N	N	N	-	110
14	Discharged																		
15	+++	++	++	++	-	-	55	N	101.4	112	N	28	N	N	N	N	N	-	110
16	++	++	++	++	-	-	60	N	101.8	92	N	34	N	N	N	N	N	-	110
17	Discharged																		
18	++	++	++	++	-	-	60	N	101.2	90	N	35	N	N	N	N	N	-	110
19	+++	++	++	++	-	-	55	N	101.8	90	N	30	N	N	N	N	N	-	110
20	-	++	++	+	++	-	45	--	102.2	130	N	28	N	Pale	N	N	N	+++	65

# INTESTINAL FOREIGN BODY CASES RESULTS

## 72 Hours post-operatively

## Ancillary Aids

No.	Urea	Na	K	Cl	TSP	PCV	Hb	WBC	SCORE	HECAscore
1	3.8	142	4.0	113	56	42	14.2	14800	70	235
2	2.6	141	3.3	110	57	39	13.2	33000	60	220
3	3.7	160	4.6	109	58	42	13.6	14000	75	230
4	4.2	148	3.3	110	51	42	13.1	12000	70	240
5	4.4	148	3.3	110	51	42	13.1	12200	70	240
6	9.7	153	3.8	93	75	50	16.2	23400	35	185
7	7.5	142	3.2	101	61	36	11.4	26000	55	195
8	Discharged									
9	Discharged									
10	Discharged									
11	Discharged									
12	6.3	159	3.1	92	63	42	13.1	16200	60	225
13	2.4	151	4.0	112	60	40	12.9	14200	75	245
14	Discharged									
15	5.4	151	4.2	103	60	42	12.2	8400	80	245
16	6.2	142	3.4	105	62	41	12.4	17600	65	235
17	Discharged									
18	3.6	150	3.8	115	58	41	12.8	22300	70	240
19	6.7	147	3.5	104	52	41	13.2	11000	70	235
20	2.1	158	4.3	109	44	24	6.6	27500	25	135

No.	Food	Fluid	Urine	Faeces	Vomit	Treat.	SCORE	App.	Temp.	Pulse rate	Pulse vol.	Resp. rate	Resp. nat.	M.M. col.	M.M. cons.	CAT.	T.T.	Abd. pain	SCORE
1	Discharged																		
2	+++	++	++	++	-	-	55	N	101.4	110	N	30	N	N	N	N	N	-	110
3	++	+++	++	++	-	-	55	N	101.4	110	N	30	N	N	N	N	N	-	110
4	Discharged																		
5	++	++	+	++	-	-	55	N	101.0	100	N	30	N	N	N	N	N	-	110
6	+++	+++	++	++	-	-	50	N	101.2	120	N	32	N	N	N	N	N	-	110
7	+	++	++	-	-	-	50	N	101.5	92	N	24	N	N	N	N	N	-	110
8																			
9																			
10																			
11																			
12	Discharged																		
13	Discharged																		
14																			
15	Discharged																		
16	+++	+++	++	++	-	-	50	N	101.4	90	N	32	N	N	N	N	N	-	110
17																			
18	++	++	++	++	-	-	60	N	101.4	80	N	32	N	N	N	N	N	-	110
19	Discharged																		
20	-	-	+	++	++++	+	5	----	96.4	90	-	26	N	Pale	N	N	N	+++	50

subsequently died.

INTESTINAL FOREIGN BODY CASES RESULTS

96 Hours post-operatively

Ancillary Aids

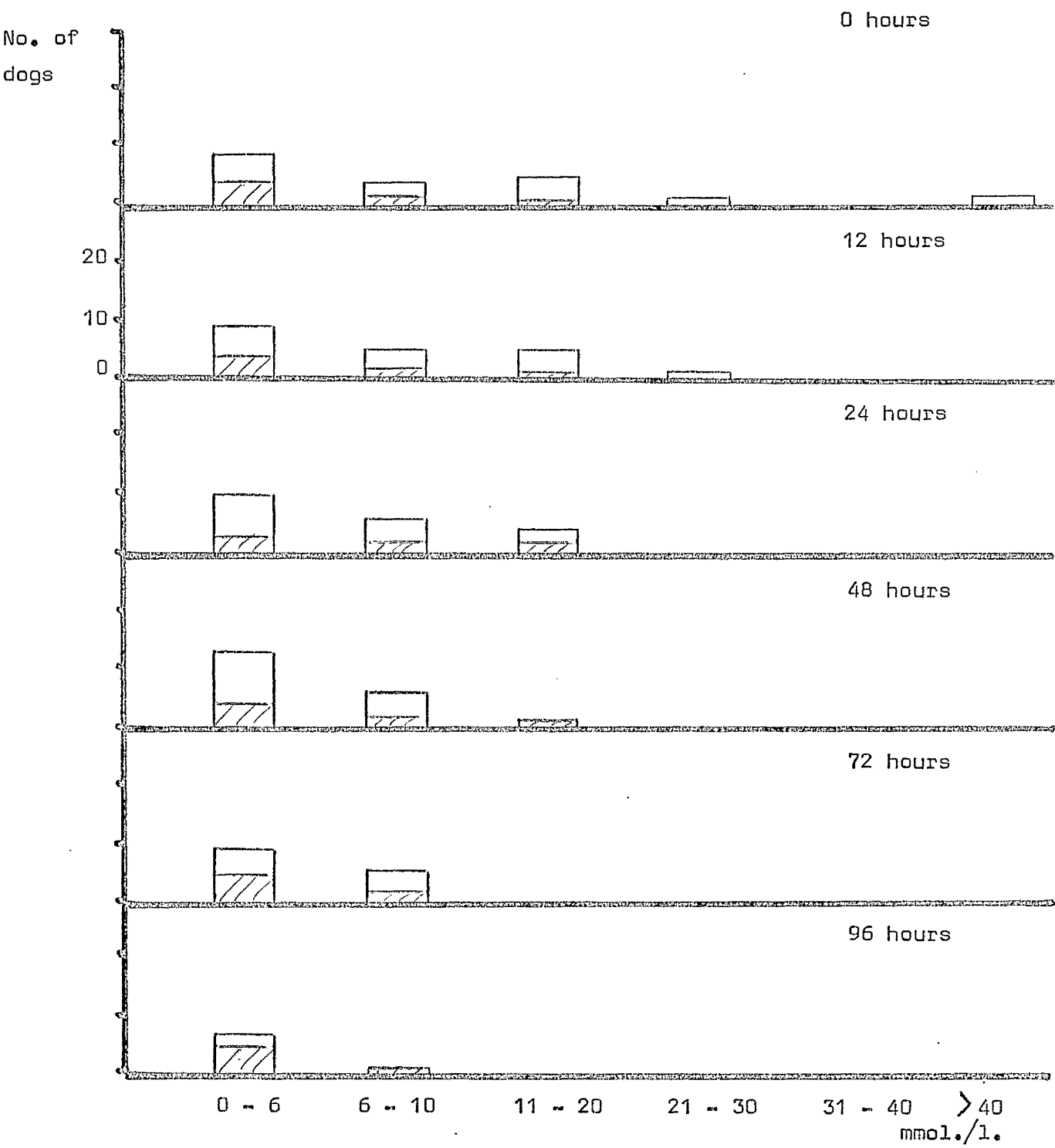
No.	Urea	Na	K	Cl	TSP	PCV	Hb	WBC	SCORE	HECAscore
1	Discharged									
2	3.2	140	3.0	107	58	40	13.4	25800	70	235
3	3.4	151	4.2	108	57	42	13.2	11000	75	240
4	Discharged									
5	2.8	151	3.6	108	58	42	13.5	15700	75	240
6	6.2	151	4.1	105	64	43	12.8	15600	65	225
7	5.1	151	3.8	116	58	32	9.4	23000	65	225
8										
9										
10										
11										
12	Discharged									
13	Discharged									
14										
15	Discharged									
16	5.3	142	3.8	103	61	39	12.2	10800	75	235
17										
18										
19	Discharged									
20	4.2	163	4.3	116	44	23	6.8	3600	25	80



Histograms of Ancillary aids - intestinal foreign body cases

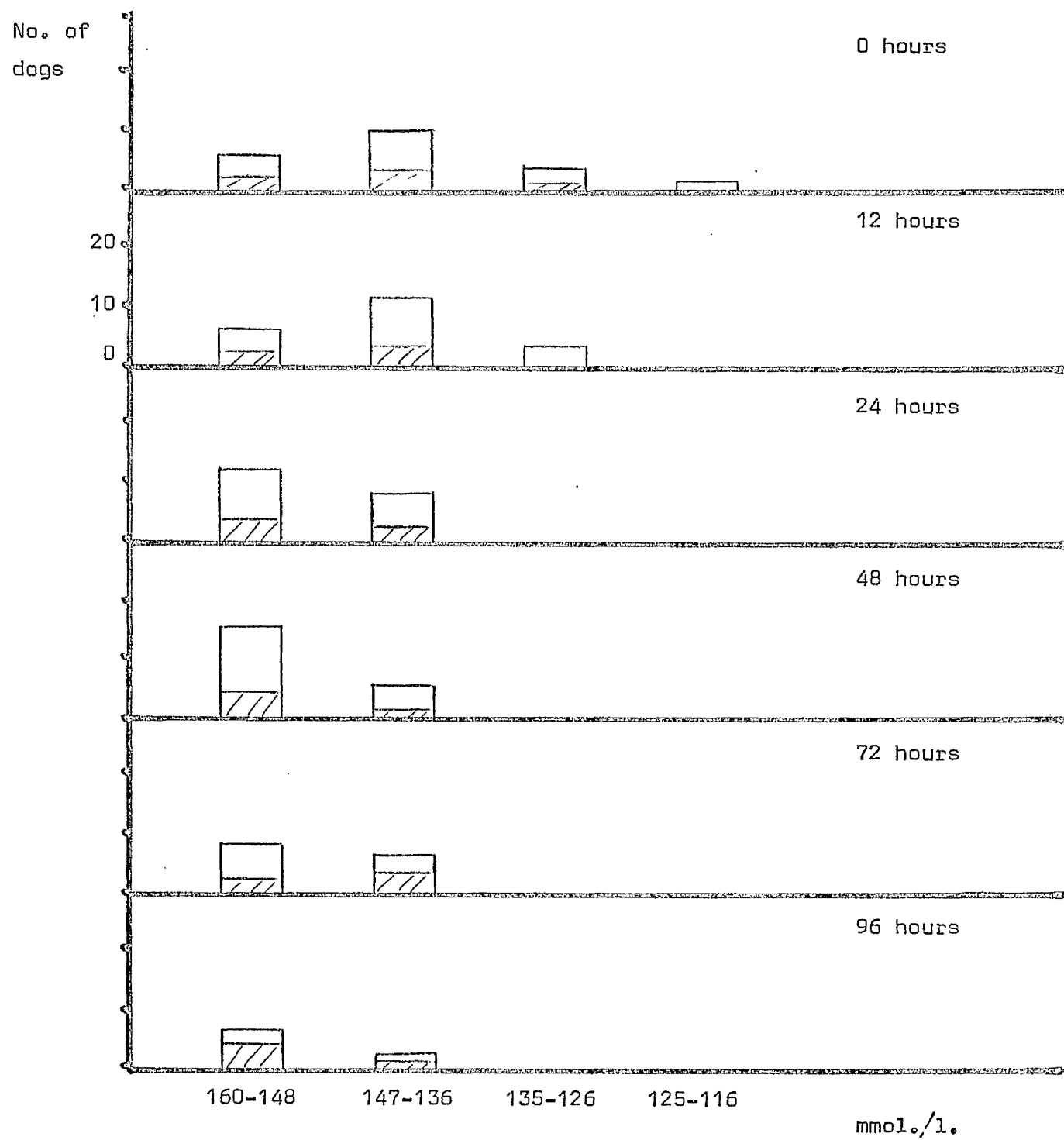
Histogram 1.

Blood urea



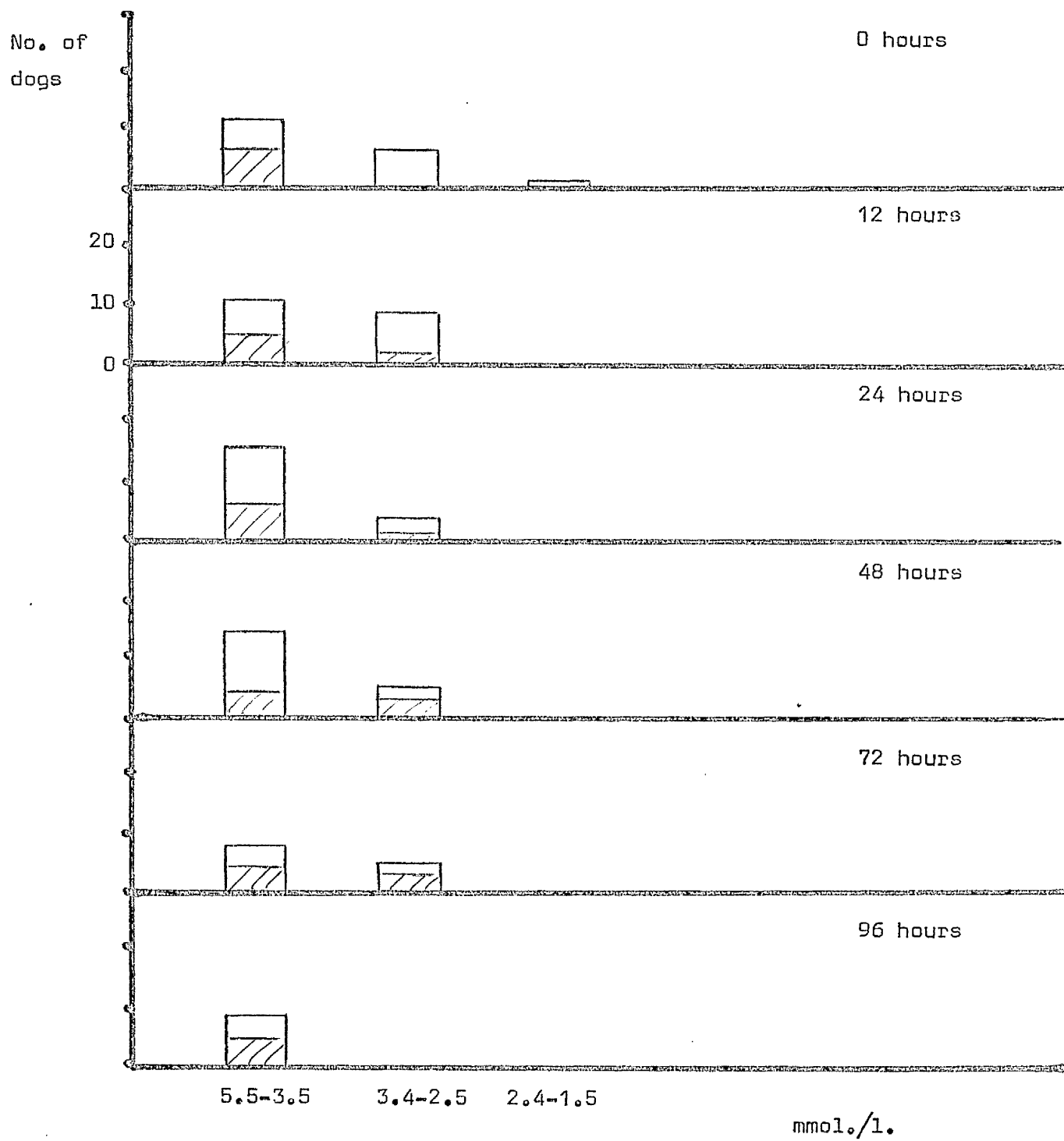
Histogram 2.

Plasma sodium



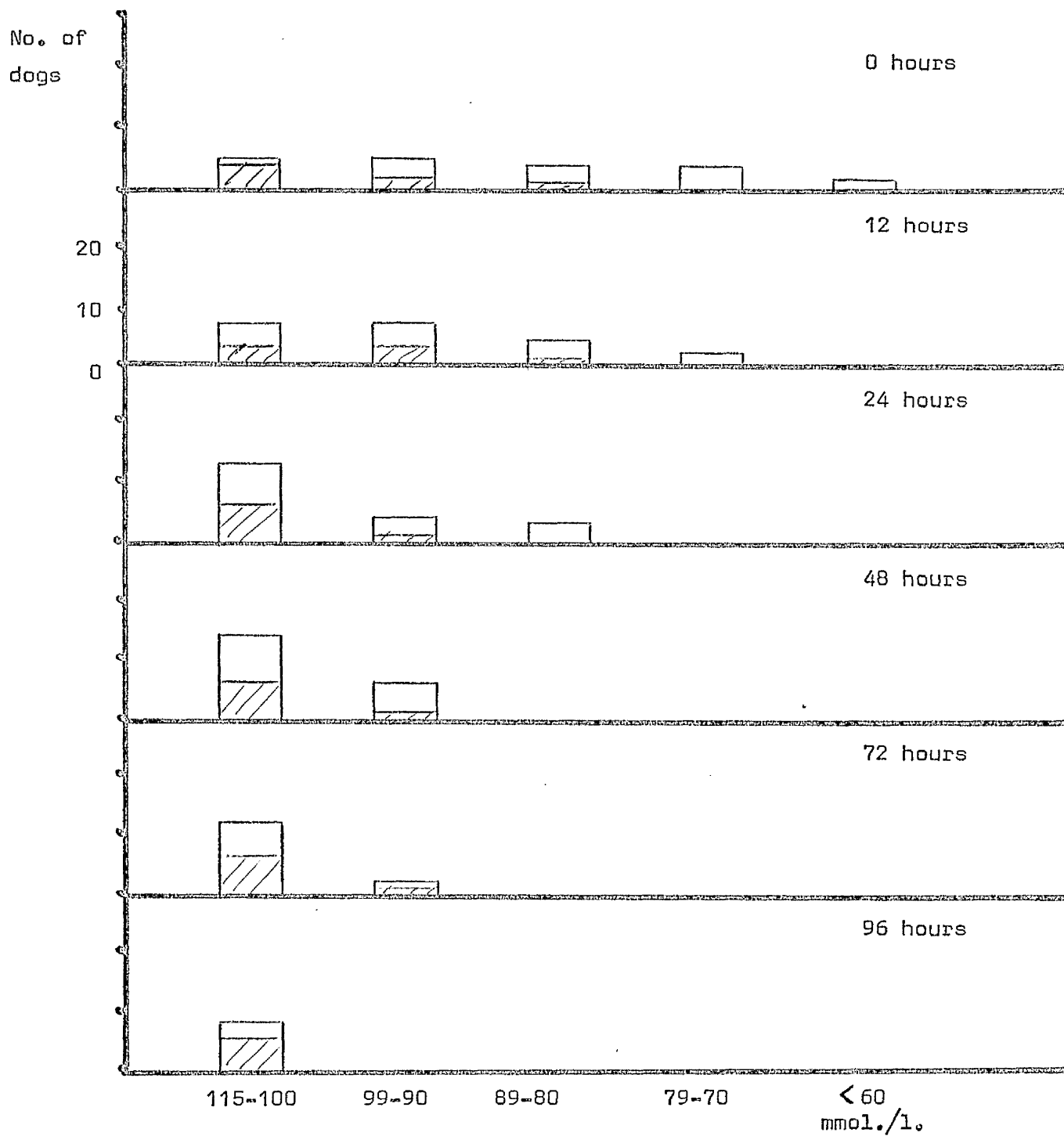
Histogram 3.

Plasma potassium



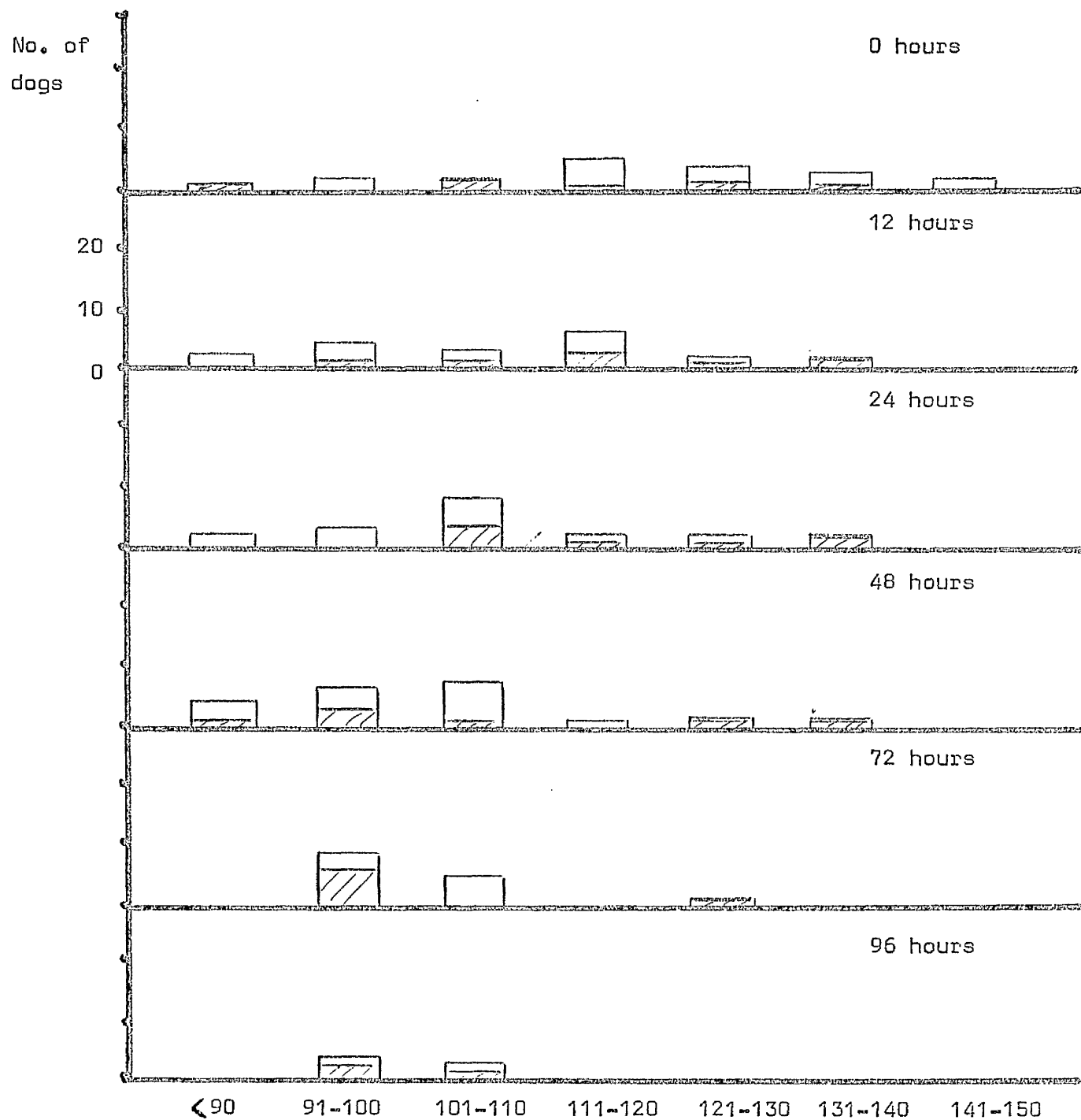
Histogram 4.

Plasma chloride



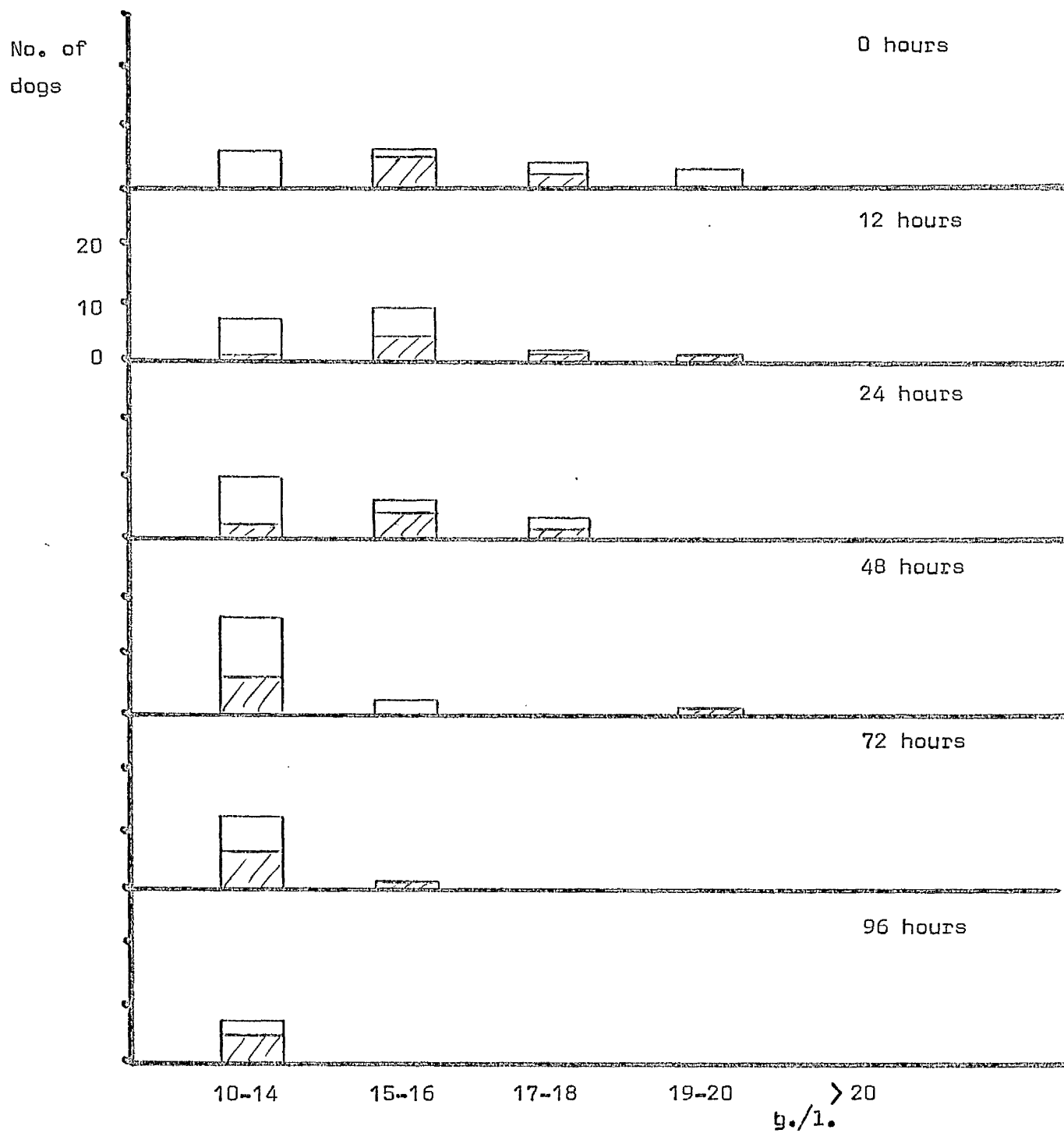
Histogram 5.

T.S.P.+ P.C.V.



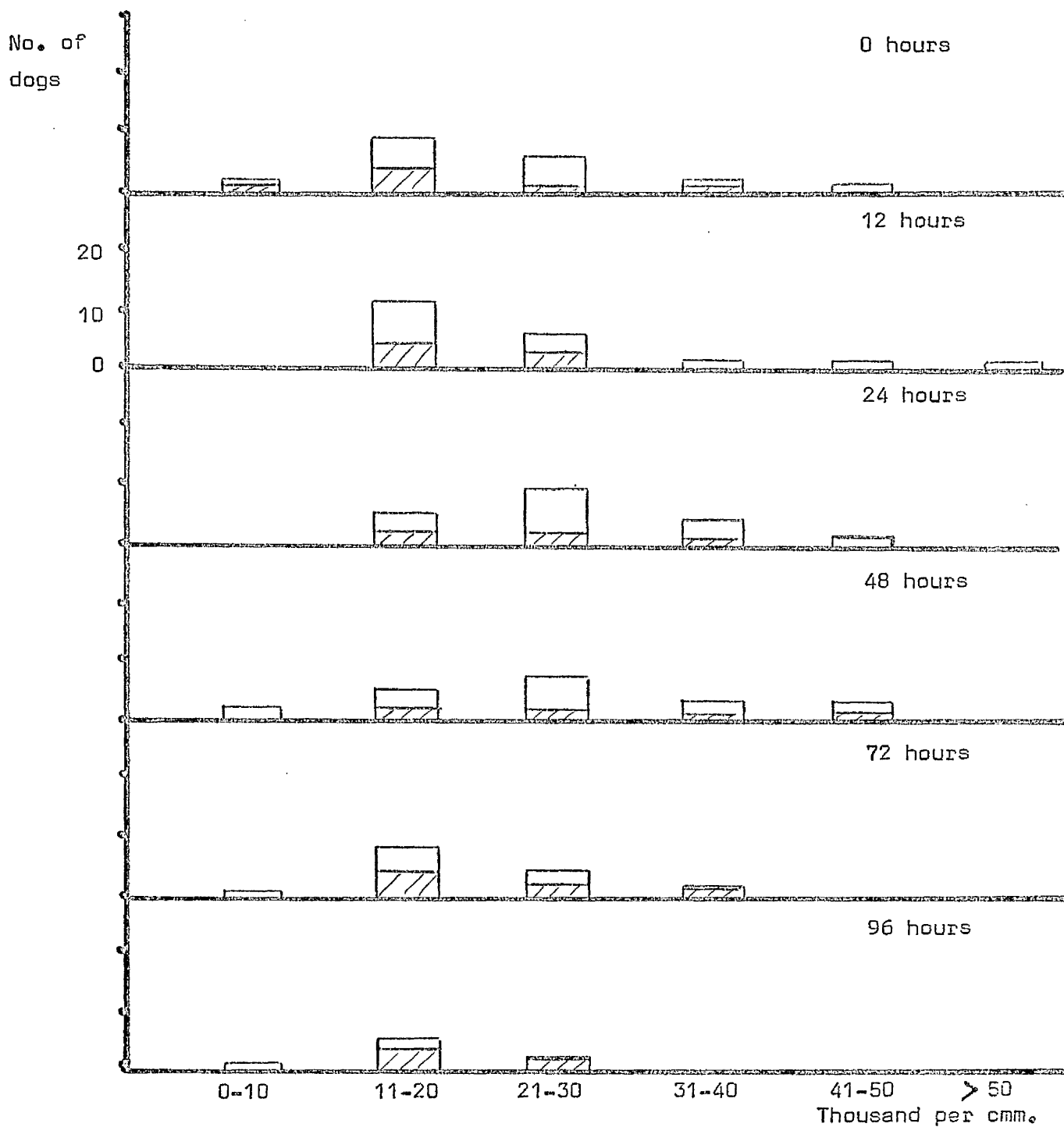
Histogram 6.

Haemoglobin



Histogram 7.

White blood cell count





Results of Pyometritis cases

PYOMETRITIS CASES RESULTS

0 Hours (On Admission)

Ancillary Aids

No.	Urea	Na	K	Cl	TSP	PCV	Hb	WBC	SCORE	HECAscore
1	5.3	158	4.6	115	75	38	11.9	21400	65	205
2	10.9	150	4.1	107	77	35	11.7	20600	50	185
3	4.7	152	4.0	116	87	26	7.9	34600	30	160
4	4.7	158	3.8	107	72	36	12.5	37600	55	150
5	4.1	142	3.1	93	68	30	10.7	16300	55	130
6	5.6	148	3.8	103	82	44	16.1	14100	50	130
7	5.6	151	4.0	105	82	35	11.6	59700	40	125
8	5.9	155	4.4	113	89	44	14.5	33400	45	90
9	7.2	145	3.4	105	73	34	14.7	21100	45	180
10	6.5	141	2.9	93	73	33	12.6	19500	50	170
11	3.3	166	4.3	115	71	33	11.3	47000	50	150
12	2.7	162	3.3	106	74	34	10.7	33400	50	130
13	4.9	146	3.6	106	71	46	21.4	20000	45	125
14	4.6	153	4.3	118	69	40	11.9	17100	75	125
15	4.4	158	4.1	104	70	42	14.4	54000	50	115
16	10.1	150	4.1	107	53	49	14.8	53900	30	100
17	18.1	154	5.1	119	90	35	11.9	27700	45	95
18	5.9	142	3.7	104	78	50	16.8	85500	30	65
19	6.5	157	4.5	112	71	30	9.1	61300	30	55
20	9.2	149	3.8	100	67	49	15.4	38300	45	40
21	44.0	147	3.1	80	98	52	19.2	55400	-25	-10
22	15.1	144	3.0	112	61	47	15.7	111000	30	-15
23	51.4	133	4.1	89	92	47	16.0	80400	-10	-20

PYOMETRITIS CASES RESULTS

## 12 Hours post-operatively

## Ancillary Aids

No.	Urea	Na	K	Cl	TSP	PCV	Hb	WBC	SCORE	HECAscore
1	3.0	154	3.3	103	76	39	12.3	28200	60	180
2	7.3	150	4.2	107	83	31	9.7	35000	35	175
3	1.6	146	3.8	106	88	20	6.0	30700	15	145
4	4.4	156	3.5	115	68	27	9.4	19900	60	135
5	4.3	142	3.6	103	66	33	11.0	41000	55	150
6	3.2	147	3.5	108	60	32	10.4	14500	65	155
7	3.7	143	3.6	112	77	29	9.7	41600	40	140
8	5.7	151	4.6	109	84	42	14.1	52000	40	100
9	4.2	151	4.4	109	64	38	12.0	68000	55	140
10	4.8	143	3.2	97	74	36	14.2	28200	45	180
11	3.6	157	4.4	118	72	35	12.4	28100	60	180
12	3.2	151	3.6	112	81	36	11.9	22100	60	160
13	5.2	151	3.8	115	70	42	18.6	32000	50	155
14	5.6	151	4.6	108	70	42	12.3	29300	70	145
15	4.2	151	4.4	109	64	38	12.0	68000	55	140
16	5.7	147	3.5	105	70	36	10.4	75400	50	160
17	16.4	152	3.8	105	80	30	8.4	38000	30	90
18	5.4	159	4.1	107	83	45	14.7	76800	35	65
19	6.8	148	4.8	109	73	33	11.3	94200	40	85
20	11.3	151	3.4	101	69	50	14.2	21800	45	55
21	46.5	139	2.1	88	92	51	18.6	84200	-30	50
22	17.2	146	5.8	113	60	48	16.2	132000	25	-30
23	50.5	140	5.4	98	75	40	13.2	65100	15	45

PYOMETRITIS CASES RESULTS

## 48 Hours post-operatively

## Ancillary Aids

No.	Urea	Na	K	Cl	TSP	PCV	Hb	WBC	SCORE	HECAscore
1	5.7	151	3.9	100	70	33	11.0	50000	55	170
2	6.2	148	4.0	108	74	33	11.6	31000	55	185
3	4.6	149	4.7	110	81	25	7.2	23700	35	165
4	4.4	151	2.9	119	68	29	9.8	79000	35	170
5	4.5	146	3.7	104	65	38	12.2	37900	65	200
6	4.6	148	3.4	110	63	38	13.2	18400	70	205
7	4.8	141	3.8	92	76	26	8.5	98400	25	170
8	4.5	151	3.8	109	73	38	11.8	43900	55	145
9	Discharged									
10	4.1	152	3.6	108	62	34	10.8	6200	75	245
11	2.9	157	4.7	113	65	34	8.2	17600	65	235
12	5.4	147	3.7	113	71	35	9.1	52900	40	205
13	3.9	162	4.3	105	70	40	14.7	20800	65	230
14	4.1	150	4.4	111	68	38	12.2	18900	75	245
15	5.1	156	4.3	115	68	40	14.7	31000	65	225
16	4.3	156	4.6	115	57	39	12.8	21700	70	235
17	3.2	156	4.4	120	70	33	10.0	14200	70	220
18	5.4	157	4.6	115	78	40	12.6	25400	65	210
19	9.6	150	4.7	108	67	28	7.5	71900	30	170
20	Discharged									
21	14.6	139	2.8	103	84	40	10.8	69000	30	150
22	Discharged									
23	34.5	151	3.4	92	81	45	14.4	98400	10	130

No.	Food	Fluid	Urine	Faeces	Vomit	Treat.	SCORE	App.	Temp.	Pulse rate	Pulse vol.	Resp. rate	Resp. nat.	M.M. col.	M.M. cons.	CRT.	T.T.	Abd. pain	SCORE
1	+	++	++	+	-	-	50	-	102.0	90	N	36	N	N	N	N	N	-	105
2	-	++	++	++	-	-	40	--	101.0	110	N	30	N	N	N	N	N	-	90
3	++	+++	++	++	-	-	55	-	101.6	120	N	22	N	Pale	N	N	N	-	100
4	-	+++	++	-	-	-	35	--	100.0	115	N	32	N	Pale	N	N	N	-	95
5	++	++	++	++	-	-	60	N	101.4	118	N	30	N	N	N	N	N	-	110
6	++	+++	++	++	-	-	55	-	102.2	140	N	32	N	N	N	N	N	-	95
7	+	++	++	+	-	-	50	-	101.4	90	N	20	N	Pale	N	N	N	-	100
8	+	+++	++	-	-	-	40	-	101.8	120	N	20	N	N	N	N	N	-	105
9																			
10	Discharged																		
11	Discharged																		
12	Discharged																		
13	Discharged																		
14	Discharged																		
15	Discharged																		
16	Discharged																		
17	Discharged																		
18	++	+++	++	++	-	-	55	N	101.3	95	N	24	N	N	N	N	N	-	110
19	++	+++	++	++	-	-	55	N	101.8	84	N	36	N	N	N	N	N	-	110
20																			
21	++	+++	+++	++	-	-	50	N	102.4	98	N	24	N	N	N	N	N	-	105
22																			
23	+	++++	+++	+	-	-	35	-	101.5	95	N	38	N	N	N	N	N	-	105

PYOMETRITIS CASES RESULTS

72 Hours post-operatively Ancillary Aids

No.	Urea	Na	K	Cl	TSP	PCV	Hb	WBC	SCORE	HECAscore
1	5.3	136	3.7	105	67	31	10.0	26800	60	215
2	5.2	155	5.3	106	74	27	8.2	61000	30	160
3	2.0	141	3.8	107	80	20	7.5	15600	40	195
4	4.6	148	3.3	111	70	31	10.2	64200	40	170
5	4.0	144	4.5	104	66	33	11.6	10700	70	240
6	3.2	147	3.4	107	67	41	13.8	28600	70	220
7	4.6	146	3.2	105	70	26	7.1	58000	35	175
8	4.2	142	4.1	119	75	35	11.3	20000	65	210
9										
10	Discharged									
11	Discharged									
12	Discharged									
13	Discharged									
14	Discharged									
15	Discharged									
16	Discharged									
17	Discharged									
18	4.6	152	4.8	111	70	38	12.2	36000	65	230
19	6.9	159	4.1	118	70	28	9.5	50400	35	200
20										
21	8.2	153	3.1	112	85	41	11.9	80000	35	190
22										
23	25.3	138	2.6	105	74	41	12.2	85300	30	170

PYOMETRITIS CASES RESULTS

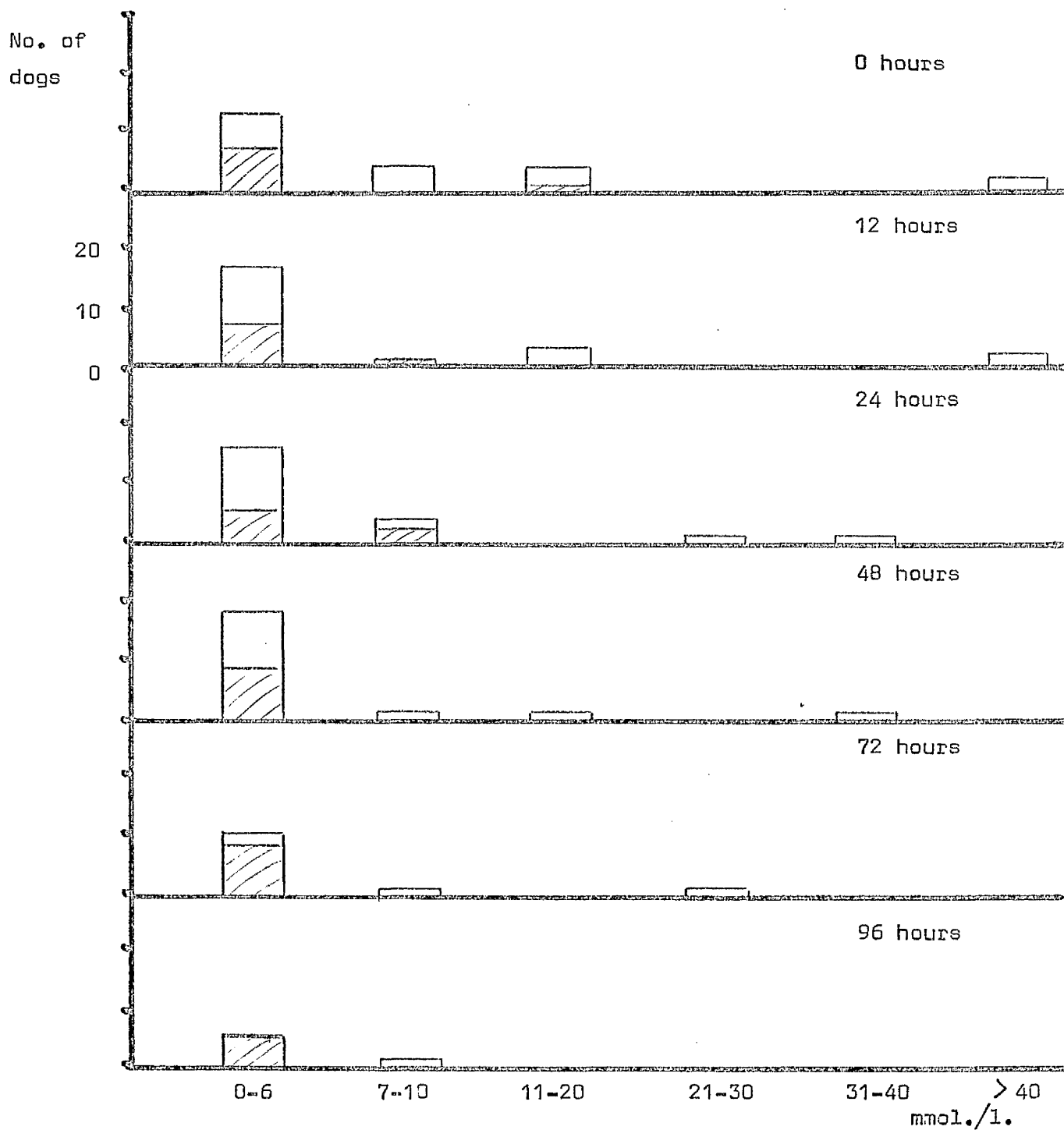
96 Hours post-operatively										Ancillary Aids	
<u>UROMETRITIS CASES RESULTS</u>											
No.	Urea	Na	K	Cl	TSP	PCV	Hb	WBC	SCORE	HECAscore	
1	Discharged										
2	4.8	150	4.6	113	68	26	8.2	16900	50	200	
3	2.2	147	3.8	112	80	21	8.1	14400	35	190	
4	Discharged										
5	3.8	146	4.8	112	61	33	11.8	14600	70	240	
6	2.3	156	3.8	104	63	38	12.2	26800	70	240	
7	4.8	141	3.7	115	68	25	6.9	38000	40	195	
8	Discharged										
9											
10											
11											
12											
13											
14											
15											
16											
17											
18	Discharged										
19	Discharged										
20											
21	Discharged										
22											
23	7.4	158	3.1	109	71	37	11.0	24500	50	200	

Histograms of Ancillary aids - pyometritis cases



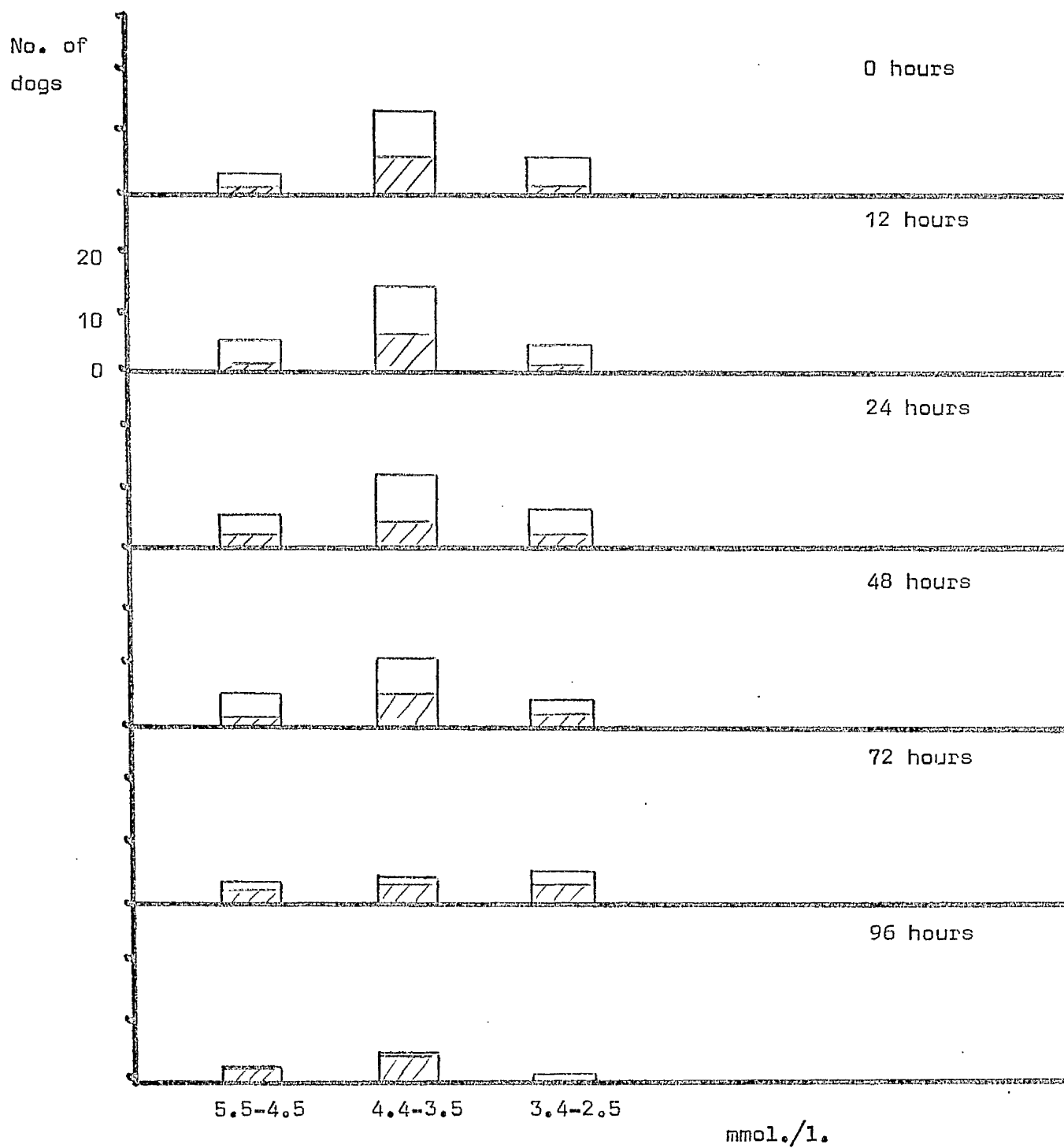
Histogram 8.

blood urea



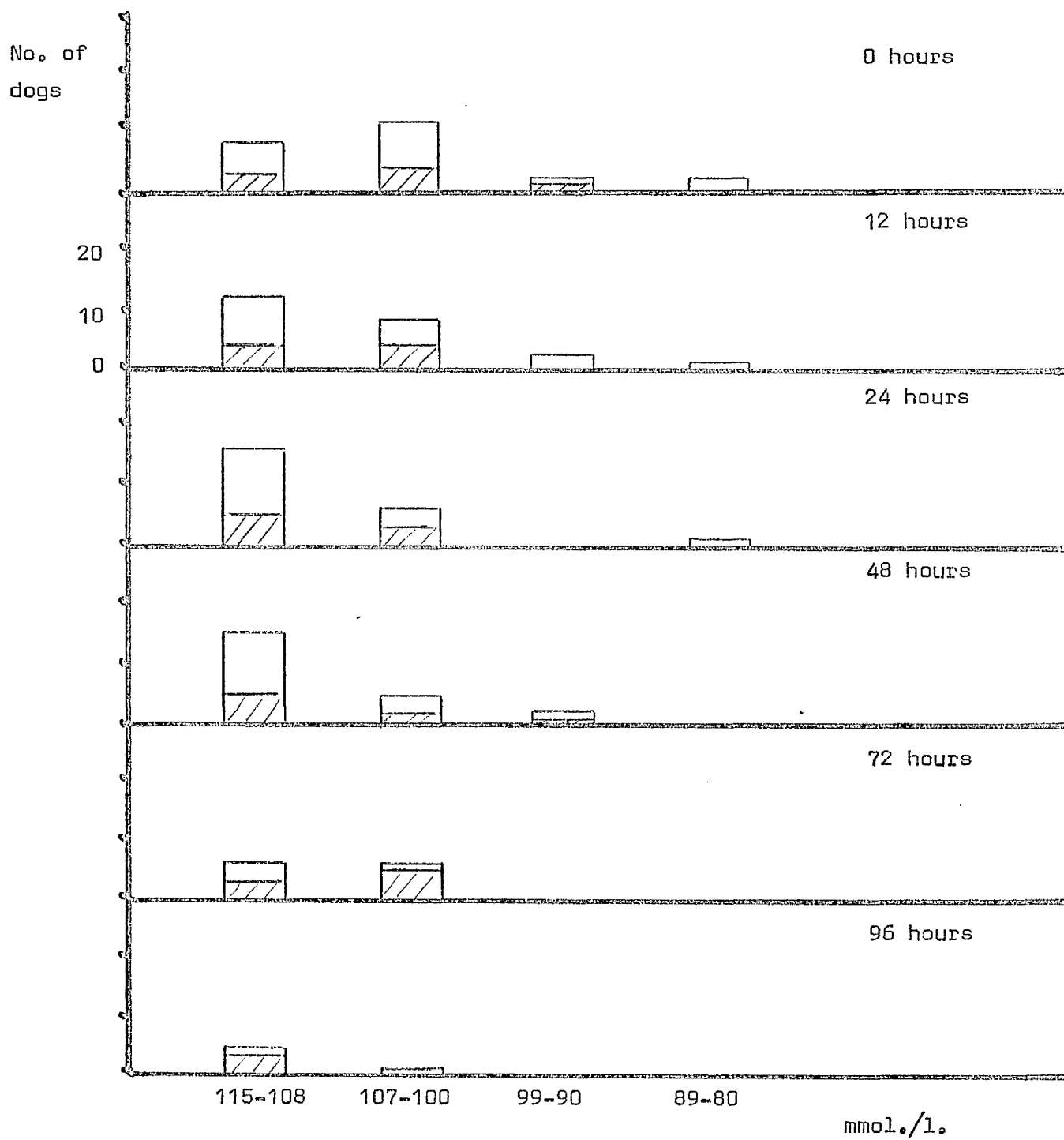
Histogram 9.

plasma potassium



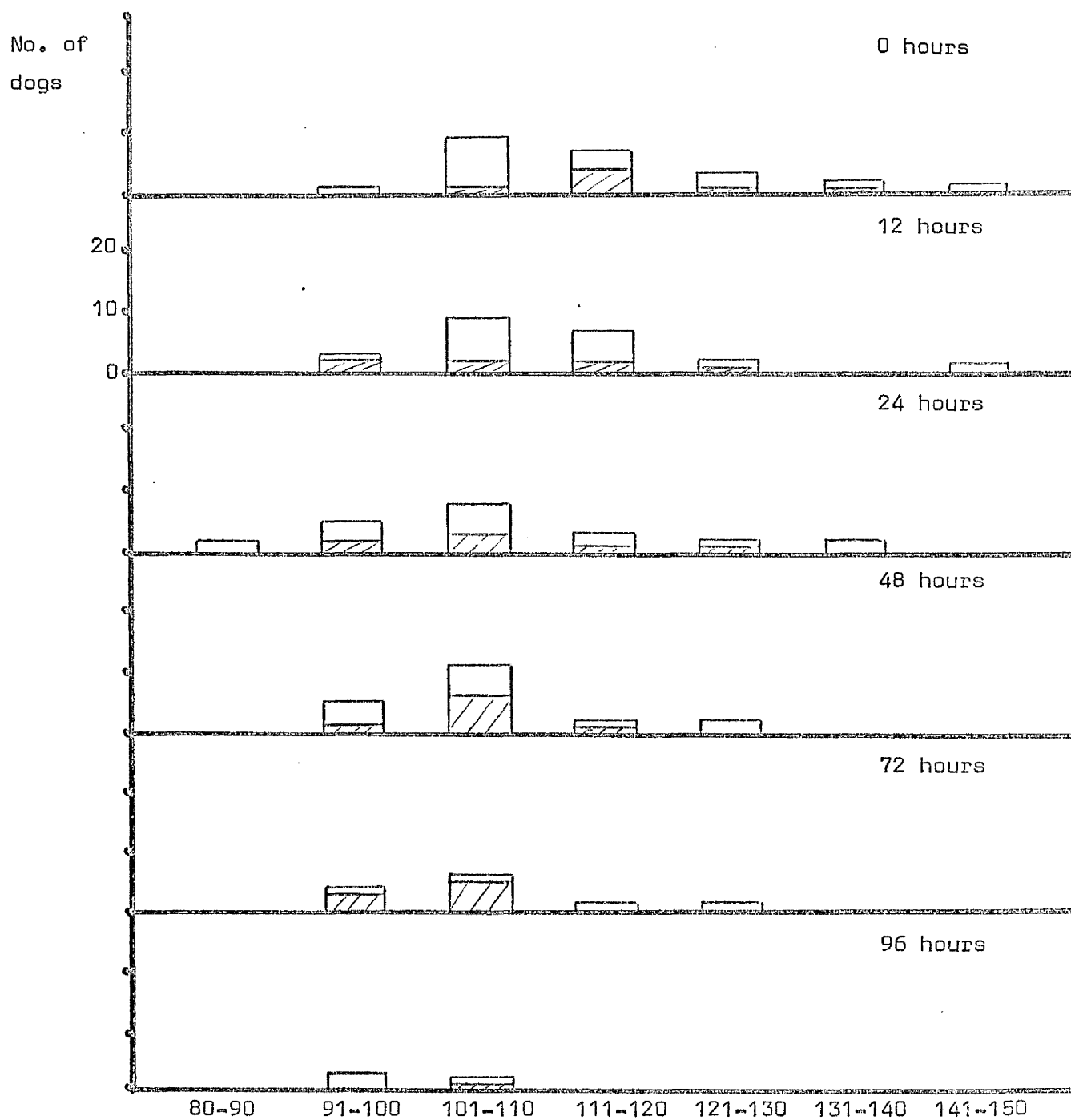
Histogram 10.

plasma chloride



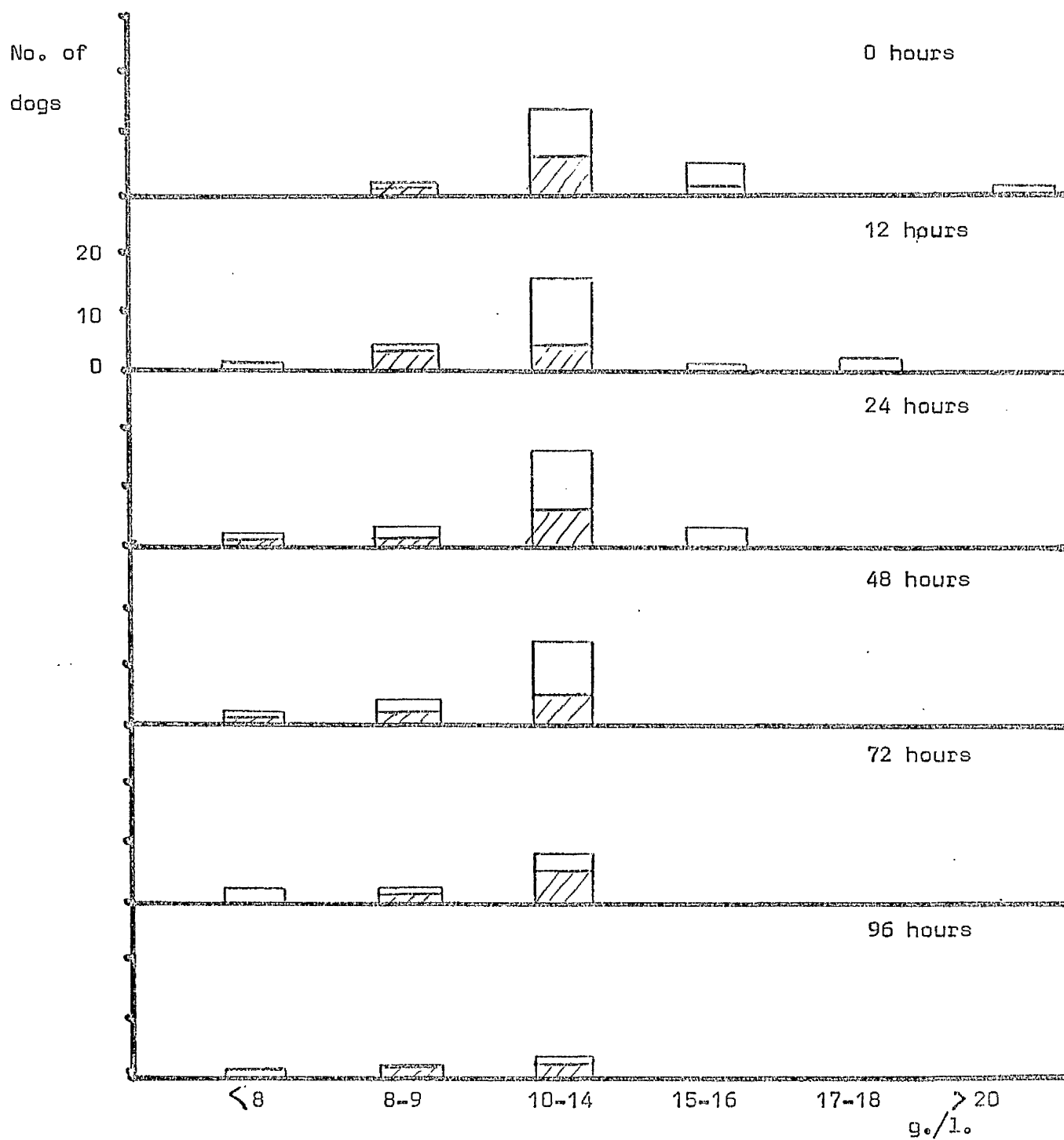
Histogram 11.

T.S.P. + P.C.V.



Histogram 12.

Haemoglobin



Histogram 13.

White blood cell count

