CERVICO-VAGINAL PROLAPSE IN THE PREPARTURIENT EWE:

AN EPIDEMIOLOGICAL AND CLINICAL STUDY.

by

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ABSTRACT OF THESIS

Title of Thesis: CERVICO-VAGINAL PROLAPSE IN THE PRE-PARTURIENT EWE:
AN EPIDEMIOLOGICAL AND CLINICAL STUDY

Two hundred and four cases of pre-parturient cervico-vaginal prolapse (CVP) were examined in general practice over three years. An overall incidence of 1.34% was found with some annual variation within flocks which was most evident in upland or hill flocks. There was no true breed disposition but the Kerry, Llyn, Suffolk and Texel breeds (and their crosses) were slightly overrepresented. Affected ewes were of all ages and body conditions. Exercise, weather and toposcopy did not affect the incidence or the severity.

Most cases (80%) occurred in the last 7 days of gestation and overall ewe mortality was found to be 24% of affected ewes. The progeny of ewes affected by CVP showed a high incidence of CVP (19%).

While affected ewes carried significantly more lambs than control ewes (2.4 lambs/ewe respectively) the number of lambs carried did not affect the severity. Ewes affected by the more severe forms of CVP carried heavier lambs than either the control ewes or the ewes affected by the milder forms of CVP. Lamb survival was worse amongst the more severely affected ewes.

Tenesmus and rectal prolapse were often encountered. The relief of tenesmus was an important part of effective therapy. Post-mortem examination showed no ewe to have a ruptured bladder but vaginal rupture, which carried a poor prognosis for both ewes and lambs, was a complication of CVP in 13 cases.

Eleven treatment methods were compared and none was found to give better lamb or ewe survival than the others except elective caesarian section which improved ewe survival at the expense of lamb survival. Delayed caesarian section resulted in an improved lamb survival but a reduced ewe survival. Ewes subjected to elective caesarian section showed a reduced conception rate at the next breeding season.

Inadequate cervical dilation (ring womb) was more common in affected ewes than in controls (17% and 0% respectively) but there was no increase in the proportion of ewes which suffered from post-parturient uterine prolapse. Post-partum recurrence of CVP occurred in 2% of cases.

Affected ewes showed a neutrophilia with left-shift and an elevated hydroxybutyrate concentration when compared to control ewes. Serum calcium and inorganic phosphate were reduced in proportion to the severity of CVP. Blood urea was frequently elevated and 80% of ewes with urea concentrations above 20 mmol/l died.

Plasma progesterone levels were higher than in control animals but oestrogen concentrations were lower. The possibility of an hormonal aetiology related to fecundity, environment, management and anatomy is not discounted.
ABSTRACT

Over three years 204 cases of Cervico-Vaginal Prolapse (CVP) were encountered in general practice. Affected ewes were of many breeds and crossbreeds, all ages and all bodily conditions. There was an overall incidence of 1.34%, which varied annually within individual flocks without apparent explanation. Seasonal and climatic differences did not appear to be responsible for the difference in incidence or severity of the disease. The availability of exercise, and the topography of the environment during the last 6 weeks of gestation had no influence on the incidence although there was a greater annual variation on upland or hill farms. There was no true breed predisposition although the Suffolk, Llyn, Kerry and Texel (and their crosses) were, relatively, slightly overrepresented in the study.

Most cases (80%) occurred within 7 days of lambing but some were recorded up to 27 days before parturition.

Thirteen ewes (6.4%) were found dead or in extremis, all suffered from vaginal rupture and varying degrees of herniation of abdominal viscera and/or severe vaginal haemorrhage. Following treatment 16 ewes (8.4%) died before lambing and 19 ewes (14%) died at or after parturition. The overall ewe mortality was found to be 24% of the original number examined.

Affected ewes were found to be carrying a higher number of lambs than the control ewes (2.4 lambs/ewe and 1.8 lambs/ewe respectively) \( (p = 0.006) \), and were found to be in slightly poorer bodily condition. The number of lambs carried by ewes had no effect upon the severity of CVP.

The more severely affected ewes carried heavier lambs than the less affected ewes (mean lamb weights of 4.12 kg and 3.46 kg respectively) \( (p = 0.026) \), but overall the control
animals were found to carry lambs of approximately the same weight (mean lamb weight of 4.31 kg) as the overall cases of CVP \( (p = 0.420) \).

Ewes with severe prolapses produced progressively fewer live lambs (1.25, 0.58, and 0.88 live lambs/ewe in second, third and fourth degree prolapses respectively) and only those with the mild (first degree) CVP yielded an improved overall lamb survival rate (1.56 live lambs/ewe), in spite of the higher uterine burden. Clinical examination revealed that tenesmus, possibly as a result of bladder obstruction was commonly encountered and rectal prolapse was occasionally present. The prolapsed tissue was frequently contaminated and/or lacerated and this was most evident in closely confined ewes.

Bladder obstruction was reflected in the high number of cases showing marked elevation of serum urea (mean 7.63 mmoles/l, range 1.17 - 47.9 mmoles/l), the passage of haemorrhagic urine once the prolapse was reduced, and the damage to the bladder found at post mortem. No post mortem examination revealed a grossly distended bladder but the bladder was found to be displaced posteriorly. Bladder rupture was not encountered, possibly because of prompt treatment. Ewes with serum urea concentrations over 20 mmoles/l seldom survived (80% mortality).

Eleven treatment methods were compared and none showed any significant advantage over the others in terms of ewe or lamb survival. The relief of tenesmus is an important aspect of therapy. Any treatment with which the veterinary surgeon is familiar should be reasonably effective if it is applied early in the pathogenesis of the condition.

Early elective caesarian section improved ewe survival but at the expense of lamb survival. However, when caesarian section was delayed with the use of other treatment methods, until signs of imminent parturition were apparent, lamb survival improved but ewe survival was worse.

Elective caesarian section immediately CVP was noticed, regardless of the gestational age, and retention methods which close the vulva, appeared to increase the possibility of ewes failing to conceive next season. Amongst ewes retained for breeding following CVP 15%
failed to conceive at the next season.

Affected ewes showed a higher incidence of inadequate cervical dilatation (ringwomb) than the control ewes (17% and 0% respectively).

Post-parturient uterine prolapse was no more frequent amongst cases of CVP than in control ewes (2% and 2.5% respectively). Recurrence of CVP in the post-partum period occurred in only five ewes (2%) and no case was seen outside the lambing season. One case occurred post-partum without, apparently, having been affected pre-partum.

A high rate of recurrence of CVP (55%) in the subsequent pregnancy was found but recurrence was not invariable (30% of affected ewes did not prolapse in the subsequent year). The progeny of ewes which developed CVP showed an increased tendency to CVP (19% of progeny bred developed CVP within 4 years).

Affected ewes showed a marked neutrophilia and left shift but red cell parameters were unaffected in spite of haemorrhage.

The serum beta-hydroxybutyrate concentrations were slightly elevated in CVP cases when compared to control sheep (p = 0.000) but were not considered to be pathologically raised.

Significant reductions in the mean serum calcium levels and inorganic phosphate were found but in no case was the depression of calcium sufficient to result in clinical hypocalcaemia. The depression in serum calcium was proportional to the severity of the condition.

A marked agonal rise in plasma glucose concentration was found.

The biochemical changes could be explained by inappetance, urinary obstruction, infection, inflammation and stress.

Plasma progesterone concentrations were found to be significantly higher in the diseased animals than the controls (median value of 6.75 ng/ml and 3.80 ng/ml respectively) (p = 0.007) but oestrogen concentrations were lower than control sheep (median 180 pg/ml and 320 pg/ml respectively) (p = 0.009). The increased fecundity may result in increased
peripheral plasma progesterone concentrations which may be responsible for an increased capacity of the vagina to dilate and a laxity of the vaginal supporting structures. The possibility of an hormonal aetiology is not discounted.
1.1: ACKNOWLEDGEMENTS:

The work of this thesis would not have been possible without the untiring guidance and encouragement of my wife, Morna, and Professors D.E.Noakes, F.W.G.Hill and A.Brand. My partners in practice, Messrs. B.Needham and P.W.Moody, accepted the time and expense given to the investigation over a five-year period with good humour and I am deeply grateful to them for their tolerance and understanding. The stalwart support of the practice nursing staff was invaluable.

As a consequence of the investigation I was honoured to receive the MacKellar Award from the Clinical Studies Trust Fund of the Royal College of Veterinary Surgeons and I am also indebted to Mrs. Anne Mackellar who has, since the award was received in 1981, maintained an active interest in the study. It is perhaps worth noting that Mr. John C. Mackellar, in whose memory the award was instigated, obtained his Fellowship of the Royal College of Veterinary Surgeons from practice on clinical work with sheep.

Dr. David Doxey of the Department of Veterinary Clinical Studies, Royal (Dick) School of Veterinary Studies, University of Edinburgh, took on the role of supervisor with a willingness and enthusiasm which have served as an inspiration to me.

I am indebted to the farmers of Warwickshire for their persistent help at a time when they would rather not have carried any extra burden!

The Veterinary Investigation Centre, Worcester, the Department of Obstetrics, Royal Veterinary College, London, and Dr. Th. Elsinghorst of the Department of Veterinary Pathology, University of Utrecht, The Netherlands, provided technical assistance and guidance which enabled far more information to be gained than was originally envisaged.
Dr. Marianne Sloet von Oldruitenborg-Oosterbaan of the Internal Medicine Department, University of Utrecht, The Netherlands, was a tremendous help in the preparation of the thesis and a persistent and enthusiastic mentor.

I am also indebted to Professor D. Zwart and Eng. B. Brouwer of the Department of Tropical Animal Production, Agriculture University of Wageningen, The Netherlands, and Professor J. Sheppard of the Computer Centre, University of Zimbabwe, for their help and advice with the computer analysis of the results and the preparation of the manuscript. The mountain of statistical analysis which derived from the work had previously seemed insurmountable!

My father, Alfred Knottenbelt, gave me tremendous help with the proof reading of the text.

Finally, I would like to extend my most sincere thanks to Professor D.E.Noakes for his continued tremendous support and encouragement from the outset of the investigation. He encouraged and criticised the work from its inception and there is little doubt in my mind that, without his support, it would not have been completed.
1.2 : DEDICATION

This thesis is dedicated to my wife Morna, my two patient and understanding daughters, Clare and Julie, and to my father and late mother who gave me the original opportunity to study at the Royal (Dick) School of Veterinary Studies, without regard for the financial difficulties which such a venture inevitably thrust upon them. I hope, in some small way, that this thesis will show that it was worth it in the end!
1.3: DECLARATION:

With the exception of those items described below, all the work involved in the thesis was carried out by myself.

The Department of Obstetrics, Royal Veterinary College, London, carried out the determination of the plasma progesterone and total oestrogen. They also prepared the haematoxylin and eosin stained sections from the biopsy material.

The Veterinary Investigation Centre, Worcester, carried out the estimations of glutathione peroxidase and provided the serum selenium levels which were derived from these. They also undertook bacteriological cultures and sensitivities which were, in the end, not included in this thesis.

Dr. Th. Elsinghorst of the Department of Veterinary Pathology, University of Utrecht, The Netherlands, taught me how to examine the sections of cervical tissue and interpret them usefully.

Messrs. Boehringer Corporation, Bell Lane, Lewes, Sussex, provided some of the clinical pathology kits and gave guidance as to their effective use. They also provided, gratis, the quality control reagents and some of the laboratory equipment which were used.

Professor D. Zwart of the Department of Tropical Animal Production of the Agriculture University of Wageningen, The Netherlands, allowed me access to the computer facilites and the technical expertise of Eng. B. Brouwer.
Professor J.Cox and the Editor of the Australian Journal of Zoology gave me permission to use the anatomical diagrams from their publications as a basis for those included in this thesis and I am most grateful to them for this help.

Mrs Sylvia Welsford and Mrs Pat Roper gave me assistance with preparation of the diagrams and tables.

Date: 29.5.87.

Date:
2.0 : INTRODUCTION :

The word prolapse is derived from the Latin word "PROLABI" meaning "to fall before". As Watts (1954) remarked, "this is not the most appropriate translation because the protrusion occurs posteriorly and falls behind the unfortunate animal". The Webster's New International Dictionary (1981) gives the meaning of prolapse as :

i) the slipping of a body part from its usual position in relation to other parts.

ii) the falling down of an internal part of the body.

It is the preparturient eversion and prolapse of the vaginal wall and/or the cervix in the ewe, with which this thesis is concerned. Many definitions have been proposed in order to describe and define the development and appearance of this prolapse of the vaginal wall and cervix. For the purposes of this thesis the prolapse of the vaginal wall will be referred to as vaginal prolapse. This is a necessary distinction, for the vagina is the space enclosed by the vaginal walls and the cervix posteriorly and as such, in theory, cannot be prolapsed.

Thus McLean (1956) and Fielden (1980) have independently defined it as an "eversion of part or the whole of the vaginal wall in such a manner that the mucosa is visible between the lips of the vulva". Merck's (1973) definition includes the word "eversion" which was a term used frequently by early veterinary surgeons including Bayly, Hankin and Haugh (1936), Laing (1949) and Edgar (1952), who variously called the disorder "Vaginal Eversion", "Eversion of the Vagina" or "Vaginal Displacement". It is pertinent, at this point, to make a distinction between vaginal prolapse as defined in the pre- and post-parturient ewe, and the condition known as vaginal hyperplasia such as is seen in
the dog (Arthur et al., 1982; Moreno, 1971) and in pigs (Cox, 1981; Bollwahn, 1980; Arthur et al., 1982). The latter disorder is the appearance of hypertrophied vaginal mucous membrane at the vulva without any concomitant posterior displacement of the vagina or cervix. The definitions given for vaginal prolapse do not all emphasise the essential differences between the two types of vaginal protrusions and consequently I will adopt McLean's (1956) and Fielden's (1980) identical definition which best describes the disorder included in this study. It is the essential facts of eversion and displacement which distinguishes vaginal prolapse from vaginal hyperplasia. The visibility of the vaginal mucosa between the vulval lips was stated by McLean (1956) to be an indication of abnormal displacement. Prolapse of the cervix resulting from vaginal displacement is merely an extension, aggravation or development of the pre-existing vaginal prolapse (Cox, 1981). It is appropriate to refer to the condition overall as cervico-vaginal prolapse (CVP) and the term will be used throughout this thesis to cover the range of the disorder in pregnant ewes.

Very similar conditions are described post-partum and during the non-breeding season in ewes (Mclean, 1956; Edgar, 1952, Watson, 1959) and although reference will be made to these conditions it is not intended to investigate them. Amongst shepherds and farmers pre-parturient cervico-vaginal prolapse is known by a wide variety of colloquial names such as "throwing the reed", "pushed the weather", "popped the rose", "red bag", "split the slit", "bearing disease", "Rosie" and, as Edgar (1952) pointed out, "no doubt many others". Prolapse and eversion of the vagina and/or the cervix occurs in a variety of animals including the horse (Richardson, Honey, Karns, McCoy, and Keck, 1983) in which species it is very rare (Vanderplassche, personal communication), pigs (Bollwahn, 1980; Arthur, Noakes and Pearson, 1983), dogs (Moreno, 1971; Arthur et al., 1983), and man (Shaw, 1941) but by far the most frequent occurrence is in ruminants in the last third of pregnancy. Amongst the ruminants the disorder is seen frequently in cattle (Watts, 1954; Buhner, 1958; Gyorgy 1968; Cox, 1981; Arthur et al., 1983; and others); occasionally in
goats (Tawab and Othman, 1985; Franklin, 1986) and most frequently in sheep, in the last two to four weeks of gestation, in which species the disorder attains its greatest numerical significance (Edgar, 1952; McLean, 1956; Cox, 1981; Arthur et al., 1983). In horses, dogs and humans the condition is not necessarily related to pregnancy but in the ruminant it is frequently regarded as a condition of the pregnant animal. In the ruminant species it may, however, also occur in maiden and non-pregnant animals under certain conditions where genetic or particular management practices or endocrine abnormalities result in an excess of circulating oestrogens in the animal (McLean, 1956; Cox, 1981; Arthur et al., 1983).

Attempts to study cervico-vaginal prolapse have caused frustration since the first clear descriptions of the disorder by Bayly et al. (1936), and Fethers (1939) in New Zealand and England respectively. Hartigan (1961) summarised these frustrations as follows: "...the economic loss is immense, the incidence is erratic, the aetiology is obscure, the treatment is vexatious, and the literature is at once immense, erratic, obscure and vexatious". McLean (1956) noted that "...few observers have made any serious attempt to substantiate their views by critical experiment" and "...little is known about the fundamental nature of the disease or its causes". Some thirty years later Sobiraj, Busse, Gips and Bostedt (1986) stated: "...there is little knowledge of the causes of vaginal prolapse, and the explanations for the development of the condition are, on the whole, unsatisfactory". It appears that only limited progress has been made despite the continuing efforts of numerous researchers.

It is clear that CVP is a difficult and universal disease. It probably occurs wherever sheep are raised, particularly under intensive and semi-intensive systems of management. Reports of significant numbers of cases in New Zealand have been made by Bayly et al. (1936), Laing (1945), and McLean (1956); in the United Kingdom by Edgar (1952) and Watson, (1959); in Tasmania by Hardy (1959); in Argentina by Moreno (1971); in the Netherlands by Pastoor and Weitenberg (1979); in Mexico by Martinez and Mendez (1985); in Germany by Sobiraj et al. (1986) and in America by Eagelman, Beebe and Leman.
Fowler and Evans (1957) stated that the economic loss was considerable because of the high mortality in both ewes and lambs. Edgar (1952), McLean (1959) and Schwarz (1975) also found a significant mortality amongst ewes affected with CVP with a high incidence of lamb mortality and dystocia. McLean (1956, 1959) described an aggressive attitude on the part of some farmers towards cases of CVP in that some would summarily kill all cases without any attempt at treatment. The loss to the sheep industry in general, and to some farmers in particular, is high due to the consequences of:

a) ewe mortality,

b) lamb mortality,

c) the culling of affected ewes,

d) time, effort and money spent in treatment and aftercare,

e) veterinary fees and drugs used either directly for treatment of the CVP, or indirectly through dystocia, salvage slaughter and the certification of animals to be salvaged for human consumption.

Shepherds and veterinary surgeons are aware of its existence throughout the world but there has been relatively little research into most aspects of the disorder. Contrary to Hartigan’s (1961) opinion, the literature is by no means immense although it is certainly vexatious and erratic and arguably obscure. Hindson and Turner (1972) illustrated the problem of investigating CVP in a survey of several hundred ewes when they found that there were almost as many suggested aetiological factors as there were answers to their questionnaire. Fielden (1980) also stated that, in spite of all the knowledge available, it was still not clear why some ewes were affected while others were not. Several reports of epidemic outbreaks have been published (Bayly et al., 1936; Laing, 1939, 1945, 1949; McLean, 1956; Edgar, 1952) but there has been little investigative work undertaken since that of Edgar (1952), McLean (1956, 1957, 1959), McLean and Claxton (1958, 1959, 1960), Stubbings (1971) and Sobiraj et al. (1986).
The aim of this thesis was to investigate the disorder from clinical and epidemiological aspects on farms in Warwickshire, within the constraints of general practice, and to review critically the published work. The objectives may be summarised as follows:

1) to determine the incidence of CVP in commercial flocks in Warwickshire, and to estimate from these figures the potential loss from this disease to the British sheep industry as a result of the disease;
2) to determine if any environmental, managemental or specific individual factors affected the development of CVP;
3) to improve the understanding of the pathogenesis of the condition;
4) to determine the effects of CVP on the health status of affected ewes by comparisons with unaffected sheep of haematological and biochemical data;
5) to determine if there is any particular medical approach by which a practising veterinary surgeon may improve the prognosis for both the ewe and her lamb(s) by a comparison of some of the different methods of treatment which have been described;
6) to review the published research which has been carried out, particularly in view of the paucity of recent investigations;
7) to stimulate research interest in a largely forgotten and under-researched disorder about which the veterinary profession appears to know little.

No experimental work has been carried out in this investigation for legal and personal reasons. All surgical and other procedures described are normally used in veterinary practice, and all the work, with a few exceptions, was carried out by myself. These exceptions have been acknowledged.
3.0: A CRITICAL REVIEW OF THE LITERATURE:
3.1 : DISTRIBUTION :

There are published reports of CVP from across the world including New Zealand (Bayly, Hankin and Haugh, 1936; Laing, 1939, 1945, 1949; McLean, 1956 et passim.); Tasmania (Hardy, 1959); United Kingdom (Fethers, 1939; Edgar, 1952; Walsby, 1952; Fowler and Evans, 1957; Jones, 1958; Hughes Ellis, 1958; Watson, 1959; Fowler, 1962; Stubbings, 1971); Australia (Bennetts, 1944; Bennetts, Underwood and Shier, 1946); Germany (Schwartz, 1973; Hertrampf, Wilkins Fiedler and Bollwahn, 1978; Rusch and Berchtold, 1978; Sobiraj, Busse, Gips and Bustedt, 1986); the Netherlands (Pastoor and Weitenberg, 1979); America (Eagelman, Beebe and Leman, 1967); Argentina (Moreno, 1971); Eastern Europe (Klauzer and Brestansky, 1973); Mexico (Martinez and Mendez, 1985) and almost certainly many others. From these reports, it is reasonable to assume that CVP occurs in most sheep-producing countries. Most of the countries from which the disease has been reported maintain intensive or semi-intensive sheep rearing methods. Indeed, all the literature, except Sobiraj et al. (1986), refers to the disease as it occurs in that kind of system, involving exclusively the wool producing breeds of sheep. Sobiraj et al. (1986), however, describe the finding of CVP in a variety of mutton sheep and in a mountain sheep. It is therefore reasonable to assume that the condition is not exclusively associated with intensification.
3.2: INCIDENCE:

Edgar (1952) stated that "little is known of the incidence (of CVP) in sheep" and he quotes Bayly et al. (1936) who described an "unusually frequent occurrence of the disease" in certain localised areas of New Zealand in the 1935 lambing season. The investigations by Bayly et al. (1936) represents the first enquiry into the incidence of CVP in pregnant ewes, although Laing (1945) pointed out that the disease had become a significant feature in New Zealand in the late nineteenth century. The incidence found by Bayly et al. (1936) varied widely amongst the nineteen farms surveyed. Five reported an incidence of 1% or less, three reported 2% and 4%, five reported 3%, and other individual farms reported incidences which varied between 5% and 8%. Laing (1945), quoted also by Edgar (1952), reported that in his experience the incidence may rise, on occasion, to 11% with 4% being an average incidence in some areas.

In the United Kingdom the first descriptions of the condition were made by Fethers (1939) who, although not quantifying the numbers involved, stated that "a good deal of trouble is being experienced with the ewes", thus giving the impression that more than the usual number of cases were being found. This, in turn, might suggest that the incidence was variable from year to year. An extensive enquiry into the incidence was made by Edgar (1952) in the United Kingdom. This covered some 21,660 breeding ewes over a two-year period on all types of farms and involving 20 different breeds and cross-breeds of sheep. He was able to calculate an overall incidence of 0.53% but, like Bayly et al. (1936) and Laing (1945) before him, found a wide variation in the incidence (from 0% in half the farms to a "particularly serious outbreak" of CVP involving 20% in one flock in one particular year). There were numerous flocks in which the incidence varied slightly
from year to year but, in general, statistical analysis revealed that there was no significant variation between years, except in flocks where "significant outbreaks" occurred. The incidence appeared to increase with advancing age of the ewes, and decreased as the lambing season progressed, i.e. there was a lower incidence in late-lambing ewes. Some breeds, such as the Romney Marsh, were apparently more often affected than others. A similar survey in New Zealand was carried out by McLean (1957) primarily to test the effect of topography upon the incidence. He found an overall incidence of 0.346% being 390 cases from a population of 112,885 ewes.

Watson (1959), reporting his experiences in New Zealand, stated that he found the disease showed a variable incidence which was possibly due to changes in weather, feed, geographical conditions and the age of the ewes.

Blackmore (1960) found that the incidence amongst obstetrical cases seen in his practice in England was 2 out of 52 cases, but does not indicate the total number of ewes this was drawn from or whether he had attended every case. However, Stubbings (1971) stated that 51 cases of CVP were treated by his practice from a "flock size" of 10,500 ewes; this indicated an incidence of 0.5% but he acknowledges that only the worst of the treatable cases would have been presented to him. Presumably the mildest would have been treated by the farmer and the most severe, untreatable cases might have been euthanased and both such cases would not, perhaps, have been presented to the veterinary surgeon. His figure does, however, approximate to that of Edgar (1952). Fielden (1980) found up to 12% affected ewes in specific outbreaks, but gives no figures for the overall population incidence. Martinez and Mendez (1985) found an overall incidence of 1.41% over 3 lambing seasons in Mexico.
3.3 : ANATOMY :

The anatomy of the pelvic and perineal structures of the non-pregnant ewe has been described in detail by Bassett (1965). The anatomical changes associated with normal pregnancy have been reported by Bassett and Phillips (1955a) In order to understand the pathogenesis and treatment of CVP, it is pertinent to describe some of the significant anatomical features in both the non-pregnant and the pregnant ewe with respect to the perineal and pelvic tissues. The most important tissues are those of the genito-urinary tract itself, but the supporting structures and those tissues which form the limits of the pelvic cavity are intimately related to the understanding of the anatomy of the area.

Figure 1 is derived from Bassett (1965) and shows a sagittal section through the pelvic region of a normal non-pregnant ewe. The rectal and urogenital attachments and the supportive structures of the pelvic cavity are shown with the associated soft tissues. The bony structures are shown in Figure 2, again modified from Bassett (1965), and illustrate the main attachments of the pelvis to the axial skeleton. Lateral dissected views of the pelvis giving the gross muscular features are shown in Figures 3, 4 and 5. The anatomical location of the major nerve trunks of the hind-quarters, including the course of sacral nerves (S ii, S iii and S iv), forming the pudendal plexus, is shown in Figure 6 which is derived from Bassett (1965), and in parts from Hertrampf et al. (1978). The caudal dissected view of the perineum and the ventral dissection of the urogenital tract of the ewe derived from Bassett (1965) are shown in Figures 7 and 8 respectively.

These anatomical features are important in the application of local anaesthesia to control tenesmus (straining) or to facilitate surgical or obstetrical manipulations within the birth canal. The muscles of the vulva and vagina, and the gluteal muscles of the hindquarters
Figure 1: Sagittal section of a normal non-pregnant ewe showing the major pouches of the peritoneum and the relationships of the major pelvic organs (after Cox, 1981).
Figure 2: Lateral view of the sacro-iliac joint showing the sacro-iliac ligament and the angle the internal angle of the ilium makes with the sacrum (after Bassett, 1965).
Figure 3: Superficial dissection of the lateral pelvis of the ewe (after Bassett, 1965).
Figure 4: Partial deep dissection of the lateral pelvic region of the ewe showing the major muscle blocks and the course of the sciatic nerve (after Bassett, 1965).
Figure 5: Deep dissection of the lateral pelvis in the ewe (After Bassett, 1965).
Figure 6: Diagram showing the origin and course of the sacral nerves after dissection of the lateral muscles of the pelvis (after Bassett, 1965).
Figure 7: Caudal dissected view of the structures forming the perineum of the ewe (after Bassett, 1965).

Figure 8: Ventral dissection of the urogenital tract of the ewe (after Bassett, 1965).
are important in some of the methods of surgical reduction of CVP. Bassett and Phillips (1955a) concluded that there were considerable species differences between cattle and sheep in the changes associated with pregnancy and attempted to describe these in the ewe. They suggested that the hormone ‘relaxin’ was responsible for some of the changes which precede the onset of parturition, such as the softening of connective tissue, and these could be involved in the development of CVP in the event of these changes occurring prematurely or being excessive.

An investigation into the normal anatomical changes revealed that sacro-iliac relaxation occurred without any apparent change in the pubic symphysis. Changes in the dimensions of the pelvic inlet therefore could not occur without a vertical pivoting of the sacrum and elongation of the sacro-sciatic ligaments. The greatest effect apparently occurred at the sacro-lumbar joints. A much greater effect was noted in the pelvic ligaments and muscle fasciae as pregnancy advanced. Thus, under the influence of relaxin or oestrogens or both, the tissues relaxed and became more flexible or elastic. This increased the potential for displacement of the reproductive tract and would have increased the vaginal circumference as pregnancy advanced.

The comparative anatomy of pregnant ewes and those affected by CVP was investigated by Bassett and Phillips (1955b) using both deep frozen and living specimens. Fifty live ewes with a history of CVP were followed throughout pregnancy and the puerperium. Measurements were taken of the capacity of the vagina to dilate, tract displacement and intra-abdominal pressure. These were then compared with 50 normal pregnant ewes with no history of CVP. The live cases showed a greater vaginal circumference. The mean vaginal circumference in non-pregnant ewes was found to be 91 mm (+/- 7 mm) while in normal pregnant ewes at full term the value was found to be 117 mm (+/- 12 mm). In cases of CVP the mean vaginal circumference was found to be 135 mm (+/- 28 mm). These authors also measured the "tract displacement" which represented the extent to which the peritoneal pouches were elongated or stretched and found that there was a significant
increase in the ewes affected with CVP. In the normal non-pregnant ewe a value of 158 mm (+/- 19 mm) was recorded and this rose to 216 mm (+/- 26 mm) in normal pregnancies. In ewes which had been affected by CVP the value in non-pregnant animals was found to be 171 mm (+/- 18 mm) and this rose progressively through pregnancy until at term it was found to be 247 mm (+/- 68 mm). Relaxation of the tissues was found to be more marked in the earlier stages of pregnancy than was found in normal ewes. Little further difference between the two groups was apparent as pregnancy advanced.

It is interesting that some of the ewes with a history of CVP had developed thickened vaginal walls, adhesions and scarring which actually appeared to help reduce the tendency for the cervix to displace. Such cases however, even when the thickening was marked to the extent of a vaginal stricture, showed the characteristic relaxation as pregnancy advanced. It is clear that even fibrous tissue may be affected by whatever causes the softening of the tissues. Bassett and Phillips (1955b) considered this to be an "hormonal effect".

Frozen sagittal sections of ewes affected with CVP were examined by Bassett and Phillips (1955b) and were compared with ewes in which there was no history of the disease. Ewes were frozen as near to the normal living posture as possible, but Bassett and Phillips (1955b) acknowledge that there may be variations as a consequence of the procedure which required ewes to be in dorsal recumbency at the time of freezing. In normal ewes considerable lengths of intestine were present in the pelvic cavity. The uterus, vagina and bladder were compressed caudally against the peritoneum. The vesico-genital and recto-genital pouches of the peritoneum were found at the level of the third and fourth coccygeal vertebrae. In cases of CVP, where the ewes were carrying twins or single lambs and the vagina was completely everted, the bladder and part of the uterus with cotyledons and foetal fluid were displaced posteriorly. As a consequence they were present caudal to the vulva. This meant that the urethra was displaced likewise and was kinked. All the tissues of the perineum, including the vulva, were distended, and visible "lines of stress"
were apparent in the peritoneum on the lateral aspect of the pelvic inlet. The head of one foetus was located inside the pelvic cavity. Prolapse was accompanied by considerable haemorrhage on the dorsal surface of the pelvic cavity and many cases showed gross haemorrhage into the peritoneal cavity itself.

An excellent, simple illustration and description of the anatomy of the pelvic structures of the ewe was given by Cox (1981), who also described the development and progression of the prolapsing organs from 'mild' to 'severe' CVP (Figures 10, 11 and 12). The apparent kinking of the urethra is important to note since it was a widely held belief that CVP was, partially at least, caused by a mechanical obstruction of the urinary tract (Laing, 1945, 1949; Hardy, 1959).

The anatomical descriptions given by Bassett and Phillips (1955b) and by Cox (1981) had been described previously by Gilruth (cited by Bayly et al., 1936). In this early description of the pelvic anatomy the posterior displacement of the urinary bladder and uterus, with the consequent constriction of the urethra resulting in straining and dysuria, was identified.

McLean (1956) and McLean and Claxton (1958) also investigated some anatomical and physiological changes in normal ewes throughout the annual breeding cycle, and in ewes affected by CVP. They measured the capacity for vaginal dilatation and the extent of vulval relaxation in the two classes of ewes. There was a marked but regular increase in the volume of the vagina as normal pregnancy advanced.

McLean (1956) postulated that for CVP to develop:

i) the vagina must be soft, full and relaxed and that prolapse would be facilitated by the existence of distendable and relaxed vestibular and vulval tissues,

ii) a force should be exerted upon the tract, which he concluded was possibly an increase in intra-abdominal pressure.

The dilatability of the vulva, vestibule and vagina was measured using an intravaginal non-elastic balloon which was attached to a water manometer and the pressure that was
required to dilate the vagina to a standard degree was measured in 2 ewes at intervals throughout the year. The results indicated a significant cyclical nature in the dilatability of the vagina and distensibility of the vulva and vestibule. McLean (1956) did not find any dramatic changes but rather a gradual and progressive change as pregnancy advanced. This is contrary to the suggestions by Bassett and Phillips (1955b) who found that most of the natural changes in normal and CVP cases occurred in the early stages of pregnancy. It would appear, therefore, that the significant differences in anatomical relationships between normal ewes and CVP cases are merely a matter of degree. Affected ewes showed no significant increase in the rate of increase of the vaginal circumference or the ability of the vagina to dilate in either the work reported by Bassett and Phillips (1955b) or McLean and Claxton (1958). While these authors have somewhat different findings, neither has shown any significant feature which may develop during pregnancy. Both showed that CVP cases have changes which were present in the normal pregnant ewe and even in the non-pregnant stages of the breeding cycle. McLean (1956) concluded that a degree of 'inversion', such that the cervix was moved posteriorly as far as the constrictor vestibulæ muscle, was a normal occurrence in non-pregnant and pregnant ewes. McLean and Claxton (1958) confirmed the cyclical nature of these changes.

Cox (1981) emphasised that the vestibule was firmly attached to the pelvic floor and to the anal ligaments and thus was much less readily displaced than the vagina, whose attachments to the pelvic floor are "very loose". McLean (1956) also suggested that the involvement of the vestibule was likely to be restricted by its firmer attachments.

An understanding of the above points will greatly assist in the comprehension of the pathogenesis and treatment of CVP which follows.
3.4 : PATHOGENESIS :

Early studies suggested that urinary obstruction was an important primary factor in the development of CVP (Bayly et al., 1936; Laing, 1945, 1949). Interviews with farmers reported by Bayly et al. (1936) suggested that there were two different types of prolapse: the first being characteristic of those developing well before lambing without urinary obstruction and the other being seen at or around parturition and invariably associated with dysuria. The authors added that, in their experience, they merely represented different stages in the development of CVP and that once the bladder moved into the pelvis and became involved in the prolapse, clinical signs of urinary obstruction may appear. Fethers (1939) did not consider that straining was induced by urinary obstruction and suggested that diarrhoea was possibly the cause. However, straining was proposed by both early workers as a primary factor in the pathogenesis.

Laing (1945) listed 12 points to explain the pathogenesis of CVP, which are summarised in Figure 9. There are difficulties with these proposals including the reasons for the recumbency, the "spasm" of the bladder neck and the continual straining which, supposedly, followed urination. The proposal that urinary obstruction was a primary factor in pathogenesis has been widely accepted. It does not really explain why, as Watson (1959) remarked, "some ewes are affected while others are not" when their feeding regimen and bodily condition were generally the same in any particular farm situation. The 'inherent laziness' of ewes with a full rumen may not be enough to explain an individual recumbency.

McLean's (1957) work on the effect of topography and posture upon the intra-abdominal pressure was a major step in the understanding of the pathogenesis of CVP. Ewes which
Figure 9: The pathogenesis of CVP deduced from the description by Laing (1945).
were carrying multiple lambs were not significantly different in this respect from either non-pregnant or pregnant ewes carrying one lamb only. The most significant increases were created by the presence of large amounts of bulk food in the alimentary tract. There seems little doubt that increased intra-abdominal pressure applies a posterior force upon the attachments of the genital tract. Such forces must be present in most, if not all, ewes at some time during the later stages of pregnancy and yet only a relatively few cases of CVP develop. Palsson (1952) quoted by Edgar (1952) used the postural effects, later investigated by McLean (1957) and McLean and Claxton (1960), to reduce the incidence of CVP by raising the back of the standing. Like Laing (1945, 1949) he used the reverse effect to good advantage in the treatment of CVP. Thus gravity, intra-abdominal pressure and tenesmus may be important and may be linked in the pathogenesis.

Tissue weakness, particularly relating to the supporting structures of the genital tract and the pelvic cavity contents, has been suggested as a possible factor in the pathogenesis of CVP perhaps as a consequence of mineral or hormonal changes (Leslie, cited in Bayly et al., 1936; Edgar, 1952; McLean, 1956; Hindson and Turner, 1962; Stubbings, 1971; Sobiraj et al., 1986). There are no studies reported on the histological structure of tissues apart from Hindson and Turner (1962) whose investigations were primarily directed towards "ringwomb" (failure of cervical dilatation). McLean (1956) and McLean and Claxton (1959) found that the genital tract became progressively more easy to displace as pregnancy advanced and this tendency was more pronounced in ewes affected by CVP. Bassett and Phillips (1955a, 1955b) found that the displacement of the genital tract was accompanied by haemorrhages and lines of stress and it is possible that the presence of such tissue damage has a further weakening effect upon the structures of support. This may, in part, explain rupture of the dorsal vaginal wall which is sometimes accompanied by herniation of abdominal contents McLean (1956), White (1961), Cox (1981); a condition described by Knottenbelt (1988) as a possible complication of CVP.
The necessity for tenesmus in the pathogenesis of CVP was illustrated by Edgar (1952). McLean (1956) and Watson (1959) noted that bladder obstructions were often severe enough to result in rupture of that organ associated with straining. This would seem to suggest that urinary obstruction is a major component in the development of CVP. Tenesmus is perhaps not as likely to follow diarrhoea (as suggested by Fethers, 1939) or vaginal irritation as was proposed by Hardy (1959), and only Knottenbelt (1988) has described the presence of diarrhoea associated with the vaginal rupture syndrome. No authors have either described a high incidence of diarrhoea or have corroborated the opinion that tenesmus may result directly from vaginal irritation. It would seem logical, however, that damage and inflammation of the vaginal mucosa may result in significant tenesmus as most authors describe damage of varying degree being present. Vaginal contamination and inflammation were found in some ewes in which no apparent CVP was present, by Bassett and Phillips (1955b).

Once the cycle of pathogenesis had started, progressive urinary obstruction, inflammation, oedema and irritation of the prolapsed tissue may then result in an aggravation of the tenesmus. This, however, does not explain McLean's (1956) findings that apparently milder forms of CVP may be accompanied by severe straining, while what appear to be very severe, inflamed and lacerated prolapses may not be characterised by marked tenesmus. Laing (1949) noted varying degrees of inflammation including necrosis, bruising and even gangrene of the prolapsed tissues. There are no later suggestions as to why this should be the case and it was possibly related to nerve irritation/neuritis as most authors including Bayly et al. (1936), Laing (1945, 1949), Arthur et al. (1982), and Knottenbelt (1987) have emphasised the need to apply some form of soothing salve or local anaestheisa to the prolapse where tenesmus is marked. Where the cervix and uterus are displaced, McLean (1956) found that the ewe showed little discomfort until pathological changes were advanced, while those ewes, in which only the vaginal wall was involved, showed extreme discomfort. This implies that dysuria was a consequence of CVP.
Figure 10: Saggital section of pelvic organs as illustrated by Cox (1981) in a ewe with 'simple' prolapse, showing reflection of bladder into the vesico genital pouch and displacing the ventral vaginal wall which appears externally below the posteriorly displaced cervix and uterus (after Cox, 1981).
Figure II: Sagittal section of pelvic organs in pregnant ewe with 'moderate' vaginal prolapse (after Cox, 1981).
Figure 12: Sagittal section of pelvis of pregnant ewe affected by severe vaginal prolapse (drawn from Bassett & Phillips, 1955) showing rectal prolapse and prolapse of vestibule such that urethra lies posterior to vulval lips. The vesico-genital pouch is severely stretched and foetal cotyledons or extremeties may be palpated.
and that the initial straining may be due to other causes such as the posterior displacement of bowel or bladder as suggested by McLean (1956). He also found that prolapses which affect the dorsal wall of the vagina were frequently associated with bowel displacement, while those which affected the ventral wall resulted more often in the displacement of the bladder. Why the discomfort should be apparently most severe in certain milder cases of CVP was, perhaps, explained by Cox (1981), who showed that the maximum obstruction of the pelvic viscera occurred before the appearance of the more severe forms of CVP. Thereafter the bladder may continue to fill but was unable to empty (Figures 10, 11 and 12).

Bayly et al. (1936) found that the 'long sausage' type of prolapse, which farmers associated with early cases and were not generally characterised by tenesmus, were those in which the bladder may not have been displaced and therefore urinary or other obstruction may not have existed.

The involvement of, and the course followed by, the ureters has received little attention but in view of the displacement of the bladder in a posterior direction these must be greatly stretched in order to reach the bladder.

Bassett and Phillips (1955b) noted the presence of bowel in the pelvis which, because it was trapped, may have resulted in a physical obstruction to the rectal contents. They illustrated a case of CVP in which the head of a lamb was forced into the prolapse inside the gravid horn of the uterus. It might be expected that this would have severely obstructed the pelvic cavity. No other authors have recorded the presence of lambs in the prolapse, but Fox (1962) described the appearance of a single gravid horn through a tear in the dorsal vaginal wall. He did, however, note that the ewe had not apparently been affected by CVP. Knottenbelt (1988) also described a similar but more dramatic occurrence where the entire genital tract, including three foetuses, was prolapsed through a large tear in the dorsal vaginal wall and had become detached at each end of the tract which had certainly been preceded by CVP. This illustrates that the gravid uterus may be
partly responsible for the pelvic obstruction.

The progression of CVP was described by Cox (1981) (Figures 10, 11 and 12) but he did not mention the most severe form of CVP which involved displacement of the vestibule. McLean (1956), however, described this occurrence in cases where continuous severe tenesmus caused bruising and sub-mucosal haemorrhages and a full prolapse of the vestibule (Figure 12). A diagrammatic description of the pathogenesis of CVP in ewes derived from the drawings of Bassett and Phillips (1955b) and Cox (1981) is shown in Figures 10, 11 and 12. Figure 12 shows the appearance of the complete prolapse involving the vestibule which is derived from the dissected specimen illustrated in Bassett and Phillips (1955b).

The explanation for the characteristic prolonged and difficult parturition, found by McLean (1956) in many CVP cases, was suggested by this author as being the possible scarring, infection and retarded cervical dilatation.

Severe cases of CVP are reported to be accompanied by a marked stretching of the recto-genital pouch of the peritoneum (Bassett and Phillips, 1955b; Cox, 1981). Because the bladder is frequently, but not invariably, incorporated in the displacement, the vesico-genital pouch must also be stretched. Bassett and Phillips (1955b) found that these stretched pouches were frequently present in ewes with twin lambs. This was probably significant in view of the high incidence of multiple pregnancies found in most investigations (Laing, 1945, 1949; Edgar, 1952). These authors found that the sacro-rectal and the vesico-pubic pouches remained largely unaffected unless the rectum was prolapsed in addition. Such a state was found by Bassett and Phillips (1955b) in a ewe carrying only a single lamb, from which it can be deduced that the severity of the CVP was not necessarily related to the presence of a twin pregnancy.

From the descriptions of the dissected specimens shown by Bassett and Phillips (1955a, 1955b) it is easy to appreciate the development and progression of the disorder in the ewe. However, there are a number of unanswered questions:
a) Is the bladder drawn into the prolapse as a consequence of the tension exerted upon it by the vesico-genital pouch following the posterior displacement of the cervix and vagina?

b) Is rectal prolapse a primary problem or does it arise as a consequence of the straining?

c) Does rupture of the vagina occur almost exclusively in the dorsal vaginal wall and is it invariably related to CVP (McLean, 1956; White, 1961; Fox, 1962; Clarkson and Faull, 1983; Knottenbelt, 1987)?

d) Does the prolapse of the vestibule result in the external urethral meatus being found as part of the prolapse as described by Bassett and Phillips (1955b)? The attachments of the vestibule were found by Cox (1981) to be firm, and McLean (1956) noted that involvement of the vestibule was unusual. In view of Cox's (1981) finding, it may be that, once the vestibule is involved in the prolapse, the associated anal ligaments and muscles are pathologically affected allowing rectal prolapse to develop as straining persists. Displacement of the more firmly attached vestibule must involve a greater degree of tissue trauma than the movement of the more mobile parts of the tubular genital tract, particularly when this is suitably relaxed as appears to occur in late pregnancy (McLean and Claxton, 1958; Bassett and Phillips, 1955).

There are numerous opinions concerning the recurrence of CVP in the subsequent pregnancy(ies), its appearance in the post-partum period and its relationship to dystocia and uterine prolapse. Thus Filmer (quoted in McLean, 1956) found a frequency of recurrence of about 40%. Fowler and Evans (1957) noted that "......it is believed that the condition will (re)occur at subsequent lambings". Watson (1959), however, could find no evidence of significant recurrence. McLean (1956), McLean and Claxton (1959) and Bassett and Phillips (1955b) found in recovered ewes significant anatomical and physiological changes, particularly in vaginal capacity and the length and strength of the
Figure 13: Schematic diagram of the pathogenesis of CVP deduced from the literature.
supporting structures of the genital tract. The published findings of these workers contain some apparently contradictory statements. They suggested that tissue slackness encourages recurrence, but that ewes affected by CVP have frequently been found to have more scar and fibrous tissue which may be expected to reduce the chances of recurrence. It is apparent that no really satisfactory explanation of the pathogenesis is available. Figure 13 is an attempt to combine the theories outlined in the literature. It is far more complex than the scheme proposed by Laing (1945) shown in Figure 9. Regrettably, a comparison between the two schemes serves only to illustrate how little more we have learned about the pathogenesis of CVP since 1945.
3.5: AETIOLOGY:

From the earliest descriptions of CVP quoted by Bayly et al. (1936) to the most recent investigation by Sobiraj et al. (1986) there have been suggestions as to the aetiology of CVP. Indeed the underlying frustrations are apparent in most reports and are typically expressed by Watson (1959) and by Hindson and Turner (1972). Watson (1959) comments that "...from time to time one may have thought that there was some definite pattern of incidence only to have the picture suddenly change with, perhaps, a sudden variation in the weather, which would in turn, affect feed, feeding habits and duration of pregnancy", and he concluded that it was almost impossible to build up a definite picture, so varied were the farmers' experiences of the disorder. A survey carried out by Hindson and Turner (1972) indicated "... about as many aetiological factors as there were answers to the questionnaires".

Despite these difficulties many workers have, however, attempted to clarify at least some of the possible aetiological factors. It will become apparent that many of these are inter-related (Watson, 1959).

A statement by Gilruth (quoted in Bayly et al., 1936) stressed the possibility of several aetiological factors. These included:

i) the presence of twin lambs
ii) the presence of abdominal fat
iii) laziness on the part of the ewe
iv) urinary obstruction
v) inflammation and / or irritation of the exposed mucous membrane.
One of these factors may lead to another and it was not very clear which may be the most significant or primary factor. Laing (1945) suggested that "overconditioning of the ewe", an excess of feed and recumbency for long periods with consequent urinary obstruction were major aetiological events.

Field Officers in the New Zealand Livestock Division, cited by Bayly et al. (1936), were in no doubt that the feeding conditions were intimately involved, particularly when there was an abundance of feed in the late autumn and early winter. In 1925, Barry (quoted by Bayly et al., 1936) stated, however, that CVP "is not always due to an overfat bodily condition" and reported that he had seen significant numbers of cases in ewes on poor quality grazing which were not considered fat. Both Leslie (quoted in McLean, 1956) and the reports from the Ruakura Animal Research Station (quoted by McLean 1956) indicated that ewes in poor condition (up to 20 pounds underweight) may be seriously affected.

Leslie (quoted in Bayly et al., 1936), like Fraser (quoted in Edgar, 1952), found that the feeding of turnips or sugar beet tops was significant and the former also emphasised the significance of suddenly providing poorly fed ewes with an abundance of good food. This resulted in the theory that the most important underlying aetiological factors were related to increased intra-abdominal pressure or to the distention of the abdomen, in particular where the ewes were in an unfit muscular state. Fethers (1939) also found the highest incidence when ewes were transferred from poor pastures to rich pastures and concluded that the primary factors were related to the volume of abdominal contents (whether due to lamb numbers, size of lambs or to food). He suggested that this pressure would act upon "flaccid musculature", causing CVP. He also proposed that such changes in diet might induce diarrhoea and that this would then be responsible for the straining which was noted at an early stage in the development of CVP. Once the straining was induced the condition was progressive. This suggested a multi-factorial aetiology. He did not mention the possibility of urinary obstruction. Further work by McLean (1957), and McLean and
Claxton (1960) found that intra-abdominal pressure varied with the posture of the animal, and was particularly increased in recumbency and that this effect might be further aggravated by sloping ground. It appeared that the nature of the abdominal contents (uterine burden and/or rumen contents) was of secondary importance and that the number of lambs exerted very little effect. This led the authors to conclude that the pressure exerted by the abdominal contents upon the pelvic-supporting structures was of primary aetiological significance. This introduced a mechanical aetiological aspect to CVP, the mechanical component being, supposedly, displacement of the urinary bladder, which in turn instigated the chain of events culminating in CVP. Hardy (1959) was also of the opinion that abdominal pressure combined with the natural tendency for a ewe to lie with her head uphill and a possible muscle flaccidity might be the initial events. Nutritional factors were also suggested in that hill pastures, being of poorer nutritional value, might induce a poor physical and physiological condition.

An extensive survey in England by Edgar (1952), resulted in the finding that many outbreaks of CVP were associated with nutritional variations which manifest as "endocrine imbalances" and which were possibly associated with a heritable predisposition. The overall effect of this, as suggested by Edgar (1952) was a loss of tone in the supporting structures of the genital tract. His investigation led him to conclude that, like Koch and Haisch (1943), a complex hormonal aetiology may have been involved. However, Edgar (1952) was unable to induce CVP with oral and parenteral stilboestrol without the additional influence of abnormally high abdominal pressure (by the application of a tight belt around the abdomen) and straining (induced by the administration of oxytocin). He found it difficult to suggest an aetiological factor which was common to all the outbreaks of CVP he investigated.

Bayly et al. (1936) and Leslie (1938) (quoted in McLean, 1956) reported that hypocalcaemia might be of significance in inducing a secondary flaccidity of muscle. Chavance (1944) proposed that CVP was due directly to a subclinical "rachitic state" in
view of his very high success rate of treatment and prevention with Vitamin D, calcium and phosphate supplementation. Numerous more recent publications have stressed the importance of the mineral status of affected ewes. Stubbings (1971) felt that calcium deficiency was important and demonstrated a statistically significant depression of serum calcium in ewes affected by CVP. This was also noted by Sobiraj et al. (1986). Klauzer and Brestansky (1973) published serum calcium concentrations in affected ewes (Figure 16) but did not obtain comparative samples from normal ewes. However, when compared to the normal values given in the standard texts (Benjamin, 1961; Schalm, Jains and Carroll, 1975; Doxey, 1983), the values obtained by Klauzer and Brestansky (1973) appear to be low. In all these investigations it should be noted that the samples were obtained from ewes already affected by CVP of varying severity.

The latest theory on aetiology proposed by Sobiraj et al. (1986) concerned the hormonal status of affected ewes. They concluded that, after analysis of blood samples, the levels of circulating 17-oestradiol were significantly elevated in cases of CVP. They suggested that under the influence of this hormone the tissues of the birth canal become relaxed and oedematous and that this was a possible primary factor in the development of the disorder. This supported earlier proposals by Koch and Haisch (1943), Richter and Gotz (1950), Fowler (1962), Merck (1973), and Schwarz (1975) that sheep affected by CVP may suffer from an hormonal imbalance and Edgar (1952) suggested that this may be inherited. In his attempts to induce CVP under experimental conditions, however, Edgar (1952) was unable to induce any detectable significant pathological changes even with excessive doses of oestrogens (stilboestrol).

Schulz, Balzascak and Richter (1985) indicated that in their series of cases the ewes were often at full term or overdue and carried multiple lambs which were overweight.

All the factors mentioned so far are, however, merely suggestions as to where the primary causes may lie and, indeed, it is possible to find authors who describe significant outbreaks of CVP in other systems, other breeds and under other topographical conditions.
Environmental, hormonal and management factors appear then to be the most favoured aetiological possibilities amongst the literature reports. While most workers have attempted to group together more than one factor in a complex of aetiological events, only Chavance (1944) has proposed a single aetiological possibility. Most opinion seems to be reflected in the statement made by Watson (1959), who wrote: "It would appear that flock management, weather, breed, twinning, terrain and type of feed all play their part in producing this perplexing condition".
3.6 : CLINICAL FINDINGS :

Two forms of the disease, represented by apparently
different clinically recognisable entities, were noticed by farmers and reported by Bayly
et al. (1936). These were:

a) a "long sausage shaped eversion" which occurred some two weeks or more
before lambing. It was not thought to involve urinary obstruction, so presumably
tenesmus was not common (Figure 14).

b) a "rounder more swollen shape" eversion which
occurred in the immediate prepartum period (two to five days) and was invariably
associated with complete urinary blockage. So serious was this obstruction that
urine had to be drawn off before reduction was possible (Figure 15).

The impression that there were two different forms of the same clinical entity was
thought by Bayly et al. (1936) to be a false premise and they preferred to classify them as
different degrees of the same condition. Laing (1945) described the appearance of "a large
reddish swelling at the vulva which appears some 7 to 28 days prior to lambing". The
ewe was characteristically listless, uneasy and grazed
little; she separated herself from the flock and gradually the swelling increased in size.
This was accompanied by straining and by congestion of the prolapsed tissue.
Inflammation was described as setting in, and the ewe often died as a consequence of
urinary obstruction.

McLean (1956) described the clinical signs in considerable detail. He noted that at first
the pathological displacement of the vagina began only when the vaginal wall was
protruded through the constrictor vestibulae muscle. Any displacement which was not as
Figure 14: Photograph showing the 'long sausage shaped' CVP described by Bayly et al. (1936).

Figure 15: The 'rounded-type' of CVP described by Bayly et al. (1936). The shape of the protrusion being imparted by the enclosed dilated bladder.
severe as this he regarded as being a normal feature of pregnancy and indeed of the non-pregnant state. Once the vaginal mucosa was visible at the vulva, either intermittently or permanently, the condition was said by McLean (1956) to be clinically significant. He described three forms of CVP which were different in appearance and associated with different complicating factors. They were:

i) mild prolapse: In these cases the pink, glistening and moist vaginal mucous membrane was visible permanently or intermittently between the lips of the vulva. Distress was minimal and little straining was noted unless a portion of the bowel or the bladder was associated with the displacement. In this event the clinical signs of distress, straining and apparent pain were often found to be severe.

In summary, the signs were marked while the degree of prolapse was slight or even not apparent.

ii) sub-acute prolapse: The mild form progressed rapidly to the state where the passively congested vaginal wall became dried, dark in colour, lacerated, cracked, contaminated and possibly even gangrenous. The shape of the sub-acute CVP was described as being roughly hemispherical to cylindrical, with a base of about 12 cm diameter and about 13 cm long. Oedema of the vaginal wall was a prominent clinical feature but it is important to note that McLean (1956) found the vestibule was not usually involved.

iii) acute prolapse: These were found to develop rapidly and were associated with almost continuous straining. The CVP was large, being some 18 to 20 cm in diameter, tense and showing extensive sub-mucosal haemorrhages. A possible complication of this type is the rupture of the dorsal vaginal wall and the eventration of variable amounts of large and small intestine or other abdominal organs. This was often accompanied by severe haemorrhage, and death may ensue.

Many authors (Mayor, 1958), Hughes Ellis (1958) and Straiton (1973) noted that clinically many cases present initially as dry, swollen, hard, lacerated and oedematous protrusions
often with gross contamination.

Straining was a commonly quoted sign and according to Hughes Ellis (1958) may be mistaken for the onset of labour. Belschner (1976) reported that there were usually no premonitory signs, that the prolapse occurred rapidly and there was little discomfort. He suggested that, as sheep were placid by nature, straining occurred only when the swelling and contamination with faecal and other matter caused discomfort and pain. Urinary obstruction preceded death which he considered was due to gangrene and/or uraemia. Mitchell (1983) found that the progression from mild to severe CVP may take several days.

Sudden rupture of the vaginal wall is suggested as being responsible for the entration of the intestines and death of some ewes (McLean, 1956; Roberts, 1971; Fielden, 1980; Mitchell, 1983; Knottenbelt, 1988). White (1961), Fox (1962), Hanson and Plant (1980), Knottenbelt (1988) and Anderson (1988) described cases of vaginal rupture which were not apparently associated with CVP. Bassett and Phillips (1955b) found however that vaginal contamination was present in ewes without any apparent CVP when these ewes had a history of CVP during a previous pregnancy and it may be that in some cases vaginal prolapse was not apparent before vaginal rupture. McLean (1956) found that CVP may be mild while the straining was severe and this may have been involved in the vaginal rupture syndrome.

The variations in clinical signs described in sheep are further confused as they may individually resemble conditions unassociated with CVP. The following conditions should be included in the differential diagnosis:

i) Urinary obstruction or dysuria arising from infection or inflammation of the urinary or genital tract, or peritonitis.

ii) Parturient conditions including

a) uterine torsion
b) "Ringwomb" or failure of cervical dilatation  c) maternal or foetal dystocia

iii) Constipation

iv) Neoplastic and granulomatous conditions of the cervix, vagina, vestibule or vulva.

v) Post-parturient conditions including
   a) uterine prolapse
   b) post-parturient cervico-vaginal prolapse
   either as a disease in its own right or as a recurrence of a preparturient CVP.

vi) The appearance of the foetal membranes.

vii) The prolapse of the urinary bladder.

In view of the difficulty encountered by many workers in classifying the various forms of CVP it is perhaps relevant to include a description of that used by Hudson (1980) for bovine vaginal prolapse. A modification of this, incorporating some of the features described by Bayly et al. (1936), McLean (1956) and Cox (1981), is used later on in this thesis in an attempt to incorporate all the various types of CVP which can be seen.

Hudson (1980) used a system of four degrees. These were:

i) First Degree: The floor of the vagina protruded intermittently from the vulva, usually when the cow was recumbent. This may not be noticed but the vaginal wall was irritated by exposure to wind, sun, dust, cold, faeces and wet ground. This induced tenesmus.

ii) Second Degree: The floor of the vagina was permanently prolapsed. The bladder may have been included in the prolapse, resulting in tenesmus and dysuria. Exposure is obvious even to the casual observer.

iii) Third Degree: The entire vagina and cervix were prolapsed. This stage may be reached apparently without progression through the milder forms, depending on
the flaccidity of the supporting structures of the urogenital tract.

iv) **Fourth Degree**: Prolonged exposure of the cervix and the vagina resulted in necrosis of the deep tissue followed by the development of adhesions around the vagina. Peritonitis was occasionally present and there may have been a rectal prolapse. Tenesmus was often severe.
3.7 : CLINICAL PATHOLOGY :

3.7.1 : Biochemistry :

Investigations into the clinical pathology of CVP have not been undertaken to any great extent. Stubbings (1971) estimated serum levels of calcium, inorganic phosphate and magnesium in affected ewes and compared these with samples from unaffected animals. His results, summarised in Figure 16, showed that there was a significant reduction in the calcium but that there were no differences in the levels of inorganic phosphate or magnesium. Hindson and Turner (1972), commenting upon these findings, questioned the significance of the results, stating that the degree of depression of the serum calcium demonstrated was unlikely to be physiologically significant. It is worth noting that Stubbings' (1971) work related to CVP while the results obtained by Hindson and Turner (1962) related primarily to cases of incomplete cervical dilatation. It is perhaps unwise to assume, like Hindson and Turner (1972), that the two diseases are necessarily related or have a common aetiology.

That Stubbings (1971) results are significant must be undisputed from the statistics shown. The same effect was demonstrated subsequently by Sobiraj et al. (1986) (Figure 16) (p < 0.01).

Klauzer and Brestansky (1973) measured calcium, inorganic phosphate, magnesium and total protein concentrations in the blood of 22 cases of CVP. The mean values for these parameters are also shown in Figure 16. These workers did not record control values however and accepted values for normal ewes (Doxey, 1983) are shown for comparative purposes. The indications were, however, that the serum calcium levels were lower than
<table>
<thead>
<tr>
<th>Author</th>
<th>No. of ewes</th>
<th>Serum calcium (m moles/l)</th>
<th>Serum magnesium (m moles/l)</th>
<th>Serum inorganic phosphate (m moles/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stubbings (1971)</td>
<td>28</td>
<td>Mean 2.35, Range 2.00 - 2.75</td>
<td>Mean 1.14, Range 0.85 - 1.85</td>
<td>Mean 3.05, Range 1.56 - 3.56</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3* Mean 2.35, Range 2.00 - 2.75</td>
<td>2.17, Range 0.85 - 1.60</td>
<td>2.15, Range 0.95 - 3.00</td>
</tr>
<tr>
<td>Klauzer and Brestansky (1973)</td>
<td>22</td>
<td>Mean —, Range 1.78, 1.58 - 2.33</td>
<td>Mean —, Range 0.80, 0.45 - 1.40</td>
<td>Mean —, Range 2.45, 1.56 - 4.17</td>
</tr>
<tr>
<td>Bostedt and Hausman (1980)</td>
<td>not stated</td>
<td>Mean &gt;2.0, Range —</td>
<td>Mean &gt;0.82, Range —</td>
<td>Mean &gt;2.26, Range —</td>
</tr>
<tr>
<td>Sobiraj et al. (1985)</td>
<td>16</td>
<td>Mean 2.1 - 2.5, Range 1.8 - 2.2</td>
<td>Mean —, Range —</td>
<td>Mean 1.2 - 2.1, Range 1.3 - 1.5</td>
</tr>
<tr>
<td>Doxey (1983)</td>
<td></td>
<td>Mean 2.5*, Range 2.4 - 2.6*</td>
<td>Mean 0.94*, Range 0.85 - 1.03</td>
<td>Mean 2.02*, Range 1.42 - 2.68*</td>
</tr>
</tbody>
</table>

*cases of CVP at parturition

not necessarily pregnant

Figure 6: Serum calcium, inorganic phosphate and magnesium concentrations found by previous workers in cases of CVP. Normal reference values are also given.
normal while the magnesium, inorganic phosphate and total protein were not significantly changed.

The significance of calcium levels in cases of CVP was first proposed by Chavance (1944) who found that both sheep and cattle responded well to vitamin D therapy.

Moseley and Axford (1973) found a significant depression of serum calcium levels in ewes in response to physiological stress. As the samples obtained by all the investigators were from existing cases of CVP which might be expected to be under stress, the depression in serum calcium seen in cases of CVP may be of no aetiological significance. This can only be determined by sampling a large number of sheep and attempting, thereby, to predict cases of CVP, a procedure not so far undertaken.

Sobiraj et al. (1986) measured plasma levels of sodium (Na⁺), potassium (K⁺) and inorganic phosphate (PO₄⁻) and found that the levels were not statistically significantly different from normal ewes (probabilities were not stated). They did, however, gain an impression that affected sheep were in the lower normal range for all these parameters. Again, what effects may be the cause of the problem and what may be the result of the CVP is uncertain. All these ewes had been treated by the Buhner technique (Buhner, 1958) and had been kept in a hospital where presumably the diet, environment and climate would have been significantly different from normal. It is possible that the changes were secondary to the effect of the prolapse. Sobiraj et al. (1986) did not indicate the severity of the condition at their initial examination but stated that all the ewes survived to lamb normally and the average lambing rate was 1.8 lambs per ewe. It may be that the prolapses in their study were therefore not severe.

There have been few other investigations into the biochemical status of affected ewes.

3.7.2: Haematology:

The results of Klauzer and Brestansky (1973) are the only apparent, published haematological values for ewes affected by CVP. The results did not provide comparative
or control values for normal ewes unaffected by CVP. In spite of this, the values given may be an indication of some of the changes. These workers found that the mean value (and the range) for the haemoglobin, erythrocyte and haematocrit in 22 ewes affected by CVP were 11.0 (9.6 - 11.8) grams/dl, 6.20 (3.8 - 9.4) x 10^{12}/l and 26 (22 - 39)% respectively. These values are generally within the normally accepted ranges (Schalm et al., 1975; Doxey, 1983).

A mean total leucocyte count of 8.1 x 10^9/l (range 4.9 - 16.5) was found by Klauzer and Brestansky. No control or reference values were stated but by comparison to the normal means and ranges quoted in Schalm et al. (1975) and Doxey (1983), the range is high. They demonstrated a moderate leucocytosis amongst ewes affected by CVP. Unfortunately they published neither the results of differential leucocyte counts which might have indicated which particular cells were increased or decreased nor any statistical analysis.

3.7.3: Hormones:

The changes in 17-βoestradiol and progesterone concentrations as ewes affected by CVP approach parturition have been published by Sobiraj et al. (1986). The changes which occur in normal unaffected ewes have been the subject of considerable investigation (Short, 1960; Rawlings and Ward, 1972, 1976, 1978; Tsang, 1974, 1978; Cox, 1975). Bassett, Oxborrow, Smith and Thorburn (1969) quantified the progesterone levels and found that the concentrations rose significantly between 50 and 120 days gestation, reaching a maximum at this point which was maintained until the last week of pregnancy when there was usually, but not invariably, a fall in the progesterone concentration. This change is depicted in Figure 17 which is derived from the work of some of these authors. They also demonstrated that concentrations in ewes carrying twins were approximately twice those in ewes with single lambs. Stabenfeldt, Drost and Franti (1972) also found that there was a statistically significant difference in ewes carrying twins as opposed to single
Figure 17: Diagrammatic representation of the hormonal changes which occur in the last weeks of pregnancy deduced from the published material (Bassett et al., 1969; Chamley et al., 1973; Tsang, 1978 and Sobiraj 1986).
lambs, possibly, they suggested, due to the placental production of progesterone. Challis, Harrison and Heap (1971) also noted a difference as pregnancy advanced and between those ewes carrying single and multiple foetuses. Chamley, Buckmaster, Cerini, Cumming, Goding, Obst, Williams and Winfield (1973) found progesterone levels to be maximal 10 days before lambing and, like Rawlings and Ward (1972, 1976, 1978), noted a fall in the last 10 days of gestation both in the blood and the myometrial levels. Tsang (1978) demonstrated a sharp rise from 60 days prepartum with a peak progesterone concentration of 16–22 ng/ml (50–70 p.moles/l) in the last 10–20 days of pregnancy. Subsequently Tsang (1978) found there was a variable reduction with a steep decline in levels two days prepartum.

The normal changes in progesterone are therefore well established, and the pattern of variation through pregnancy and differences in uterine burden are clear. The most significant features would appear to be:

a) peak levels occur at approx 130–140 days gestation,

b) a higher peak level in ewes with twin pregnancies than for singletons,

c) a decline in the last 10 days, particularly in the last 2–3 days.

Oestrogen levels have also been investigated in depth largely by the same authors and again a pattern of change is apparent (Figure 17). Challis (1971) noted in 7 pregnancies from 5 sheep that concentrations were very low (mean approx. 20 pg/ml) in the early stages of pregnancy and remained low up to the day before parturition when a mean tenfold increase was noted (the maximum increase being up to 411 pg/ml). This occurred at the time when the concentration of progesterone was decreasing and with it the progesterone–induced inhibition of the myometrium. The same pattern has been described by Challis, Harrison and Heap (1971), Robertson and Smeaton (1973), Chamley et al. (1973), Tsang (1974) (1978), and Rawlings and Ward (1976). None of these workers have established any significant difference between twin and single pregnancies with respect to the oestrogen concentrations.
Sobiraj et al. (1986) compared the levels in normal ewes with those in ewes affected by CVP. They reported no significant variation in progesterone levels, but noted that CVP-affected ewes had higher concentrations of the biologically active 17-βoestradiol than normal ewes up to the characteristic and well described preparturient rise. These authors proposed a causal relationship between this higher oestrogen level and the clinical development of CVP.

No work appears to have been done to verify the long held view that CVP is the result of an endocrine abnormality (Koch and Haisch, 1943; Richter and Gotz 1950, Edgar, 1952 Schulz et al., 1985).
3.8: TREATMENT:

Several different forms of treatment have been advocated over the years by shepherds and veterinary surgeons. Laing (1945) found that many of these are best regarded as barbaric practices, such as the use of bran to fill the vagina or short sticks inserted therein. The more acceptable methods range from simple management or handling techniques to complex surgical approaches. Most shepherds and veterinary surgeons claim success from one or other particular method with which they have become familiar. McLean (1957) was in little doubt that most cases could be saved by expert veterinary attention given in the early stages, but economic factors needed to be considered. Bayley et al. (1936) noted the urgency of diagnosis and more particularly of treatment for successful results. There have been many treatments advocated and most authors had one which they promoted strongly (Chavance, 1944; Laing, 1945, 1949; Hughes Ellis, 1958; Mayor, 1958; Hardy, 1959; Watson, 1959; Blackmore, 1960; Hartigan, 1961; Fowler, 1962; Eagelman, Beebe and Leman, 1967; Belschner, 1976; Rusch and Berchtold, 1978; Pastoor and Weitenberg, 1979, and Arthur et al., 1982). Several authors have given an adequate summary of some of these methods (Merck 1973; Cox, 1981; Arthur et al., 1982; Knottenbelt, 1987) but most presented a common initial approach to the treatment of CVP which were minor variations on the following:

i) the tissue should be cleansed carefully using non-irritant solutions and dressed with antibacterial substances,

ii) the prolapse should be fully reduced,

iii) the vagina should be retained in its correct position.
Figure 19: Table showing the suggested treatment protocols (+) of various authors and those aspects which were not successful (0) in the hands of the authors.
Some variations at each stage and several extra stages have also been advocated. Figure 18 summarises each worker's suggested approach.

There are few reports of the consequences of withholding treatment completely. Laing (1945) and McLean (1956) reported occasional spontaneous resolutions. In view of the further statements made by McLean (1956) that most cases died if not treated and most cases survived if given suitable veterinary care, this would not appear to be a satisfactory approach in practice.

Bayly et al. (1936) and Mclean (1957) found that farmers showed a strong selection for favourable cases before treatment was attempted. Severely affected animals considered to be beyond treatment were killed. In some cases all affected ewes were slaughtered regardless of the extent of CVP. Both these authors found that farmers generally advocated gentle cleansing and possibly the application of an astringent such as dilute vinegar. Olive oil was then used as a lubricant. Having reduced the prolapse, wool strands from the perineal area and the posterior thighs were tied across the vulva (as advocated later by Laing, 1945; Belschner, 1976; Cox, 1981; Mitchell, 1983, and Clarkson and Faull, 1983). Safety pins or sutures across the vulval labiae were also advocated. Exercise was used as an adjunct to these methods (Bayly et al. 1936; Laing, 1945). A success rate of 15-90% was claimed by Bayly et al. (1936).

The simplest method suggested by Laing (1945) was the crude but effective one of tying the ewe's hind legs to a fence "for a few hours". This employed the postural effect which has also been shown to be an effective method of treatment by Palsson (quoted in Edgar, 1952) who suggested that by allowing the standing to build up at the back it was possible to exert sufficient force on the perineum to prevent and to control CVP. Bruere (1956) and Slatter (1981) (personal communication) suggested that an effective method of treatment for the milder forms of CVP was to restrain the ewe in such a way that the hindquarters of the patient were elevated some 10 - 20 cm above the forequarters and with this objective the latter made several crates which were developed from
3. Pass lateral to vulva, crossing below.

2. Join together, knot at base of tail.

1. Tie round girth

4. Tie over abdominal spine

Figure 19: Drawing showing the method of construction of a string truss for the retention of CVP as described by Fowler (1962) and Cox (1981).
commercially available lamb adopter pens.

In addition to the basic approach mentioned above, Fethers (1939) advocated the elevating of the hindquarters to aid the actual reduction, helped by gentle pressure from a flat hand and a slight rotatory movement. He also advocated leaving the hand inside the vagina to ensure full reduction and, once completed, the ewe should be restrained so that her hindquarters be elevated relative to her forequarters in the standing position. Ewes which strained sometimes required vulval suturing with coarse silk sutures which were removed after 4 or 5 days. Laing (1945) made a point of avoiding the use of strong irritant antiseptics as he considered that they were inclined to cause straining. Personal hygiene and sympathetic handling of the tissues were emphasised. He suggested the use of chloral hydrate per os to control straining. Stitches, pins and other vulval closure apparatus were to be reserved for the more severe or chronic cases. Merck (1973) advocated the use of Laudanum to control straining.

Laing (1945), Marsh (1958), Fowler (1962), Cox (1981) and Clarkson and Faull (1983) advocated the use of externally applied trusses made of rubber, string or canvas, which act by applying pressure to the perineum lateral to the vulval lips (Figure 19). Laing (1945) used a piece of car inner tube with a hole cut in it which was tied with binder twine to the wool on the flanks. A string and canvas harness, as proposed by Marsh (1958), exerted a restraining force upon the tissues of the perineum and so retained the prolapse.

There are a number of further methods proposed in the literature which do not involve surgical interference. The first of these, suggested by Hartigan (1961), involved the induction of an artificial pneumoperitoneum. This technique had previously been used by Esperson (1962) for the control of straining in cattle. Filtered air was pumped into the peritoneum via the left paralumbar fossa using a sterile hypodermic needle and an enema pump. Once a moderate degree of abdominal distention had been induced, Hartigan (1961) found that the ewe did not strain and that, as an indirect consequence, the prolapse did not recur. As he pointed out, it was not clear why this was effective and in view of
Figure 20: Diagram with dimensions of the 'home-made' wire intravaginal prosthesis advocated by Laing (1949) and Fowler and Evans (1957).

Figure 21: Photograph of the commercially available Daltons Spoon (Arnolds Ltd., UK) advocated by Merk (1973), Rusch and Berchtold (1978), Cox (1981) and Mitchell (1983).
Figure 22: Diagram of the modification of the Daltons Spoon proposed by Pastoor and Weizenberg (1979) showing the trans-vulval threaded locking pin.
McLean's (1956) and McLean and Claxton's (1960) work on intra-abdominal pressure, it appears even more difficult to explain. Intra-abdominal pressure would be expected under such circumstances to be abnormally high but Hartigan's (1961) results appear to be excellent. However, he reported the treatment of only 3 ewes in this way. Jones (1958) found that where ruminal bloat existed the incidence was higher and this would seem to confirm that when the intra-abdominal pressure is elevated there is an increased tendency for the development of CVP. The simplicity and lack of tissue interference in Hartigan's (1961) method suggests that it should have been widely adopted but this has not apparently been the case.

A further method was proposed by Mayor (1958). This entailed the use of an inflated football-type bladder inserted in the vagina for several days after the reduction of the CVP. He reported a high success rate but the method was not tried extensively and has not gained universal appeal. Hartigan (1961) found this method to be unsatisfactory although he does not state why. There are no published results from tests of this method.

Work in 1949 by Laing proposed the use of an intravaginal device made from no. 8 wire in the form of a 'U' such as is illustrated in Figure 20. The apparatus was inserted into the vagina after reduction of the CVP in such a way that the rounded end rested in the anterior vagina or fornix and the arms were tied on each side to the wool of the flank. The apparatus had been in use for many years in his district and had gained a good reputation. This method was also proposed by Fowler and Evans (1957), by Hardy (1959) and by Jones (1958) following its availability on a commercial basis. Again, good results were reported. The apparatus has subsequently been modified in various ways including the plastic Dalton's spoon by Merck (1973), Rusch and Berchtold (1978), Cox (1981) and Mitchell (1983) which is shown in Figure 21. A modification widely used in Holland which sutures the device to the perivulval skin using eyelets located at the neck was described by Pastoor and Weitenberg (1979). This modification is illustrated in Figure 22. However, Watson (1959) found no great advantage in the use of the Dalton's spoon.
Figure 23a and b: Schematic diagram showing the placement of the intra-vaginal buttons advocated by Eagelman et al. (1967) for the retention of CVP.
Hartigan (1961), Straiton (1973) and Fowler and Evans (1957) found that the results were sometimes gratifying and sometimes disappointing and that ewes, contrary to the widely held belief, were not always able to lamb past the device, an advantage which had always been a major stimulus to the use of such devices.

Following work by Hentschal (1961) on the use of an intravaginal anchoring method in cattle, Eagelman, Beebe and Leman (1967) developed a similar method for use in sheep. This involved the suturing of discs of plastic from the anterior dorso-lateral aspect of the vagina to similar discs applied externally over the gluteal muscles. The technique is illustrated in Figure 23 (a and b) and required the use of epidural anaesthesia. The method obviously needed a good knowledge of surgical anatomy and is not without hazard in terms of infection and serious trauma such as interference with the sciatic nerve or the major blood vessels of the pelvis. Eagelman et al. (1967), however, found no complications with locomotion, defaecation or parturition following its use. The major advantage of Eagelman’s et al. (1967) method, as well as of the other intra-vaginal devices was supposedly that lambing would occur without the need for interference. In the case of Eagelman’s et al. (1967) technique, this seemed to be true although it was based only on a small sample. A further interesting side effect of the ‘Eagelman’ technique derived from the development of adhesions and scarring along the lines of the sutures which appeared to reduce the tendency for recurrence within the limited numbers followed. This may be difficult to confirm in view of the findings of several workers (Watson, 1959; Hardy, 1959) that recurrence does not invariably take place in subsequent pregnancies.

From the earliest reports of Bayly et al. (1936) various methods of vulval closure have been suggested. Various suture patterns have been advocated and these are illustrated in Figures 24, 25, 26 and 27. There are basically two different patterns and one surgical procedure. The first pattern includes safety pins of the locking ‘nappy type’ (Laing, 1945, 1949) (Figure 24a), the Flessa type pins with screw on ends (Gyorgy, 1968; Pastoor and Weitenberg, 1979) (Figure 24 b), the simple transverse interrupted sutures (Bayly et al.
SAFETY PIN TECHNIQUE (LAING 1945)


SIMPLE INTERRUPTED TRANS VULVAL SUTURES (BAYLY et al 1936 et passim)

FIGURE-OF-EIGHT SUTURE (BLACKMORE 1973)

HORIZONTAL MATTRESS SUTURE (STRAITON 1973)

"QUILL-SUPPORTED" HORIZONTAL MATTRESS SUTURE (MORENO 1971)

Figure 24 a, b, c, d, e and f: Diagrammatic representation of the suture patterns advocated by various authors for the retention of CVP in ewes.
Figure 25 a and b: Diagrams of the vulval inversion suture methods used to retain CVP.
1936; Fethers, 1939; Laing, 1945, 1949; Walsby, 1952; Marsh, 1958) (Figure 24c), the figure-of-eight suture (Blackmore, 1960) (Figure 24d), horizontal mattress sutures (Straiton, 1973) (Figure 24e) and supported mattress sutures (Moreno, 1971; Rusch and Berchtold, 1978; Mitchell, 1983) (Figure 24f). All of these appose the vulval lips or the vestibular walls.

The second type of vulval closure involves the methods which tend to invert the vulval lips and which therefore tend to appose the perivulvar skin on either side of the vulva itself. These methods include the quill-supported inversion suture (Gyorgy, 1968; Cox, 1981) (Figure 25a) and the lace-up method (Merck, 1973) (Figure 25b).

The surgical methods for vulval closure include the Buhner subcutaneous perivulvar purse-string suture (Buhner, 1958; Kubicek, 1977; Fielden, 1980; Arthur et al., 1982) and the Caslick's sub-mucous resection method (Roberts, 1971; Merck, 1973; Franklin, 1986).

Buhner's method in cattle (Buhner, 1958) has been adapted for use in the ewe. A subcutaneous purse-string suture was laid around the vulva in such a way that it reinforced the action of the constrictor vestibulae muscle. Local infiltration analgesia or caudal epidural anaesthesia was applied before two horizontal 1 to 2 cm long incisions were made, one between the anus and the dorsal commissure of the vulva and the other some 4 to 5 cm below the ventral commissure. A special Buhner needle was used to insert the suture material which several authors suggest could be umbilical tape (Fielden, 1980; Arthur et al., 1982; Mitchell, 1983) or any heavy gauge suture material (Arthur et al., 1982). The Buhner method and the equipment required is illustrated in Figure 26.

A full surgical submucous resection technique (Caslick's Operation), such as is frequently used for the treatment of pneumovagina in horses (Arthur et al., 1982) and vaginal prolapse in cattle (Farquharson, 1949), has been advocated by Roberts (1971), Merck (1973) and Arthur et al. (1982). This involved the resection of a strip of the vaginal mucous membrane from each side of the vulva at the muco-cutaneous junction from about 1/2 to 1/3 of the vulva dorsally and suturing the raw edges so obtained in 2 layers
Figure 27: Diagram of the method of sub-mucus resection for the treatment of CVP (Roberts, 1971; Merk, 1973; Arthur et al., 1982).
(Figure 27). The vulval aperture was much smaller after healing. It was then necessary to perform a surgical episiotomy at parturition. The method relies upon primary union healing and this would be expected to occur over 7 - 10 days. As most workers have found that CVP occurs within the last 7 - 10 days of gestation the method would not seem to be a logical approach to the problem in most cases.

The object of the vulval suture methods and Buhner's method was to mimic the effects of the constrictor vestibulae muscle and most authors emphasised that the sutures need not be pulled very tight (Fethers, 1939; Hughes Ellis, 1958). Cox (1981) stated that Buhner's suture should be tightened only sufficiently to allow the passage of a lubricated upright flat hand into the vulva. Straiton (1973) advocated the use of a horizontal mattress suture applied through the lips of the vulva and tied as tightly as possible. This is a dangerous statement, open to misinterpretation by those reading his book. Even the earliest of authors (Fethers, 1939, Laing, 1945) advocated the use of loose sutures.

The proponents of the use of transvulval sutures have an almost equal number of opponents. Thus Fowler and Evans (1957), Mayor (1958), Watson (1959), Hartigan (1961), Fowler (1962), Schwarz (1975) and Belschner (1976) considered that they might result in vulval lacerations in the event of straining being continuous or strong. Such lacerations could become infected although the main reason for the rejection of the methods was probably that ewes required more attention at parturition.

Opinions are divided concerning the efficacy of intravaginal devices depending upon the requirement that ewes be able to lamb unaided and at the same time to ensure firm retention of the prolapse. Certainly the latter methods rendered normal lambing impossible when they were applied sufficiently tightly to retain a CVP. Lambing therefore has to be closely supervised. Whether this was a disadvantage or not is perhaps a matter for debate, but there can be little outward sign of internal problems associated with the vagina such as vaginal rupture, necrosis, dystocia, or even relapse of the CVP when vulval sutures are in place. Almost all the authors who support the use of vulval sutures pay particular attention
to the need for cleanliness and avoiding the placing of the sutures through the vulval skin. Cox (1981), like others, suggested that the sutures should be located at or beyond the wool line. Schwarz (1975) also stated that it is preferable not to pierce the labiae vulvae.

The use of caesarian section as a method of treatment was first advocated by Walsby (1952). Up to that time the results of such surgery were poor (Benesch and Wright, 1950) but Walsby (1952) developed a satisfactory surgical technique and suggested that it could ensure an improved prognosis for both the ewe and her lamb(s). He pointed out that 'section' of ewes effected by CVP which were not yet at term may result in complications, more for the lambs than the ewe. Premature lambs may not be viable or may not be able to be mothered properly due to the absence of milk. He advocated, therefore, that caesarian section should be delayed as long as possible up to full term by the use of any other means of retention of the CVP, but that in any event the technique was an effective alternative form of therapy. This was emphasised subsequently by de Bois (1958), Blackmore (1960), Rusch and Berchtold (1978), Fielden (1980), Mitchell (1983) and by Schulz et al. (1985). The latter authors stated that caesarian section was the "only way to keep ewes and lambs alive". Mitchell (1983) suggested that this surgical approach was satisfactory from 142 days gestation. From Blackmore's (1960) results the ewe was more often saved following caesarian section than were the lambs.

Another unusual method of treatment was used by Watson (1959). This was the parenteral injection of penicillin and stilboestrol in ewes which would not tolerate other methods of treatment, including the wire retainer, balloon or stitching. He found that those ewes which did not abort within 24 hours had usually stopped straining and would then accept a retainer. Whether this author was advocating the induction of abortion as a means of treatment is not certain, and the rationale behind this treatment must be questioned, particularly in view of Edgar's (1952) attempts to induce CVP with parenteral stilboestrol. Chavance (1944), following his 'rachitic theory' as a cause of CVP, obtained a high success rate with the use of oral Vitamin D, calcium and phosphate therapy. There are no
further reports of the use of this approach but Bayly et al. (1936) and Watson (1959) noted that the oral supplementation of minerals did not appear to have a significant effect. Chavance (1944), however, claimed good results both for treatment and prophylaxis. Investigations into the effects of treatment of CVP is limited to Edgar (1952) who tested the use of human chorionic gonadotrophin by sub-cutaneous injection in 8 affected ewes. This followed the suggestion by Koch and Haisch (1943) that such an approach was effective in treating cows with vaginal prolapse. Edgar's (1952) results were emphatic in that such therapy had no effect, either upon the clinical entity or upon the outcome.

Amongst all the suggestions for treatment a common theme is the need for the control of straining. It would appear that the major reason for the failure of many of the methods of retention is disruption and tearing, or the displacement of the retaining device. Only the football bladder approach (Mayor, 1958) and Hartigan's (1961) artificial pneumoperitoneum appear to be unaffected by straining. Since it would seem that the success or failure of most of the methods hinge upon the presence or absence of straining, 'soothing oils' were advocated by early workers including Bayly et al. (1936) and Laing (1945) and systemic drugs such as chloral hydrate (Laing, 1945) and laudanum (Merck, 1973) have also been advocated.

The use of local anaesthesia was first mentioned by Hughes Ellis (1958), although it is not clear whether his infiltration of the vulval labiae was to allow the insertion of sutures alone. Surface application of anaesthetic agents to the 'membrane' is mentioned by Roberts (1971) but was not found to be very successful. Eagelman et al. (1967) used a mixture of 95% ethanol and procaine to induce epidural anaesthesia in order to apply their retention buttons but noted that it reduced severe straining after reduction of the prolapse. Roberts (1971) suggested the use of epidural anaesthesia or the regional block of the sacral nerves using lignocaine or ethanol. The use of alcohol as an epidural local anaesthetic was proposed by Roberts (1971) to induce prolonged anaesthesia (for up to 2 months). Merck (1973) suggested that short-term replacement and retention might be achieved with
epidural anaesthesia.

Anaesthesia of the sacral nerve plexus (pudendal plexus) was used by Hertrampf et al. (1978) as an adjunct to other forms of therapy. By using 22-25% ethanol as a perineural anaesthetic agent applied to the sacral nerves they found that pain, discomfort and severe straining were successfully controlled in 30 out of 32 cases. Twenty-five (78%) of the ewes were cured by this treatment alone. There were no undesirable effects upon locomotion, rumination, defaecation or parturition. Arthur et al. (1982) also advocated the use of epidural anaesthesia but did not describe the use of ethanol.

The methods of administration of epidural anaesthesia were described by Hall and Clarke (1983). An anterior route at the lumbo-sacral junction and a posterior approach at the junction between the first two coccygeal vertebrae were described. The former method appears to be more complex and required that the ewe be restrained in lateral recumbency with the spine flexed dorsally as far as possible. The latter method appears easier but it was apparently not always easy to locate the foramen. A method for regional block of the sacral nerves was described by Hertrampf et al. (1978) who used this as a form of treatment and advocated its use specifically for CVP. However, Mitchell (1983) noted that local anaesthesia was seldom necessary under any circumstances.
3.9: PROGNOSIS:

In considering the veterinary approach to any commercial animal suffering from a disease the prognosis is always of paramount importance, and as would be expected, various researchers have presented assessments of this factor. Bayly et al. (1936) did not present any opinion on the prognosis for untreated animals, but they did mention that the prognosis would be expected to vary according to how soon the prolapse was treated after eversion. They implied that the prognosis may be better if the sheep were closely attended. An assessment of the prognosis for a disease where farmers have an aggressive attitude to cases is likely to be the most difficult. Fethers (1939) stated that if the trouble occurred just before delivery of the lamb, the outlook was all the more serious. By this it is assumed that CVP close to lambing would have a worse prognosis. Chavance (1944) recorded excellent therapeutic and prophylactic results following treatment with vitamin D, calcium and phosphate, but did not quantify them, nor did he state the severity or degree of CVP.

Laing (1945) found that, if early sympathetic treatment were under-taken, "a good percentage of cures can be obtained", but about half the affected ewes died or had dead lambs. He noted the need to observe cases for relapse after lambing.

In his work on the indications for caesarian section in the ewe, Walsby (1952) noted that the prognosis for the lambs was improved when such surgery was delayed until parturition was imminent. Rusch and Berchtold (1978) also suggested that the prognosis for the ewe may be improved by caesarian section. The earliest reports were by Bayly et al. (1936), who found that "rather perfunctory" treatments given by farmers produced a success rate of between 15% and 90%, but noted that this related only to the mildest cases, all others
being killed. In 1952 Edgar published the first quantitative results on the prognosis and concluded that nearly half the ewes died (43%) and one-third of the lambs of affected surviving ewes also died (33%). The latter figure included some 9% of lambs which died in utero and 24% which were born dead.

Laing (1945), McLean (1956) and Merck (1973) agreed that mild cases may recover spontaneously but the majority die if left untreated. Belschner (1976) found that untreated ewes generally died after a few days. This implied that the condition was progressive and that the prognosis for untreated ewes was always poor. A further study by McLean (1959) investigated in detail the mortality rate in ewes affected by CVP and their lambs. Excluded from his results were a number of cases where farmers killed the ewes without contemplating treatment. It was calculated that 61% of cases recovered, presumably with some form of treatment, 27% died and 13% of ewes were killed in extremis. Thirty-two percent of single lambs born to ewes with CVP were dead, while 58 cases produced one or more dead lambs from twin births. Only 154 live lambs were produced from a possible 256, which represents a 40% mortality in ewes surviving until full term.

Up to 50% of ewes were found by Fowler and Evans (1957) to die or suffer from retarded cervical dilatation (ringwomb) or have dead lambs. Like McLean (1959), these authors suggested that in real terms the prognosis was in practice much worse for the ewe in the long term, as farmers generally culled affected sheep either immediately, in the event of no live lambs being born, or at the end of the season in any case. Thus the prognosis in purely medical terms may be very different from the loss the farmer suffers as a consequence of the disease. Jones (1958) also suggested that the mortality was about 50%, but it is noticeable that most investigations appear to have relied heavily upon the findings of Edgar (1952) and McLean (1956, 1959). Watson (1959) recorded that, in 50% of cases, treatment worked well. Presumably this meant that the ewes recovered sufficiently to lamb normally and raise their lambs without complications.
In 1971, Roberts reported that the prognosis was "generally good but that 20 to 30% of CVP cases may die or expel dead foetuses" and Schwarz (1975) suggested that, in spite of treatment, "losses of up to 40% must yet be taken into account". It is not clear to which "losses" he was referring.

Rusch and Berchtold (1978), in Switzerland, found that 35 out of 54 CVP cases were affected by dystocia and that most of these involved 'ringwomb'. There was a significantly higher mortality in the lambs when compared to normal lambings. Their results showed that out of 95 lambs born only 66 survived (a mortality of 35%) while in normal ewes a survival rate of 92% was suggested.

The relationship between CVP and "ringwomb" has been the subject of considerable debate in the veterinary literature (Straiton, 1957; Edwards and Jones, 1957; Walsby, 1952; Kenworthy, 1957; Hindson and Turner, 1962; Kubicek, 1977, Rusch and Berchtold, 1978) and no satisfactory outcome of this debate is yet apparent. However, if we accept the majority decision that the two diseases at least occur under the same conditions (Arthur et al., 1982), then such complications may indirectly affect the prognosis of CVP. Secondary factors, such as incomplete cervical dilatation or intercurrent disease such as pregnancy toxaemia may also affect the overall prognosis.

Prognostic figures for CVP may also be confused by the attitudes and prejudices on the part of the farmers and, indeed, some veterinary surgeons. Thus Slatter, (1980, personal communication), after 40 years experience with sheep, made a habit of culling affected ewes either at lambing, in the event that no live lambs were born, or in any case at weaning. As the financial return was negligible for these ewes he, like Fowler and Evans (1957), suggested that the loss was total.

Post-parturient problems following CVP, including relapse (Bayly et al.,1936; Laing, 1945, 1949), prolapsed uterus (Marsh, 1958; Vandeplassche et al. 1963) and myiasis of the perineal tissues (Watson, 1959) have been recorded. Each of these conditions would ordinarily carry a poor prognosis in their own right, and in the event would probably be
regarded equally seriously by the farmer. Generally, investigators have found that the outlook for these animals was poor as they were frequently complicated by severe tenesmus, debility and infection.

In the opinion of Vandeplassche and Spincemaile (1963) CVP is definitely a predisposing factor for uterine prolapse which occurs frequently in the puerperium in ewes. The prognosis for this condition would, therefore, have a bearing on the prognosis for CVP when one preceded the other but Roberts (1971) suggested that there is no increase in the incidence of prolapsed uterus in CVP cases.

From the literature above it can be seen that the prognosis for either ewes or lambs is most difficult to assess. The speed and efficiency of treatment and the possibility of related preparturient and parturient problems are all likely to affect the survival of the ewe and/or the lambs. Perhaps it is for these reasons that few results have ever been published apart from those of Edgar (1952), McLean (1956, 1959) and Rusch and Berchtold (1978).
3.10 : POST MORTEM FINDINGS :

The post-mortem findings in animals which have died directly from CVP or from conditions secondary to the development of a prolapse have not been reported widely. Bayly et al. (1936) carried out a few such examinations which confirmed that the only abnormality identified was the eversion of the vagina. They noted, however, a number of blood clots, with considerable congestion, about the neck of the bladder which they presumed were lesions resulting from the disturbances of the circulation which followed the vaginal eversion and bladder displacement. Evidence of constipation was not a consistent finding. Where urinary obstruction was complete, possibly from the kinking of the urethra as already described, they noted serious back pressure upon the kidneys. They presumed that death in such cases was a result of obstructive uraemia.

Laing (1945) found that, at the post-mortem examination of CVP cases, there was much abdominal fat present in the organs. The liver was pale and greasy and 90% of the ewes subjected to necropsy carried twin lambs. The bladder was apparently always enormously distended with urine.

McLean (1956) reported that the majority of untreated cases died from uraemia, rupture of the bladder, toxaemia from secondary infection and occasionally intestinal strangulation. However he did not indicate whether these findings were the results of post-mortem examinations or whether, if so, any other organs were involved. Watson (1959) found that a frequent cause of death was rupture of the bladder.

Some of the pathological findings in freshly killed animals used for investigation by Bassett and Phillips (1955b) have been discussed in previous chapters (anatomy and pathogenesis) and are indicative of the kind of lesions which may be seen at post-mortem.
In 1961, White described an unexplained condition in heavily pregnant ewes in which rupture of the vagina and herniation of abdominal viscera were the prominent signs. Fox (1962) also reported one such case where a gravid uterine horn was prolapsed through a tear in the dorsal wall of the vagina. Fox (1962) did not perform a necropsy, but all three cases seen by White (1961) revealed a rent in the dorsal vaginal wall. No other changes were reported. While neither Fox (1962) nor White (1961) found these ewes to be affected by CVP, McLean (1956), Mitchell (1983) and Knottenbelt (1988) found that such a condition represented a possible complication of CVP. Roberts (1971) also suggested that vaginal rupture was invariably the result of CVP.
3.11: PREVENTION AND CONTROL:

The possibilities for the prevention and control of a disease about which little is known are likely to be fraught with difficulties. A disease entity, as has been shown in the Pathogenesis section, is most difficult to prevent and control when the factors of its apparently multifactorial aetiology are seemingly interlinked and possibly interdependent.

As will become apparent, unless the disease can be reproduced experimentally and the specific aetiological factors identified, such advice is likely to be vague at best, and at worst misleading. In spite of this, several authors have proposed means of prevention and of control.

Both Edgar (1952) and McLean (1956), after carrying out extensive work on CVP, concluded that for reasons of heredity and recurrence, all affected ewes should be culled from the breeding flock if any preventive action was going to be effective. The fact that Bayly et al. (1936) and other workers, including Laing (1945, 1949), Edgar (1952) and McLean (1956), have pointed out the significant incidence in certain breeds of sheep which have a wide, roomy pelvis, selective breeding policies may be sound practice in attempting to control CVP. Thus, the wider stanced Romney Marsh type of sheep may, because of its anatomical conformation, be responsible for the appearance of a significant incidence of CVP when crossed with a narrower type of ewe such as the Merino. This occurrence was reported by Bayly et al. (1936) where the introduction of a Romney Marsh ram resulted in an increase in the incidence of CVP in flocks of Merino ewes. When a Merino ram was re-introduced the number of cases of CVP again fell to normal levels. The farmers reported to these authors that the disease did not occur in the pure Merino but that crosses with Romney, Southdown and Lincoln, either as first or second cross,
were affected. The significance of this type of inherited characteristic was also commented upon by Laing (1945, 1949), Edgar (1952) and McLean (1956).

The fact that a high incidence of twin and multiple pregnancies has been found (Bayly et al., 1936; Fethers, 1939; Laing, 1945, 1949) suggests that an inherited or managementally manipulated fecundity would possibly exert an indirect effect. Clarke (quoted in McLean 1959) has also confirmed that heredity was an important factor in the aetiology of CVP. The advice to cull affected ewes has subsequently been given by some authors as an effective preventive measure (Fowler and Evans, 1957; Merck, 1973; Schwarz, 1975; Fielden, 1980). Indeed, Fielden (1980) was most assertive that the culling of ewes and their lambs provided the cornerstone of prevention and control policy for CVP. There is not, however, any apparent published experimental work which has proved the beneficial effect of breeding policy and culling as a means of prevention or control, although Watson (1959) stated that work was being undertaken at the Ruakura Research Station in New Zealand. Some 100 breeding ewes, the daughters of ewes which had themselves been affected by CVP, were at that time being managed under controlled conditions of management, specifically to establish the question of heritability.

Regular exercise for heavily pregnant ewes as a means of reducing the incidence of CVP was suggested by Bayly et al. (1936), Laing (1945), Belchner (1971) and Fielden (1980). This was said to increase muscle tone and thereby decrease the opportunities for the development of CVP, and the results reported by Bayly et al. (1936) would certainly seem to confirm this. These authors described one farmer who, by driving the ewes about a mile in the morning and evening, was successful in controlling the disorder. The effect of this approach may not have been directly upon muscle fitness but upon the reduction in the number of ewes that were recumbent for extended periods or other undefined factors. The absence of exercise was also proposed by Bayly et al. (1936) and Laing (1945, 1949) as an aetiological factor. It is difficult, therefore, to separate the individual aetiological factors and thereby enable one to draw conclusions as to the effect exercise might be
expected to have on the possible control of the disorder. Mitchell (1983) suggested that movement of sheep should be unhurried and jostling avoided and this might be taken to mean that exercise as a means of control should be limited rather than encouraged.

The findings of veterinary surgeons quoted by Bayly et al. (1936), their own findings, and those of Laing (1945, 1949) Fethers (1939), Jones (1958), Fraser (quoted in Edgar, 1952), Edgar (1952), Hardy (1959) and others, that variations in incidence occurred under different nutritional regimens indicated that judicious feeding management, particularly in the last trimester of pregnancy, may provide some hope of control. Belchner (1971), Straiton (1973), Merck (1973), Fielden (1980), and Mitchell (1983) all suggested that the control of CVP may be aided by careful attention to the feeding policies. However, it is not a simple equation and the nutrition may be linked to exercise, environment and to climatic conditions prevailing at the time and at other stages in the year (Watson, 1959).

The feeding of certain materials has, however, been closely correlated with major outbreaks of CVP such as that described by Fraser (quoted in Edgar, 1952) when large quantities of turnips were fed to heavily pregnant ewes. Hardy (1959) reported a similar occurrence when ewes were given access to large amounts of kale, and Leslie (quoted in Bayly et al., 1936) reported a further outbreak following the use of wheat and pea straw. It is possible to conclude from these findings that dietary controls may have a direct influence on the prevention and control of CVP.

The work by McLean (1957) and McLean and Claxton (1959) may partially explain the reasons for the increase in incidence in ewes fed high bulk diets and thereby inducing an increased intra-abdominal pressure. Since the reverse might then be expected to reduce the incidence of CVP, it has been advocated that a high-concentrate ration should be fed to heavily pregnant ewes (Straiton, 1973; Mitchell, 1983). However, more severe problems have occurred from sudden changes in the feeding regimen in an attempt to control an outbreak of CVP (Bayly et al., 1936). The starving of ewes in the face of an outbreak was found by these authors to result in a severe outbreak of pregnancy toxaemia although the
incidence of CVP was noted to be reduced.

However, while most opinions have supported the importance of high-bulk diets in the aetiology of CVP, Dayus (quoted in Bayly et al. 1936) noted that even sheep on a rich and succulent pasture were affected. That ewes need not be overweight is noted by Barry (quoted in Bayly et al. 1936), and keeping ewes in good bodily condition may not therefore, on its own, necessarily prevent CVP.

The effect of grazing oestrogenic pastures was again raised by Straiton (1973) and Merck (1973), who advocated the removal of heavily pregnant ewes from such pastures as a means of prevention and control. This assumes that oestrogens are implicated as a cause of the condition, which is contradicted by the findings of Bennetts (1944) and Bennetts, Underwood and Shier (1946). They categorically stated that CVP, as defined in this thesis, is not found in any greater numbers under such conditions. McLean (1956) also emphasised that oestrogenic pastures were not responsible for higher than normal numbers of ewes affected by CVP, but, in spite of this, Roberts (1971) recorded that this circumstance was a significant aetiological factor.

It seems unlikely, therefore, that nutritional measures could on their own, be effective in preventing or controlling CVP.

Apart from the provision of exercise and the control of the diet there seems little that can help in the control of CVP. Palsson (cited by Edgar, 1952), however, found that it was beneficial to alter the ewes' posture. This could be achieved by allowing the standing to build up at the back so that the hindquarters of the ewes were raised while they were at the trough. McLean (1957) and McLean and Claxton (1960) found that where the ewes were lying, or even grazing, on hill pastures the incidence of CVP was higher than on flat ground and although the reverse effect can occur in a confined yard (Palsson, cited in Edgar, 1952) it is difficult to see how such a management procedure could be used as a means of control on a hill farm. Straiton (1973), Merck (1973) and Fielden (1980) have all advocated the use of level ground pastures for heavily pregnant ewes in an attempt to
limit the number of cases of CVP occurring in a flock. This may not always be a very practical suggestion!

It is already apparent that many authors consider that more than one, and possibly many, different factors of environment, management and individual characteristics are involved in the aetiology and, this being so, the prevention and control will be most difficult. Belchner (1971) stated broadly that "prevention is a question of management" and no further elaboration was given presumably because this was sufficiently vague as to include all the possibilities!
4.0: MATERIALS AND METHODS:
4.1. : CASE MATERIAL :

Cases were obtained from commercial farms served normally by my own practice based in Stratford-on-Avon which participated in the practice Flock Health Programme. The farms were situated mainly in Warwickshire but some were in the counties of Gloucestershire, Oxfordshire, West Midlands and Worcestershire. Farmers were chosen on the basis of their willingness to allow every case of CVP to be examined and treated, irrespective of severity. Details of the flock size, breed composition, management system and feeding regimen were sought. This provided the source of the series of cases over 3 lambing seasons ie. 1981, 1982 and 1983.

4.2. : FARM TYPE :

4.2.1 : Topographical type :

Farms were classified in terms of topography as described by Mclean (1957) into two basic categories. These were:

a) flat

b) hilly or upland

This rather arbitrary classification was thought reasonable and flat farms required to be totally flat or nearly so with the possible exception of relatively steep-sided water courses. All others were classified, as by Mclean (1957), as hilly without any attempt to classify them further (Figure 28).

Occasionally ewes had been moved from hill ground to flatter home paddocks in the autumn, or had been bought in from indeterminate sources in the pre-tupping season. It
Figure 28: Photograph showing a generally flat farm where the ewes were grazed on the more hilly areas during the last 6 weeks of pregnancy. Such a farm was classified as a hill or upland farm for the purposes of this study.

Figure 29: Photograph showing the perineum of a ewe with a very short docked tail, where the length of the remaining portion of tail would not cover the vulva.
was not possible to obtain individual histories of the animals regarding the topography of their original environment but the farms were classified according to the topography or management system prevailing over the last 6 weeks of gestation, i.e. from the time supplementary feeding was started. It is accepted that this is an arbitrary distinction but it provided a significant changing point in the management of the ewes and, in all cases, the environment remained constant from this point on.

4.2.2: Housing systems:

Housed sheep were divided into two groups:

a) Covered and enclosed yards in which exercise was restricted,

b) Covered open yards in which housing resulted in the ewes being required to take exercise in the normal course of their behaviour. In this group ewes had free access between covered yards and flat pastures.

Sheep were considered to be 'housed without exercise' if their available floor space was less than about 3 square metres per ewe. Trough space was considered adequate in all cases as it was observed that all the ewes could feed simultaneously without undue hindrance from the others in the group.

Ewes kept closely confined for the greater part of the day and given access to larger areas or forcibly exercised for even limited periods were considered to be housed with open yards.

The available air space was considered to be adequate on all farms where the ewes were housed.

4.2.3: Breed distribution within flocks:

Recording the breed distributions and flock sizes proved difficult.

Most farmers knew how many ewes they had in their flock but could not provide details of the exact breed distribution unless pure-bred ewes were involved. The predominant
breed(s) were noted with approximate percentages given by the farmers. Most flocks contained several distinct breeds and crosses.

4.3. : FEEDING REGIMENS, ENVIRONMENT AND POPULATION :

4.3.1 : Diet :

All farms began supplementary feeding of pregnant ewes between 5 and 7 weeks prepartum although the amounts and quality varied somewhat. Hill farms fed hay on the ground and concentrates in open troughs, or directly on the ground, once daily. Flat ground farms tended to provide hay in covered racks and concentrates in open troughs, while housed sheep received all foodstuffs from racks or troughs in covered, dry conditions. Hay was of variable quality, and concentrates were either commercially available pencils or nuts (16% - 18% protein) or home-formulated feeds consisting largely of home-grown cereals (wheat, oats, barley) and protein supplements milled with variable amounts of chopped straw, hay or other bulk forage with molasses.

4.3.2 : Mineral supplementation :

Mineral supplementation was provided on all farms, either by mixture with the concentrate in commercial or home-formulated concentrates or by free access to specific mineral supplements in the form of blocks or powders. A high magnesium and high calcium content, or both, were invariably the aim of the farmer. However, mineral supplementation was always empirical. No farm used the "self service cafeteria" type of mineral supplementation which relies upon a degree of self-interest from the ewes.

4.3.3 : The environment :

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Details of the prevailing climatic conditions over the three years were obtained from Her Majesty's Meteorological Office, London Road, Bracknell, Berkshire, and related to the weather station at Wellesbourne, Warwickshire. This is situated some 8km east of Stratford-on-Avon and is slightly to the north of most of the farms involved, but it was taken as a guide for the overall state of the weather which varied slightly over the three years.

4.3.4: Overall population of sheep in United Kingdom:

Details of the population of breeding ewes in the United Kingdom over the three years were obtained from the Agricultural Census Branch of the Ministry of Agriculture Fisheries and Food, Epsom Road, Guildford, Surrey.
4.4 : PARTURITION MANAGEMENT :

4.4.1 : Preparturient prophylactic care of the ewes :

All ewes in the study had been vaccinated for the clostridial infections with commercially available vaccines. Some had received vaccination against Orf (contagious pustular dermatitis), Pasteurellosis and/or Foot Rot. No further attention was paid to this aspect of the management.

All ewes had received commercial anthelminitics at least once during the 4 weeks prior to lambing, and several flocks had been treated twice. No flock had received treatment for liver fluke as it was not considered to be a problem in the area.

Flocks where there was a history of intercurrent disease were noted accordingly. These conditions included parasitism (ectoparasites or endoparasites), Foot Rot, Twin Lamb Disease (pregnancy toxaemia/ketosis syndrome), hypocalcaemia (milk fever), hypomagnesaemia (lactation tetany), Orf (contagious pustular dermatitis), malnutrition, copper deficiency (swayback), and selenium deficiency (enzootic muscular dystrophy).

Some farmers were reluctant to acknowledge the existence of these conditions but practice records and their own, eventual, co-operation enabled this to be recorded.

Individual cases of disease on the farms were not regarded as significant but where repeated cases occurred over 2 or more years, these were recorded.

Farmers were asked for their own subjective appraisal of the current health of the flock at each visit and this often produced important information such as "not as good as last week" or "they've got too heavy" or "they don't like the feed" or "they are eating too much mineral block". In this way it was hoped to relate the general health status of the flock to the occurrence of CVP. This also provided an index of the typicality of the case being examined by comparison with the other ewes in the flock.
4.4.2: Parturient ewes:

The normal management of the pregnant and parturient ewes as lambing approached was as follows:

Ewes were fed and observed once or twice (and occasionally several times) a day and confined individually just before, or just after parturition on all properties except the hill farms where the ewes were left largely to their own devices and were examined closely only if a problem was apparent. Housed ewes, generally, were better monitored. This meant that cases of CVP tended to be noticed sooner on flat farms and in housed flocks and that parturient ewes which had been treated for CVP were more carefully nursed and monitored in these flocks.

4.4.3: Lambing pattern: The lambing pattern was noted for each farm and depending on the pattern classified as follows:

a) Random lambing.

b) Synchronised lambing. No farmer had used any aid to increase the fecundity of the ewes but one had used intravaginal progesterone-impregnated sponges (VERAMIX, Upjohn Ltd. U.K.) to allow a synchronous early lambing.

The extent of the lambing season and the three-week period of maximum lambing activity for each farm was noted and from this information the extent and period of maximum activity for each farm type was obtained.

4.5: EXAMINATION OF CVP CASES:

4.5.1: Notification of cases:

Farmers were asked to report cases of CVP as soon as possible, irrespective of the severity or the time of day. The cases were attended to as soon as practicable. Most ewes were examined within 1 hour and all were attended to within 6 hours.
Shepherds were asked not to move the ewe unless the disorder was severe; in which case she was to be confined in a pen and observed until she could be examined. This proved satisfactory and permitted a record of the progress of some cases of CVP to be made. Farmers, shepherds or assistants were asked to note tenesmus, vaginal rupture, impending parturition or any other significant clinical features.

4.5.2: **Clinical examination of cases:**

4.5.2.1: A brief clinical examination was carried out, with particular note being made of age, breed, body condition (according to Hughes, 1981) and the presence of any obvious intercurrent disease.

4.5.2.2: The age was determined according to Miller and Robertson (1947). All ewes over the age of 5 years were grouped together.

4.5.2.3: Note was made of the length of the tail and classified as:

i) **short**, when the tail could not reach the dorsal commissure of the vulva (Figure 29);

ii) **moderate**, when sufficient to cover the vulva (approximately 1/2 of the tail removed);

iii) **long**, where the tail was long enough to extend beyond the vulva (approximately 1/4 removed) and

iv) **undocked**.

This was generally noted after completion of the treatment.

4.5.2.4: Parity information was obtained from the shepherd which was usually assessed after the age of the ewe had been determined. Few shepherds were inclined to retain barren ewes for the next season, or to retain ewes which had some complication of pregnancy such as dystocia, dead lambs or CVP. The estimated parity was probably accurate in most cases although all those assumed to be of the third or higher parities were classified together as multiparous. Accurate information was generally available for the
Figure 30 (a): Photograph showing the insertion of the special applicator pin used to insert the plastic identity tag (Ritchie Tags Ltd., U.K.).

Figure 30 (b): Photograph showing the inserted tags in left and right ears following the completion of treatment.
first and second pregnancies. However, pedigree and small flocks provided more accurate information on the breeding histories.

4.5.2.5: Any significant development of mammary glands and vulva was noted but no record of this was made unless parturition appeared to be imminent; in which case a different approach may have been required eg. assisted delivery or caesarian section.

4.5.3: Identification of ewes:

Ewes were identified using a plastic tag, numbered with indelible ink, inserted in the right ear using the special applicator provided (Ritchie Tags Ltd., England) (Figure 30a and b). An identicaly marked tag was then held against the prolapse and a photograph of the case was taken. This tag was then inserted into the left ear in the same way. This reduced the danger of misidentification of ewes due to loss of one tag or fading of the ink. The photographic record was therefore permanently held against the identity number. Most ewes remained identifiable throughout the study period although several individual tags were lost, mostly through tearing or infections at the site of insertion.

Photographs were taken using an Olympus OM2n camera with a 50mm lens and an Olympus T20 dedicated flash. A variety of commercial film (ASA 64, 100 or 400) was used.

4.5.4: Blood samples:

Blood samples were obtained by jugular puncture, with the ewe standing, with aseptic precautions, using Vacutainers (Becton and Dickinson Ltd., England) and 21 gauge 1.5 ins needles. Blood was collected into lithium heparin (10 ml), EDTA (7ml), fluoride oxalate (7ml) and plain tubes (10ml). The samples were immediately identified with the same number as the ear tag.

4.5.5: Degree of prolapse:
Figure 31 (a and b): Photographs illustrating the intermittent (Simple) First Degree prolapse of the dorsal vaginal wall. Photograph (b) was taken within minutes of (a).
Figure 32 (a and b): Photographs illustrating the intermittent prolapse of the lateral vaginal walls typical of First Degree CVP. Photograph (b) was taken first some moments before (a).
Figure 33 (a and b): Photographs showing the intermittent prolapse of all the walls of the vagina giving a rounded appearance to the protruding tissue.
Figure 34: Persistent prolapse of the dorsal vaginal wall (First Degree CVP). Some oedema and inflammation are apparent.

Figure 35: Mild (Second Degree) prolapse of the vaginal wall showing superficial contamination, oedema and inflammation. The round appearance of the prolapse may be created by the incarcerated bladder or other abdominal structures such as omentum or gut.
Figure 36: Photograph illustrating the appearance of the Second Degree (Moderate) prolapse of the vaginal wall. There is a slight rectal prolapse and the vestibular wall is also slightly prolapsed (*). Contamination is minimal but congestion is apparent. The perineal area is swollen due to the entrapped bladder.

Figure 37: A congested, oedematous, inflamed and contaminated Second Degree prolapse of the vaginal wall. A prolapse of the vestibular wall is also apparent (*).
Figure 38 a: Third Degree (Severe) prolapse showing complete prolapse of the vagina with the cervix visible. There is slight contamination, marked mucosal sloughing, oedema and inflammation. The prolapse is rounded due to the inclusion of the bladder.

Figure 38 b: Third Degree (severe) CVP showing some contamination, congestion, oedema and inflammation. The external os of the cervix is visible and the rounded shape indicates the inclusion of the bladder.
Figure 39 a : Photograph showing extensive contamination, necrosis, infection, oedema and laceration of the Third Degree (severe) CVP.

Figure 39 b : Third degree CVP showing marked drying of the exposed mucous membrane. Contamination and laceration are not obvious.
Figure 40: Photograph of Third Degree CVP characterised by deep passive congestion and some contamination.

Figure 41: Photograph of a Third Degree (severe) CVP in which severe cyanosis and marked sub-mucosal haemorrhage can be seen.
The degree of prolapse was assessed according to the following classification which closely follows that used by Cox (1981) and Hudson (1980) for the condition in cattle.

i) FIRST DEGREE (or SIMPLE PROLAPSE): A portion of the vaginal wall was protruding intermittently or persistently from the vulval lips. It may only have been noticeable when the animal was recumbent. The exposed mucosa may have been mildly irritated by exposure and by contamination. Examples of this type of prolapse are shown in Figures 31 (a and b), 32 (a and b) 33 (a and b) and 34.

ii) SECOND DEGREE (or MODERATE PROLAPSE): The vaginal wall was persistently prolapsed and may have contained the bladder or other internal organs. There may have been some evidence of interference in urination, and tenesmus may have been present. The exposed mucous membrane may have been inflamed and possibly congested, lacerated or infected. Figures 35, 36 and 37 show the appearance of this type.

iii) THIRD DEGREE (or SEVERE PROLAPSE): The vaginal wall was completely everted and the cervix was visible beyond the vulval lips (Figures 38 and 39). The continued exposure resulted in the mucosa becoming lacerated, contaminated infected and dried. Continuous straining was present, resulting in the appearance of deep, passive congestion (Figure 40), sub-mucosal haemorrhages (Figure 41) and eventually ischaemia over more or less of the prolapsed tissue (Figure 42). It was frequently possible to palpate either the grossly distended bladder (Figure 38), foetal extremities (Figure 43, 44 and 45) or other abdominal contents within the prolapse. No apparent organ involvement (Figure 46) may have been present.

iv) FOURTH DEGREE (or COMPLICATED PROLAPSE): Prolonged exposure and severe tenesmus resulted in severe degeneration and devitalisation of the tissue of the prolapse (Figure 47). There may have been almost continuous straining. The ewe may have been systemically affected and separated herself from the flock.
Figure 42: Photograph showing a Third Degree CVP in which extensive ischaemia, cyanosis and tissue degeneration are apparent.

Figure 43: Photograph showing a Third Degree prolapse with congestion, inflammation, oedema, ischaemia and cyanosis, and focal necrosis (†). The prolapse contains the head of foetus in the gravid horn.
Figure 44: Third degree (severe) CVP without any apparent inclusion. Inflammation and oedema of the external os is apparent but contamination and laceration are absent.

Figure 45: Photograph showing the appearance of a Third Degree CVP when soft tissues such as omentum is included in the prolapse.
Figure 46: Third degree CVP with the cervix protruding from the vulva without any apparent vaginal wall. It is easy to understand the reference in the colloquial names for CVP to roses!

Figure 47: Fourth Degree CVP showing severe drying, gangrene, and severe contamination of the prolapsed vagina. The prolapse contained only soft tissues (either intestine or omentum).
Figure 48: Photograph showing the typical appearance of a ewe afflicted by the ruptured vagina syndrome.

Figure 49: Photographs showing the ruptured vagina complication of CVP (Fourth Degree) with herniation of abdominal contents.
Figure 50: Fourth degree CVP complicated by a bilateral perineal hernia in an old ewe.

Figure 51: Photograph of a ewe affected by CVP showing a mild rectal prolapse.
Often she did not eat and was in obvious pain. The dorsal wall of the vagina may have split allowing the herniation of abdominal contents (Figure 48 and 49).

There may have been a rectal prolapse (Figure 51) or other complications such as a perineal hernia (Figure 50).

Invariably abdominal organs were enclosed in the prolapse and these may have been pathologically affected by the restrictions placed upon them by the prolapse.

The classification into these categories was sometimes difficult because of the gradual transition from one category to another. Furthermore, it was possible that a prolapse from one category may have shown some of the features of another, but it was considered important to attempt to classify them. The photographic record of almost all the cases was kept to prevent any major errors of classification and to ensure a degree of consistency.

4.5.6: Tenesmus:
The extent to which tenesmus was apparent was noted in all cases. It was classified as:

i) Absent; where no spontaneous tenesmus had been observed either before or during the clinical examination. Ewes which strained during manipulation, reduction or retention of the CYP were classified in this category provided that they did not strain spontaneously at any other time.

ii) Mild; where straining was either intermittent or did not appear to distress the ewe.

iii) Severe; where tenesmus was persistent and accompanied by apparent distress. The ewes were often heard to bleat at each strain. Urine was sometimes passed in small squirts.

4.5.7: Rectal prolapse:
The presence of a rectal prolapse was recorded and classified as:
Figure 52 a and b: Photographs of cases of CVP in which severe rectal prolapse is present. Both the rectal wall and the vaginal tissues are severely congested and contaminated.
Figure 53 a and b: Photographs showing no (a) and mild (b) contamination of the exposed tissues in cases of CVP. A case showing severe contamination is shown in Figure 47.
i) Mild; when the prolapse was either intermittent or of such an extent that defaecation was not affected (Figure 51).

ii) Severe; when the prolapsed rectal wall interfered with defaecation and was permanently prolapsed or was extensive (Figures 52 a and b).

4.5.8: Contamination: The extent of contamination of the exposed tissues was classified as:

   i) Absent (Figure 53a).
   ii) Mild (Figure 53b).
   iii) Severe (Figure 47).

4.5.9: Cervical biopsy:

A small wedge of tissue was taken from one of the folds of the cervix and placed immediately in 10% formal saline for subsequent histological examination as described by Hindson and Turner (1962). Haemorrhage from the biopsy site was not found to be significant, and the tissue could be safely and quickly obtained in all but the mildest cases from which no samples were taken. Biopsy samples were not obtained from every case of CVP, and it was not felt prudent to employ the forceable extraction of the cervix as is described by Hindson and Turner (1962) and by Malone (1957). The samples were delivered to the Department of Surgery and Obstetrics, of the Royal Veterinary College, London for processing. Sections were stained with haematoxylin and eosin.

Initially, an attempt was made to obtain a biopsy of the vaginal wall but the tissue proved extremely friable and the technique hazardous; the vaginal wall readily split, leaving a major surgical problem.

Sections were classified according to Hindson and Turner (1962) as follows:

   i) Epithelium: the extent of visible erosion of the epithelium which was graded as outlined below:
Figure 54 a, b, c and d: Photomicrographs showing the normal (a), the partially eroded (b), severely eroded (c) and the necrotic (d) changes recognised in the epithelium of the cervical biopsy specimen (x 450) (stained H & E).
Figure 55 a, b, and c: Photomicrographs showing slight (a), moderate (b) and severe (c) inflammatory responses found in the stroma of the biopsy specimens from the external os of the cervix of ewes affected with CVP (x 450) (Stained H & E).
Figure 56 a, b and c: Photomicrographs showing slight (a), moderate (b) and severe (c) oedema in the stroma of the cervical tissues found in cases of CVP. (x 450) (Stained H & E).
Figure 57: Photomicrograph showing haemorrhage into the stroma of the cervical biopsy in a case of CVP (x 450) (Stained H & E).
0 = Normal tissue
1 = Partially eroded
2 = Severely eroded
3 = Necrotic
4 = Absent from biopsy specimen

An example of each type of response is shown in Figure 54 a, b, c and d respectively.

ii) Stroma: the extent of tissue oedema and inflammation was also noted from the histological sections and graded as follows:

<table>
<thead>
<tr>
<th>OEDEMA</th>
<th>INFLAMMATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1</td>
<td>1</td>
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<tr>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>3</td>
<td>3</td>
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</tbody>
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The appearance of the various pathological changes is shown in Figures 55 (a, b and c) and 56 (a, b and c).

Haemorrhage within the tissues of the biopsy specimen such as is shown in Figure 57 was recorded.

4.6 : TREATMENT :

4.6.1 : Initial approach :

After clinical examination of each ewe and the collection of the appropriate specimens, a similar initial approach was used in all ewes except where this was considered inappropriate; for example when the CVP was not visible, where interference might have aggravated a mild, intermittent CVP, or where the ewe was dead.
Figure 58: Photograph of a ewe showing the typical posture adopted during periods of straining.
4.6.1.1: The control of tenesmus:

Where tenesmus was considered to be of significance (Figure 58), either because the ewe was distressed or because handling of the CVP was difficult as a consequence of the straining, local anaesthesia was used to control it. One of the methods listed below was then applied. Ewes which had not strained prior to treatment but which did so during or after treatment to such an extent that local analgesia was thought to be justified, were grouped together with those ewes in which anaesthesia had been used in the first instance.

i) **Topical application** using a specific surface acting ointment (Xylotox Plus Gel, Astra, U.K.) which contains lignocaine hydrochloride, or by surface application of up to 15ml of 2% lignocaine hydrochloride (Xylotox, Astra Ltd., U.K.).

ii) **Sacral plexus regional block** as described by Hertrampf et al. (1978) but using 2% lignocaine hydrochloride with adrenaline (1:100,000) (Xylotox Plus, Astra Ltd., U.K.) rather than 20% ethyl alcohol used by these authors. Three millilitres were deposited over each of the last 3 sacral nerves on each side after thorough surgical preparation of the site.

iii) **Epidural anaesthesia** using 3 to 4 ml of 2% lignocaine hydrochloride (Xylotox, Astra Ltd., U.K.) administered either at the junction of the first two coccygeal vertebrae or at the lumbo-sacral junction through a 21 gauge 1.5in hypodermic needle as described by Whitelaw (1960) and Hall and Clarke (1983). The site selected was closely clipped, and then disinfected with 1% cetrimide followed by surgical spirits prior to injection. The lumbo-sacral junction site proved awkward to use when handling facilities were not ideal, and restraint of the ewe was difficult. Hall and Clarke's (1983) method requires that the ewe be placed in lateral recumbency with the spine flexed to dilate the lumbo-sacral space. However, the intercoccygeal site which, at first sight, may seem easier, was not always reliable and one or other method was used depending on the ewe and
the facilities available.

4.6.1.2: Positioning of the ewe:

The ewe was turned onto her side and then onto her back so that an assistant could lift the hindquarters off the ground in the manner described by Straiton (1973), Hughes Ellis (1958) and Cox (1981); a straw bale was sometimes a useful aid (Straiton, 1973). The lifting of the ewe in this fashion was occasionally aided by soft cotton or nylon rope passed around the neck of the operator or assistant and fastened to the hocks of the ewe (Figures 59a and b). (Struggling was sometimes effectively reduced by tying the front legs together and/or applying a blindfold.) In this way the prolapse was lifted from the ground and was more easily handled.

One farmer would not allow restraint in this manner and in these cases the ewe was restrained over a rail as shown in Figure 59c.

4.6.1.3: Initial cleansing and disinfection:

Where appropriate, most ewes received a common basic initial approach which involved the thorough and very gentle cleansing of the tissues with a solution of 1% cetrimide (Cetavlon, ICI, UK). Gauze swabs or cotton wool were used to dislodge any foreign material and a soft towel was used to dry the exposed tissues. The only ewes which did not receive this were those in which no prolapsed tissue was visible at the time of the examination. These ewes, however, were examined using an illuminated vaginascope, to check for the presence of contamination and to ensure that the vaginal wall was healthy. The presence of contamination was taken as confirmation that the ewe was suffering from a mild degree of CVP. In all cases where the tissues were infected, inflamed, lacerated or necrotic the cleansing procedure was completed by copious irrigation with warm saline. Obstetrical lubricant (Veterinary Drug Co.Ltd., U.K.) or K-Y Jelly (Johnson and Johnson Ltd., England) was then gently applied to the surface.

The prolapse was palpated carefully to identify any enclosed organs and, if the bladder was identified and was considered to be under severe tension or so large as to make it
Figure 59 a: Diagrammatic representation of the use of cotton rope to aid restraint and reduction of the CVP as described by Cox (1981).
Figure 59b: Drawing of the method of restraint where an assistant is available to lift the hind quarters of the ewe and leave a clear unobstructed field of view for the operator.
Figure 59 c: Restraint of the ewe over a rail which was used occasionally in response to concern by one shepherd over restraint in dorsal recumbency.

Figure 60: Photograph showing a case of CVP in which the udder is enlarged and the foetal membranes are presented indicating that parturition is imminent.
difficult to return to the abdomen (Figure 50), cystocentesis was carried out using a sterile 18 gauge, 1.5in hypodermic needle attached to a length of clean but not necessarily sterile tubing. The needle was directed towards the body of the bladder and only sufficient urine was released to reduce severe distention and enable reduction to be undertaken. Repeated puncturing of the bladder was avoided. The appearance of the urine was noted, but samples were not collected.

Other organs identified within the prolapse included gravid uterine horn(s) where the cotyledons were palpable, lamb extremities (Figure 43), and soft tissues which may have been bowel, fat or omentum.

The prolapse was reduced using the flat of the hands and gentle pressure, applied with a slight twisting motion, as described by Fethers (1939). The reduction of a full bladder was undertaken with particular care, and the prolapse was initially gently lifted to allow urination.

Most of the cases were easily reduced by this method and once reduction was completely accomplished the hand was left in the vagina for a few minutes to allow for the return of normal blood circulation, as suggested by Laing (1939, 1945, 1949). This was not easy to assess. The hand was removed once the temperature of the vaginal wall had returned to normal, as was advocated by Fethers (1939), Laing (1945, 1949) and Bruere (1956). Many of the ewes started to strain if they had not received any form of anaesthesia, often with considerable force and the issue of variable volumes of urine. Where this was the case the ewe was lowered to the ground to reduce the contamination of the vagina.

Where the cervix was found to be open, either by observation and palpation, or by noting the presence of foetal membranes (Figure 60), and particularly if there was colostrum in the teats, the ewe was treated differently. An attempt was made to deliver the lambs per vaginum. This was not always possible and then a caesarian operation was performed.
4.6.2: Retention of reduced prolapses:

Following reduction of the prolapses, as described above, a number of different methods were used to retain the prolapse in situ. Initially it was intended to apply these randomly but, unfortunately, this was not always possible. A shortage of time, and the fact that some shepherds were not keen on certain methods, meant that the selection of the treatments described below could not be applied randomly or be numerically equalised. Additional problems arose concerning the severity of the prolapse and the method of retention employed. It was not possible under practice circumstances to justify a mild form of retention in the treatment of severe CVP or a severe form of retention in mild prolapses. The methods applied were as follows:

4.6.2.1: Postural restraint. The ewe was restrained in a "lamb adopter crush" by neck halter or gate such that her hindquarters were raised approximately 25 to 35 cm above the level of the head. This was achieved by the use of a tilted board or by laying a straw bed to produce such a slope as is described by Bruere (1956).

These methods were generally reserved for the mildest forms of prolapse as it was not considered humane to use this approach with the more severe cases. Sometimes it was used as an adjunct following other methods of treatment. In some very mild cases no treatment was used at all.

4.6.2.2: Trusses or retainers without closure of the vulva.

4.6.2.2.1: Canvas, string or rubber type trusses such as described by Laing (1945), Marsh (1958), Fowler (1962) and Cox (1981). In addition, this group included the tying of wool strands across the vulva from one side to the other (Laing, 1939, 1945,). These methods were not used by the author but several farmers used them. An example of a home-made truss is shown in Figure 61 a and b.

4.6.2.2.2: The Intravaginal Prosthesis. The wire or plastic truss variously known as the 'Saveve' (Arnolds Ltd, U.K.) or 'Dalton's Spoon' as described by Laing (1949), Fowler and Evans (1957), Jones (1958), Rusch and Berchtold (1978), Pastoor and Weitenberg.
Figure 61 a and b: Lateral (a) and posterior (b) views of a home-made string and sacking truss used for the retention of mild forms of CVP by one farmer.
and Cox (1981), were either made commercially or were made by the author from the pattern given by Laing (1949) and Fowler and Evans (1957).

The retainers were inserted into the vagina after reduction of the CVP, and then fastened to the wool over the gluteal region as far forward as would retain it.

4.6.2.2.3: The Eagelman Technique. This method employs trans-pelvic fixation of the anterior vagina, as described by Eagelman et al. (1967) which is a modification of the 'Minchev' technique used in the cow (Hentshal, 1961).

The perineal and gluteal regions were prepared for surgery with low epidural anaesthesia to control straining and pain during the operation. After reduction of the CVP, a clean hand was inserted into the vagina carrying a 3in quarter curved cutting suture needle to which a length of Number 2 gauge nylon suture material and a 4 cm diameter plastic disc (coat buttons were found to be satisfactory) had been attached. The sharp point of the needle was shielded by the index finger so that no accidental injury to the vaginal wall could occur. The angle between the sacrum and the caudal border of the shaft of the ilium was located, and the needle thrust through this space in an upwards and outwards direction to emerge through the skin some 5 to 6 cm lateral to the midline. A second plastic disc was attached to the two ends of the nylon after the needle had been removed. A pad of surgical gauze was placed under the gluteal disc and the suture, so created, gently tightened until the first resistance was felt. A lubricated hand was reinserted into the vagina to ensure that it was fully reduced before tying the suture. The technique was repeated on the other side. Prophylactic procaine penicillin (Propen, Glaxo Ltd, England) at 20 mg/kg bodyweight was administered intramuscularly and the ewe was released.

Eagelman et al. (1967) advocated the use of surgical steel wire suture (26 or 016 gauge) but this was found to be difficult to manipulate and no specific problems were encountered with the nylon material.

4.6.2.3: Methods which close the vulva.
Figure 62 a: Retention of CVP with the use of 3 locking nappy-type safety pins placed horizontally through the lips of the vulva. Note the oedema between the pins which developed rapidly.

Figure 62 b: Retention of CVP using 2 kilt or blanket pins placed horizontally across the vulva. The points of insertion and emergence of the pins is near to the wool line.
Figure 62 c: Trans-vestibular simple interrupted sutures placed across the vulva to retain CVP. The knots have not yet been drawn across laterally.
These include all those methods which either close the vulva surgically or which rely upon pressure on the lateral aspects of the vulva to produce such closure.

4.6.2.3.1: Safety pins. Safety pins of the self locking or "Nappy" type, such as was described by Laing (1945), and which have been widely used by shepherds over many years (Slatter, 1980, personal communication), were applied across the vulval lips horizontally as is shown in Figure 62a. Care was taken to ensure that the pins were placed as widely as possible in an attempt to avoid the vulval lips. It was not generally possible, however, to avoid the vulval lips because of the limited size of the pins. No smaller size of safety pins was used in this study.

4.6.2.3.2: Kilt pins. These were a modification of the safety pin method (above) but are considerably larger and straight. They do not generally have a locking device. The pins were inserted horizontally across the vulva in such a way that the insertion and emergent points were as wide apart as possible. Figure 62b shows how the pins were placed to maintain the reduction of CVP. There are no previous reports of their use.

4.6.2.3.3: Trans-vulval interrupted sutures. These employed heavy gauge suture material (No 4 metric nylon or catgut). Sutures were inserted using a 3in quarter-curved cutting needle from the wool line and were inserted as deeply as possible across the vulva and vestibule. The suture was tied using a surgeon's knot ensuring that tension on the tissues did not restrict the local circulation. The ideal site for the suture was taken to be the location of the constrictor vestibulae muscle. The knot was drawn to one side after being laid. Normally between 2 and 4 such stitches were laid, depending on the length of the stretched vulva. Particular care was taken to avoid interference with urination by careful placement of the most ventral suture. The appearance of the vulva after insertion of the sutures is shown in Figure 62c.

4.6.2.3.4: Buhner's technique. This involved the laying of a subcutaneous perivulval purse-string suture. It was first advocated by Buhner (1958) for cattle and subsequently for the ewe by Kubicek (1977), Rusch and Berchtold (1978), Fielden (1980), Cox (1981)
and Arthur et al. (1982). The method used in this work was identical to that described by Arthur et al. (1982). After local anaesthesia, using either local infiltration or epidural, two 1 cm long incisions were made horizontally: the first between the anus and vulva, and the second some 3 to 4 cm below the ventral commissure of the vulva. A special Buhner needle was inserted, using aseptic precautions, from the ventral incision to the dorsal incision on each side of the vulva and a length of tape which had previously been soaked in an aqueous solution of procaine penicillin or sterile heavy gauge (No 6 or 8) catgut or nylon suture material was drawn subcutaneously around the vulva. The two ends were tied at the ventral incision, with sufficient tension to allow the introduction of only two or three fingers. The effect was to mimic the tension of the constrictor vestibulae muscle. Care was taken to ensure that the tension was not excessive.

The operation necessitates a surgical approach and the suture must be cut when lambing commences but it is possible to examine the birth canal before releasing the stitch to check that parturition is indeed imminent. Routine prophylactic antibiotic in the form of long-acting procaine penicillin (Propen, Glaxo Ltd., England) at 20 mg/kg. was administered in all cases.

4.6.2.3.5 : Trans-vaginal quill aided sutures. A loop of heavy gauge suture material (such as nylon, no. 6) was inserted from the wool line across the dorsal part of the vulva and vestibule to emerge on the other side at a similar point. The needle was then cut out and another such suture laid at the ventral end of the vulva. A quill of rubber tubing or 9 to 10 cm long piece of fan belt from a motor-car was inserted in the loops on the one side and the tension taken up. A further piece of belt was then tied into the sutures. This basic method was advocated by Moreno (1971) and Rusch and Berchtold (1978), but no reports are evident on the application of fan belt. The use of the fan belt was considered advantageous because it has an "inbuilt" curvature, which meant that the tension across the ventral commissure could be reduced to a minimum and the sutures could be "locked" into the serrations on the inside of the curvature on the lateral aspects of the vulva. Only
Figure 63 a: Diagrammatic scheme for the insertion of quill-aided sutures for the retention of CVP.
Figure 63 b and c: The appearance of the vulva after the application of quill-aided sutures; in this case lengths of motor car fan belt.
Figure 64 a: Photograph showing a case of CVP which had been treated and retained using trans-vestibular simple interrupted sutures which was subsequently found to have the placental membranes presented and which proved refractory to manual cervical dilatation necessitating caesarian section.

Figure 64 b: Photograph of a case of CVP in which the foetal membranes were presented, and which proved refractory to manual cervical dilatation. The cervix and vagina are markedly inflamed and congested.
Figure 64 c: A case of CVP in which the foetal membranes were presented through the cervix. The membranes were obviously not fresh and the cervix could not be dilated manually and the case was considered to be complicated by ringwomb.
sufficient tension was applied to rest the "quill" against the vulva and so mimic the effects of the constrictor muscle of the vestibule. Correct tension was taken to mean that a vertically held flat, lubricated hand could just be introduced into the vulva past the sutures.

The technique of placement of the sutures and quills is illustrated diagrammatically in Figure 63a and the appearance after completion is shown in Figure 63 b and c.

4.6.2.4 : Immediate delivery.

4.6.2.4.1 : Delivery per vaginum. Ewes in which the placental membranes were presented through the cervix (Figure 60 and 64) were examined closely, and if possible, with suitable gentle handling the lambs were delivered. Those which strained strongly after delivery were given local anaesthesia by epidural injection, or by regional block of the sacral nerves and transvulval sutures were inserted as above. No record of this procedure was kept.

4.6.2.4.2 : Caesarian section. Ewes selected for caesarian section were those in which the operation was considered to be the only justifiable treatment. This was because of a significant obstruction to delivery, or where death was considered likely from complications of CVP, or where the placental membranes were presented and the ewe could not be delivered per vaginum.

Many of these ewes showed complete or partial failure of cervical dilatation (ringwomb). This was diagnosed by the imminence of parturition in the absence of cervical dilatation sufficient to allow vaginal delivery (Figure 64). In most cases the decision to operate was taken early and no attempt was made to postpone further interference for 24 to 36 hours as was first advocated by Straiton (1957). It is accepted that this might have increased the number of operations performed but this was not considered further.

A number of ewes were subjected to an elective operation. This was undertaken in view of the widely held opinions dating from Walsby (1952) to Schulz et al. (1985), that
Figure 65 (a): Close shaving/clipping of the wool from the left flank of a ewe in lateral recumbency in preparation for caesarian section.

Figure 65 (b): Surgical preparation of the site after local infiltration anaesthesia along the proposed incision line.
caesarian section is the only way to keep the ewes and lambs alive. The objective of this surgery was an attempt to save the lives of the ewes in the first instance and then, if possible, those of the lambs.

Surgical Method:

i) Restraint: The ewe was quickly transported to the surgery without prior starvation or withholding of water. On arrival, a "towel blindfold" was placed on the ewe which, it has been found, has a marked sedative effect. Ewes would lie in lateral recumbency for extended periods in this way although, as an added precaution, the fore and hind legs were tied together and then fastened to the operating table. No ewe was given any form of chemical restraint as it was thought to reduce the viability of the lambs and the natural mothering instincts of the ewe. The ewe was thus restrained in right lateral recumbency and the left flank and sub-lumbar fossa were prepared for surgery by close shaving (Figure 65a) and surgical disinfection with chlorhexidine (Hibiscrub, ICI. England), followed by irrigation with surgical spirits (Figure 65b).

The surgical method closely followed that described by Walsby (1952), Hughes Ellis (1958), de Bois (1958) and Straiton (1973) and Vivrette (1986).

ii) Anaesthesia: Anaesthesia was induced by local infiltration in all cases, using 10 - 15 ml of 2% lignocaine hydrochloride with 1:100000 adrenalin (Xylotox Plus, Astra Ltd., U.K.), along the proposed line of incision which was on an imaginary line joining the stifle in its normal standing position and the xiphoid, i.e. a low flank, paracostal incision. This site was chosen because it avoids the milk vein ventrally and little interference from the rumen and intestines is to be expected. Entry to the abdomen at this site involved very little muscle interference, bleeding is minimal and closure of the wound is simple.

iii) Surgical entry was gained by incising the skin and the combined aponeuroses of the oblique muscles and then entering the abdomen through section of the transverse abdominis muscle and peritoneum which were tented to avoid damaging the internal
Figure 65 (c): Uterine incision made longitudinally in the horn of the uterus over a foetal extremity.

Figure 65 (d): Continuous inverting Lembert suture used to close the uterine wound after delivery and the insertion of an antibiotic pessary.
organs. The nearest gravid horn was partially exteriorised through the abdominal wound and the uterus incised over a foetal extremity. The foetus was delivered avoiding, as far as possible, contamination of the peritoneal cavity with the foetal fluids. Other lambs, if present, were either delivered through the same wound or, more rarely, a second uterine incision was made in the same way. It was found that there was a greater lamb viability if the umbilical cord was not broken until the nose and mouth of the lamb had been cleared of mucus and fluid. This was done in every case and in general the umbilicus was left intact until several minutes after the lamb had been delivered and the uterus showed signs of involution. This required the presence of an assistant to attend to the lambs. A 400 mg pessary of amoxicillin (Clamoxyl, Beecham Ltd. England) was deposited in the uterine lumen. No attempt was made to remove the foetal membranes. The uterine incision was closed using a simple continuous suture pattern with No.1 chromic catgut and an atraumatic round-bodied needle (Ethicon, Edinburgh, UK). The uterine incision was then oversewn using the same pattern (Figure 65c and d). No antibiotic or other material was used on the wound.

The CVP was re-examined and the cervix was drawn anteriorly from within the abdomen to ensure that the prolapse was totally reduced. A superficial examination of the pelvic structures was made, noting any obvious abnormalities, such as haemorrhages and tearing of tissues in the abdomen. Particular attention was given to the size and disposition of the bladder.

The peritoneum and the transverse abdominal muscle were then closed together using a simple continuous pattern and No.4 chromic catgut (Ethicon Ltd. Edinburgh, U.K.). The aponeuroses of the oblique muscles were closed together using simple interrupted sutures of the same material. The skin wound was closed with No.1 sheathed multifilament nylon (Supramid, B.Braun, Melsingen, Germany) in a horizontal mattress suture pattern (Figure 65e).
Figure 65 (e): Final closure of the skin wound with horizontal mattress sutures using nylon material.

Figure 65 (f): Trans vulval sutures inserted in response to tenesmus following surgery.
iv) Post-operative care: The ewe was placed in a secluded pen with any live lambs until collected by the farmer. Tenesmus during the immediate post-operative period, with or without prolapse of the vagina or cervix, was an indication for the use of trans-vulval sutures which were inserted as described above using 2 to 3 ml of 2% lignocaine hydrochloride on each side of the vulva (Figure 65f). Procaine penicillin (Mylipen, Glaxo Ltd. England) was given at 20mg/kg and the farmer instructed to repeat this daily for five days. He was requested to observe the ewe closely in the meantime for the passage of the foetal membranes and any complications. Death of either the ewe or the lambs was to be reported. Some shepherds were either unwilling or unable, for other reasons, to carry out these checks, and many of the ewes were turned out at arbitrary times from the time of arrival at the farm to hours or days later.

No special instructions were given with regard to feeding of the ewe or the management of the lambs other than those following a caesarian section.

The weight of most of the lambs was obtained before feeding, but after they were dry. The sexes of the lambs were not recorded.

The surgical procedure was generally completed within an hour.

4.6.2.5: Post-treatment care and observation:

Having used the methods described above to retain the prolapses, the farmers were reminded of the signs which would indicate impending parturition and the appropriate procedure to be adopted for each method used. These included the onset of straining, the presence of foetal membranes or foetal extremities at the vulva and the presence of colostrum in an engorged udder. The shepherd was requested to notify the surgery when this occurred, particularly in the event of any abnormalities in lambing. If this occurred, the ewe was to be isolated for attention as soon as practicable. No obstetrical manipulation was to be undertaken by the shepherd. However, in spite of the offer of free attention,
this was not often adhered to and many ewes were treated adequately or, in some cases inadequately, by well-meaning shepherds. Those ewes which lambed normally without assistance were not examined again unless there were post-parturient problems. The shepherd was asked to weigh any lambs, either alive or dead, and report the numbers, the weights and the details of the survival of the ewe and the lambs. The results were tabulated as follows:

i) Number of lambs born

ii) Number of live lambs born

iii) Total weight of lambs (dry)

iv) Pre-partum complications

v) Problems with delivery

vi) Post-partum complications

The same details were recorded for any ewes which were attended to personally.

Post-parturient CVP and uterine prolapses were particularly noted and where possible attended personally.

4.6.2.6 : Retained ewes and progeny.

A number of ewe lambs born to dams with CVP were purchased and identified, using the same type of plastic tags as the dams. The farmers agreed to retain these in the flock and include them in the normal breeding programme in the following years. No special treatment was to be given with regard to the management of these animals which were monitored until the 1985/6 lambing season or until they died or were culled. Details of their lambing and the extent of any prolapses were recorded. No other information was obtained.

No attempt was made to encourage farmers to retain recovered cases of CVP in their flocks but several did, and most of these were followed annually until they were either culled or died. No blood samples were obtained in the subsequent years and only the details of the extent of any prolapses and the results of the lambings were recorded. In this
way several ewes were followed for up to four years. In the event that CVP occurred in these ewes the approach to the retention of the CVP was the same as is outlined above. At the end of each lambing season each ewe’s record card was updated with any relevant information such as death, slaughter, culling etc. While this generated useful information, it also encouraged the farmer to participate in the ensuing season. The following questions were asked:

a) Is the ewe still in the flock? YES/NO.
   If YES: is she destined to be bred this year? YES/NO. If NO: DIED/CULLED for reasons other than CVP, DIED/CULLED as a direct result of CVP

b) Is it your normal policy to CULL CVP ewes? YES/NO

4.6.2.7: Ewes found dead or ‘in extremis’:

Ewes affected with CVP or ruptured vagina which were found dead, were subjected to a brief necropsy with particular attention being paid to the uro-genital system. Any macroscopic pathological changes in other organs were noted. No blood samples were collected. Once suitable blood and tissue samples were obtained, ewes considered to be beyond treatment were humanely destroyed using either 0.75 grams of sodium pentobarbitone (Euthatal, May and Baker Ltd., Dagenham, England) followed by 100 ml 40% Magnesium Sulphate solution intravenously or with a 0.32 calibre pistol (Scott Webley Humane Killer). A laparotomy was performed immediately in an attempt to salvage the lamb(s). Routine necropsy was then performed as outlined above. In almost all cases of necropsy the number and weight of the lambs was recorded. This approach was also used when possible if animals which had been treated, died before, at or after parturition.

4.7: HAEMATOLOGY AND BIOCHEMISTRY:

Blood samples were processed by the author at the practice laboratory. All determinations undertaken at the practice were carried out within 12 hours of sampling and most within 6 hours. All parameters were expressed in SI units.
4.7.1 : Haemogram :

Using the EDTA sample of blood the red cell parameters were obtained as follows:

i) **Erythrocyte count (rbc):** A Coulter particle counter Model Fn (Coulter Electronics Inc., Hialeah, USA) and an automatic diluter were used to count the erythrocytes (x 10^12/l)

ii) **Haematocrit (Packed cell volume) (PCV):** Using a Hawksley micro-haematocrit tubes, Crystaseal (Hawksley, England), a Hawksley microhaematocrit centrifuge and the Hawksley Microhaematocrit Reader the values for the haematocrit were obtained. Results were expressed as litres/litre.

iii) **Haemoglobin (Hb):** Determined using Drabkins Reagent (Benjamin, 1961) and a spectrophotometer (Cecil 303, Cecil Instruments Ltd., Cambridge, UK.). The results were expressed in grams/litre.

iv) From the parameters above the **mean cell volume (MCV)** and the **mean corpuscular haemoglobin concentration (MCHC)** were obtained using the following formulae:

\[
\text{MCV} = \frac{\text{PCV} \times 100}{\mu\text{m}^3} \quad \text{rbc}
\]

\[
\text{MCHC} = \frac{\text{Hb} \times 100}{\%} \quad \text{PCV}
\]

4.7.2 : Leucogram :

The EDTA sample was used to obtain the parameters included in the leucogram which were the total leucocyte count and the differential leucocyte count.
i) **Total leucocyte count (wbc):** The leucocyte count was performed on the Coulter Counter as above using a saponin lysing solution (Zapoglobin, Coulter Electronics Inc., Hialeah, USA) \( \times 10^9/l \)

ii) **Differential leucocyte count:** A thin smear was stained using Modified Wright-Geimsa stain (DIF-QUIK; American Hospital Supply (UK) Ltd., Didcot, England) and, using the battlement method (Benjamin, 1961) a differential leucocyte count was obtained after counting 100 cells. Neutrophils, eosinophils, basophils, lymphoctes and monocytes were identified and expressed in absolute count \( \times 10^9/l \). Individual abnormal cells were excluded.

### 4.7.3: Metabolic parameters

This group included the plasma glucose which was obtained from the fluoride oxalate sample, and serum urea, \( \beta \) hydroxybutyrate, total protein and albumin levels which were derived from the clotted sample after centrifugation to separate the serum.

#### 4.7.3.1: Plasma glucose

The GOD - PERID method described by Werner, Rey and Weilinger (1970) was used after deproteinisation with uranyl acetate solution (Urac, Boehringer Corporation Ltd, Lewes, Sussex, England) (BCL). All the appropriate reagents and the method are marketed in kit form by BCL (Kit 124010). The tests were carried out at 37°C in a water-bath (Precitherm, BCL).

As uranyl acetate is mildly radioactive, only automatic pipettes with disposable tips were used. Quality control was maintained using Precinom S quality control solution (BCL) with each estimation.

#### 4.7.3.2: Urea:

Serum urea was determined using the kit (No.124770) available from BCL (UK) which employed a colorimetric determination using Berthelot's Reagent after the action of a
urease enzyme on the urea substrate. The method was described by Fawcett and Scott (1960) and again quality control was maintained by the simultaneous use of Precinorm S solution (BCL, UK) with every sample or group of samples. 4.7.3.3:

Beta-hydroxybutyric acid (BHBA):

The method described by Williamson and Mellanby (1974) was used to determine the serum levels of BHBA. The method is tedious and quality control was maintained through check determinations at the Veterinary Investigation Centre, Worcester.

4.7.3.4: Total Protein:

The Biuret method described by Weichselbaum (1946) was used in the form of a commercially available kit (BCL) (No. 124281). Quality control was maintained with the use of Precinorm S quality control Solution (BCL).

4.7.3.5: Albumin:

The method described by Doumas, Watson and Biggs (1971) using a citrate buffer with a pH of 3.8 (BDH Chemicals Ltd., Poole, Dorset, England) and bromocresol green (BDH Chemicals Ltd.) was used and controlled by repeated checks at the Veterinary Investigation Centre, Worcester. Latterly, commercially prepared bovine serum albumin, Fraction V, became available (BDH Chemicals, UK) and this was prepared as a standard solution and its albumin level was determined using the Biuret method outlined above. This standard solution was calibrated by determination of the albumin by the Veterinary Investigation Centre, Worcester, and was then used throughout the study.

4.7.4: Electrolyte and mineral parameters:

These included serum calcium, magnesium and copper which were determined from serum, selenium which was derived from the lithium heparin sample, and inorganic phosphate which was obtained from the fluoride oxalate plasma.

4.7.4.1: Calcium:
Using the colorimetric method described by Sarker and Chavhan (1967) without deproteinisation which forms the basis of a commercially available kit (BCL, No. 204382) the serum calcium levels were determined and controlled by Precinorm S (BCL).

4.7.4.2 : Inorganic Phosphate:

Plasma obtained from the fluoride oxalate sample of blood was deproteinised using 1.2N trichloracetic acid (BCL) and the supernatant was analysed using the BCL kit (No. 124974) which employs the method described by Zilversmit and Davis (1950). The method uses ammonium molybdate in sulphuric acid and ammonium vanadate in nitric acid and is a colorimetric estimation. Quality was checked with the use of commercial quality control solutions (Precinorm S, BCL, UK).

4.7.4.3 : Magnesium :

A commercial kit (Roche Diagnostics, Welwyn Garden City, England, Kit no. 07-1019-9) uses the colorimetric method described by Bohuon (1962) which is a xylidyl-blue reaction.

4.7.4.4 : Copper :

A kit for the determination of serum copper is marketed by BCL (No. 124834) which uses a bathocuproin method after deproteinisation of the serum with 1.2 N trichloracetic acid (Zak, 1958), and this was used as the basis for the serum copper determinations in this work. However, it was found over several years prior to the current investigation that the method described for use on human blood was not reliable, possibly due to carotenes in sheep blood and a modification employing a double deproteinisation using 1.2 N trichloracetic acid initially, and then 1N perchloric acid produced better results. Serum copper determinations were not always performed for reasons of time but sufficient samples were analysed to provide some statistical analysis.

4.7.4.5 : Selenium :

Three ml of whole blood, with lithium heparin anticoagulant, were sent to the Veterinary Investigation Centre, Worcester, for determination of the selenium which was
derived indirectly from determination of the glutathione peroxidase (GshPx). The method employed was that described by Board and Peter (1976).

4.7.5: Hormone parameters:
Using the lithium heparin blood sample, plasma was separated after centrifugation and 3 ml were stored at -20°C until the end of the season. The samples were then delivered to the Royal Veterinary College, Department of Surgery and Obstetrics, for determination of plasma progesterone and 17β-oestradiol. Both plasma progesterone and plasma oestrogen were estimated by radio-immuno assay using the method of Challis, Davies and Ryan (1973) and Challis, Heap and Illingworth (1971) respectively. The antiserum for progesterone was raised in goats (Specific Antisera Ltd, Wilmslow, England) and the intra- and inter-assay coefficients of variation were 17.4% and 18.8% at 1.4 ng/tube respectively. The oestrogen antiserum was also raised in goats (Specific Antisera Ltd.). The intra- and inter-assay coefficients of variation were 9.1% and 21.6% at 113.7 pg/tube. Progesterone was expressed as ng/ml and 17β-oestradiol as pg/ml.

4.8: LABORATORY EQUIPMENT:
In addition to the equipment mentioned so far, the laboratory was equipped with a variety of precision pipettes (Oxford Adjustable Sampler, Oxford Instruments, BCL, UK); these used plastic disposable tips which were not reused. Many of the determinations used small volumes of reagents and were carried out directly in 5ml disposable optically matched cuvettes (BCL, UK). This avoided the transfer of solutions from container to container and were found to be an advantage in terms of quality and economy. Where greater volumes of reagents were required, disposable plastic centrifuge tubes (BCL, UK) were used. They were also used where centrifugation was required for deproteinisation, such as in the methods for inorganic phosphate, glucose and copper. A Biolam binocular
microscope with oil immersion (Labpak Ltd., Coventry, England) was used.

4.9 : CONTROL EWES:

In addition to the cases of CVP seen over the three years a number of normal ewes were selected, sampled and followed in the same way as those which had been affected. Wherever possible (generally after every third case of CVP), a control ewe of similar age, bodily condition, of the same breed, and at the same stage of gestation was identified and sampled. The control ewes were not identified with particular CVP cases and were not followed in subsequent years, but they provided a group of apparently normal control ewes with which to compare the affected animals. No cervical biopsies were obtained from these animals.

4.10 : STATISTICAL METHODS:

The nature of the results obtained from this investigation necessitated the use of non-parametric statistical methods of analysis. In some areas such as the haematology and biochemistry the distribution of the continuously variable data was found to be normal while others were not. For this reason it was decided to use non-parametric analysis throughout the investigation. The effect of this decision was to bias the results in favour of non-significance as the tests are generally less sensitive than the more frequently used parametric analyses.

Where comparisons were made between 2 groups only, for example a comparison between diseased and control animals, the Wilcoxon 2 Sample Test was used and where more than
two classes were to be compared such as ewes in each of the three years, or between ewes which were suffering from different degrees of CVP or where 11 different methods of treatment were to be compared, the Meintel Haentzel or the Kruskal-Wallis test was applied (Daniel, 1983). The means and standard deviations (SD) of the continuously variable parameters such as the biochemistry and haematology results were obtained using standard formulae (Daniel, 1983). However, these indices had limited value where the data was not of normal distribution. Where these are given the distribution had been shown to be normal or near normal. In respect of the leucocyte count, the differential leucocyte count and the hormone parameters, the median and ranges are given as the standard deviation was extremely high due to the wide range of values for these parameters.

All analyses were carried out using the SAS statistical software program on an IBM mainframe computer at the Agricultural University of Wageningen, The Netherlands. Where \( p \geq 0.05 \) differences were considered to be insignificant (ns). Where \( p < 0.05 \) the results were described as significant (*), and where \( 0.01 > p > 0.001 \) the results were described as highly significant (**). If \( p < 0.001 \) then results were classified as extremely significant (***).
5.0 RESULTS
5.1 : FLOCK MANAGEMENT :

5.1.1 : Management within the Flocks :

Managemental variations from farm to farm were wide but all flocks participating had undertaken routine clostridial vaccination and anthelmintic programmes. Thus all ewes in the survey had been vaccinated within the last 4 weeks of pregnancy, and had received modern anthelmintic therapy at least once during pregnancy, the majority in the last 2 - 4 weeks of gestation. The flocks involved in this study were part of the practice Flock Health Programme in which advice and visits were made regularly at strategic times in the year. This was primarily designed to prevent the occurrence of epidemic disease and to improve productivity.

5.1.2 : Flock nutrition :

Feeding regimens varied widely according to the farm type and the availability of home-grown or commercial feeds. The majority of farms started supplementary feeding from about the 90th day of gestation. All farmers aimed to provide a maintenance ration only up to this time and generally aimed to feed a 16 - 18 % crude protein ration with a metabolisable energy level of approximately 12 MJ / kg dry matter at a steadily increasing rate to reach a maximum of 1.0 kg. per ewe per day in the last week of gestation. Several farmers formulated and prepared their own rations based on home-grown cereals and commercial protein concentrates. The greatest variations in feeding regimen was encountered in the hill farms (Farm Type 3) on which it was impossible to determine the quantity or quality of the food intake of a specific ewe or indeed of the ewes in general. Bulk forage feeding was in all cases carried out using variable qualities of hay and in the
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1 Tested mid gestation.
2 Tested spring-summer.
3 Tested in post-partum ewes.
4 Tested in pre-partum ewes.

**Figure 66:** Table showing the trace element and mineral monitoring (M) on farms studied. Deficiencies of individual elements on farms are shown as (D). Farms supplemented according to deficiencies shown.
case of the grazing ewes pasture contribution was taken as negligible. The housed or 
yarded sheep were all bedded on either barley or wheat straw. No farm used slatted floor 
houses. No other records of the feeding regimes were made.

5.1.3: Mineral supplementation:
Mineral supplementation of the ration or separate feeding of minerals was practised 
widely. The details of the mineral intake for each farm were difficult to quantify but 
generally the intention was to provide a high calcium ration up to lambing and a high 
magnesium intake thereafter. Figure 66 shows those farms where specific minerals were 
monitored annually in the normal course of events and corrected as necessary. These 
included copper and selenium, and several farms found it necessary to monitor these 
annually and decisions were made as to the advisability of supplementation by individual 
dosing or by dietary addition.

5.1.4: Intercurrent Disease:
Intercurrent disease on farms, either at the time of lambing, or historically, included 
copper deficiency (swayback) on 12 farms, selenium deficiency (muscular dystrophy) on 
5 farms, magnesium deficiency (post parturient hypomagnesaemia in ewes) on 3 farms and 
calcium deficiency (preparturient hypocalcaemia) on 2 farms. The farms which had, 
historically, suffered from the disorders tended to be those on which routine monitoring 
of flock levels was carried out as shown in Figure 66. Other disorders encountered 
sporadically in several of the flocks included ketosis (Farms 2, 7, 15, 26), contagious 
pustular dermatitis (Farm 16), foot rot (Farms 1, 4, 9, 10), and periorbital (staphylococcal) 
dermatitis (Farm 16). Where these were seen during the study period appropriate 
treatment was given.
5.2. : CASE MATERIAL :

5.2.1 : Participating Farms :

Twenty-six farms registered an interest in the investigation. Farm 17 withdrew before the start and several individual farms either were inconsistent in reporting cases or entered later in the investigation (Figure 67). Generally, those farms where the commitment was intermittent had problems associated with the availability of labour.

5.2.2 : Incidence :

Figure 67 shows the farm registration number, the number of ewes forming the breeding flock each year and the number of prolapses seen (and the percentage of the overall flock affected by CVP) in each of the three lambing seasons over the period of study (1981, 1982 and 1983). The number of cases of CVP examined and monitored in each year of the investigation is shown in Figure 68. Sixty-five cases were seen in the 1981 season, 55 in the 1982 season and 84 cases were seen in 1983.

These figures do not include one case (Ewe C108) which had a post-parturient CVP without a history of having been affected in the pre-parturient period. The number does, however, include cases which had a ruptured vagina and herniation of abdominal contents as described by Knottenbelt (1988).

Also excluded from this number are 53 ewes which, having been retained in the breeding flock the season after the initial episode, were followed through the ensuing lambing seasons and which may have prolapsed again in the future. This group of ewes is
### Lambing Season

<table>
<thead>
<tr>
<th>Farm number</th>
<th>Farm type#</th>
<th>No of ewes</th>
<th>Prolapse type 1°</th>
<th>2°</th>
<th>3°</th>
<th>4°</th>
<th>Total cases</th>
<th>No of ewes</th>
<th>Prolapse type 1°</th>
<th>2°</th>
<th>3°</th>
<th>4°</th>
<th>Total cases</th>
<th>No of ewes</th>
<th>Prolapse type 1°</th>
<th>2°</th>
<th>3°</th>
<th>4°</th>
<th>Total cases</th>
</tr>
</thead>
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<tr>
<td>1</td>
<td>3</td>
<td>33</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>--</td>
<td>3 (9%)</td>
<td>36</td>
<td>1</td>
<td>1</td>
<td>--</td>
<td>--</td>
<td>1 (3%)</td>
<td>Sold up</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>3</td>
<td>80</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>--</td>
<td>2 (2.5%)</td>
<td>66</td>
<td>1</td>
<td>1</td>
<td>--</td>
<td>--</td>
<td>1 (1.5%)</td>
<td>114</td>
<td>2</td>
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<td>2</td>
<td>--</td>
<td>5 (4.4%)</td>
</tr>
<tr>
<td>3</td>
<td>2</td>
<td>946</td>
<td>2</td>
<td>3</td>
<td>2</td>
<td>--</td>
<td>7 (0.7%)</td>
<td>745</td>
<td>-</td>
<td>1</td>
<td>1</td>
<td>--</td>
<td>2 (0.3%)</td>
<td>800</td>
<td>2</td>
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<td>1</td>
<td>5 (0.6%)</td>
</tr>
<tr>
<td>4</td>
<td>3</td>
<td>204</td>
<td>1</td>
<td>1</td>
<td>--</td>
<td>2</td>
<td>1 (1%)</td>
<td>220</td>
<td>1</td>
<td>-</td>
<td>2</td>
<td>1</td>
<td>2 (0.9%)</td>
<td>142</td>
<td>-</td>
<td>3</td>
<td>1</td>
<td>--</td>
<td>4 (2.8%)</td>
</tr>
<tr>
<td>5</td>
<td>0</td>
<td>No sheep</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>424</td>
<td>6</td>
<td>6</td>
<td>1+</td>
<td>-</td>
<td>12 (2.8%)</td>
<td>400</td>
<td>5</td>
<td>6</td>
<td>4</td>
<td>2</td>
<td>17 (4.3%)</td>
</tr>
<tr>
<td>6</td>
<td>2</td>
<td>163</td>
<td>4</td>
<td>3</td>
<td>1</td>
<td>--</td>
<td>8 (5%)</td>
<td>144</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>-</td>
<td>1 (0.7%)</td>
<td>174</td>
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<td>0</td>
<td>1</td>
<td>2 (1.2%)</td>
</tr>
<tr>
<td>7</td>
<td>2</td>
<td>217</td>
<td>3</td>
<td>1</td>
<td>--</td>
<td>4</td>
<td>2 (2.0%)</td>
<td>209</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>4 (1.9%)</td>
<td>201</td>
<td>1</td>
<td>1</td>
<td>--</td>
<td>--</td>
<td>2 (1.0%)</td>
</tr>
<tr>
<td>8</td>
<td>1</td>
<td>217</td>
<td>-</td>
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<td>--</td>
<td>-</td>
<td>1 (1%)</td>
<td>265</td>
<td>-</td>
<td>3</td>
<td>-</td>
<td>1</td>
<td>4 (1.5%)</td>
<td>235</td>
<td>1</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>3 (1.3%)</td>
</tr>
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<td>152</td>
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<td>-</td>
<td>-</td>
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<td>1 (0.6%)</td>
<td>142</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>0 (0%)</td>
<td>140</td>
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<td>2</td>
<td>1</td>
<td>5 (3.6%)</td>
</tr>
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<td>167</td>
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<td>-</td>
<td>No results</td>
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<td>108</td>
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<tr>
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<td>3</td>
<td>105</td>
<td>-</td>
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<td>1</td>
<td>1</td>
<td>4 (3.8%)</td>
<td>Sold up</td>
<td></td>
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<td>-</td>
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<td>-</td>
<td>-</td>
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</tr>
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<td>7 (2.3%)</td>
<td>294</td>
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<td>6 (2.0%)</td>
<td>274</td>
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<td>-</td>
<td>--</td>
<td>4 (1.5%)</td>
</tr>
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<td>13</td>
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<td>700</td>
<td>2</td>
<td>8</td>
<td>-</td>
<td>11</td>
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<td>680</td>
<td>-</td>
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<td>1</td>
<td>1 (0.2%)</td>
<td>640</td>
<td>1</td>
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<td>--</td>
<td>2 (0.3%)</td>
</tr>
<tr>
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<td>3**</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>0</td>
<td>0 (0%)</td>
<td>Sold up</td>
<td></td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
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<td>450</td>
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<td>4 (1.8%)</td>
<td>340</td>
<td>-</td>
<td>4</td>
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<td>1</td>
<td>7 (2.1%)</td>
<td>340</td>
<td>-</td>
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<td>1</td>
<td>6</td>
<td>16 (1.8%)</td>
</tr>
<tr>
<td>17</td>
<td>3</td>
<td>Uncoop-operative farmer</td>
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</tr>
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<td>60</td>
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<td>1 (1.7%)</td>
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<td>-</td>
<td>-</td>
<td>360</td>
<td>1</td>
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<td>-</td>
<td>5 (1.4%)</td>
</tr>
<tr>
<td>19</td>
<td>2</td>
<td>350</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>-</td>
<td>1 (0.3%)</td>
<td>No results</td>
<td>200</td>
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<td>1</td>
<td>3</td>
<td>1 (1.5%)</td>
<td>230</td>
<td>3</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>4 (1.7%)</td>
</tr>
<tr>
<td>20</td>
<td>3</td>
<td>143</td>
<td>0</td>
<td>0</td>
<td>-</td>
<td>-</td>
<td>0 (0%)</td>
<td>Sold up</td>
<td>41</td>
<td>0</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
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<td>-</td>
<td>-</td>
<td>-</td>
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</tr>
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<td>0</td>
<td>3</td>
<td>1 (1.7%)</td>
<td>99</td>
<td>1</td>
<td>-</td>
<td>1</td>
<td>1</td>
<td>1 (1.1%)</td>
<td>83</td>
<td>0</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>0 (0%)</td>
</tr>
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<td>120</td>
<td>2</td>
<td>-</td>
<td>-</td>
<td>2</td>
<td>1 (1.7%)</td>
<td>92</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>2</td>
<td>1 (1.1%)</td>
<td>88</td>
<td>0</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>23</td>
<td>3</td>
<td>240</td>
<td>1</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>2 (0.8%)</td>
<td>350</td>
<td>-</td>
<td>3</td>
<td>-</td>
<td>3</td>
<td>0 (0.9%)</td>
<td>309</td>
<td>1</td>
<td>-</td>
<td>1</td>
<td>-</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>24</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>2 (0.8%)</td>
<td>106</td>
<td>2</td>
<td>1</td>
<td>-</td>
<td>3</td>
<td>2 (8.6%)</td>
<td>15</td>
<td>0</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>25</td>
<td>3</td>
<td>No results</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>1 (0.3%)</td>
<td>No results</td>
<td>406</td>
<td>-</td>
<td>-</td>
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<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>26</td>
<td>3</td>
<td>104</td>
<td>0</td>
<td>0</td>
<td>-</td>
<td>-</td>
<td>0 (0%)</td>
<td>103</td>
<td>0</td>
<td>190</td>
<td>-</td>
<td>1</td>
<td>1 (0.9%)</td>
<td>119</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td>4748</td>
<td>19</td>
<td>29</td>
<td>10</td>
<td>7</td>
<td>6 (1.37)</td>
<td>5316</td>
<td>10</td>
<td>27</td>
<td>9</td>
<td>55</td>
<td>1 (0.37)</td>
<td>5144</td>
<td>20</td>
<td>22</td>
<td>26</td>
<td>15</td>
<td>84 (1.63)</td>
</tr>
</tbody>
</table>

---

*# 0= Housed, closed yards, minimal exercise. † Case C108 post-partum case.  
1= Housed, open yards, voluntary exercise.  
2= Flat grazed.  
3= Hill grazed.

**Figure 67**: Table showing the annual incidence of CVP (by degree and total) on the farms studied. Farm type of each farm involved is also shown.
### Table 1: Number of CV Cases

<table>
<thead>
<tr>
<th>Year</th>
<th>Number of CVP Cases</th>
<th>Percentage of Total CVP Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1981</td>
<td>65</td>
<td>32</td>
</tr>
<tr>
<td>1982</td>
<td>55</td>
<td>27</td>
</tr>
<tr>
<td>1983</td>
<td>84</td>
<td>41</td>
</tr>
<tr>
<td>TOTAL</td>
<td>204</td>
<td>100</td>
</tr>
</tbody>
</table>

**Figure 68**: Total annual number of cases of CVP investigated.

### Table 2: Farm Type Distribution

<table>
<thead>
<tr>
<th>Year</th>
<th>Farm type</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>1981</td>
<td></td>
<td>344(2)</td>
<td>655(3)</td>
<td>3184(11)</td>
<td>565(5)</td>
</tr>
<tr>
<td>1982</td>
<td></td>
<td>877(3)</td>
<td>718(3)</td>
<td>2452(9)</td>
<td>1268(6)</td>
</tr>
<tr>
<td>1983</td>
<td></td>
<td>818(3)</td>
<td>705(3)</td>
<td>2795(10)</td>
<td>826(4)</td>
</tr>
</tbody>
</table>

Mean flock size: 255, 231, 281, 177

**Figure 69**: Table showing the total number of ewes and the number of farms (in brackets) on each farm type for each year and the mean flock size for each farm type.
<table>
<thead>
<tr>
<th>Year</th>
<th>Farm type O</th>
<th>Farm type 1</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of ewes</td>
<td>Prolapse type</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1° 2° 3° 4°</td>
</tr>
<tr>
<td>1981</td>
<td>344</td>
<td>1 1 0 0 2</td>
</tr>
<tr>
<td>1982</td>
<td>877</td>
<td>6 9 0 0 15</td>
</tr>
<tr>
<td>1983</td>
<td>818</td>
<td>5 7 5 2 19</td>
</tr>
<tr>
<td>Total</td>
<td>2039</td>
<td>12 17 5 2 36</td>
</tr>
<tr>
<td></td>
<td>% of cases</td>
<td>(33) (47) (14) (6) (100)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Year</th>
<th>Farm type 2</th>
<th>Farm type 3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of ewes</td>
<td>Prolapse type</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1° 2° 3° 4°</td>
</tr>
<tr>
<td>1981</td>
<td>3184</td>
<td>14 18 7 6 45</td>
</tr>
<tr>
<td>1982</td>
<td>2453</td>
<td>3 6 5 5 19</td>
</tr>
<tr>
<td>1983</td>
<td>2795</td>
<td>8 3 8 6 25</td>
</tr>
<tr>
<td>Total</td>
<td>8432</td>
<td>25 27 20 17 89</td>
</tr>
<tr>
<td></td>
<td>% of cases</td>
<td>(28) (30) (22) (19) (100)</td>
</tr>
</tbody>
</table>

* Farm type O = housed, closed yards, minimal exercise
* Farm type 1 = housed, open yards, voluntary exercise
* Farm type 2 = no housing, flat grazing, exercise natural
* Farm type 3 = no housing, hill grazing, exercise natural

Figure 70a: Table showing the annual distribution of CVP cases seen on each farm type, and the types of prolapse seen. The overall flock size and the total (and percentage of the total flock affected in brackets) number of cases seen is also shown.
Figure 70b: Histogram showing the distribution of prolapse types on each farm type.
<table>
<thead>
<tr>
<th>Month</th>
<th>January</th>
<th>February</th>
<th>March</th>
<th>April</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Farm type</td>
<td>Farm type</td>
<td>Farm type</td>
<td>Farm type</td>
</tr>
<tr>
<td>Year</td>
<td>0 1 2 3 Total</td>
<td>0 1 2 3 Total</td>
<td>0 1 2 3 Total</td>
<td>0 1 2 3 Total</td>
</tr>
<tr>
<td>1981</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0 0 2 2 (3%)</td>
<td>0 6 10 8 24 (37%)</td>
<td>2 0 25 1 29 (45%)</td>
<td>0 0 10 0 10 (15%)</td>
</tr>
<tr>
<td>1982*</td>
<td>0 0 1 1 2 (4%)</td>
<td>9 2 7 7 25 (46%)</td>
<td>3 4 10 6 23 (42%)</td>
<td>3 0 1 5 (9%)</td>
</tr>
<tr>
<td>1983</td>
<td>2 1 0 1 4 (5%)</td>
<td>8 8 7 5 28 (34%)</td>
<td>7 7 15 6 35 (42%)</td>
<td>2 5 3 16 (19%)</td>
</tr>
<tr>
<td>Total</td>
<td>2 1 1 4 8 (40%)</td>
<td>17 16 24 20 77 (38%)</td>
<td>12 11 51 13 87 (43%)</td>
<td>5 5 14 7 31 (15%)</td>
</tr>
</tbody>
</table>

* one case seen in May

**Figure 7**: Table showing the monthly distribution of cases seen on each farm type in each year.
<table>
<thead>
<tr>
<th>FARM TYPE</th>
<th>Season</th>
<th>1981</th>
<th>1982</th>
<th>1983</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Extent of Season</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Max. lambing activity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Max. cases</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>Extent of Season</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Max. lambing activity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Max. cases</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Extent of Season</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Max. lambing activity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Max. cases</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Extent of Season</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Max. lambing activity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Max. cases</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Figure 72: Table showing the extent of the lambing season, the three week period of maximal lambing activity and the three week period during which most of CVP were seen for each farm type for each year.
considered separately, later.

An overall incidence of 1.3% was found with the annual incidence in each of the years being 1.4%, 1.0% and 1.6% respectively.

5.2.3: Farm Types:

Each farm was classified into one of four types (Figure 67), as described previously, and similar farm types were then grouped together. Figure 69 shows the number of farms of each type in each year and the total number of ewes on the combined farms with the mean flock size for each farm type. The number of cases of CVP seen in each year on each type of farm is shown in Figure 70.

5.2.4: Monthly cases:

Figure 71 shows the monthly distribution of cases in each year on each farm type and the total number of cases of CVP encountered in each month. The extent of the lambing season, the three-week period of greatest lambing activity and the three-week period in which most of the CVP cases were seen on each farm type in each year are shown in Figure 72.

Farm types 0 and 1 (on which ewes were housed either partially or completely) tended to lamb earlier in the season and the season was shorter than either of the other two types. The period of maximum lambing activity invariably coincided with the period when most of the prolapses were seen. There was no apparent pattern of incidence in relation to the start or finish of lambing between the years. It is not possible to apply a simple statistical test to determine this relationship but Figure 72 shows, clearly, the distribution of cases. A separate analysis of the individual farm data was not carried out.

5.2.5: Exercise and housing:

172
<table>
<thead>
<tr>
<th>Year</th>
<th>No. of ewes</th>
<th>1°</th>
<th>2°</th>
<th>3°</th>
<th>4°</th>
<th>Total (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1981</td>
<td>344</td>
<td>18</td>
<td>14</td>
<td>1</td>
<td>2</td>
<td>(0.6)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>9</td>
<td>14</td>
<td>1</td>
<td>2</td>
<td>(1.4)</td>
</tr>
<tr>
<td>1982</td>
<td>877</td>
<td>6</td>
<td>10</td>
<td>14</td>
<td>15</td>
<td>(1.7)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>9</td>
<td>13</td>
<td>12</td>
<td>11</td>
<td>(0.9)</td>
</tr>
<tr>
<td>1983</td>
<td>818</td>
<td>10</td>
<td>12</td>
<td>9</td>
<td>9</td>
<td>(2.6)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>10</td>
<td>17</td>
<td>7</td>
<td>11</td>
<td>(1.2)</td>
</tr>
<tr>
<td>Total</td>
<td>2039</td>
<td>31</td>
<td>39</td>
<td>27</td>
<td>27</td>
<td>(1.8)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>31</td>
<td>46</td>
<td>35</td>
<td>33</td>
<td>(1.2)</td>
</tr>
</tbody>
</table>

Figure 73: Table showing the number and percentage (in brackets) of prolapse type seen annually and total in housed and non-housed flocks.

<table>
<thead>
<tr>
<th>Year</th>
<th>No. of ewes</th>
<th>1°</th>
<th>2°</th>
<th>3°</th>
<th>4°</th>
<th>Total (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1981</td>
<td>344</td>
<td>2</td>
<td>6</td>
<td>1</td>
<td>0</td>
<td>(0.9)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>17</td>
<td>23</td>
<td>9</td>
<td>7</td>
<td>(2.6)</td>
</tr>
<tr>
<td>1982</td>
<td>877</td>
<td>6</td>
<td>14</td>
<td>0</td>
<td>1</td>
<td>(1.3)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4</td>
<td>13</td>
<td>9</td>
<td>8</td>
<td>(0.9)</td>
</tr>
<tr>
<td>1983</td>
<td>818</td>
<td>10</td>
<td>12</td>
<td>9</td>
<td>9</td>
<td>(2.6)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>10</td>
<td>17</td>
<td>7</td>
<td>11</td>
<td>(1.2)</td>
</tr>
<tr>
<td>Total</td>
<td>2039</td>
<td>17</td>
<td>39</td>
<td>27</td>
<td>27</td>
<td>(1.8)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>17</td>
<td>46</td>
<td>35</td>
<td>33</td>
<td>(1.2)</td>
</tr>
</tbody>
</table>

Figure 74: Table showing the annual and total number of cases and percentage in brackets of each prolapse type seen in flocks considered to be un-exercised and in flocks in which exercise was normally available.

<table>
<thead>
<tr>
<th>Year</th>
<th>No. of ewes</th>
<th>1°</th>
<th>2°</th>
<th>3°</th>
<th>4°</th>
<th>Total (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1981</td>
<td>344</td>
<td>2</td>
<td>6</td>
<td>1</td>
<td>0</td>
<td>(0.9)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>17</td>
<td>23</td>
<td>9</td>
<td>7</td>
<td>(2.6)</td>
</tr>
<tr>
<td>1982</td>
<td>877</td>
<td>6</td>
<td>14</td>
<td>0</td>
<td>1</td>
<td>(1.3)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4</td>
<td>13</td>
<td>9</td>
<td>8</td>
<td>(0.9)</td>
</tr>
<tr>
<td>1983</td>
<td>818</td>
<td>10</td>
<td>12</td>
<td>9</td>
<td>9</td>
<td>(2.6)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>10</td>
<td>17</td>
<td>7</td>
<td>11</td>
<td>(1.2)</td>
</tr>
<tr>
<td>Total</td>
<td>2039</td>
<td>17</td>
<td>39</td>
<td>27</td>
<td>27</td>
<td>(1.8)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>17</td>
<td>46</td>
<td>35</td>
<td>33</td>
<td>(1.2)</td>
</tr>
</tbody>
</table>
Those ewes which were unable to exercise freely and extensively were classified together (which in reality included only those in confined yards) and they were compared to the remaining ewes which were considered to be exercised adequately. Classification of the flocks in terms of their exercise is shown in Figure 73 with the number of cases of each degree of CVP seen in each year. Statistically there is no difference in the incidence of CVP between the two types of management ($p = 0.64$). Figure 74 shows a similar comparison using the criterion of the availability of housing to the ewe. Statistically, there is no difference between the housed and non-housed flocks in the incidence of CVP ($p = 0.24$) although the highest annual incidence occurred in the housed flocks in the third year of the study (2.6%).

5.3: THE EWES:

5.3.1: Control (normal) Ewes:

The number of control (normal) ewes examined and monitored each year is shown in Figure 75. The control ewes were not followed specifically beyond the year in which they were first selected and no cases of CVP were encountered in ewes which had previously been used as control animals.

5.3.3: Flock Breed Distribution:

The estimated breed distribution within the flocks is shown in Figure 76. This reflects the information given by farmers as to the rough percentage of breed distribution in the flocks involved in the investigation. As this was an estimated figure it was not felt advisable to subject the figures to statistical analysis. Most flocks consisted of a mixture of breeds and their crosses. One farm only (Farm 22) carried ewes of only one breed (pedigree Suffolk) and this farm did not encounter an exceptional number of prolapses.
<table>
<thead>
<tr>
<th>Breed of ewe</th>
<th>No. of cases (%)</th>
<th>Estimated* prevalence of breed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suffolk</td>
<td>6 (2.9%)</td>
<td>1%</td>
</tr>
<tr>
<td>Suffolk cross</td>
<td>20 (9.8%)</td>
<td>4%</td>
</tr>
<tr>
<td>Scotch - half bred</td>
<td>33 (16.2%)</td>
<td>23%</td>
</tr>
<tr>
<td>Mule</td>
<td>71 (34.8%)</td>
<td>41%</td>
</tr>
<tr>
<td>Masham</td>
<td>21 (10.3%)</td>
<td>12%</td>
</tr>
<tr>
<td>Border Leicester cross</td>
<td>14 (6.9%)</td>
<td>4%</td>
</tr>
<tr>
<td>Kerry cross and Kerry</td>
<td>4 (2%)</td>
<td>0.5%</td>
</tr>
<tr>
<td>Llein and Llein cross</td>
<td>4 (2%)</td>
<td>0.5%</td>
</tr>
<tr>
<td>Texel and Texel cross</td>
<td>14 (6.9%)</td>
<td>3%</td>
</tr>
<tr>
<td>Welsh half bred and Beulah cross</td>
<td>10 (4.9%)</td>
<td>7%</td>
</tr>
<tr>
<td>Welsh half bred and Beulah cross</td>
<td>4 (2.0%)</td>
<td>3%</td>
</tr>
<tr>
<td>Other</td>
<td>2 (1.0%)</td>
<td>0.5%</td>
</tr>
<tr>
<td>Unknown breed</td>
<td>1 (0.5%)</td>
<td>0.5%</td>
</tr>
<tr>
<td>TOTAL</td>
<td>204 (100%)</td>
<td>100%</td>
</tr>
</tbody>
</table>

* estimated from figures given by farmers.

Figure 76: Table showing the number (and percentage) of cases of CVP seen in each breed type of sheep and the estimated prevalence of breeds in the overall flock.

![Histogram showing the age distribution of CVP cases and control ewes.](image)

Figure 77: Histogram showing the age distribution of CVP cases and control ewes.
One flock (Flock 14) consisted of only three ewes of the Mule breed which were raised as orphans and treated as pets.

5.4. RESULTS OF SPECIFIC CASES:

5.4.1: Breed of ewe:

Twelve breeds/types were identified during the study and the number of cases seen within each of these is shown in Figure 76. The most frequently affected breeds were the Mule, the Scotch Half-bred, and the Masham, which together represented 61% of cases seen. The estimated prevalence in the total flock for all these breeds combined was 76%. As these breeds appeared to be over-represented in the population these were probably not significant (Figure 76).

The Suffolk and its crosses accounted for 12.7% of cases and the Texel and Border Leicester (and their crosses) each showed an incidence of 6.9%. Comparison between the number of cases seen in each breed-type and the estimated overall proportion of the flock occupied by these breeds indicated that the breeds were relatively over represented. As there were no exact population distribution figures for the individual flocks a statistical comparison could not be effectively derived.

5.4.2: Age of affected ewes:

The age distribution of affected and control ewes are shown in Figure 77. All ages of sheep were represented, including ewes aged 1 year (2.9% of cases) but a majority of cases were encountered in ewes aged 5 years and over (33%). The 5-year old animals formed the largest single age group in the overall flock (approximately 37%). The mean age of the control ewes was 3 years while that of the affected ewes was 4 years. The difference is probably a result of the relatively lower number of five year old control ewes sampled.
Figure 78: Body scores of ewes affected by CVP and control ewes. (Median body score for cases 2.5). (Median body score for controls 3.0).

Figure 79: Case of CVP in which Safety pins were found inserted into the vulva. One pin was effectively in position but the other had torn though the vulval lips. The ewe had been bought through the market 2 weeks previously.
This was inadvertent, and became apparent only when the statistical analysis was performed. Statistically, however, there is only a slight difference between the ages of the two groups (\( p = 0.046 \)).

5.4.3 : Parity of affected ewes:

Ewes in their first pregnancy were affected in significant numbers (18.6% of cases) and approximately the same number of ewes lambing for the second time were affected (18.1%). The majority of affected ewes (63.3%) were third parity or more. (This would be expected from the age distribution as most ewes in commercial flocks were expected to lamb each year of their breeding lives).

5.4.4 : Body score of affected ewes:

The histogram shown in Figure 78 illustrates the distribution of the various body scores in affected ewes. Body condition varied between 1.0 and 4.5. The mean body score of affected ewes was 2.8 (SD 0.66) (range 1.0 - 4.5) while the mean for the control ewes was 3.10 (SD 0.72) (range 1.5 - 5.0). The distribution of the results is normal with the majority of ewes in body condition 2.5 - 3.0, and an almost equal number of cases on either side of the mean.

There was no significant difference between the body condition scores of the affected ewes and those of the control ewes. In spite of the preponderance of cases within the range 2.0 - 4.0, cases of CVP occurred in ewes in both poor and good condition (\( p = 0.08 \)).

5.4.5 : Typicality of ewe:

Ninety two ewes (45%) were considered by the shepherds to be in typical condition for their flock, 43 (21%) were thought to be in better than average condition, while 69 (34%) were in poorer condition than their fellows. This is purely subjective and is obviously
open to bias by the shepherd.

No opinion was obtained concerning the control ewes.

5.4.6: Tail Length of Affected ewes:

Only one case (Case C44) was seen in a ewe with an undocked tail and 15 cases (7.4%) were encountered in ewes with a long, but docked tail (ie.an estimated ¼ of the tail removed). 105 cases (52%) were seen in ewes with an estimated ½ of the tail docked (the tail being sufficiently long to cover the vulva) and 82 cases (40%) were seen in ewes with ¾ of the tail docked (the tail would not reach the dorsal commissure of the vulva). Thus a preponderance of cases (187 ewes or 92%) was seen in ewes with half or more of the tail removed.

No record of the tail length of the control ewes was made. It would be expected however that the tails of the control ewes would have been docked.

5.4.7: Evidence of previous CVP:

193 ewes showed no evidence of any previous prolapse though it was difficult to be certain. Eight cases showed visible vulval scars and were considered to be possible cases of prolapse but perineal lacerations at lambing, post parturient prolapse, uterine prolapse after lambing or prolapse of the vagina at times other than the prepartum period could not be excluded. Three animals had a certain history of CVP either from the shepherd or from the appearance of the vulval tissues. One animal still carried two safety pins in the vulval lips (Figure 79) although it was not possible to be certain whether this had been used to treat CVP in the present or previous pregnancy or one of the other conditions mentioned above. All the ewes were, in any case, treated as first-time prolapses at the first examination in this investigation.

None of the control ewes showed any evidence of vulval damage or any other evidence of having been affected by CVP.
<table>
<thead>
<tr>
<th>Year</th>
<th>No. of ewes found dead with vaginal rupture</th>
<th>No. of ewes euthanased or died vaginal rupture</th>
<th>No. of ewes found dead other causes incl. CVP</th>
<th>No. of ewes euthanased other causes incl. CVP</th>
<th>Total*</th>
<th>No. of post mortem examinations</th>
</tr>
</thead>
<tbody>
<tr>
<td>1981</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>1982</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>1983</td>
<td>5</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>TOTAL</td>
<td>7</td>
<td>6</td>
<td>0</td>
<td>0</td>
<td>13</td>
<td>13</td>
</tr>
</tbody>
</table>

* not including those which died after treatment or up to 7 days post partum.

Figure 30: Table showing the number of cases of vaginal rupture and/or CVP which were found dead, the number which either died immediately or were euthanased and the number of post mortem examinations carried out.

Figure 31: Histogram showing the number of cases seen on the days before lambing.
5.4.8: Cases found dead or in extremis:

The number of cases found dead, the number found in extremis and the number of ewes in which the ruptured vagina syndrome was seen is shown in Figure 80. The number of cases euthanased immediately on humane grounds and the number of these subjected to post-mortem examination are also shown.

One case of ruptured vagina, which was considered to be less severe, was treated surgically by repair of the vaginal tear after thorough cleansing and careful reduction of the herniated 45 cm of ileum. Amoxycillin (Clamoxyl, Beecham, UK) at 7mg/kg and multivitamins (Vitatonic, Form Laboratories, UK) were given parenterally twice daily and 30 mg trenbolone acetate (Finajet 30, Hoescht, Germany) was administered on the first day only. The ewe appeared to respond to the surgery and was in reasonable condition over the following two days but was found dead, without apparent cause, some 72 hours after surgery. Post mortem examination was however only performed after a further 24 hours and autolysis was severe.

No cases affected with CVP only were found dead or in extremis at the first examination. This was probably due to the willingness and enthusiasm of the farmers to detect cases of CVP early and the speed with which they were examined and treated. More cases were probably, therefore, detected than might have been the case under normal conditions. They were also, therefore, possibly treated earlier.

One control ewe died prior to delivery having been affected by severe ketosis which did not respond to the normal treatment.

5.4.9: Degree of prolapse:

A total of 204 cases of CVP were seen. The overall and annual distribution of the prolapse types is shown in Figure 67.

The distribution of the types of CVP on each farm is shown in Figure 67, and on each farm type in Figure 70. The relationship between exercised and non-exercised and
Figure 82: Histogram showing the number of CVP cases which died after treatment and before lambing.

<table>
<thead>
<tr>
<th>Year</th>
<th>Prolapse degree</th>
<th>Rectal prolapse</th>
<th>Tenesmus</th>
<th>Vaginal contamination/laceration</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of cases</td>
<td>Total</td>
<td>No. of cases</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1981</td>
<td>1982</td>
<td>1983</td>
</tr>
<tr>
<td></td>
<td>1°</td>
<td>1</td>
<td></td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>2°</td>
<td>2</td>
<td>2</td>
<td>18</td>
</tr>
<tr>
<td></td>
<td>3°</td>
<td>4</td>
<td>(11%)</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td>4°</td>
<td>1</td>
<td></td>
<td>11</td>
</tr>
<tr>
<td></td>
<td>1982</td>
<td>1</td>
<td></td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>2°</td>
<td>4</td>
<td>2</td>
<td>21</td>
</tr>
<tr>
<td></td>
<td>3°</td>
<td>3</td>
<td>(15%)</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>4°</td>
<td>1</td>
<td></td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>1983</td>
<td>1</td>
<td></td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>2°</td>
<td>5</td>
<td>12</td>
<td>22</td>
</tr>
<tr>
<td></td>
<td>3°</td>
<td>4</td>
<td>(14%)</td>
<td>23</td>
</tr>
<tr>
<td></td>
<td>4°</td>
<td>2</td>
<td></td>
<td>14</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>1</td>
<td></td>
<td>19</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2°</td>
<td></td>
<td>6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>11</td>
<td>11</td>
<td>27</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3°</td>
<td></td>
<td>11</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4°</td>
<td></td>
<td>4</td>
</tr>
</tbody>
</table>

Figure 83: Table showing the number of cases of CVP (overall and for each prolapse degree) in each year and overall, in which rectal prolapse, tenesmus and vaginal contamination/laceration were prominent clinical features.
between housed and non-housed sheep with respect to the number of cases of each type of 
CVP is shown in Figures 73 and 74 respectively.

5.4.10: Days to delivery:

The number of days between the appearance of CVP and the onset of parturition is 
illustrated in Figure 81. The 22 cases seen on the day of parturition include 10 ewes in 
which elective caesarian section was performed and 3 ewes where caesarian section was 
specifically indicated at the initial examination. The mean number of days before lambing 
on which CVP first occurred was 5.6 days (range 0 - 27 days). While some cases were 
encountered as early as 27 days pre-partum, most cases (80%) occurred within the last 7 
days of gestation.

The mean number of days between the examination and delivery for the control ewes was 
5.0 which was not significantly different from the CVP cases (p = 0.87). This indicated 
that the selection of the ewes was adequate in this respect.

5.4.11: Days to pre-partum death:

The number of ewes which died between treatment and the onset of lambing was 18 
(being 7, 4 and 7 cases in the respective years) with a mean days to death of 4.4 (range 1 -
12 days) (Figure 82). Only 12 of these were subjected to post-mortem examination which 
was primarily directed to determine the number of lambs present in utero. The remaining 
6 ewes were unfortunately lost to the survey for a variety of reasons. Figure 85 shows the 
overall ewe mortality and includes the number of ewes which died between treatment and 
parturition.

5.4.12: Rectal prolapse:

Rectal prolapse was seen in a total of 27 ewes (13%) while 164 ewes (86%) were 
unaffected (Figure 83). Of the affected ewes 23 (12%) were mildly affected and 4 cases
showed severe and persistent rectal prolapse.

Twenty two cases out of the 27 showing rectal prolapse (82%) were affected by second or third degree prolapses. Only one case of first degree prolapse was noted to have a rectal prolapse and 4 cases were noted in the fourth degree group (Figure 83).

Rectal prolapse was not encountered in any of the control ewes.

5.4.13: Tenesmus:

Tenesmus (mild or severe) was present in 76% of CVP cases and Figure 83 shows the number of ewes in which this was seen in each year and the relationship between this and the degree of CVP.

Severe tenesmus was encountered in 54 cases (27% of live cases seen) and of these, 4 (7%) were affected by first degree prolapses, 21 (39%) were second degree, 15 (28%) were third degree and 14 (26%) were fourth degree.

In contrast, tenesmus was not encountered at all amongst the control ewes and was not an invariable complication of CVP.

5.4.14: Vaginal contamination:

Mild or severe contamination and/or laceration of the prolapsed tissues were present in 138 cases from a total of 204 (68%). It was considered to be mild in 118 cases (86% of contaminated ewes) and severe in 20 (14%).

The relationship between contamination and/or laceration and the severity of the prolapse, is shown in Figure 83. Of the 20 ewes which showed severe vaginal contamination and/or laceration none was affected by first degree CVP while 4, 9 and 7 ewes were affected by second, third and fourth degree respectively.

Two vaginal examinations, at approximately fourteen and seven days prepartum were performed on all 235 ewes forming the flock from Farm 8 in the 1983 season. These examinations revealed no vaginal contamination in any of the ewes, including 3 which
subsequently developed CVP. Control ewes were not examined for the presence of vaginal contamination.
5.5: CLINICAL APPROACH:

5.5.1: Local Analgesia:

Eighty cases were considered to require local analgesic as an essential part of their treatment. These included those with severe degrees of CVP, severe contamination of the vagina or those in which tenesmus was not resolved by reduction and retention of CVP. Ewes which passed urine during or after reduction generally did not require anaesthesia. No record of the number of cases which fell into each of the categories mentioned was, however, kept. Of the 80 cases which received analgesia 12 (15%) received sacral regional anaesthesia using 2% lignocaine with adrenalin (1:100,000)(Xylotox, Astra Ltd. UK), and 56 (70%) received epidural anaesthesia via the lumbosacral space (40 cases) or the first intercoccygeal space (16 cases) using 2% lignocaine without adrenaline(Lignotox, Astra Ltd., UK). Surface analgesia was used in 12 cases by local application of lignocaine hydrochloride gel (Xylotox Gel, Astra Ltd., U.K.) to the exposed mucosa.

The use of epidural or regional anaesthesia appeared to be totally effective in abolishing tenesmus for more than 2 hours in every case and most frequently (60 cases out of the 68 so treated) for more than 12 - 24 hours or permanently. However, the use of surface analgesia was almost totally ineffective in the control of tenesmus and frequently (11 out of the twelve cases) either epidural or sacral block was necessary immediately.

5.5.2: Initial approach:

All the affected ewes, except those where the prolapse was so mild as to warrant no interference, or those which were either dead or euthanased immediately, were subjected
to an identical regimen of cleansing and irrigation of the prolapsed tissue as outlined previously.

5.5.3. Treatment methods:

Eleven different approaches to treatment were used, with 4 basic principles involved. Figure 84 shows the number of cases to which these were applied, the number and percentage of effective / successful treatments (ie. those which having been applied on the first occasion were still in force on the day of lambing or death whichever came first) and the number of ewes which were considered to be cases of dystocia at parturition. The normal removal of the retention device where necessary was not considered to constitute dystocia.

As there was no significant difference between the methods within each category of treatment (p = 0.32 for Group 1, 0.19 for Group 2, and 0.08 for Group 3) it was thought justifiable to group them together into the four basic treatment methods.

Figure 85 shows the number of cases for which each of the four basic methods was used and the proportion of cases in which delivery was normal and the number of ewes in each group requiring assisted delivery. The number of cases which were affected by dystocia, the number affected by ringwomb and the number of successful treatments are shown in Figure 86. The table also shows the significance of the difference between the various groups. Dystocia was recorded (with the proportion of ringwomb cases shown in addition), in which post-partum complications (including CVP, prolapsed uterus, death or euthanasia) were found, the proportion culled due to CVP and the proportion of ewes which, having been retained, were barren in the following season.

There was no significant difference in the percentage of successful treatments between the three treatment groups (1, 2 and 3) (group 4 was, as would be expected, 100% successful; delivery by caesarian section or per vaginum being carried out immediately).

There was a significant difference (p = 0.024), however, between the groups in respect of
<table>
<thead>
<tr>
<th>Treatment group</th>
<th>Treatment method</th>
<th>No. of ewes</th>
<th>Percentage of total cases treated</th>
<th>No. (%) of * successful treatments</th>
<th>Parturition</th>
<th>No. of ewes with no significant problems</th>
<th>No. of ewes with dystocia</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Postural restraint</td>
<td>8</td>
<td>4%</td>
<td>6 (75%)</td>
<td>6 (75%)</td>
<td>0 (%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>String/canvass truss</td>
<td>4</td>
<td>2%</td>
<td>1 (25%)**</td>
<td>4 (100%)</td>
<td>0 (%)</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Safety pins</td>
<td>15</td>
<td>8%</td>
<td>12 (80%)</td>
<td>10 (67%)</td>
<td>5 (33%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Kilt pins</td>
<td>24</td>
<td>13%</td>
<td>23 (96%)</td>
<td>13 (54%)</td>
<td>11 (46%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Quill supported vulval sutures</td>
<td>9</td>
<td>5%</td>
<td>9 (100%)</td>
<td>6 (67%)</td>
<td>3 (33%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Buhner method</td>
<td>14</td>
<td>8%</td>
<td>11 (79%)</td>
<td>12 (86%)</td>
<td>2 (14%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Vulval sutures</td>
<td>57</td>
<td>31%</td>
<td>48 (84%)</td>
<td>32 (56%)</td>
<td>25 (44%)</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Eagelman method</td>
<td>7</td>
<td>4%</td>
<td>6 (86%)</td>
<td>4 (57%)</td>
<td>3 (43%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>&quot;Dalton’s spoon&quot;</td>
<td>18</td>
<td>10%</td>
<td>11 (61%)</td>
<td>10 (56%)</td>
<td>7 (44%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>&quot;Savewe&quot;</td>
<td>18</td>
<td>10%</td>
<td>11 (61%)</td>
<td>10 (56%)</td>
<td>7 (44%)</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Lambed immediately</td>
<td>5</td>
<td>3%</td>
<td>n/a</td>
<td>1 (20%)</td>
<td>4 (80%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Caesarian section*</td>
<td>25ª</td>
<td>13%ª</td>
<td>(25)</td>
<td>n/a</td>
<td>n/a</td>
<td></td>
</tr>
<tr>
<td>Total cases treated</td>
<td></td>
<td>186</td>
<td>91%</td>
<td>127 (68%)</td>
<td>98 (53%)</td>
<td>60 (32%)</td>
<td></td>
</tr>
<tr>
<td>Control ewes</td>
<td></td>
<td>80</td>
<td>n/a</td>
<td>n/a</td>
<td>77 (96%)</td>
<td>3 (4%)¹&quot;</td>
<td></td>
</tr>
</tbody>
</table>

¹ treatment in force on day of lambing without repetition or change after last visit.

² includes 10 ewes subjected to elective Caesarian Section immediately.

" minimal assistance only.

Figure 8.4: Table showing the number of ewes subjected to each treatment method, the proportion of these which were successful and the extent of normal and dystocic lambings for each type. The number of control ewes which lambed is also shown.
<table>
<thead>
<tr>
<th>Treatment group</th>
<th>No. of ewes</th>
<th>No. of ewes which died between treatment assisted and parturition</th>
<th>No. of normal deliveries (incl. assisted)</th>
<th>No. of ewes which died or euthanised at or within 7 days of delivery</th>
<th>Total ewes died (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>12</td>
<td>2 (17%)</td>
<td>10 (83%)</td>
<td>0 (0%)</td>
<td>2 (17%)</td>
</tr>
<tr>
<td>2</td>
<td>119</td>
<td>6 (6%)</td>
<td>97 (79%)</td>
<td>16 (16%)</td>
<td>22 (18%)</td>
</tr>
<tr>
<td>3</td>
<td>25</td>
<td>3 (12%)</td>
<td>20 (80%)</td>
<td>2 (8%)</td>
<td>5 (20%)</td>
</tr>
<tr>
<td>4</td>
<td>30*</td>
<td>0 (0%)</td>
<td>5 (16%)</td>
<td>1 (3%)</td>
<td>1 (3%)</td>
</tr>
<tr>
<td>TOTAL</td>
<td>169</td>
<td>11 (7%)</td>
<td>132 (78%)</td>
<td>19 (26%)</td>
<td>30 (18%)</td>
</tr>
<tr>
<td>Control</td>
<td>82</td>
<td>1 (1%)</td>
<td>79 (96%)</td>
<td>2 (3%)</td>
<td></td>
</tr>
</tbody>
</table>

* includes 10 cases elective Caesarian Section.

**Figure 85**: Table showing the number and percentage of ewes from each treatment group which died before delivery, were delivered normally, and which died within 7 days of delivery. The statistical significance of the differences between the groups and between the CVP cases and the control ewes is shown.
<table>
<thead>
<tr>
<th>Treatment group</th>
<th>No. of ewes</th>
<th>No. (and%) of successful treatments</th>
<th>No. (and%) with prepartum problems</th>
<th>No. (and%) with normal delivery</th>
<th>No. (and%) with dystocia cases</th>
<th>No. (and%) ringwomb cases</th>
<th>No. (and%) cases culled due to CVP</th>
<th>No. (and%) with post-partum problems</th>
<th>No. (and%) barren next season</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>12</td>
<td>7 (58%)</td>
<td>5 (42%)</td>
<td>10 (83%)</td>
<td>0 (0%)</td>
<td>2 (13%)</td>
<td></td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>2</td>
<td>119</td>
<td>103 (87%)</td>
<td>33 (28%)</td>
<td>73 (67%)</td>
<td>46 (39%)</td>
<td>16 (13%)</td>
<td></td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>3</td>
<td>25</td>
<td>17 (68%)</td>
<td>12 (48%)</td>
<td>14 (56%)</td>
<td>10 (40%)</td>
<td>4 (16%)</td>
<td></td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>4</td>
<td>30</td>
<td>n/a</td>
<td>n/a</td>
<td>n/a</td>
<td>n/a</td>
<td></td>
<td></td>
<td>n/a</td>
<td>n/a</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td>178</td>
<td>127 (87%)</td>
<td>52 (34%)</td>
<td>118 (77%)</td>
<td>69 (45%)</td>
<td>22 (14%)</td>
<td>50 (29%)</td>
<td>41 (23%)</td>
<td>9 (17%)</td>
</tr>
<tr>
<td>Control</td>
<td>80</td>
<td>n/a</td>
<td>5 (6%)</td>
<td>79 (98%)</td>
<td>3 (4%)</td>
<td>0</td>
<td>n/a</td>
<td>2 (2%)</td>
<td>n/a</td>
</tr>
<tr>
<td>Significance*</td>
<td></td>
<td>n/a</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>**</td>
<td></td>
<td>ns</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

† treatments in force on day of lambing.

* All pregnancy toxaemia.

* Ewes showing ringwomb subjected to Caesarian Section or lambed.

* From 171 ewes.

** Wilcoxin 2 sample test.

* Of ewes retained (53).

* Including death, tenesmus, pregnancy toxaemia, hypocalcaemia.

* Uncertain data.

Figure 86: Table showing the efficacy of each treatment group with the statistical significance between treatment groups and between control and diseased ewes overall.
the proportion with pre-partum complications which, again, was entirely attributable to
group 4 in which no pre-partum complications could obviously arise. There was no
statistical difference between the other three groups analysed together (excluding group 4)
(p = 0.21).

All four groups showed an almost equal proportion of ewes with normal delivery (p =
0.536).

Considering groups 2 and 3 only there was no difference between them with respect to
the proportion of dystocia cases, although between groups 1, 2 and 3 there was a marked
difference (p = 0.024). This was possibly attributable to the low proportion of the group 1
cases which presented with dystocia. This, in turn, was probably attributable to the
restricted use of group 1 treatments for mild forms of CVP only.

There were no significant differences in the number of cases seen in each treatment
group which were culled as a consequence of CVP alone or which suffered from post-
partum complications. However, in spite of no overall significance between the four
groups (p = 0.109), in respect of the proportion of barren ewes in the next pregnancy, the
highest proportion was in those ewes subjected to caesarian section. There was a
significant difference between this group and the combined groups 1, 2 and 3 (p = 0.005.)

5.5.4 : Success of treatment method :

The success of individual treatments varied and Figures 84 and 86 show the number of
successful treatments for each treatment method ie. those which, having been applied at
the first examination, were still in force at parturition. Of the 29 ewes in which treatment
was not successful, 12 (41%) were subjected to caesarian section. The other 17 cases were
treated by whichever method was considered to be the most appropriate.

There was a relatively high failure rate in the few cases treated by the string/truss
method (75%) and with the Dalton’s Spoon (39%). This latter result was particularly
disappointing in view of the favour with which this instrument is viewed worldwide. Failure, generally, resulted from a high proportion of cases (5) in which tenesmus became unacceptably forceful and could not be resolved by the use of epidural anaesthesia for any meaningful length of time. All the other methods showed a success rate of over 75%, including the group 1, which were treated posturally, with trusses applied by the farmer or were not treated at all.

The group 4 ewes, as expected, showed 100% success but the sub-group in which ewes were lambed immediately were more liable to dystocia (80% compared to 39% and 40% in groups 2 and 3 respectively). This was expected as the group included the ewes which were found to be lambing while affected with CVP. Group 4 also included those ewes which were subjected to caesarian section, even when there had been another method in force prior to the onset of parturition (12 cases) and those subjected to elective caesarian section (10 cases). Only 3 ewes affected by CVP were delivered by caesarian section on the first day. These ewes were presented with all the normal signs of full term but were not delivered per vaginum because either they were physically obstructed (ringwomb or foetal dystocia) or the prolapse was considered to be too fragile to permit obstetric manipulation.
5.6 : PARTURITION :

5.6.1 : Deaths up to lambing :

The number of ewes which died between treatment and lambing in each treatment group is shown in Figure 85 and compared to the control group. The highest proportion of deaths (17%) occurred in the Group 1 ewes, ie. those which were treated posturally, or by the truss methods. The proportion of deaths between treatment and parturition in groups 2 and 3 was 6% and 21% respectively while in group 4 it was, of course, 0%.

One control ewe died due to non-responsive ketosis.
Statistically there was a significant difference between the diseased and the control ewes (p = 0.013) with respect to the number of deaths prior to lambing.

5.6.2 : Delivery :

The number of ewes in each treatment group and the control ewes which lambed satisfactorily (including those which required some assistance) is shown in Figures 85 and 86.

The lambing process in 78 affected ewes (47%) was considered to be normal, requiring little or no interference while 89 (53%) needed skilled assistance from either veterinarian or shepherd.

In contrast, none of the control ewes required any significant obstetrical assistance at lambing.

A significant number of cases of delayed or inadequate cervical dilatation (ringwomb) was encountered in each treatment group, but there was no apparent difference between
<table>
<thead>
<tr>
<th>Year</th>
<th>No. of ewes</th>
<th>No. and percentage of ewes which did not require caesarian section</th>
<th>Elective caesarian section</th>
<th>Indicated caesarian section</th>
<th>Caesarian section at subsequent pregnancy</th>
<th>No. of retained ewes</th>
<th>Caesarian section</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of retained ewes</td>
<td>Caesarian section</td>
<td>Retained ewes</td>
<td>Retained ewes</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1981</td>
<td>53 (100%)</td>
<td>0 (0%)</td>
<td>16</td>
<td>0 (0%)</td>
<td>1 (6%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1982</td>
<td>48 (79%)</td>
<td>3 (6%)</td>
<td>7 (15%)</td>
<td>16</td>
<td>0 (0%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1983</td>
<td>48 (76%)</td>
<td>7 (11%)</td>
<td>8 (13%)</td>
<td>21</td>
<td>3 (14%)</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>164</td>
<td>81 (100%)</td>
<td>10 (6%)</td>
<td>15 (9%)</td>
<td>53</td>
<td>4 (8%)</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>81 (100%)</td>
<td></td>
<td>0 (0%)</td>
<td>n/a</td>
<td>n/a</td>
<td>n/a</td>
</tr>
</tbody>
</table>

Figure 87: Table showing the annual (and total) numbers (and percentages) of ewes subjected to caesarian section and compared to the control ewes. Also shown is the number of ewes which were retained into the following season(s) which required caesarian section whether or not they had been subjected to caesarian section on the first occasion.
the different treatment groups in the proportions of ringwomb cases \((p = 0.260)\) (Figure 86).

There was, however, a marked difference between the affected and control ewes. Ringwomb was seen in 22 ewes (14%) of the CVP cases and in none of the control ewes \((p < 0.001)\).

5.6.3 : Caesarian section :

A total of 25 ewes were subjected to caesarian section. Of these, 10 were carried out electively on the day CVP was noticed without regard for the stage of pregnancy or the presence or absence of signs of imminent parturition.

Three ewes were delivered by caesarian section because they showed immediate indications that this procedure was necessary. Vaginal laceration/contamination was so severe in one case that vaginal delivery was not thought to be safe and the 2 others could not be delivered per vaginum due to ringwomb.

In 12 cases caesarian section followed the use of one or other of the retention methods. Of the latter cases 5 suffered almost continuous tenesmus and a consequent deterioration in metabolic status. Three cases were presented lambing but were considered to be poor candidates for vaginal delivery because the vaginal wall was necrotic and/or badly inflamed. The remaining 4 cases had shown various maternal and foetal dystocia complications (one being a case of ringwomb).

A table of the annual numbers subjected to this procedure is shown in Figure 87, which also shows the number of ewes which required caesarian section at a subsequent pregnancy. A total of 4 ewes (8%) from 53 retained ewes needed subsequent caesarian section at one of the following lambings.

Figure 87 illustrates that the overall requirement for caesarian section was 9% of cases of CVP at the first occasion and 8% in ewes which prolapse again in a subsequent pregnancy (excluding those in which surgery was elective). This was an insignificant difference.
<table>
<thead>
<tr>
<th></th>
<th>No. of ewes</th>
<th>No. of premature births</th>
<th>Estimated* prematurity (days) (mean)</th>
<th>Mean no. of lambs/ewe</th>
<th>Mean no. of live lambs per ewe**</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elective caesarian section</td>
<td>10</td>
<td>7 (70%)</td>
<td>5.5</td>
<td>2.0</td>
<td>0.7</td>
</tr>
<tr>
<td>Indicated caesarian section</td>
<td>15¹</td>
<td>9 (60%)</td>
<td>3.0</td>
<td>2.0</td>
<td>0.9</td>
</tr>
</tbody>
</table>

* estimated from maternal and foetal features
** live at 48 hrs post surgery
¹ includes 12 ewes which had received other forms of treatment prior to Caesarian Section.

Figure 88: Table showing the number of premature deliveries and the mean estimated prematurity of the foetuses in ewes subjected to elective and indicated Caesarian Section.
<table>
<thead>
<tr>
<th>Year</th>
<th>Prolapse degree</th>
<th>No. significant problems</th>
<th>Outcome post partum (No. of ewes)</th>
<th>Post partum CVP</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of ewes</td>
<td>Total</td>
<td>Death &lt;7 days post partum</td>
</tr>
<tr>
<td>1981</td>
<td>1°</td>
<td>15</td>
<td>44</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>2°</td>
<td>22</td>
<td></td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>3°</td>
<td>7</td>
<td></td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>4°</td>
<td>0</td>
<td></td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>42</td>
<td>51</td>
<td>3</td>
</tr>
<tr>
<td>1982</td>
<td>1°</td>
<td>10</td>
<td></td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>2°</td>
<td>19</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>3°</td>
<td>5</td>
<td></td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>4°</td>
<td>8</td>
<td>42</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>36</td>
<td>51</td>
<td>3</td>
</tr>
<tr>
<td>1983</td>
<td>1°</td>
<td>17</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>2°</td>
<td>15</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>3°</td>
<td>13</td>
<td></td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>4°</td>
<td>6</td>
<td>51</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>42</td>
<td>51</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>1°</td>
<td>15</td>
<td></td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>2°</td>
<td>19</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>3°</td>
<td>7</td>
<td></td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>4°</td>
<td>14</td>
<td>51</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>42</td>
<td>51</td>
<td>3</td>
</tr>
</tbody>
</table>

Figure 99: Table showing the number of ewes in each year for each degree of CVP (and overall) which were known to suffer from post parturient complications (death, euthanasia, prolapsed uterus, post-partum CVP) and compared to the control ewes. The statistical significance of the differences between the diseased and control animals is shown.
Figure 90 a and b: Post-parturient recurrences of CVP showing extensive necrosis, inflammation and infection of the prolapsed cervix and vagina. The cases were characterised by continuous severe tenesmus following normal spontaneous delivery in (a) and an assisted delivery in (b).
Amongst the control ewes no caesarian section was found to be necessary and the difference between the diseased and control ewes was significant (p < 0.001).

Figure 88 shows the estimated number of premature births and the number of days premature the lambs were thought to be on delivery by caesarian section, either elective or indicated. The prematurity was based purely on clinical experience of lambing ewes without measurement of the foetuses and may be subject to criticism in that respect. No statistical analysis was therefore carried out.

A table showing the outcome in terms of survival, lambs born and live lambs produced is shown in Figure 99.

5.6.4: Problems arising in the puerperium:

The outcome for the ewes within 7 days post-partum is summarised in Figure 89 which also shows the annual incidence of post-parturient problems such as post-parturient recurrence of CVP (Figure 90a and b) and uterine prolapse (Figure 91a and b), in relation to the degree of CVP. Thus, 26 ewes (out of 163) died within 7 days of delivery and 4 were euthanased on humane grounds. An overall mortality of 22% was obtained in the CVP cases, while, in the control ewes, 2 ewes died out of a total of 81 which lambed (mortality = 3%). There was a significant increase in the post-partum mortality in affected ewes compared to control ewes (p < 0.01). Three cases of prolapsed uterus occurred in the diseased sheep (2%) (Figures 91a and b) while 2 cases occurred amongst the control ewes (2.5%). The numbers are not adequate to show a statistical analysis but the indications are that there is no increase in the incidence of prolapsed uterus in CVP cases. Post-parturient CVP, as shown in Figures 90a and b, was recorded in 5 cases (4%). One case (C108) developed post-parturient CVP without a history of having been affected pre-partum but it is not certain that she had not been affected and may merely not have been detected by the shepherd. Alternatively, this may represent a genuine case of post-partum CVP. In any case, post-partum CVP was not encountered in any of the
Figure 91 a and b: Post-parturient uterine prolapse in ewes which had been affected by CVP pre-partum. Delivery had been complicated by delayed cervical dilatation (ringwomb) and a particularly large, single foetus.
control ewes.

The low numbers preclude any statistical analysis but it is noticeable that 5 cases of post-partum CVP were certainly recurrences of the prepartum condition.
5.7 : THE LAMBS :

5.7.1 : The uterine burden of affected ewes :

The number of lambs carried by ewes affected by CVP in each year compared to the
number born to the control ewes (in total) is shown in Figure 92.

The total number of diseased ewes (information obtained from 186 cases) carried a total
of 421 lambs giving a lambing rate of 2.26 lambs/ewe compared to 1.86 lambs/ewe in the
control ewes (Figure 93). There is a statistically significant difference between the two
classes of ewe (p = 0.004).

There was no significant difference between the different degrees of prolapse with
respect to the the uterine burden of affected ewes (p = 0.18) (Figure 93). Each prolapse
degree group carried a mean of between 2.19 and 2.37 lambs per ewe. Figure 94 shows
the number of affected ewes in each year and overall and Figure 95 shows the number of
live lambs born to affected ewes in each year without regard for the degree of CVP.

Only 8% of cases carried singletons compared to 37% of control ewes and 59% of cases
carried twins compared to 41% in controls. Triplets were carried by 32% of CVP cases
carried and only 21% of control animals. Both control and diseased ewes showed a 1%
incidence of quadruplet lambs.

Figure 96 shows the number of ewes affected by each prolapse degree, the overall
number and the number of control ewes which carried singleton, twin, triplet and
quadruplet lambs. There is a significant increase in the number of multiple births in CVP
cases (92% of affected ewes and only 63% of control ewes carried multiple lambs) (p =
0.01) but no difference between ewes affected by different degrees of CVP ( (p = 0.66).
<table>
<thead>
<tr>
<th>Year</th>
<th>No. of ewes</th>
<th>No. of lambs</th>
<th>Mean no of lambs/ewe (SD)</th>
<th>No. of live lambs born*</th>
<th>Mean no of live lambs/ewe (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1981 CVP cases</td>
<td>58</td>
<td>142</td>
<td>2.45 (1.21)</td>
<td>70</td>
<td>1.21 (0.99)</td>
</tr>
<tr>
<td>1982</td>
<td>54</td>
<td>115</td>
<td>2.13 (1.06)</td>
<td>60</td>
<td>1.11 (1.02)</td>
</tr>
<tr>
<td>1983</td>
<td>74</td>
<td>164</td>
<td>2.22 (0.90)</td>
<td>78</td>
<td>1.05 (0.90)</td>
</tr>
<tr>
<td>Total</td>
<td>186</td>
<td>421</td>
<td>2.26 (0.78)</td>
<td>208</td>
<td>1.12 (1.08)</td>
</tr>
<tr>
<td>Control ewes</td>
<td>81</td>
<td>151</td>
<td>1.86 (0.79)</td>
<td>117</td>
<td>1.44 (0.91)</td>
</tr>
</tbody>
</table>

* lambs alive after 48 hours.

**Figure 92**: Table showing the number of lambs carried, the mean (and S.D.) number of lambs per ewe and the number and mean (and S.D.) of the lambs born alive and alive after 48 hours in the cases and control ewes. The significance of the differences between the cases and control ewes is shown.
<table>
<thead>
<tr>
<th>Prolapse degree</th>
<th>Lambs carried</th>
<th>Litter weight</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of ewes</td>
<td>No. of lambs</td>
</tr>
<tr>
<td>CVP cases</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>43</td>
<td>102</td>
</tr>
<tr>
<td>2</td>
<td>73</td>
<td>160</td>
</tr>
<tr>
<td>3</td>
<td>38</td>
<td>86</td>
</tr>
<tr>
<td>4</td>
<td>32</td>
<td>73</td>
</tr>
<tr>
<td>TOTAL</td>
<td>186</td>
<td>421</td>
</tr>
<tr>
<td>CONTROL</td>
<td>81</td>
<td>151</td>
</tr>
</tbody>
</table>

\* Lambs alive 48 hours after birth.

\*\* Includes 10 ewes subjected to elective Caesarian Section.

**Figure 93**: Table showing the distribution of lambs carried, live lambs born, the total weight and the mean individual lamb weight for each degree of prolapse. The standard deviations are shown (in brackets). The same values for the control group are shown with the significance of the differences.
<table>
<thead>
<tr>
<th>Year</th>
<th>n</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>1981</td>
<td>58</td>
<td>5 (9%)</td>
<td>24 (41%)</td>
<td>27 (47%)</td>
<td>2 (3%)</td>
</tr>
<tr>
<td>1982</td>
<td>54</td>
<td>4 (8%)</td>
<td>39 (72%)</td>
<td>11 (21%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>1983</td>
<td>74</td>
<td>5 (7%)</td>
<td>48 (65%)</td>
<td>21 (28%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Total</td>
<td>186</td>
<td>14 (8%)</td>
<td>110 (59%)</td>
<td>59 (32%)</td>
<td>2 (1%)</td>
</tr>
<tr>
<td>Control</td>
<td>81</td>
<td>30 (37%)</td>
<td>33 (41%)</td>
<td>17 (21%)</td>
<td>1 (1%)</td>
</tr>
</tbody>
</table>

Figure 94: Table showing the uterine burden and the percentage of the total number cases of CVP in each year overall and for the control ewes (combined).

<table>
<thead>
<tr>
<th>Year</th>
<th>n</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>1981</td>
<td>60</td>
<td>23 (38%)</td>
<td>11 (18%)</td>
<td>17 (28%)</td>
<td>8 (13%)</td>
<td>1 (1.0%)</td>
</tr>
<tr>
<td>1982</td>
<td>53</td>
<td>15 (28%)</td>
<td>15 (28%)</td>
<td>19 (36%)</td>
<td>4 (8%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>1983</td>
<td>73</td>
<td>35 (48%)</td>
<td>13 (18%)</td>
<td>16 (22%)</td>
<td>9 (12%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Total</td>
<td>186</td>
<td>73 (40%)</td>
<td>39 (21%)</td>
<td>52 (28%)</td>
<td>21 (11%)</td>
<td>1 (0.5%)</td>
</tr>
<tr>
<td>Control</td>
<td>81</td>
<td>11 (14%)</td>
<td>32 (40%)</td>
<td>27 (33%)</td>
<td>11 (14%)</td>
<td>0 (0%)</td>
</tr>
</tbody>
</table>

* Lamb alive 48 hours after delivery

Figure 95: Table showing the number of ewes in each year which delivered no live lambs, a single live lamb, live twins, live triplets and live quadruplets and the corresponding percentage of the lambing ewes in each group.
<table>
<thead>
<tr>
<th>Degree of prolapse</th>
<th>No. of lambs carried</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 (%)</td>
</tr>
<tr>
<td>CVP ewes</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>2</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>3</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>4</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Total</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Control ewes'</td>
<td>0 (0%)</td>
</tr>
</tbody>
</table>

* Ewe found to be barren excluded from results.

**Figure 96:** Table showing the number (and proportion) of ewes affected by 1, 2, 3, 4th degree prolapses which carried 1, 2, 3, & 4 lambs. The total number of CVP cases and control ewes which carried 1, 2, 3, & 4 lambs is also shown.

<table>
<thead>
<tr>
<th>Degree of prolapse</th>
<th>No of ewes delivering</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 live lambs</td>
</tr>
<tr>
<td>CVP cases</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>8 (19%)</td>
</tr>
<tr>
<td>2</td>
<td>24 (32%)</td>
</tr>
<tr>
<td>3</td>
<td>24 (62%)</td>
</tr>
<tr>
<td>4</td>
<td>17 (68%)</td>
</tr>
<tr>
<td>Total</td>
<td>73 (40%)</td>
</tr>
<tr>
<td>Control ewes</td>
<td>12 (15%)</td>
</tr>
</tbody>
</table>

* 1 missing result — ewe barren.

**Figure 97:** Table showing the number and percentage of ewes in each prolapse degree which produced one, two, three or four live lambs (i.e. still alive at 48 hrs. post partum). The corresponding numbers for control ewes are also shown.
5.7.2: Lamb mortality:

The number of live lambs delivered and the number of live lambs born per affected ewe in each year is shown in Figure 95 and compared to the lambs born to the control ewes. The number of ewes in each year which produced 0, 1, 2, 3 and 4 live lambs is also shown in Figure 97. Thus, 40% of CVP cases and 15% of control ewes produced no live lambs.

The proportion of control ewes which produced 1, 2 and 3 live lambs from multiple births was 39%, 33% and 13% respectively, while in the diseased ewes the respective proportions were 19%, 29% and 12%.

A total of 208 lambs (mortality of 50.6%) were born alive to 186 ewes originally examined (Figure 93). (The uterine burden of 18 ewes, some of which were lost to follow up and some of which died, was not known). Thus, overall, 1.12 live lambs were produced per affected ewe, while in the control group the rate was 1.42 live lambs per ewe (Figures 93 and 94). This is a significant difference (p = 0.001).

There is, also, a significant difference between the control and affected ewes in the mortality of the lambs. In the control ewes 23% of lambs died, while in the CVP cases the mortality was 50.6% (p = 0.001).

Amongst first, second, third and fourth degree prolapse cases the lamb mortality was found to be 34, 43%, 74% and 62% respectively (compared to an overall mortality in the control ewes of only 23%). Ewes affected by first degree prolapses produced 1.56 live lambs per ewe on average (compared to 1.42 live lambs/ewe from controls), but second degree prolapses produced only 1.25 live lambs per ewe. Third and fourth degree prolapses yielded a mean of 0.57 and 0.88 live lambs per ewe respectively. There is therefore an increasing mortality as CVP becomes more severe up to Third Degree CVP. This is statistically significant (p = 0.007) (Figure 98).

The reduced mortality of lambs from ewes with fourth degree prolapses was possibly due to the extra care with which these animals were treated on the farms and to the fact that 3 ewes were subjected to caesarian section immediately and 5 more were lambed
Figure 98: Table showing the percentage mortality of lambs from ewes affected by various degrees of prolapse and the corresponding mortality in control ewes.
<table>
<thead>
<tr>
<th></th>
<th>No. of ewes</th>
<th>No. of ewes alive 7 days post surgery</th>
<th>Total no. of lambs carried</th>
<th>Mean no. of lambs/ewe</th>
<th>Total no. of live lambs</th>
<th>Lamb mortality (%)</th>
<th>No. of ewes with no live lambs</th>
<th>Percentage of pregnancies producing no live lambs</th>
<th>No. of ewes carrying multiple foetuses and percentage of total no.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elective caesarian section**</td>
<td>10</td>
<td>10 (100%)</td>
<td>20</td>
<td>2.0</td>
<td>7</td>
<td>65</td>
<td>7</td>
<td>70</td>
<td>8 (80%)</td>
</tr>
<tr>
<td>Indicated caesarian section†</td>
<td>15</td>
<td>14 (93%)</td>
<td>30</td>
<td>2.0</td>
<td>13</td>
<td>57</td>
<td>7</td>
<td>47</td>
<td>15 (100%)</td>
</tr>
<tr>
<td>Total</td>
<td>25</td>
<td>24 (96%)</td>
<td>50</td>
<td>2.0</td>
<td>20</td>
<td>60</td>
<td>14</td>
<td>56</td>
<td>23 (92%)</td>
</tr>
<tr>
<td>Control ewes</td>
<td>81</td>
<td>80 (99%)</td>
<td>151</td>
<td>1.86</td>
<td>119</td>
<td>21</td>
<td>12</td>
<td>15</td>
<td>51 (63%)</td>
</tr>
<tr>
<td>CVP cases overall</td>
<td>185</td>
<td>160 (86%)</td>
<td>419</td>
<td>2.27</td>
<td>210</td>
<td>50</td>
<td>73</td>
<td>40</td>
<td>171 (92%)</td>
</tr>
</tbody>
</table>

* live lambs at 48 hrs post partum
** Caesarian section performed immediately regardless of stage of pregnancy
† Caesarian section performed when specifically indicated including ewes which had previously been treated

Figure 99: Table showing the number of ewes subjected to Caesarian section (elective and indicated) and the number of lambs, the number of live lambs, the lamb mortality, the proportion of pregnancies which failed to produce any live lambs and the proportion of pregnancies which carried multiple foetuses. The corresponding values for control ewes (and the statistical significance of the differences between affected ewes and the controls) and the total CVP population (and the significance of the differences between the total population of cases and the control ewes) are shown.
immediately. Figure 97 shows the number of ewes affected by each degree of CVP which delivered 0, 1, 2, 3, and four live lambs. An increasing mortality became apparent as the severity of CVP became greater. The highest proportion of single live lambs born was found amongst ewes affected by first degree CVP with a gradual reduction to the fourth degree CVP cases where no ewes produced one live lamb i.e. they either all died or more than one survived. The same trend was apparent in ewes which produced 2 and 3 live lambs.

The survival of lambs delivered by indicated and elective caesarian section is shown in Figure 99 which also shows, for comparative purposes, the results of the control ewes and the overall results of the affected ewes. It is apparent that the lambing rate for these animals is almost exactly 2 lambs/ewe in both caesarian section classes but the mortality amongst ewes subjected to elective surgery was higher (7%) than those in the elective group (0%).

A much higher proportion of the elective group failed to produce any live lambs (70%) as opposed to 47% in the cases in which surgery was indicated. Thus, 70% of pregnancies in the elective group could be considered a total failure. As in the case of the the overall group of ewes affected with CVP a high proportion of ewes (92%) subjected to caesarian section carried multiple foetuses (compared to 92% amongst CVP cases and only 63% in the control ewes).

5.7.3: Lamb weight:

The mean litter weight of the lambs born to the overall group of ewes affected by CVP, to ewes affected by different degrees of CVP and to the control ewes is shown in Figure 93. Unfortunately not every case provided this information but from 98 cases an average litter weight of 7.87 kilograms with a mean individual lamb weight of 3.64 kilograms was obtained. The corresponding values for 52 control ewes were 7.49 kilograms and 4.31 kilograms. There was an apparently slightly higher mean litter weight but a lower mean
individual lamb weight amongst ewes affected by CVP but the former result was not significant \( p = 0.13 \) and the latter only marginally so \( p = 0.04 \). Combining the cases of CVP from First and Second Degree ie. those in which the cervix was not prolapsed, resulted in a significantly different mean lamb weight from the combined Third and Fourth Degree CV cases (in which the cervix was prolapsed) \( p = 0.001 \).

The control ewes were not generally monitored closely at parturition and while the lambs were dry when weighed they may have fed prior to being weighed. The diseased ewes, however, were almost always monitored closely and the lambs weighed immediately, so that generally they were unfed but may have been wet.

This group also included the weights of lambs which were dead at birth (including the premature), those which were weighed at post mortem examination of the dam and some which were left after death for some hours before weighing. The interpretation of these results should therefore be treated with caution.

The litter weight of those ewes affected by more severe degrees of CVP tended to be greater but this was not statistically significant \( p = 0.25 \). Likewise the individual lamb size became higher as the degree of prolapse became worse. Indeed, the milder forms of CVP showed a reduced lamb birth weight when compared to the control ewes. Only those ewes affected by the most severe forms of CVP carried lambs which were on average the same weight as the control ewes (Figure 93).

An analysis of the litter weight variations revealed that there was no significant difference from year to year in the mean litter weight of ewes affected by CVP \( p = 0.092 \).
5.8 : EWE PROGNOSIS :

5.8.1 : Rebred ewes :

A total of 53 ewes were retained in their flocks and were rebred in the first season after the initial episode of CVP. The individual histories of the retained ewes are shown in Figure 100. There were 9 affected ewes (17%) which did not fall pregnant in the subsequent lambing season, i.e. were considered barren, although they had been running with the ram in the expectation of pregnancy. There was no specific information as to whether they had, in fact, been mated. However, most farmers used raddle keels on the rams and, had the ewes not been marked they would most probably have been culled. It is therefore assumed, tentatively, that oestrus had been normal. No information as to the frequency of mating was obtained.

Figures 100 and 101 show the number of ewes which did become pregnant, the number affected by CVP in the ensuing season, the number which were not affected by recurrence of the CVP, and the number of lambs carried with the number of live lambs delivered. It is apparent that the retained ewes which fell pregnant maintained the higher than normal fecundity (between 2.1 and 2.3 lambs per ewe compared to 1.86 in the control group). Statistically there was no difference in the lambing rate over the ensuing years for the retained ewes (p = 0.48) or between the number of live lambs delivered per ewe (p = 0.78).

Of the 53 retained sheep 29 (55%) prolapsed the following year and 9 ewes were barren (17%); the proportion of ewes which suffered from a recurrence of CVP in the next year was, in reality, 66% when the barren ewes were excluded from the number of ewes retained. Ewes did not necessarily prolapse the following year as 16 ewes (36%) out of 44
<table>
<thead>
<tr>
<th>Case no.</th>
<th>Initial episode</th>
<th>Second year</th>
<th>Third year</th>
<th>Fourth year</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Prolapse Degree</td>
<td>No. of live lambs</td>
<td>Prolapse Degree</td>
<td>No. of live lambs</td>
</tr>
<tr>
<td>2</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>3</td>
<td>3</td>
<td>0</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>4</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>8</td>
<td>1</td>
<td>3</td>
<td>2</td>
<td>2</td>
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<tr>
<td>11</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>2</td>
</tr>
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<td>17</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>18</td>
<td>3</td>
<td>2**</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>20</td>
<td>1</td>
<td>3</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>28</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
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<td>2</td>
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<td>2</td>
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<td>1</td>
<td>3</td>
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<td>3</td>
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<td>4*</td>
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<td>3</td>
<td>0</td>
<td>2</td>
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<td>0</td>
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<td>4</td>
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<td>180</td>
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<td>nil</td>
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<td>194</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>198</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>3</td>
</tr>
</tbody>
</table>

* died  
† culled  
* post partum CVP only  
n/k lost to follow up  
* lamb retained for breeding

Figure 10: Individual case records from ewes which were retained for breeding after suffering from CVP. The degree of prolapse, the number of lambs and the number of live lambs is shown for each breeding season surveyed.
<table>
<thead>
<tr>
<th>Year</th>
<th>First season CVP affected</th>
<th>No. of ewes</th>
<th>Lambs carried</th>
<th>Mean no. of lambs per ewe</th>
<th>Live lambs</th>
<th>Live lambs per ewe</th>
<th>No. of barren ewes</th>
<th>Second season CVP affected</th>
<th>No. of ewes</th>
<th>Lambs carried</th>
<th>Mean no. of lambs per ewe</th>
<th>Live lambs</th>
<th>Live lambs per ewe</th>
<th>No. of barren ewes</th>
<th>Third season CVP affected</th>
<th>No. of ewes</th>
<th>Lambs carried</th>
<th>Mean no. of lambs per ewe</th>
<th>Live lambs</th>
<th>Live lambs per ewe</th>
</tr>
</thead>
<tbody>
<tr>
<td>1981</td>
<td>16</td>
<td>40</td>
<td>2.5</td>
<td>32</td>
<td>2.0</td>
<td></td>
<td>0</td>
<td>15</td>
<td>36</td>
<td>2.4</td>
<td>17</td>
<td>1.1</td>
<td></td>
<td>0</td>
<td>2</td>
<td>4</td>
<td>2.0</td>
<td>2</td>
<td>1.0</td>
<td></td>
</tr>
<tr>
<td>1982</td>
<td>16'</td>
<td>31</td>
<td>2</td>
<td>28</td>
<td>1.8</td>
<td></td>
<td>3</td>
<td>5</td>
<td>10</td>
<td>2.0</td>
<td>8</td>
<td>1.6</td>
<td></td>
<td>0</td>
<td>10</td>
<td>4</td>
<td>2.0</td>
<td>3</td>
<td>1.5</td>
<td></td>
</tr>
<tr>
<td>1983</td>
<td>21</td>
<td>41</td>
<td>2</td>
<td>2</td>
<td>15</td>
<td>0.7</td>
<td>6</td>
<td>9</td>
<td>18*</td>
<td>2.3*</td>
<td>15</td>
<td>1.9</td>
<td></td>
<td>0</td>
<td>3</td>
<td>13</td>
<td>2.2</td>
<td>10</td>
<td>1.7</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>53</td>
<td>112</td>
<td>2.1</td>
<td>75</td>
<td>1.4</td>
<td>9</td>
<td>29*</td>
<td>64</td>
<td>2.3</td>
<td>40</td>
<td>1.4</td>
<td></td>
<td>0</td>
<td>15</td>
<td>21</td>
<td>2.1</td>
<td>15</td>
<td>1.5</td>
<td></td>
</tr>
</tbody>
</table>

1 includes case C108 which presented as post-parturient CVP in first season.
2 includes barren ewes from previous year.

Figure 10: Table showing the number of lambs carried, the number of lambs per ewe, the number of live lambs born (overall and per ewe) born to the ewes retained for further lambing seasons.
<table>
<thead>
<tr>
<th>Year</th>
<th>No. of ewes retained in flock</th>
<th>No. of ewes culled due to CVP</th>
<th>No. of ewes culled for other reasons</th>
<th>No. of ewes died/unknown or not applicable</th>
<th>Mantel-Haenszel* Chi² (P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1981</td>
<td>56</td>
<td>16 (29%)</td>
<td>19 (34%)</td>
<td>4 (7%)</td>
<td>&lt;0.05 (*)</td>
</tr>
<tr>
<td>1982</td>
<td>44</td>
<td>16 (36%)</td>
<td>14 (32%)</td>
<td>1 (2%)</td>
<td>&lt;0.01 (**)</td>
</tr>
<tr>
<td>1983</td>
<td>71</td>
<td>21 (30%)</td>
<td>17 (24%)</td>
<td>8 (11%)</td>
<td>&lt;0.001 (*** )</td>
</tr>
<tr>
<td>TOTAL</td>
<td>171</td>
<td>53 (31%)</td>
<td>50 (29%)</td>
<td>13 (8%)</td>
<td>&lt;0.001 (*** )</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Year</th>
<th>No. of ewes retained in flock</th>
<th>No. of ewes culled due to CVP</th>
<th>No. of ewes culled for other reasons</th>
<th>No. of ewes died/unknown or not applicable</th>
<th>Mantel-Haenszel* Chi² (P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1981</td>
<td>30</td>
<td>24</td>
<td>0</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>1982</td>
<td>21</td>
<td>14</td>
<td>0</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>1983</td>
<td>32</td>
<td>23</td>
<td>0</td>
<td>8</td>
<td>1</td>
</tr>
<tr>
<td>TOTAL</td>
<td>83</td>
<td>61 (74%)</td>
<td>0</td>
<td>18 (22%)</td>
<td>4 (5%)</td>
</tr>
</tbody>
</table>

* with reference to annual normal.

' difference between control total and CVP case total.

Figure**: Table showing the number and proportion of ewes retained, culled for reasons of CVP, culled for other reasons. The statistical significance of the differences annually and overall is shown.
which were pregnant showed no evidence of CVP. The individual histories of twelve cases (C77, C80, C82, C97, C105, C105, C114, C117, C118, C147, C165 and C172) illustrate that recurrence may never follow or may occur at any subsequent pregnancy regardless of being normal in the intervening years. Thus, for example, ewe C42, having first prolapsed in 1981, then completed two successive normal pregnancies thereafter but in her fourth pregnancy showed a typical CVP after which she was culled.

The only case of CVP post-partum which was not definitely related to the pre-partum condition (Ewe C108) proved to be barren in the first subsequent year and then did not show any evidence of CVP at the next two fertile pregnancies.

5.8.2 : Culled ewes :

The number of ewes which were culled from the flocks as a consequence of CVP is shown in Figure 102. Also shown are the numbers of ewes which were culled for other reasons and those which were retained for breeding purposes. A total of 55 ewes were unfortunately lost to follow-up or died.

There was a significant increase in the culling rate in the diseased ewes when compared with the control ewes on an annual basis (p =< 0.01, < 0.05 and < 0.001 for 1981, 1982 and 1983 respectively) and overall (p < 0.001). Although there is a marked significance the nature of this investigation meant that some farmers had retained affected ewes for the purposes of the investigation only, and would ordinarily have culled many if not all the affected ewes. This would have resulted in an even greater significance to the differences between the control and affected sheep. Conversely some flocks were composed almost exclusively of older ewes which would reflect in a normally higher annual culling rate.

There was, however, a marked similarity in the culling percentage and the retained proportion between each year (approximately 30% and 70% respectively in each year). The retention of some of the ewes was regarded as a significant advantage in spite of confusing the culling data.
5.9: RETAINED LAMBS:

5.9.1: Source of lambs:
A total of 16 female progeny of CVP cases were retained by three farmers and were bred normally. The animals were identified at birth in the same way as cases of CVP.

5.9.2: Individual histories of retained lambs:
The individual histories of these animals are shown in Figure 103. Three (19%) developed typical CVP at some stage in the ensuing years. One ewe prolapsed twice in successive years having been affected initially in her first lambing season at 1 year of age. One ewe aged 4 years prolapsed for the first time in her third pregnancy, and one two year old prolapsed in her second pregnancy. There were insufficient cases to allow meaningful statistical analyses but the apparently high rate of CVP might indicate the need for further investigation.

5.9.3: Lambing rate and prognosis:
The overall lambing rate of the retained lambs was 22 lambs delivered to the 16 primiparous ewes of which 12 were one year old (a rate of 1.4 lambs per ewe). The primiparous yearling ewes produced 15 lambs (1.25 lambs per ewe) all of which were alive at 48 hours old. This compares with a rate of 17 lambs born to 13 primiparous one year old control ewes (1.31 lambs per ewe) and 4 primiparous one year old cases of CVP (for which lambing details were known) which produced a total of 5 lambs (a rate of 1.25 lambs per ewe) (Figure 103).
<table>
<thead>
<tr>
<th>Lamb No.</th>
<th>Dam case no.</th>
<th>Year of birth</th>
<th>One year old</th>
<th>Two years old</th>
<th>Three years old</th>
<th>Four years old</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>CVP Carried</td>
<td>Lambs live</td>
<td>CVP Carried</td>
<td>Lambs live</td>
</tr>
<tr>
<td>RB1</td>
<td>18</td>
<td>1981</td>
<td>Barren*</td>
<td>nil 2 2</td>
<td>nil 1 1</td>
<td>+ 2 1</td>
</tr>
<tr>
<td>RB2</td>
<td>18</td>
<td>1981</td>
<td>nil 1 1</td>
<td>nil 2 2</td>
<td>nil 3 3</td>
<td>nil 2 1</td>
</tr>
<tr>
<td>RB3</td>
<td>18</td>
<td>1982</td>
<td>Barren*</td>
<td>nil 1 1</td>
<td>nil 2 0</td>
<td>n/k</td>
</tr>
<tr>
<td>RB4</td>
<td>18</td>
<td>1983</td>
<td>Not bred</td>
<td>nil 1 1</td>
<td>n/k</td>
<td>n/k</td>
</tr>
<tr>
<td>RT1</td>
<td>28</td>
<td>1982</td>
<td>nil 1 1</td>
<td>nil 2 2</td>
<td>nil 2 2</td>
<td>n/k</td>
</tr>
<tr>
<td>RT2</td>
<td>28</td>
<td>1983</td>
<td>nil 1 1</td>
<td>nil 3 3</td>
<td>n/k</td>
<td>n/k</td>
</tr>
<tr>
<td>RR1</td>
<td>110</td>
<td>1982</td>
<td>nil 1 1</td>
<td>nil 3 3</td>
<td>n/k</td>
<td>n/k</td>
</tr>
<tr>
<td>RR2</td>
<td>110</td>
<td>1982</td>
<td>nil 1 1</td>
<td>+ 2 0*</td>
<td>Culled</td>
<td></td>
</tr>
<tr>
<td>RR3</td>
<td>110</td>
<td>1983</td>
<td>nil 2 2</td>
<td>nil 3 3</td>
<td>n/k</td>
<td>n/k</td>
</tr>
<tr>
<td>RR4</td>
<td>110</td>
<td>1984</td>
<td>nil 2 2</td>
<td>n/k</td>
<td>n/k</td>
<td>n/k</td>
</tr>
<tr>
<td>RS1</td>
<td>115</td>
<td>1982</td>
<td>Barren*</td>
<td>nil 1 1*</td>
<td>nil 1 1</td>
<td>n/k</td>
</tr>
<tr>
<td>RR6</td>
<td>118</td>
<td>1982</td>
<td>nil 1 1</td>
<td>nil 1 1</td>
<td>nil 2 2</td>
<td>n/k</td>
</tr>
<tr>
<td>RR7</td>
<td>118</td>
<td>1983</td>
<td>+ 2 0</td>
<td>+ 2 1</td>
<td>Died**</td>
<td></td>
</tr>
<tr>
<td>RR8</td>
<td>118</td>
<td>1983</td>
<td>nil 2 2</td>
<td>nil 3 3</td>
<td>n/k</td>
<td>n/k</td>
</tr>
<tr>
<td>RR9</td>
<td>118</td>
<td>1984</td>
<td>nil 1 1</td>
<td>n/k</td>
<td>n/k</td>
<td>n/k</td>
</tr>
<tr>
<td>RA1</td>
<td>165</td>
<td>1984</td>
<td>nil 2 2</td>
<td>n/k</td>
<td>n/k</td>
<td>n/k</td>
</tr>
</tbody>
</table>

* put to ram.
** Death unrelated to CVP.
* Caesarian Section.

Figure 2.1: Table showing the individual breeding history of each of the retained progeny ewe lambs whose mothers had been affected by CVP. Ewes which suffered from CVP are shown as + .
<table>
<thead>
<tr>
<th>No. of lambs retained</th>
<th>First Lambing</th>
<th>Second Lambing</th>
<th>Third Lambing</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of ewes</td>
<td>No. of CVP cases</td>
<td>No. of lambs carried</td>
</tr>
<tr>
<td>16**</td>
<td>16**</td>
<td>1</td>
<td>22</td>
</tr>
<tr>
<td>Control ewes</td>
<td>13</td>
<td>0</td>
<td>17</td>
</tr>
</tbody>
</table>

** 3 ewe lambs put to ram barren in first year
* total control group

**Figure 1(a)**: Table showing the combined results of successive lambings in retained ewes compared to control ewes.

<table>
<thead>
<tr>
<th>No. of CVP cases</th>
<th>No. of repeat cases</th>
<th>No. of lambs carried</th>
<th>Mean lambs per ewe</th>
<th>No. of live lambs born</th>
<th>Mean live lambs per ewe</th>
<th>No. of cases requiring C.S.</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>1</td>
<td>6</td>
<td>2.0</td>
<td>1.1</td>
<td>0.3</td>
<td>2*</td>
</tr>
</tbody>
</table>

* one Caesarian section performed at first episode of CVP and one at second episode.

**Figure 1(b)**: Table showing the uterine burden, survival and no. of ewes requiring Caesarian Section in cases of CVP seen in retained lambs.
Eighteen of the 22 lambs born to the primiparous progeny group (82%) were alive at 48 hrs old (1.13 live lambs per ewe), 13 of the control lambs were alive (1.0 live lambs per ewe) and only 1 lamb from the 5 CVP cases was alive (0.2 live lambs per ewe). Combining all the pregnancies for the progeny group (33 pregnancies) 58 lambs were born (an overall rate of 1.76 lambs per ewe).

Figure 104 shows an analysis of the progeny group over the period of the study. The lambing rate rose from 1.3 lambs/ewe, as primipara, to 2.3 lambs/ewe at third parity. The latter rate agrees closely with that of the CVP cases referred to earlier.

5.10. : PATHOLOGY MATERIAL :

5.10.1 : Biopsy specimens :

Samples were obtained from 124 cases but two were impossible to interpret due to marked degeneration, possibly due to inadequate formalin. A table showing the relationship of the various changes in the epithelium and stroma to the extent of CVP is shown in Figure 105.

Most cases of CVP (83%) showed some epithelial damage unless the CVP was very mild (first degree). Amongst this latter group 56% showed no apparent pathological change in the epithelial integrity.

Oedema was consistently present (92%) and frequently this was moderate to severe.

An inflammatory response was present in all but two cases and varied from mild to severe.

The milder forms of CVP were characterised by milder responses and from Second degree CVP the more apparent was the inflammatory response.

Significant haemorrhage into the tissues (Figure 57) was seen in 16 cases (13%).

5.10.2 : Post-mortem examinations :
<table>
<thead>
<tr>
<th>Tissue</th>
<th>Epithelium</th>
<th>Stroma</th>
<th>Inflammation</th>
<th>Haemorrhage</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Degree</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>18</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>3</td>
<td>53</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>3</td>
<td>32</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>0</td>
<td>19</td>
<td>0</td>
</tr>
<tr>
<td>TOTAL</td>
<td>16</td>
<td>11</td>
<td>122</td>
<td>7</td>
</tr>
</tbody>
</table>

Significance *** ns ***

* missing on biopsy specimen

Figure 105: Table showing the major pathological features present in the biopsy samples obtained from the external cervical os. The number and percentage (in brackets) of ewes affected by each prolapse type in which the changes were seen are shown. The statistical significance of the differences between the degrees of prolapse is shown for each pathological parameter.
Post-mortem examinations were performed on 13 ewes which were either found dead or euthanased immediately, of which 3 were in 1981, 3 in 1982 and 7 in 1983. All these cases were of the vaginal rupture syndrome and were subjected to post mortem examination. Ewes which died between treatment and lambing (16 cases) were also examined. The major purpose of this was to determine the number of lambs in utero and to examine the pelvic structures, including the bladder, for abnormality. Data relating to the uterine burden of these ewes has already been shown.

The cases of vaginal rupture showed that the tear was invariably situated in the dorsal vaginal wall and was longtitudinal (varying from 8 - 15 cms in length) but it was difficult to establish any pattern as the bruising and haemorrhage was extensive. Vaginal contamination was noted in 4 cases out of 13 (31%) where there had been no history of CVP.

In all the post-mortem examinations in pre-parturient ewes affected with either the vaginal rupture syndrome or CVP alone, intrapelvic haemorrhage was noted and was particularly prominent in the vesico-genital pouch of the peritoneum. There were lesser haemorrhages under the peritoneum on the abdominal wall. In only 4 cases was the bladder found to be severely distended, although in 23 cases (out of 29) the bladder was thought to be out of position being reflected back into the pelvic cavity. Haematuria and/or haemoglobulinuria were found in 9 cases. The bladder wall in all of these was haemorrhagic. In no case, however, was any gross pathology evident in the kidneys or ureters.

Post-mortem examination was somewhat cursory and little other data was obtained. In the case of 5 ewes which were found in extremis (all with the ruptured vagina syndrome) and euthanased immediately an instant laparotomy was performed in an attempt to obtain live lambs. Only one produced a live viable lamb out of a total number of 11 lambs in utero (a mortality of 91%).

No ewe with CVP was found dead without vaginal rupture.
5.11 : CLIMATE :

The weather conditions in each of the survey years was obtained from Her Majesty's Meteorological Office and is summarised in Figure 106. The mean monthly temperature and the temperature range are shown. The number of wet days and the number of snow days as defined by the Meteorological Office are shown for each of the survey years. No effect of climate on the incidence or extent of CVP was immediately apparent but analysis was not detailed and the conclusion is unreliable.
<table>
<thead>
<tr>
<th>Year</th>
<th>January</th>
<th>February</th>
<th>March</th>
<th>April</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean daily temp. (range)</td>
<td>Rain(1) days</td>
<td>Snow(a) days</td>
<td>Mean daily temp. (range)</td>
</tr>
<tr>
<td>1981</td>
<td>4.9°C (-2.7/12.2)</td>
<td>16</td>
<td>0</td>
<td>2.9°C (-5/13)</td>
</tr>
<tr>
<td>1982</td>
<td>2.0°C (-8.2/12.8)</td>
<td>14</td>
<td>10</td>
<td>5.1°C (-4.5/14)</td>
</tr>
<tr>
<td>1983</td>
<td>6.7°C (-3.5/13.5)</td>
<td>15</td>
<td>0</td>
<td>1.5°C (-6.8/10.9)</td>
</tr>
</tbody>
</table>

\(1\) Rain day — a period of 24 hours in which 0.2mm or more of rain fell.
\(a\) Snow day — a day when over half of the ground is covered with snow at 0900 GMT.

**Figure 106**: Table of abbreviated weather conditions in 1981, 1982, 1983 from the Wellesbourne Weather Station.
5.12: HAEMATOLOGY:

5.12.1: Haemogram:

The mean results for the erythrocyte parameters including haemoglobin (Hb), erythrocyte count (RBC), haematocrit (PCV) and the derived Mean Cell Volume (MCV), and Mean Cell Haemoglobin Concentration (MCHC) are shown in Figure 107 for the overall CVP cases and are compared to the overall results from the control ewes. The statistical analysis of these results is shown. Cases of CVP were not, apparently, affected significantly as far as the erythroid parameters were concerned.

5.12.2: Leucogram:

There was a marked and significant increase in the leucocyte count of affected ewes with a prominent shift to the left (Figure 108). The change in leucocyte count is almost entirely explicable by an increase in the neutrophil count (Figure 108). The median values, ranges and statistical significance of the variations from the control ewes are shown. There was a significant increase in the total leucocyte count as the extent of CVP became more severe ($p = 0.03$). There was also a significant variation from year to year but an explanation for this could be not found.

5.13: BIOCHEMISTRY:

5.13.1: Metabolic Parameters:
<table>
<thead>
<tr>
<th>Year</th>
<th>Ewes</th>
<th>Haemoglobin (g/l)</th>
<th>Total erythrocyte count ($\times 10^{12}$/l)</th>
<th>Haematocrit (l/l)</th>
<th>Mean cell volume ($\mu^3$)</th>
<th>Mean cell haemoglobin concentration (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CVP cases (n=64)</td>
<td>12.1 (1.5)</td>
<td>8.7 (1.3)</td>
<td>36.0 (4.1)</td>
<td>39.1 (5.3)</td>
<td>36.1 (2.2)</td>
</tr>
<tr>
<td></td>
<td>Control ewes (n=30)</td>
<td>11.6 (1.1)</td>
<td>9.2 (1.3)</td>
<td>35.8 (3.2)</td>
<td>37.5 (3.9)</td>
<td>34.4 (2.2)</td>
</tr>
<tr>
<td>1981</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>CVP cases (n=52)</td>
<td>11.8 (1.1)</td>
<td>9.2 (1.1)</td>
<td>36.2 (4.9)</td>
<td>37.0 (3.1)</td>
<td>34.0 (3.3)</td>
</tr>
<tr>
<td></td>
<td>Control ewes (n=21)</td>
<td>10.2 (0.9)</td>
<td>8.3 (0.6)</td>
<td>31.1 (4.1)</td>
<td>38.8 (2.3)</td>
<td>32.4 (1.8)</td>
</tr>
<tr>
<td>1982</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>CVP cases (n=72)</td>
<td>10.1 (1.8)</td>
<td>8.2 (1.9)</td>
<td>31.9 (3.2)</td>
<td>39.1 (6.3)</td>
<td>32.1 (2.3)</td>
</tr>
<tr>
<td></td>
<td>Control ewes (n=32)</td>
<td>10.8 (1.8)</td>
<td>9.0 (1.1)</td>
<td>32.9 (2.7)</td>
<td>36.8 (3.0)</td>
<td>32.1 (2.3)</td>
</tr>
<tr>
<td>1983</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>CVP cases (n=188)</td>
<td>11.2 (1.6)</td>
<td>8.6 (1.4)</td>
<td>34.6 (3.9)</td>
<td>38.7 (4.2)</td>
<td>34.1 (3.0)</td>
</tr>
<tr>
<td></td>
<td>Control ewes (n=83)</td>
<td>10.9 (1.3)</td>
<td>8.9 (1.1)</td>
<td>33.2 (3.1)</td>
<td>37.6 (3.1)</td>
<td>33.9 (2.2)</td>
</tr>
</tbody>
</table>

**Figure 107:** Table showing the annual and overall mean (and standard deviation) of the erythrocyte parameters (haemoglobin, erythrocyte count, haematocrit, mean cell volume and mean cell haemoglobin concentration) for affected ewes and control ewes. The statistical significance of the differences between the two groups is shown for each year and overall.
<table>
<thead>
<tr>
<th>Year</th>
<th>Ewes</th>
<th>Mean leukocyte count (×10^9/l) (SD)</th>
<th>Median differential leukocyte count</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Neutrophils (×10^9/l) (SD)</td>
<td>Lymphocytes (×10^9/l) (SD)</td>
</tr>
<tr>
<td>1981</td>
<td>CVP cases (n=64)</td>
<td>9.02 (2.62)</td>
<td>5.61 (1.42)</td>
</tr>
<tr>
<td></td>
<td>Control ewes (n=30)</td>
<td>7.20 (1.42)</td>
<td>3.19 (0.94)</td>
</tr>
<tr>
<td>1982</td>
<td>CVP cases (n=52)</td>
<td>8.50 (2.35)</td>
<td>6.21 (1.68)</td>
</tr>
<tr>
<td></td>
<td>Control ewes (n=21)</td>
<td>7.03 (1.79)</td>
<td>4.66 (1.30)</td>
</tr>
<tr>
<td>1983</td>
<td>CVP cases (n=72)</td>
<td>7.98 (2.42)</td>
<td>6.70 (2.30)</td>
</tr>
<tr>
<td></td>
<td>Control ewes (n=32)</td>
<td>6.11 (1.55)</td>
<td>4.25 (1.70)</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>8.48 (2.49)</td>
<td>6.41 (1.96)</td>
</tr>
<tr>
<td></td>
<td>Control ewes (n=83)</td>
<td>6.74 (1.63)</td>
<td>4.20 (1.67)</td>
</tr>
</tbody>
</table>

**Figure 108:** Table showing the annual and total mean (and standard deviation) of the leukogram of affected ewes and control ewes. The statistical significance of the annual differences and overall are shown.
<table>
<thead>
<tr>
<th>Year</th>
<th>Ewes</th>
<th>Total serum protein (g/l)(SD)</th>
<th>Serum albumin (g/l)(SD)</th>
<th>Serum globulin (g/l)(SD)</th>
<th>Serum β hydroxybutyrate (mmol/l)(SD)</th>
<th>Serum urea (mmol/l)(SD)</th>
<th>Plasma glucose (mmol/l) (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1981</td>
<td>CVP cases</td>
<td>69.7 (7.8)</td>
<td>30.2 (4.1)</td>
<td>39.1 (7.1)</td>
<td>0.66 (0.16)</td>
<td>7.79 (5.11)</td>
<td>2.68 (0.83)</td>
</tr>
<tr>
<td></td>
<td>n=64</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control ewes</td>
<td>65.9 (6.8)</td>
<td>32.2 (3.9)</td>
<td>33.7 (8.3)</td>
<td>0.44 (0.11)</td>
<td>5.12 (1.57)</td>
<td>2.57 (0.46)</td>
</tr>
<tr>
<td>1982</td>
<td>CVP cases</td>
<td>62.3 (8.6)</td>
<td>32.6 (4.4)</td>
<td>29.7 (8.0)</td>
<td>0.63 (0.17)</td>
<td>7.60 (6.40)</td>
<td>2.85 (1.00)</td>
</tr>
<tr>
<td></td>
<td>n=53</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control ewes</td>
<td>63.4 (4.1)</td>
<td>33.1 (3.4)</td>
<td>30.3 (5.0)</td>
<td>0.42 (0.09)</td>
<td>3.69 (0.96)</td>
<td>2.99 (0.57)</td>
</tr>
<tr>
<td>1983</td>
<td>CVP cases</td>
<td>59.0 (8.4)</td>
<td>29.3 (5.4)</td>
<td>29.6 (7.2)</td>
<td>0.59 (0.16)</td>
<td>6.75 (3.63)</td>
<td>2.69 (0.63)</td>
</tr>
<tr>
<td></td>
<td>n=72</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control ewes</td>
<td>61.7 (7.0)</td>
<td>30.3 (5.6)</td>
<td>31.4 (5.7)</td>
<td>0.40 (0.13)</td>
<td>4.48 (1.22)</td>
<td>3.10 (0.46)</td>
</tr>
<tr>
<td>1983</td>
<td>CVP cases</td>
<td>63.5 (9.4)</td>
<td>30.5 (4.9)</td>
<td>32.8 (8.7)</td>
<td>0.63 (0.16)</td>
<td>7.63 (6.38)</td>
<td>2.77 (0.99)</td>
</tr>
<tr>
<td></td>
<td>n=187</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control ewes</td>
<td>63.7 (6.5)</td>
<td>31.7 (4.7)</td>
<td>31.9 (6.7)</td>
<td>0.42 (0.11)</td>
<td>4.51 (1.40)</td>
<td>2.88 (0.54)</td>
</tr>
</tbody>
</table>

**Figure 109:** Mean annual and overall values for total protein, albumin, hydroxybutyrate, urea and glucose, and the standard deviation (in brackets) for control and diseased ewes. The statistical significance of the annual and overall differences is shown.
<table>
<thead>
<tr>
<th></th>
<th>Total protein</th>
<th>Albumin</th>
<th>β Hydroxybutyrate</th>
<th>Urea</th>
<th>Glucose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Degree of prolapse</td>
<td>***</td>
<td>***</td>
<td>***</td>
<td>***</td>
<td>**</td>
</tr>
<tr>
<td>Farm type</td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
</tr>
<tr>
<td>Year</td>
<td>***</td>
<td>***</td>
<td>*</td>
<td>ns</td>
<td>ns</td>
</tr>
</tbody>
</table>

Figure 10: Probability – significance (Wilcoxon 2 sample tests) for biochemical (metabolic) parameters by degree of prolapse, farm type and year.
<table>
<thead>
<tr>
<th></th>
<th>Degree of CVP</th>
<th>Control ewes</th>
<th>Significance of difference between degree of prolapse</th>
<th>Significance of difference between control ewes and cases overall</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1°</td>
<td>2°</td>
<td>3°</td>
<td>4°</td>
</tr>
<tr>
<td>Mean glucose</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(mmol /l) (SD)</td>
<td>2.49</td>
<td>2.71</td>
<td>2.74</td>
<td>3.05</td>
</tr>
<tr>
<td></td>
<td>(0.60)</td>
<td>(0.61)</td>
<td>(0.92)</td>
<td>(1.17)</td>
</tr>
<tr>
<td>Mean urea</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(mmol /l) (SD)</td>
<td>6.19†</td>
<td>5.91†</td>
<td>8.89†</td>
<td>8.83†</td>
</tr>
<tr>
<td></td>
<td>(2.27)</td>
<td>(2.33)</td>
<td>(5.10)</td>
<td>(3.14)</td>
</tr>
<tr>
<td>Mean β hydroxybutyrate</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(mmol /l) (SD)</td>
<td>0.61</td>
<td>0.58</td>
<td>0.66</td>
<td>0.60</td>
</tr>
<tr>
<td></td>
<td>(0.16)</td>
<td>(0.17)</td>
<td>(0.13)</td>
<td>(0.18)</td>
</tr>
</tbody>
</table>

* Significant difference between the mean of 1°+2° and 3°+4° CVP cases but no difference between 1° and 2° and between 3° and 4° individually.

Figure III: The mean values for glucose, urea and β hydroxybutyrate for each prolapse degree (and standard deviation in brackets). The significance of the differences between the degrees and between control and affected ewes overall are shown.
These included serum glucose (Glu), urea, hydroxybutyrate (BHBA), total protein (TP) and albumin (Alb) levels. The mean values and the standard deviations of these indices for the total population of affected sheep and the controls are shown in Figure 109. The statistical significance of the differences between the control and affected ewes is also shown.

The significance of the variations in the metabolic parameters between different degrees of CYP are shown in Figure 110. This also shows the significance of farm type and year on each of the parameters.

Figure 111 shows the variations with degree of CVP in more detail without regard for the annual variations which were not statistically significant (p = 0.12 for glucose, p = 0.17 for urea, p = 0.13 for BHBA, p = 0.10 for total protein and p = 0.15 for albumin). In spite of the marginally significant annual differences in the levels of glucose, BHBA, total protein and albumin it was felt justified for simplicity to pool all the results from each year and analyse them as a single unit.

The most noticeable variation from the normal was seen in urea levels where the diseased ewes showed a mean value of 7.63 mmoles/l (SD = 6.38) while the normal ewes showed a mean value of 4.51 mmoles/l and a SD of only 1.40 mmoles/l. The difference between the two groups is highly significant (p = 0.00). The overall and the annual results showed the same order of significance. Of 5 ewes with urea levels over 20 mmoles/l only one survived (a mortality of 80%).

A significant elevation in the BHBA in the affected ewes was found (p = 0.00) when compared to the control ewes. The mean overall value for the BHBA was found to be 0.63 mmoles/l (SD = 0.16) compared to the control ewes value of 0.42 mmoles/l (SD = 0.11).

There were marginal or insignificant differences in the glucose total protein, albumin, and globulin levels between the diseased and control ewes.

The variations noted in the values as a consequence of differing degrees of CVP are shown in Figure 111 which also shows the statistical significance of these differences.
## Table 11: Mean annual and overall values (and standard deviations, in brackets) for mineral and electrolyte parameters in affected and control ewes. The significance of the annual and overall differences between cases and control ewes is shown.

<table>
<thead>
<tr>
<th>Year</th>
<th>Copper (mmol/l)(SD)</th>
<th>Selenium (μmol/l)(SD)</th>
<th>Magnesium (mmol/l)(SD)</th>
<th>Inorganic Phosphate (mmol/l)(SD)</th>
<th>Calcium (mmol/l)(SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1981</td>
<td>15.16 (4.79)</td>
<td>0.088 (0.034)</td>
<td>1.13 (0.21)</td>
<td>4.49 (1.71)</td>
<td>2.18 (0.21)</td>
</tr>
<tr>
<td></td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>1982</td>
<td>2.31 (0.29)</td>
<td>0.082 (0.026)</td>
<td>1.09 (0.18)</td>
<td>3.55 (1.09)</td>
<td>2.31 (0.29)</td>
</tr>
<tr>
<td></td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>1983</td>
<td>4.07 (2.33)</td>
<td>0.079 (0.029)</td>
<td>1.12 (0.22)</td>
<td>4.07 (2.33)</td>
<td>2.15 (0.73)</td>
</tr>
<tr>
<td></td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Total</td>
<td>2.12 (0.44)</td>
<td>0.077 (0.032)</td>
<td>1.13 (0.21)</td>
<td>4.07 (2.33)</td>
<td>2.04 (0.27)</td>
</tr>
<tr>
<td></td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
</tr>
</tbody>
</table>

Legend:
- *: Reduced number of samples - none in 1983.
There was a variation in both glucose and urea between the various degrees of prolapse which indicated that the more severe forms were accompanied by a rise in blood urea which tends to be exaggerated in 3rd and 4th degree cases, and a progressively rising blood glucose level as the severity of CVP increases.

There was no apparent difference in the levels of BHBA between the various degrees of prolapse (p = 0.82). The variation in these parameters seen in the ewes on different farm types indicated that the farm type is insignificant (Figure 110).

Thus in the more severe forms of CVP, the glucose and the urea rose significantly, while the total protein and albumin fell slightly. The concentration of BHBA showed an increase when first and second degree CVP cases were considered together and compared with third and fourth degree CVP combined.

A difference was noted in the BHBA, the total protein and the albumin between the individual years (p = 0.046, and 0.05 respectively) but no explanation for this was apparent.

5.13.2: Electrolyte parameters:

The serum electrolyte levels include calcium (Ca\(^{2+}\)) inorganic phosphate (PO\(_4\)^{3-}\) and magnesium (Mg\(^{2+}\)). Figure 112 shows the overall means and the standard deviation of each parameter for both the affected and control groups.

Statistical analysis reveals that there is no annual difference between ewes affected by CVP over the three years (p = 0.27 for Ca\(^{2+}\), p = 0.16 for PO\(_4\)^{3-}\) and p = 0.56 for Mg\(^{2+}\)) and it is therefore justified to use the total population as a single comparative group which may then be compared effectively with the control group which, likewise, can be combined.

The Ca\(^{2+}\) and PO\(_4\)^{3-}\) levels did not vary significantly between the different farm types in the diseased ewes (p = 0.44, p = 0.43 respectively).
<table>
<thead>
<tr>
<th></th>
<th>Degree of CVP</th>
<th>Significance of difference between CVP degrees</th>
<th>Mean for control ewes</th>
<th>Significance of difference between CVP cases and controls</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1° 2° 3° 4°</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean calcium (mmol l⁻¹) (SD)</td>
<td>2.24 (0.63) 2.15 (0.14) 2.03 (0.27) 1.91 (0.26)</td>
<td>***</td>
<td>2.31 (0.29)</td>
<td>***</td>
</tr>
<tr>
<td>Mean inorganic phosphate (mmol l⁻¹) (SD)</td>
<td>4.36 (1.41) 4.23 (1.96) 3.59 (1.74) 3.47 (2.99)</td>
<td>**</td>
<td>4.64 (1.22)</td>
<td>***</td>
</tr>
<tr>
<td>Mean magnesium (mmol l⁻¹) (SD)</td>
<td>1.15 (0.18) 1.10 (0.20) 1.14 (0.21) 1.15 (0.20)</td>
<td>ns</td>
<td>1.13 (0.21)</td>
<td>ns</td>
</tr>
</tbody>
</table>

Figure 1: Mean values (and standard deviation in brackets) for calcium, inorganic phosphate, and magnesium for each prolapse degree and for the control ewes overall. The significance of the differences between prolapse degrees and the overall significance between diseased and control ewes for each parameter is shown.
<table>
<thead>
<tr>
<th>Year</th>
<th>Ewes</th>
<th>Progesterone (ng/ml)</th>
<th>Oestrogen (ng/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Median (Range)</td>
<td>Median (Range)</td>
<td></td>
</tr>
<tr>
<td>1981</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CVP</td>
<td>n=59</td>
<td>6.6 (0.6-20)</td>
<td>120 (30-860)</td>
</tr>
<tr>
<td></td>
<td>**</td>
<td>***</td>
<td>***</td>
</tr>
<tr>
<td>Control</td>
<td>n=30</td>
<td>3.75 (0.5-9.3)</td>
<td>290 (140-900)</td>
</tr>
<tr>
<td>1982</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CVP</td>
<td>n=51</td>
<td>12.0 (0.6-20)</td>
<td>170 (70-610)</td>
</tr>
<tr>
<td></td>
<td>**</td>
<td>***</td>
<td>***</td>
</tr>
<tr>
<td>Control</td>
<td>n=21</td>
<td>3.20 (0.9-8.3)</td>
<td>320 (120-900)</td>
</tr>
<tr>
<td>1983</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CVP</td>
<td>n=71</td>
<td>5.25 (0.3-20)</td>
<td>325 (60-900)</td>
</tr>
<tr>
<td></td>
<td>***</td>
<td>ns</td>
<td>ns</td>
</tr>
<tr>
<td>Control</td>
<td>n=32</td>
<td>3.90 (0.9.0)</td>
<td>360 (49-950)</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CVP</td>
<td>n = 181</td>
<td>6.75 (0.3-20)</td>
<td>180 (30-950)</td>
</tr>
<tr>
<td></td>
<td>***</td>
<td>***</td>
<td>***</td>
</tr>
<tr>
<td>Control</td>
<td>n=83</td>
<td>3.80 (0.48-8.7)</td>
<td>320 (49-950)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Significance of annual differences</th>
<th>Cases of CVP</th>
<th>Cases of CVP</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>control ewes</td>
<td>control ewes</td>
</tr>
<tr>
<td></td>
<td>ns</td>
<td>ns</td>
</tr>
</tbody>
</table>

Figure 4: Median values (and range in brackets) for progesterone and oestrogen for control and affected ewes in each year and overall. The significance of the annual differences, the differences between the years and the overall differences is shown.
<table>
<thead>
<tr>
<th>Degree of CVP</th>
<th>Significance of difference between degree of CVP</th>
<th>Significance of difference between control ewes and CVP cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1°</td>
<td>7.21 (4.54)</td>
<td>•</td>
</tr>
<tr>
<td>2°</td>
<td>9.00 (5.30)</td>
<td>4.20 (1.98)</td>
</tr>
<tr>
<td>3°</td>
<td>8.86 (5.20)</td>
<td>•</td>
</tr>
<tr>
<td>4°</td>
<td>6.78 (6.29)</td>
<td>•</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Mean plasma progesterone (ng/ml) (SD)</th>
<th>Mean plasma oestrogen (pg/ml) (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>187 (142)</td>
<td>358 (162)</td>
</tr>
<tr>
<td>242 (179)</td>
<td></td>
</tr>
<tr>
<td>293 (199)</td>
<td></td>
</tr>
<tr>
<td>286 (163)</td>
<td></td>
</tr>
</tbody>
</table>

Figure 15a: Table showing the mean values (and standard deviation in brackets) of plasma progesterone and oestrogen in ewes affected by different degrees of CVP. The significance of the annual differences and of the overall differences is shown.

Figure 15b: Distribution of mean hormone levels in ewes affected by different types of CVP.
The effect of the degree of prolapse on the $\text{Ca}^{2+}$, $\text{PO}_4^{4-}$ and $\text{Mg}^{2+}$ levels is shown in Figure 113.

The degree of CVP had a marked effect on the $\text{Ca}^{2+}$ level ($p = 0.0001$). There was a progressive reduction in the mean $\text{Ca}^{2+}$ from first degree prolapses to the more severe forms. A parallel effect was also noted in the $\text{PO}_4^{4-}$ values and in this case the variation was also significant ($p = 0.006$). The more severe forms of CVP are, therefore, apparently accompanied by a progressive reduction in serum calcium and inorganic phosphate.

There was no difference in the magnesium levels between ewes affected by different degrees of CVP ($p = 0.31$).

5.13.3: Trace element parameters:

Those included in this study were copper and selenium. The values obtained are shown in Figure 112 with the statistical analysis giving the mean and the standard deviations. The statistical significance of these compared to the control ewes is shown.

5.13.4: Hormone levels:

The median value, the range and the statistical difference between the affected and control ewes in each year group and overall is shown in Figure 114. The range of values for both progesterone and oestrogen was high in both affected and control ewes but statistically the affected animals (without regard for the degree of CVP) showed an elevated progesterone and a reduced oestrogen when compared to the control ewes.

The degree of CVP was a significant factor in the progesterone and oestrogen levels (Figure 115a and b) ($p = 0.001$ in both cases). More severely affected ewes showed a relative elevation in the levels of oestrogen while, in ewes affected by milder degrees of CVP, the progesterone was relatively raised.

Farm type did not exert any detectable effect upon the hormone levels ($p = 0.38$ for progesterone and 0.56 for oestrogen).
6.0: DISCUSSION:
6.1. THE FARMS AND MANAGEMENT SYSTEMS:

During the course of the three year investigation several farms were unable to continue full participation in the project and some cases were lost as a consequence but this problem was accepted as almost inevitable from general practice. This influenced the distribution of the cases that were evaluated and reduced the validity of statistical analyses that could be performed on some of the farm data.

All the flocks involved in this investigation were well managed and were involved with a limited flock health programme which was run from the practice. Without the cooperation and help of the farmers the work could not have taken place or would at the very least have been very much more limited in its scope.

All the animals were adequately wormed and vaccinated, and the feeding regimen was predetermined using the resources at the disposal of the farmer. Although feeding methods varied widely, all the flocks were adequately nourished. The only standardisation of the feeding was in the time of the start of the supplementary feeding which was from about the 90th day of gestation.

The supplementation of the diet with minerals, particularly with calcium and phosphate, did not appear to be beneficial although the empirical administration of minerals made it difficult to assess possible responses. Chavance (1944) proposed that the disorder was both treatable and preventable by the use of calcium and vitamin D supplementation although in this investigation this aspect was not explored.

During the study several significant findings were made with regard to the mineral and nutritional status of the affected ewes but it was not practicable to withdraw mineral supplementation from particular farms. Several farmers altered the mineral
supplementation method, and while this appeared to make little or no difference to the incidence on the farms concerned, no really satisfactory conclusions could be drawn. It may represent an aspect of CVP which could be further researched especially as Chavance (1944) suggested a strong relationship to the calcium status.

The status of flocks in the study in respect of magnesium, copper and selenium revealed that there were no significant differences between the incidence on those farms in which monitoring and supplementation was, or was not, practised.

The problem of carrying out such an investigation in general practice is clearly illustrated by this. Obviously, had any of the fourteen farms routinely tested for the copper status of pregnant ewes been found deficient, treatment would have been instituted. Generally those farms with a history of deficiency were supplemented accordingly. In any case the results suggested that the status of these minerals was of no material significance in the disorder. Likewise, the presence of intercurrent disease on the farm was not found to be a significant factor in the development of CVP. Few ewes were found to be affected by other diseases concurrently although, interestingly, five of the control ewes developed ovine ketosis (Twin-lamb disease). No flock was affected during the period of study with any form of epidemic disease which apparently altered the number or type of case seen.

6.2: INCIDENCE:

Bayly et al. (1936) found the incidence of CVP to vary (between 1% and 8%) between flocks and between different seasons in the same flock. Laing (1945) reported that the average incidence in some areas of New Zealand was about 4% with the highest about 11%. Edgar (1952), in the United Kingdom, determined that the overall incidence was 0.53% of the lambing ewes. In contrast to Edgar's (1952) work, the results of the present study indicate that the average annual incidence may be between 1.0% and 1.6%. This may indicate that the incidence in the area of the United Kingdom in which this work was carried out might have been inordinately high or, as is possibly more likely, that this study
would produce more accurate results than those which involved postal surveys. Earlier, milder forms of CVP were recognised and examined promptly in the present work and this represents a considerable improvement on previous work on the estimation of the incidence. It is likely that Edgar (1952) and McLean (1956), in carrying out their surveys on the incidence of CVP in the United Kingdom and New Zealand respectively, might not have included those cases where the CVP was either intermittent/mild or had been so severe that the animal died before CVP had been identified. The results of the present investigation may therefore provide a more accurate figure for the incidence of CVP in this area of the United Kingdom.

The overall breeding ewe population of the United Kingdom in 1981, 1982 and 1983 was 14.8 million, 15.7 million and 16.1 million respectively (Ministry of Agriculture Livestock Census Data, 1984). The number of cases of CVP occurring annually in the United Kingdom could be as high as 250,000 in an average year. This represents a serious clinical disorder which almost certainly results in a significant loss to the sheep industry. This is obviously only a rough estimate but the disorder is reported from all areas of the United Kingdom and while there may be local variations in the numbers of ewes affected, the overall number may be used as an indication of its possible clinical significance.

The effect of the farm type on the incidence of CVP was apparently minimal but it was noticeable that mild cases (first degree) were more frequently reported in totally confined ewes. This may reflect the better observation normally expected on these farms. Where no housing was provided and the ewes were grazing on flat ground, there appeared to be a more even distribution of the various degrees of CVP. The reasons for this were not clear and statistically it was difficult to demonstrate any significant difference between the farm types in respect of the severity of CVP. Nevertheless, Edgar (1952) found one flock which showed a very significant annual variation ($p < 0.1$), but overall his results indicated that there was no significant variation from year to year. The present work suggests that there is a difference in the incidence in different years on
particular farms. There were annual variations in the number of cases seen on particular farms, although the breed of ewe, the feeding and the management was very similar from year to year. McLean's (1956) proposal that CVP was an inevitable occurrence and a natural accident of pregnancy in flocks of sheep was not confirmed as there were flocks in which the incidence varied markedly. Thus, for example, Farm 9 had only one case in the first season (0.7%), while in the next two seasons the incidence was 0% and 3.6% respectively.

Many of the farms experienced one or two years in which no cases were seen, but no flock was free of CVP for all three years of the study. The maximum incidence encountered in this study was 9.1% (excluding the pet flock of three ewes in which CVP occurred in two ewes in the third year). This was in a small flock of mixed breeds where the sheep were grazing very steep hill pastures and this would seem to agree with McLean (1957) and McLean and Claxton (1959) that the incidence is highest on hill farms. However, there was a significant number of cases in one of the housed flocks (Farm 5) which had started farming sheep in the 1982 season, and had bought all the ewes some 7 months previously. The entire flock of Scotch half-bred ewes was aged 2 years at the time of lambing and all were primiparous in the 1982 season. The incidence on Farm 5 rose from 2.8% in the first year (primiparous ewes) to 4.3% in 1983 (ewes lambing for the second time). The feeding and management of the ewes on Farm 5 was apparently the same during both years and it is, therefore, apparent that variations may be found without any obvious reason. This unexplained variation is one of the factors which have frustrated previous workers including Watson (1959) and Hartigan (1961).

The classification of the farms into the different types was made to test whether there was any significant effect exerted by the topography, or the muscular fitness of the ewes on the incidence of CVP, as was suggested by McLean (1957, 1959). Although McLean (1957) suggested that the long-term history of the ewes was important some farms were both flat and hilly in parts, it was thought reasonable to consider only the last 6 weeks of
gestation. This was done because on all the participating farms the ewes remained in the same environment from this point onward. It is accepted that the possible muscular stretching which might arise when a ewe is in an uphill position, either recumbent or standing (McLean, 1957), may indeed be significant at an earlier stage of pregnancy, but this was impossible to quantify or regulate under commercial conditions.

Comparisons were made between farms where ewes received little or no exercise during the last 6 weeks of pregnancy (housed flocks in confined yards) and those in which exercise was either freely available or obligatory, to test the theory proposed by Bayly et al. (1936), Fethers (1939), Laing (1945, 1949) and Belschner (1976), that exercise and muscular fitness were important factors in the aetiology of CVP. Laing (1945) suggested that fat, lazy and unexercised ewes were more likely to be affected than others. Indeed, Bayly et al. (1936), Laing (1945, 1949) and Belschner (1976) have suggested that forced exercising of "forward-in-lamb" ewes might prevent the disorder. In this study there were only 3 farms (2 in the first year of the survey) where the ewes were totally confined in enclosed yards with limited scope for exercise. The results obtained from these farms showed that there was no overall difference in the proportion or severity of cases when compared to those farms where exercise was available indicating that the provision of exercising facilities may not be an important factor in the prevention or control of CVP.

The possible curative effect of exercise on cases of CVP was not tested. The non-exercised flocks, however, showed a wide annual variation (0.65% - 2.3%) compared to the variation on the other farms (0.9% - 1.5%). The significance of this is not clear from this work.

Although there was no significant difference between the incidence amongst housed ewes (including those confined and those where shelter was available on a voluntary basis) and those maintained outside regardless of the weather, a higher proportion of cases did appear to occur in the housed animals (1.7% compared to 1.2% in the non-housed animals). The provision of housing did not appear to affect the degree to which a ewe
might prolapse even though the hill flocks were generally less closely observed, and the prolapses were often discovered and examined later than those in closely confined animals. These aspects may require further investigation with greater numbers of ewes in each category.

Edgar (1952) found that, over two years, 21% of cases were seen in January and 21% in February while 36% were seen in March and 20% were seen in April. His survey indicated an almost equal monthly proportion of the ewes were affected by CVP (1% in January and 0.5% in each of the next three months). The greatest frequency of cases of CVP in the present work coincided with the period of maximum lambing activity with 80% of cases seen in February and March. It is possible to conclude, like Edgar (1952), that the condition occurs at all stages of the lambing season. Fewer cases may be expected when fewer ewes were lambing which does not entirely agree with Edgar's (1952) survey in which the highest incidence occurred in January but the results of a postal survey may be misleading because some cases may have been missed or ignored when lambing activity was at its height.

The extent of the lambing season varied between farm types. The upland farms tended to lamb later than the housed farms and have longer lambing seasons. This meant that cases of CVP were more concentrated in the housed than in the hill flocks. Amongst the farms surveyed, few ewes lambed later than the end of April although amongst the general sheep population in the area there were lambs born throughout May and occasionally in June. CVP is not, apparently, a disease which is affected directly by the climatic conditions. Minor variations in the prevailing climate over the three-year period made no material difference to the proportion of cases occurring in a particular month.

6.3: THE EWES:
Before considering the detailed results of the affected ewes, it is pertinent to consider the way in which the control ewes were selected. Normal healthy ewes were selected to coincide with approximately every third case. The selection of control ewes was carried out so that they should be as near as possible to the same bodily condition, age, parity and gestation as the affected animals. Ideally, it would have been better to pair each case with a normal ewe of the same type, age, body condition and gestation but due primarily to the financial and time constraints, this was not possible. Unfortunately the age distribution and body condition scores of the two groups was ultimately different. This is perhaps a hazard of inadequate selection of the control ewes, which should have been derived either to coincide with every case, or an attempt to balance the ages and body conditions of the two groups should have been made. More control ewes should have been selected to correspond with the slightly older ewes in better condition from the CVP group. One ewe in the control group was ultimately found to be barren leaving 82 pregnant ewes in the control group. This control group does not necessarily correspond in every respect to the overall group of CVP cases. Notwithstanding these difficulties, comparisons have been made between the two groups. Indeed, since no other research has been reported in which extensive comparisons have been made between a group of CVP cases and a group of apparently normal ewes, it was considered to be a justifiable exercise. The control ewes were not followed over subsequent seasons and it is not known whether any of them eventually developed CVP. However, no identified control ewe was subsequently treated as a case of CVP during the study period.

A statistically significant variation between breeds was found by Edgar (1952); in particular he found the Romney Marsh breed was most likely to be affected. This agreed with the earlier impression of Bayly et al. (1936) that certain of the broader pelvis types of ewes were more often affected. The breed distribution within the flocks proved difficult to determine accurately in the present work, as farmers were generally uncertain of the breed proportions and could only provide a rough estimate. However the breed of all the
affected ewes was recorded and while the majority of cases (35%) were Mules, this breed was estimated to comprise about 41% of the total population. The Suffolk (and its crosses), the Border Leicester (and crosses), the Kerry, Llyn and the Texel (and their crosses) appear to be over-represented in the population of ewes affected with CVP. These breeds are considered to be those with a broader pelvis and generally a higher fecundity. It was impossible to analyse the breed incidence more fully in view of the uncertain overall distribution of breeds.

The highest incidence of CVP in the investigation was found in a pet flock of only three ewes, all of the Mule type. Two of these developed CVP in the third season. These ewes were maintained in a small garden and were regularly taken for walks, were housed in the centrally heated house and were fed heavily, but not very wisely, on a wide variety of foods including domestic waste and were hand-grazed along road verges and lawns! It is of course impossible to extrapolate from this to commercial flocks but it may indicate that the management of the pregnant ewe is an important factor.

All the ewes in this survey were of the wool type and no case was seen in any other breed. Indeed, apart from a small flock of Jacob ewes, no other breeds were attended by the practice. Sobiraj et al. (1986), however, recorded one case in a German Mutton ewe and another in a mountain sheep. It is thus possible that the disorder occurs in all breeds under the appropriate managemental conditions. Most previous opinions have supported the view that there is a breed disposition. While this may be so, cases are found in breeds other than the normal wide pelvis type of ewe, such as the mule. The mule breed for example featured strongly in this and other work (Hanson and Plant, 1980; Hosie, 1988, unpublished data, personal communication). Factors other than anatomical features therefore may be involved and may be at least as significant as the genetic factors.

The maximum incidence occurred amongst four-year old ewes with an overall mean age of the affected ewes of 3 years. This latter figure is somewhat misleading as all ewes over five years of age were grouped together and some cases were probably very much older
than that! Many ewes were found to be broken-mouthed and it was impossible to be certain of the ages over 6 in many of the cases. An accurate age distribution of the population in the flocks surveyed was not known, so it is not possible to conclude that one particular age group is more likely to be affected; this warrants more detailed investigation. It is worth noting that Laing (1945) reported that the majority of affected ewes were 2 years old and over, and that one-year old ewes were very rarely affected. The reported impression that it is older ewes which are more often affected (Bayly et al., 1936; Laing 1945, 1949; Clarkson and Faull, 1983), is borne out by this work but one-year old ewes may also be affected. Yearling ewes lambing for the first time represented 3% of the total number of cases. Watson (1959), however, noted that there was an impression of age-related incidence, but remarked that this might be artificial due to the habit of culling of cases of CVP. The simple conclusion which can be drawn from the results of this investigation is that CVP may occur in all ages of ewe.

Accurate recording of parity was difficult and it was only possible to be certain of the first and second parity ewes. Beyond this the information was unreliable and it was decided to restrict the classification and to group all the remaining ewes together as multiparous. Most cases were encountered in the latter group which, as older ewes were found to be at least as frequently affected, formed the largest parity group. Significant numbers of cases were, however, recorded amongst first and second parity ewes. From the data obtained on Farm 5 it is apparent that CVP can occur as a significant outbreak among primipara. Overall 23% of cases were found to be primiparous ewes (regardless of age). This finding agrees with several previous workers, including Edgar (1952) and McLean (1956), although it was commonly believed that the disorder occurred most frequently in multipara (Bayly et al., 1936; Laing, 1945, 1949). Sobiraj et al. (1986) found 30% of cases were primiparous but did not record the ages of the ewes.

There was a significant difference in the body-condition scores in affected and control ewes (mean body scores being 2.8 and 3.1 respectively) (p = 0.04), which reflected the
inadequate selection of the control ewes. Amongst the CVP cases the majority of ewes were found to be in good lambing condition as described by Hughes (1981) and Russell (1985), i.e. body score 2.5 - 3.5. The latter author suggested that ewes in better condition than body score 2 at lambing were adequately fed. In this study only 13 ewes were gauged to be in poorer condition than body score 2 at the time of examination and 6 were considered to be overweight (body score > 3.5). This meant that 91% of affected ewes were between body score 2.0 and 3.5 while in the control animals 82% were between score 2.0 and 3.5.

Fethers (1939) considered that the changes in nutrition from autumn to winter which were associated with a dramatic increase in abdominal fat and growth of the lamb may have been responsible for the development of CVP. This opinion was contradicted by the present study since the affected ewes tended to be in slightly poorer condition than the unaffected but the obvious deficiency in the selection of the control ewes may not justify this interpretation. Clinically the majority of affected ewes were reported by the farmers to be typical of the flock to which they belonged, and this might indicate that it is not body condition alone which is responsible for the onset and progression of CVP but the subjective opinion of the farmer may be misleading.

Only one case of CVP was encountered in an undocked ewe and 92% of cases were found in ewes in which the tail had been docked at or shorter than the ventral commissure of the vulva. The inadequacy of this part of the study arises from the absence of information on the proportion of ewes with full or long docked tails in the flock at large. It was not possible therefore to draw conclusions from this data. Most farmers preferred docked tails and it is probable that a similar very small proportion of the total population had undocked or long tails. Data on the tail length of the control ewes was not recorded. The only reference in the literature to the significance of tail docking is that of Bailey (1986), who suggested that overshort docking of tails might result in a neuritis, presumably of the cauda equina, and that this might be responsible, at least in part, for CVP. No author has suggested that there may be a mechanical retaining effect of a long tail but some
shepherds consider this to be an important aspect (Slatter, 1981, personal communication). If this were of such concern to shepherds it seems strange that many breeding ewes are still docked very short, a finding frequently encountered in this work. The possible significance of the length of the tail requires further investigation because, if the disorder is, in fact, rare in undocked animals, control may involve a relatively simple managerial manipulation.

During the course of the project only 13 cases were found dead or in extremis and all were cases of the ruptured-vagina syndrome. No case of CVP alone was found dead or in extremis. Of the 13 cases, 5 were known to have been affected by CVP and a further 4 animals revealed vaginal contamination which was suggestive that CVP had occurred prior to the rupture. Thus 70% of ruptured vagina cases were considered to have been affected by CVP prior to the rupture. Only one of these animals was treated surgically and this proved in vain, since the ewe died 48 hours later. These findings lead to the conclusion that ruptured vagina is a highly fatal condition and is frequently a complication of CVP. It may apparently also occur without any visible CVP. McLean (1956) and Roberts (1971) suggested that rupture of the vagina was a possible sequel to CVP but White (1961), Fox (1962), Hanson and Plant (1980), Knottenbelt (1988) and Anderson (1988) have described cases of the vaginal rupture syndrome in which there was no outward evidence of CVP.

All but one of the ewes found in extremis were euthanased immediately and laparotomy was performed in an attempt to deliver live lambs. Only one lamb survived from a possible 13 and this indicates that laparotomy is not justified in these cases. It appears that the ewe is unable to sustain the lambs even for relatively short periods after the vagina has ruptured. This may be the result of toxaemia, anaemia, exhaustion, shock or uraemia but other possibilities may be considered.

Examination of the vagina in these cases revealed that the tear was invariably situated in the dorsal wall and, as far as could be judged, seemed to run longitudinally. The injury was invariably accompanied by considerable haemorrhage and this may be a reason for the
poor survival of the lambs although routine haematology indicated that the animals were compensating adequately. Variable lengths of small and/or large bowel were herniated through the tear in most cases. In one case the entire uterus had been herniated and had broken away from its ovarian and cervical attachments (Figure 116). In another the herniation of one gravid horn closely resembled the case described by Fox (1962). One of the most significant factors described by Knottenbelt (1988) in cases of ruptured vagina was the high pregnancy rate amongst affected ewes. In his series of 17 cases there was a mean of 2.7 lambs per ewe. In the 13 cases in this study (which are included in the 17 cases described by Knottenbelt, 1988), the lambing rate was 2.42 lambs/ewe. This is still high and corresponds closely to the rate in the CVP cases overall. Hanson and Plant (1980) found, in 7 cases of the syndrome in Australia, that the ewes were generally in poor condition and there was a low prevalence of multiple births. These authors also proposed that the condition was a result of excessive tension of the vagina against the floor of the pelvis but it would seem a contradiction that excessive tension would arise with a low uterine burden and a reduced overall abdominal content.

The presence of vaginal/cervical bruising and laceration seen in some cases of CVP did not seem to be related to rupture of the vagina and, indeed, the animals so affected showed no increased mortality. It appears that the vaginal wall is particularly resilient to such an insult and it was noticeable how quickly the ewes returned to normal behaviour once the prolapsed tissue had been reduced and retained.

Anderson (1988) described cases of the ruptured-vagina syndrome which occurred without any warning, being only preceded by a cough or hiccup. It is hard to understand how this could take place, and it would seem more logical that some tissue weakness preceded the movement of the cervix and vagina and it is suggested that most, if not all, cases of the syndrome are associated with the presence of CVP (apparent or inapparent) although quite plainly the mere presence of CVP may not necessarily result in rupture.
Figure 116: A case of CVP which was found dead affected by the ruptured vagina syndrome in which the entire uterus had become prolapsed and then detached from the ewe prior to death. The tear in the vagina occurred in the dorsal wall and was extensive. Heavy haemorrhage can be seen at one ovarian ligament and at the cervix.
All thirteen cases (including those in which no CVP was reported and no vaginal contamination was apparent) were subjected to a post-mortem examination and the findings agreed closely with those described by Bassett and Phillips (1955b) in which extensive sub-peritoneal and pelvic haemorrhages were present. The urinary bladder of all affected ewes was found to be intact, but frequently contained dark, blood-stained urine and the bladder wall was invariably markedly inflamed. No case was found at post mortem examination with a ruptured bladder so urine might have been voided after the vaginal rupture occurred. The urinary bladder might then return into the pelvic cavity, so relieving the urethral obstruction.

Post-mortem examinations showed that the ewes were otherwise apparently unaffected by the disorder, the visible lesions being confined to the pelvic peritoneal pouches and the urogenital tract. In any case, the prognosis for ewes in which the vagina is ruptured is, apparently, hopeless.

There has been considerable debate as to whether CVP does occur as a distinct clinical entity in the post-parturient ewe, or outside the breeding season. Bayly et al. (1936) suggested that cases of CVP occurring in the non-breeding season, or in the immediate post-lambing period, were likely to have been recurrences of unnoticed CVP which had occurred pre-partum. Here, only one case (Case C108) was encountered in the post-parturient period without any history of having been affected prior to parturition. It is possible that this animal had a mild pre-parturient CVP which had not been recognised. Alternatively it is possible that this represented a genuine post-partum CVP. Most of the recurrences presented with extensive necrosis and inflammation of the cervical os such as is illustrated in Figure 90, and were characterised by severe and continuous straining.

From this study one can conclude that most, if not all, cases of CVP seen in the post-lambing period are recurrences of pre-parturient CVP.

No cases of CVP were found outwith the lambing season. The occasional cases seen at shearing by Bayly et al. (1936) were considered by these workers to be recurrences rather
than a new event, and perhaps it is justified to state that CVP does not occur as a primary condition outwith the last weeks of pregnancy. Factors associated with pregnancy alone, therefore, should be considered in determining its aetiology and pathogenesis.

In the flock of Border Leicester-cross ewes examined by illuminated vaginal speculum, none of the ewes examined showed evidence of vaginal contamination such as straw, faeces or inflammation, although three cases of CVP were subsequently encountered. The presence of vaginal contamination was described by Bassett and Phillips (1955b) in ewes which had suffered from CVP in the previous pregnancy. In the flock examined here, no affected ewes had been retained from the previous year so possibly a previous episode of CVP may result in a slackness of the pelvic attachments and, therefore, a tendency to CVP, as was suggested by Bassett and Phillips (1955b) and McLean (1956). It is apparent that CVP may occur spontaneously and without any warning. McLean (1956) proposed that there was a normal physiological displacement of the vagina and cervix in both pregnant and barren ewes and that only when the prolapse was visible between the vulval lips did the disorder become significant. While this may be so, the work of Bassett and Phillips (1955a, 1955b) and the present work would indicate that the milder forms of displacement may be associated with significant pathological changes and that these may progress rapidly to the point where the prognosis for the ewes worsens. This study shows that with good shepherding, it should be possible to detect all but the mildest of cases and institute therapy immediately.

No animal was found in extremis affected with CVP alone and although this situation is not likely in general practice, suitable education of shepherds may improve the prognosis markedly. The early recognition and treatment of CVP should therefore be encouraged.

The prolapse of the vagina and cervix as described in this study may take various forms ranging from the intermittent appearance of the vaginal wall (most frequently involving the dorsal wall) to severe complete prolapse of the vagina such that all or part of the vestibule is also everted. In this latter type, which was not described by Cox (1981), the
external urethral meatus may lie ventrally and be visible from the outside. These cases represent the complete prolapse of the vestibule and vagina and are fortunately rarely seen (Figures 117, 118 and 119). Diagnosis may be confirmed by attempting to pass a probe between the vulval lips and the vaginal wall. If the probe penetrates the space, it is likely that the vestibule is not prolapsed but, if there is a smooth transition and the probe cannot be inserted, then the vestibule must be prolapsed. In these cases there is frequently a constricting band created by the constrictor vestibulae muscle (Figure 119).

The appearance of the prolapses seen in the course of this work varied widely. Considering that every case was attended as promptly as possible, it was dramatic how quickly vascular compromise, contamination and necrosis developed. The prolapsed tissue often became lacerated, particularly in housed sheep where the bedding was soiled and the density of sheep was high. Most of the badly contaminated cases (Figure 39a) were seen in housed ewes, even though the cases were generally noticed and treated more quickly than those with a more extensive environment. Ewes which grazed hill pastures showed no increase in the severity of CVP when compared with the other groups and generally the extent of contamination and laceration were much less. This was contrary to the findings of McLean (1956, 1957) and it is suggested that hilly pastures have no more effect on the occurrence or the severity than flat ground.

It is suggested also that, where CVP occurs in confined sheep, early recognition and isolation of the patient is essential to reduce the extent of trauma and contamination.

The four degrees of CVP encountered in this study were divided on clinical criteria alone and, as expected, the divisions were not always clearly defined. The classification was designed to help determine whether different environmental and individual factors affected the severity of the disorder. Some annual variations in the proportions of each type of CVP were found. In the first year, there were more of the milder first degree prolapses (28% of cases) than in each subsequent year (20% and 21% in 1982 and 1983 respectively), but overall, the majority of the cases (38%) were second degree CVP (with
Figure 117: Photograph of a Second Degree CVP in which the vestibule is partially prolapsed. The muco-cutaneous junction can be seen with a small part of the vestibular mucosa exposed to the outside.
Figure 118: A case of CVP in which the vestibule is prolapsed. There is a smooth junction between the skin at the vulval lip and the prolapsed tissue and a probe could not be inserted at the muco-cutaneous junction.

Figure 119: The prolapse shown in the photograph was complicated by rectal prolapse and an obvious partial prolapse of the vestibule. The constricting band created by the muscle is clearly visible but only involves the dorsal part of the vestibule.
23% of first degree, 22% third degree and 17% fourth degree). Some milder forms of CVP may have been missed, particularly in the prevailing commercial situation. As prompt attention was given to every case, there was perhaps less opportunity for progression into the more severe forms. In practice it seems probable that a veterinarian may expect to encounter a higher proportion of the more severe forms.

Clinical examination of the cases of CVP showed that a significant proportion (76%) of the ewes showed tenesmus and 68% of the prolapses were contaminated with grass, straw, mud or faeces, and were variously lacerated and/or bruised. There was no evidence to suppose that the tenesmus was a consequence of the vaginal contamination/laceration alone as several cases showed severe straining without any signs of either form of trauma. Some ewes showed severe, but transient, tenesmus with forceful and prompt expulsion of urine on reduction of the CVP. Straining often ceased once a full flow of urine had been passed even though there was obvious haematuria so it is more likely that the bladder distention is significant in the pathogenesis than the presence of cystitis.

Straining was noticeably worse in the second degree cases which supports the findings of McLean (1956) that the severity of straining may be less if the bladder is sufficiently displaced to allow its continued dilatation i.e. when the limiting dimensions of the bones of the pelvis were no longer a restriction upon its filling. Under these circumstances the bladder may not be able to empty and may eventually rupture. This complication has been widely reported (Watson, 1959; Roberts, 1971; Fielden, 1980) although, in the present study, no animal was found with a ruptured bladder. It is easy to imagine that there would be an obstruction of the bladder and rectum if the pelvis was obstructed by either a foetus or the bladder or other abdominal organs, and that continued straining by the ewe may be expected to aggravate the disorder. Laing (1945, 1949), Watson (1959) and Fielden (1980) have all given high priority to the relief of the bladder. The present work would seem to support this attitude, and cystocentesis can be avoided by early intervention and by relieving the bladder as soon as possible by lifting the prolapse and thereby reducing the
urethral kinking.

Bladder incarceration may, however, not occur and in some cases (Figures 14, 44 and 45) no organ may be obviously involved. In such cases straining may be absent or marked. It was possible that a ruptured bladder may not have been detected at the initial examination but these cases would have died of uraemia over the following few days with uroperitoneum present post mortem. No such case was encountered, and it can be concluded that, while urethral obstruction frequently occurred, early intervention and treatment probably prevented rupture of the bladder. It is likely that a practising veterinarian would encounter cases of CVP in which bladder rupture was present, particularly in longer standing cases.

The premise that CVP is a consequence of straining alone is not invariably true and Figures 14 and 46 show cases of relatively extensive CVP in which there was no straining and indeed no other evidence of irritation. These resemble the "long sausage type of prolapse" described by Bayly et al (1936). The existence of this type of CVP indicates that tenesmus and bladder incarceration do not invariably occur and may indicate that local inflammatory changes may be involved in the onset of straining. This may lead to the thought that CVP may occur in two basic forms (a long thin type and a more rounded, tense type), a premise first proposed by some of the farmers reported by Bayly et al. (1936) but not really considered since. If bladder (or other organ) obstruction of the pelvis was a prerequisite for the development of CVP the former would not be expected to occur.

Tenesmus was found to persist in some cases, even after reduction and retention. This seemed to be particularly bad in ewes in which either the Dalton's Spoon or the Nappy Safety Pin method was used to retain the prolapse. It is suggested that the presence of either a space occupying object in the vagina which may irritate the fornix or vaginal wall, or damage to the sensitive tissues of the vulval lips, may in some cases aggravate straining.
Mayor (1958) advocated the use of an inflated football bladder in the vagina and found that there was no further tenesmus. This may indicate that it is not the presence of the foreign body in the vagina which causes the problem, but rather the shape of the retention device and/or the trauma which it induces. It is easy to visualise that the round but thin section of the Dalton's Spoon may, in some cases, cause damage to the sensitive tissues of the fornix, particularly if the ewe continues to strain after its insertion.

The control of tenesmus by the induction of an artificial pneumoperitoneum, as advocated by Hartigan (1961) and Arthur et al. (1982) in the treatment of CVP in sheep and cattle respectively, suggests that the extent of the disorder may have been more related to straining than any other single factor. Hartigan's (1961) results, admittedly on only 3 sheep, indicated that once the tenesmus was controlled there was no further need for any method of retention; indeed, Hertrampf et al. (1978) found that merely controlling tenesmus resulted in a 78% cure of CVP. In the course of the present investigation neither the football bladder technique nor the pneumoperitoneum method was employed, mainly on the grounds that they were not apparently widely used in general practice. This may be more through ignorance of the methods than through reluctance to try them.

Most opinions suggest that the presence of the bladder, in particular or other abdominal organs in the pelvic cavity, is responsible for the tenesmus which in turn seems to exacerbate the disorder (Cox, 1981 and others). The present work would seem to support this possibility.

Thirteen percent of cases showed an intercurrent rectal prolapse which was possibly the result of the tenesmus. Rectal prolapse seldom required special attention except where CVP was severe. Again, it appeared that control of tenesmus simultaneously resolved the rectal prolapse.

Epidural or sacral anaesthesia controlled tenesmus effectively, although sometimes only temporarily. This suggested that the sensory origin of the straining was in the organs innervated by the sacral nerves; these being the rectum, the bladder, the vaginal wall and
the cervix. No attempt was made to use ethanol as advocated by Eagelman et al. (1967), Roberts (1971) and Hertrampf et al. (1978), primarily because this would have involved the use of a material not licensed for the purpose and in any case it was thought to be an unnecessary complication. The prolonged analgesia without deleterious effects upon locomotion, defaecation or subsequent parturition as found by Hertrampf et al. (1978) should be further investigated. The use of morphine derivatives in the epidural space, such as is used in the human for the control of perineal pain (Behar, Magora, Olshwang, and Davidson, 1979), may also be of interest in the long term control of tenesmus particularly as it would appear that little or no effect is shown on locomotion, defaecation or parturition and the effect is apparently of considerable duration. The present work, however, supports the findings of Hertrampf et al. (1978) that once straining was controlled, and provided that it did not restart, the condition seldom recurred. It was hard to justify leaving the prolapse in an unreduced state, hoping for a spontaneous resolution following regional anaesthesia and so this approach was not tested.

One of the most striking features of this study was the distribution of cases occurring on each of the days prior to parturition. The greatest number of cases (80%) occurred in the last 7 days of gestation (and 95% occurred in the last 14 days), but a few occurred up to 27 days before delivery. A mean value of 5.6 days prepartum was obtained whereas Sobiraj et al. (1986) found a mean of 10.4 days in only 16 cases. Edgar (1952), in a survey of some 102 cases of preparturient CVP, showed that 76% of cases occurred in the 14 days prior to parturition. He also found that 10% of cases occurred between 4 and 8 weeks before lambing but in the present work only individual cases were encountered between 9 and 27 days prepartum. The findings agreed with the observations of many previous workers, including Bayly et al. (1936), Laing (1945), Edgar (1952), Watson (1959), Belchner (1976) and Sobiraj et al. (1986), that CVP occurs most frequently in the last two weeks of pregnancy but that individual cases may be seen up to 4 weeks, or more, before parturition. The indications are that the last two weeks of gestation may provide the best
information on aetiology and pathogenesis.

6.3.1: Clinical management :

It was inevitable that the clinical management of the cases of CVP in this study was influenced by practicality. Some farmers insisted on particular approaches and several well meaning individuals persistently examined and treated the cases themselves, calling for a visit thereafter. In spite of the offer of free visits and free treatment etc. this was a problem, even in the third year of the study.

It was not possible to apply experimental or unusual approaches to the cases. For example, in order to determine a true mortality rate in animals affected by CVP and a comparison with different methods of treatment, some ewes with CVP should have been left untreated. This was not considered to be ethical on purely humanitarian grounds as well as for commercial considerations. The clinical approach to the cases depended heavily on the goodwill of the farmers and I was reluctant to abuse this.

There were no apparent complications from restraint of the ewes in dorsal recumbency with elevation of the hindquarters and the assistance of gravity was helpful in reducing the prolapse quickly.

Every effort was made to ensure that the tissues were treated gently. This approach appeared to be beneficial as no animal was treated in such a way that the vagina ruptured or proved difficult to keep clean. A final irrigation with warm sterile saline and copious lubrication with a non-irritant obstetrical lubricant were considered to be of great benefit. Even in those animals in which the vagina was severely affected by contamination, laceration, bruising, or when the vagina filled with urine before reduction could be completed, the tissues appeared to be healthier after cleaning and decontamination. Since this treatment was used in all the ewes it is impossible to be certain that this is a desirable or necessary part of the therapy but as it is the normal approach to tissue cleansing, it might be expected to be beneficial.
6.3.2: Treatment and retention:

Eleven methods of treatment and retention were compared and these ranged from the simplest postural restraint, as advocated by Bruere (1956) and Slatter (personal communication), to the use of elective caesarian section. None of the methods used was experimental (with the possible exception of the use of the longer kilt-type pin rather than the nappy pin and the use of fan-belt in place of wooden quills) since they had all either been described previously, or were regularly used by farmers.

No method of treatment appeared to give consistently better results than the others. The Dalton's Spoon proved to be the most disappointing for, although some previous authors found almost total success (Laing, 1949; Fowler and Evans, 1957), only 61% of the cases treated in this way here, were satisfactorily retained. Pastoor and Weitenberg (1979) expressed some dissatisfaction with Dalton's Spoon because the attachment to the perineal wool was often unsatisfactory and they developed a modification involving Flessa-type pins to retain the prosthesis. In none of the cases in this study was attachment found to be a particular problem. Persistent tenesmus following insertion was a significant cause for dissatisfaction associated with its use, which was also the finding of Hartigan (1961) and Watson (1959).

There are few reports of the relative effectiveness of the various methods, and most veterinary surgeons and farmers appear to use their own preferred method. This would seem to indicate that no individual method has gained any better overall reputation than the others. This may be expected if no method had shown persistent advantages over years of use and this is indeed the finding in this survey. No one method or, indeed, broad type of method, seemed to have any major significant advantages in terms of the survival of the ewe or the lambs, the degree of pre- or post-lambing complications or the long term prognosis for the ewe.
The use of caesarian section as a method of treatment was approached slightly differently. Of 10 ewes subjected to an elective caesarian section, regardless of the presence or absence of signs of parturition, all survived but the lamb mortality rate was high (65%) and 70% of ewes so treated produced no viable lambs (as opposed to only 15% in the group of ewes in which surgery was specifically indicated).

Lambs delivered by elective caesarian section were found to be consistently more premature than those delivered by non-elective caesarian section as might be expected as CVP occurred mostly in the 2 - 9 day period before lambing. There was also a higher incidence of infertility in these ewes at the next lambing season. Several authors have suggested that caesarian section is indicated when straining is severe or there is difficulty with retention in the last 14 days of pregnancy (Fielden, 1980; Arthur et al., 1982). Schulz et al. (1985) found that caesarian section was the only way to keep the ewes and lambs alive. This latter work was based on a very limited number of cases. It is not, however, the finding of this study which indicated that ewe mortality may be reduced at the expense of lamb survival by the use of elective caesarian section while any delays which can be obtained by other methods produces increased lamb survival at the expense of raised ewe mortality. In this work, animals subjected to caesarian section after a delay using other methods of retention showed a reduced ewe survival when compared to the non-elective group (from 100% survival to 7 days post surgery to 93% survival respectively). On balance, it would seem that the advice given by Walsby (1952) to delay surgery until signs of parturition are present, or at least for as long as is practicable, is sound in respect of lamb rather than ewe survival.

When considering the best type of treatment it is important to assess the stage of gestation initially, and then apply an appropriate method to reduce and retain the prolapse in as near to the natural position as possible without undue pressure and without irritation or damage to the tissues. Ideally, natural unassisted delivery of the lambs should be possible but the best method in this respect (the Eagelman et al. (1967) modification of the Minchev
technique) requires more surgical interference than the other methods and may thus be of limited practical value. In theory, at least, Dalton's Spoon should satisfy the criteria; it is cheap, easy to use, and should allow unassisted lambing. However, it also proved rather disappointing in this study in that unassisted lambing was not always possible.

The postural and truss methods, combined, produced fewer cases of dystocia, which may be expected, as their use was generally limited to the mildest cases.

The supported quill method using lengths of motorcar fan-belt has several advantages. It is cheap, easy to insert and, in spite of the sutures penetrating the vaginal wall, appeared to cause little irritation. The single needle insertion carrying the double strand of suture material is possibly a better technique than those where two separate strands for each end of the quill are inserted or where vulval inversion resulted from non-invasive sutures placed at the wool line. The supported quill methods, necessarily, provide added security of retention but, in so doing, prevent any chance of normal delivery.

In the course of this work, there were several cases in which retention devices were not removed from the ewe in time while others were removed early, in response to the suspected onset of lambing. In either case there was the potential for lamb deaths, tearing of the vulva or recurrence of the prolapse. In this respect the Buhner technique (Buhner, 1958) and the lace-up method (Merck, 1973) (which was not tested in this work) have the advantage that the vulva can be released to allow vaginal examination and re-closed if necessary without further trauma.

The Buhner method is widely regarded as the method of choice (Kubicek, 1977; Fielden, 1980; Cox, 1981; Arthur et al., 1982;) and the results of its use were consistently good in this work. There were few complications and once properly inserted there is a certain satisfaction to be gained from it. Problems may arise from poor insertion of the suture (particularly if it is not placed deeply enough) or if it is drawn too tightly. It is not practical for a shepherd and it really does require the skill of a veterinarian!
There were no significant differences amongst the various treatments with respect to the number of ewes with dystocia, the number of ringwomb cases, the number with post-partum complications and the number culled as a direct consequence of CVP. As there were only slight differences between treatment methods in respect of the ewe prognosis it is not thought prudent to advise the adoption of any specific type of treatment method. There is little doubt that urgent reduction and safe retention are advisable in all cases of CVP.

6.3.3: Post-parturient complications:

Comparison between affected ewes and control animals showed that post-parturient complications were much more frequently encountered in the former group. Amongst the control ewes 95% lambed without any assistance at all (and those that required some help needed minimal interference, while in the diseased animals only 68% did so. This agrees with Edgar (1952), Kenworthy (1957), Edwards and Jones (1958), Stubbings (1971) and Schulz et al. (1985), that ewes affected with CVP have a higher incidence of dystocia than unaffected sheep. Most of these workers found that the major complication of CVP was a delayed or absent cervical dilatation (ringwomb).

6.3.3.1: Retarded cervical dilatation (ringwomb):

Edwards and Jones (1957) found that CVP and ringwomb were invariably present simultaneously.

In the present study none of the control ewes was affected by ringwomb, while 17% of affected ewes suffered to varying degrees. Unfortunately this figure was not entirely reliable because it depended partially upon the opinion of the shepherds who, in spite of appeals to the contrary, were liable to deliver their own ewes prior to examination. Most of the shepherds involved in the study were experienced and reliable, however, and their opinions were accepted! Amongst the ewes examined personally some were found to be
obviously affected by retarded cervical dilatation and while this may have been due to a somewhat premature delivery subsequent to the liquefaction of the cervical seal (Figures 64 a, b and c), the condition could not easily be corrected and a diagnosis of ringwomb was justified.

The number also included some ewes in which the cervix was dilatable, but with difficulty. Hindson and Turner (1962), contrary to Edgar (1952), Kenworthy (1957) and Edwards and Jones (1957), could not establish any correlation between the two conditions. Rusch and Berchtold (1978) found that 66% of CVP cases were demonstrably affected by ringwomb, a higher proportion than was encountered in the present work (17%). It is possible that if an attempt was made to lamb every case of CVP at initial examination that the figure for ringwomb cases would be very high, but this would represent prematurity rather than a pathological retardation of cervical dilatation.

There seems little doubt from this study that ringwomb is an important potential complication of CVP. Arthur et al. (1982) noted that the two conditions appeared to occur under the same seasonal influences but such a conclusion could not be confirmed or otherwise. Further controlled investigations of this aspect are needed but it would seem that CVP cases are more often affected by ringwomb than are unaffected ewes.

In addition to the cases of ringwomb, a number of ewes were affected by other forms of dystocia such as foetal malpresentation, and cervical and/or vaginal damage which rendered vaginal delivery dangerous. Again, the number of cases with these complications exceeded the number of control ewes so affected (45% and 4% respectively).

6.3.3.2: Post-parturient uterine prolapse.

There are divided opinions on the relationship between CVP and post-parturient prolapse of the uterus. Vanderplassche and Spincemaille (1963) suggested a definite relationship existed but Watson (1959) and Roberts (1971) were adamant that there was no connection between the two disorders. The results of this work would seem to agree more
with Watson (1959) and Roberts (1971) than with Vanderplassche and Spincemaille (1963) in that only 3 cases of uterine prolapse were encountered amongst 183 cases of CVP which lambed (2%), and 2 cases were seen in the 81 control ewes (2.5%). There was no apparent difference in the appearance or any other clinical feature of the prolapsed uterus between the CVP cases and the control ewes. If there had been some causal relationship considerably more cases may have been expected amongst the diseased animals.

6.3.3.3 : Ewe mortality.

Post-partum mortality amongst ewes with CVP was markedly higher than in the control ewes (a mortality within 7 days of delivery of 11% and 3 % respectively). This might be expected in view of the increased possibility, in affected ewes, for puerperal septicaemia, toxaemia, metabolic disorders associated with inappetence and debility.

It was noticeable that there were significant numbers of deaths in both the pre- and post-lambing period amongst the cases of CVP (7% and 14% respectively). Jones (1958), McLean (1959), Roberts (1971), Schwarz (1975) and Martinez and Mendez (1985) concluded that the overall mortality rate amongst cases of CVP may be as high as 50%.

The most extensive investigations into mortality of ewes affected by CVP was carried out by McLean (1959) in New Zealand who found an overall ewe mortality rate of 40% (including 13% of cases which were destroyed immediately). In this study the ewe mortality rate was found to be 21% of affected animals.

The mortality rate in commercial flocks may be even higher as in this study almost all cases were seen quickly and treated professionally without regard for cost or time. More usually, ewes might be treated later and/or handled by unqualified people at a time when they could be expected to be very busy attending to normal animals. Slatter (1981, personal communication) expressed little sympathy for the ewes and wasted little time in dealing with them. The ewe mortality rate can, therefore, be justifiably given as at least 20%.
6.3.3.4: Long term ewe prognosis.

The data relating to the long term prognosis for the ewes was derived from the project more by accident than design. Initially it was not planned to follow the ewes beyond the first episode of CVP as it was normal practice to cull affected ewes either immediately, if no live lambs were born, or in any case after weaning. Several farmers responded to the idea of retaining the affected ewes and this formed the basis for the extra investigation. One of the most serious long-term complications was the possibility of ewe infertility at the next breeding season. There are no previous reports of investigations into this problem.

There was generally little effect upon subsequent fertility except following elective caesarian section or vulval closure methods of retention. Both of these resulted in some ewes failing to conceive at the next breeding season. The possibility of one particular method resulting in infertility may be worth investigating more deeply.

The culling of ewes affected by CVP, either to prevent its recurrence (Edgar, 1952) or to limit the number of progeny from affected ewes because of its possible heritability, has been suggested (Laing, 1945, 1949; Edgar, 1952; Fowler and Evans, 1957; Straiton, 1973). Such elimination of the affected ewes, which has been widely practised (Slatter, 1981, personal communication) may have limited the number of ewes which would have had the opportunity to rebreed and this may explain why no records of this aspect are apparent. The present investigation has shown that subsequent infertility is not inevitable, and that careful choice of treatment method may result in a better prognosis in this respect.

In spite of the retention of some ewes which would ordinarily have been culled, there was a much higher culling rate amongst affected ewes than control animals. This difference would, of course, have been much higher without the retention of some cases of CVP. A more aggressive policy towards retention of affected ewes may have yielded some significant information.
6.3.3.5: Recurrence of CVP.

Watson's (1959) opinion that ewes need not prolapse again at the next pregnancy or, indeed, ever again, is supported by the present results. Edgar (1952) followed only two ewes into the following lambing season, and found that neither of them developed a prolapse. In spite of this and the opinion of Watson (1959) that recurrence was not invariable, there has remained a persistent notion amongst shepherds and veterinary surgeons that culling is necessary to prevent recurrence (Laing, 1949; Belschner, 1976; Edgar, 1952; Slatter, personal communication). Belschner (1976) noted, however, that recurrence was likely only if the same predisposing factors were present in the following season, thereby implying that it was not invariable.

The present results appear to be the only record of significant numbers of CVP ewes which have been followed for several pregnancies. Bassett and Phillips (1955b) examined a group of 50 ewes which had a history of CVP and found that all showed thickening of the vaginal wall and/or lesions in the anterior vagina. Eight of the cases showed no apparent signs of having been affected but were known to have a history of CVP. These ewes may have been affected with mild CVP or may have healed remarkably well. Several of the ewes, when examined by an illuminated vaginoscope, showed evidence of vaginal contamination suggestive of intermittent CVP. Their results do not indicate how many of the ewes subsequently developed CVP, except that 5 of the 8 ewes with a contaminated vagina developed CVP. These authors found that the dilatability and displacement capacity of the cervix was markedly reduced in ewes which showed scarring of the vagina, and suggested that this was the reason for the non-recurrence of CVP. Eagelman et al. (1967) suggested that their method of anterior vaginal wall fixation resulted in the non-recurrence of CVP through the development of adhesions and scar tissue along the path of the sutures.
The present study indicates that a ewe may prolapse at any age and then may either prolapse again at the next pregnancy (55% of the retained cases), or may not prolapse (30%), or may be barren at the next breeding season (15% of retained cases). This indicated that, although the chance of recurrence was high, it was not inevitable and this agrees with Filmer (quoted in McLean, 1957) and Gerring (quoted in McLean, 1959) in New Zealand.

The subsequent development of CVP need not be as severe as the first episode (7% of prolapse recurrences were found to be milder at the second occasion) but in most cases prolapse tended to be either as severe or more so (55% and 38% of recurrences in the following year, respectively). If a ewe were barren in the second year there was no greater or lesser tendency for her to prolapse in the third year than there was for those ewes which were pregnant in the second year.

Thirteen of the 53 ewes (25%) did not prolapse again during the period they were followed. The extent of vaginal scarring and consequent fibrosis was not determined and it may be that the ewes which did not reprolapse were those in which these changes were present. This possibility was suggested by Bassett and Phillips (1955b) and was alluded to by Eagelman et al. (1967). This aspect may warrant further investigation.

6.3.3.6: Lambing rates and mortality.

Amongst the retained ewes there was no significant difference in the numbers of lambs carried from year to year, nor was there any lesser or greater ewe or lamb mortality than in the overall number of CVP cases. This indicates that while the chances of recurrence are high there is no greater or lesser problem created by the prolapses than would be the case in ewes prolapsing for the first time. However, the fact that recurrence does occur suggests that the culling of affected ewes may be a necessary managemental process to reduce the number of cases and thereby reduce to a minimum the consequent mortality. It is accepted that it would have been better to retain all the affected ewes until they either
died, or were culled due to CVP but this was not possible, and this limited study has highlighted possibilities for future research.

6.3.4: The effect of climate:

The possibility of a climatic influence in the aetiology of CVP was not investigated in depth but, subjectively, the three years of the investigation were largely similar. No apparent correlation could be shown between the minor changes in climatic conditions and the occurrence of CVP in any particular flock or in the overall annual number of CVP cases. It seems unlikely, therefore, that the climatic conditions have any direct influence on either the aetiology or the pathogenesis.

6.4: THE LAMBS:

6.4.1: Lamb numbers:

A major component of the work of this thesis was directed towards the number of lambs carried by the affected ewes and their prognosis. Numerous previous workers have concluded that ewes affected by CVP carry at least twin lambs (Bayly et al., 1936; Laing, 1945, 1949; Hughes Ellis, 1958; Schulz et al., 1985). Sobiraj et al. (1986), however, found a lambing rate amongst cases of CVP of only 1.85 lambs/ewe which compares closely with the control group in this study. This work however included cases of CVP in non-woolled sheep which might be expected to be less fecund than the normal dual purpose animals included in the present study.

In spite of including in his questionnaire the number of lambs carried, Edgar (1952) did not publish any results relating to this factor and no other work appears to have been undertaken to quantify the impression that most affected ewes carry multiple lambs.
In this study there was a significantly larger number of lambs carried by the affected ewes when compared to the control group. The difference in the mean lamb burden from year to year was insignificant. An overall lambing rate of 2.26 lambs per ewe, compared to 1.86 lambs per ewe in the control ewes, indicated that ewes affected by CVP were possibly those in which there was a higher metabolic demand, and were perhaps also the ewes with a higher fecundity for which farmers were trying to breed. The higher number of lambs born means that, as genetic pressure is exerted by selection for greater fecundity, we may be selecting for those ewes with an increased tendency towards CVP. Many other factors may be related to the body condition and metabolism of a ewe which carries twins as opposed to single lambs and would need to be considered. These include the extra demands placed on the nutritional status of the dam by the foetuses and the reduced abdominal space available for the digestive tract. Such ewes may also show a greater depression of the appetite (Straiton, 1973) as full term approaches.

The number of lambs carried did not appear to have any effect upon the severity of the prolapse. It is thus possible to conclude that, while ewes with multiple lambs are more likely to be affected, the severity of the condition is not dependent upon the number of lambs. Indeed, there were significant numbers of ewes affected by all types of prolapse including the most severe while carrying only a single lamb.

6.4.2: Litter and individual lamb weight:

Fethers (1939) suggested that inordinately rapid growth of the lambs might be a causative factor and Schulz et al. (1985) found that most mature lambs were overweight (no actual weight or comparison with lambs born to control ewes was given). These appear to be the only references to the size of the lambs delivered from ewes affected by CVP.

In the course of this work many of the lambs were weighed and this provided the first apparent investigation into this aspect of CVP.
The subjective impression of overweight lambs noted by Schulz et al. (1985) was not supported by the findings. There were no significant differences between the affected and the control ewes in either the mean overall litter weights or the overall mean individual lamb weights.

The only factor which was of particular note was the increase in the weight of the lambs carried by ewes affected by the more severe degrees of CVP (3rd. and 4th. degree prolapses) (4.12 Kg/lamb), when compared to the less severely affected ewes (prolacpses of 1st. and 2nd. degree) (3.46 Kg/lamb). The significance of this requires further study but it may be that the severity, but not the initiation of CVP, is related in some way to the weight of the lambs rather than the number.

6.4.3: Lamb mortality:

Consideration only of the number of lambs carried may give a false impression of the disorder, for it has often been reported that there is also a significant mortality in the lambs (Edgar, 1952; McLean, 1959; Schulz et al., 1985). Amongst the control lambs in this study, a lambing rate of 1.86 lambs per ewe produced 1.44 live lambs per ewe. This latter figure closely resembles that published in the Agricultural Returns of the Ministry of Agriculture Fisheries and Food (1984), viz. 1.38 lambs per ewe (derived from the number of lambs sold/retained and the overall United Kingdom breeding ewe population). Data from eight of the farms in this investigation over the previous 3 years (1978 - 1981) which were extracted from the practice and farm records yielded a fecundity of 1.79 lambs per ewe with 1.48 live lambs/ewe at the end of the lambing season.

Amongst the affected ewes, however, the higher total lamb burden mentioned above resulted in an overall survival rate of only 1.12 lambs per ewe. This represented a much higher lamb mortality than in the control ewes, particularly when one considers that there were more lambs carried in cases of CVP.
Edgar (1952) and McLean (1959) both found a 33% mortality in the lambs born to CVP cases. McLean (1959) also noted that 33% of affected ewes which were carrying single or multiple lambs produced one or more dead lambs. In the present study 40% of affected ewes produced no live lambs, and 21% produced only one live lamb from multiple pregnancies.

An overall mortality of lambs (ie. those which were born dead or which died within 48 hours of birth) of almost 50% was found in this work which was higher than expected from the literature reports (Edgar, 1952; McLean, 1959). This was possibly because the cases of CVP were treated early and some ewes may have been kept alive by the earlier more aggressive treatment of the CVP than may have been the case in Edgar's (1952) and McLean's (1959) results. The figure for lamb mortality may, therefore, be considered realistic when careful treatment is available and it is obvious that this level of mortality is already unacceptable.

Combining both ewe and lamb mortality in terms of number of sheep dead as a consequence of CVP the figures in this work (43%) closely resemble those of Edgar (1952) in the United Kingdom, viz. 37%, and McLean (1959) in New Zealand, viz. 40%.

Amongst the ewes subjected to caesarian section, either elective or specifically indicated, the number of lambs carried on average was 2.0 which appears to be closer to the control group rate of 1.86 lambs per ewe. The reason for the difference between the total prolapse group and those subjected to caesarian section is unclear. It is possible that the number of ewes forming the caesarian section group was too low or that they were generally younger. However, 56% of caesarian section ewes produced dead lambs (compared to 40% in the overall prolapse group and 15% in the control group) and the lamb mortality was highest in the elective caesarian section group (65%) and lowest in the control group (21%). This compares with a mortality of 57% and 50% in the indicated caesarian section group and the overall CVP affected ewes, respectively.
A most interesting group of data was obtained from consideration of the survival of lambs from ewes with different degrees of CVP. Thus, the higher survival rate of lambs in ewes affected by first degree CVP was such that there were more lambs alive at 48 hours after birth in this group of ewes than in the control ewes (1.56 lambs per ewe and 1.42 lambs per ewe, respectively). However, as soon as the severity of CVP rose to second degree, the number of live lambs per ewe fell to below that of the control sheep (1.25 live lambs per ewe and 1.42 live lambs per ewe, respectively). The proportion of live lambs born fell still further to 0.58 live lambs per ewe in the third degree prolapse cases and amongst the fourth degree cases 0.88 live lambs per ewe were produced. This indicated that, while affected ewes carried more lambs overall, the more severe the prolapse the less the chances of survival for the lambs. The slightly better lamb survival amongst the fourth degree prolapse cases may have been due to the increased care and attention inevitably paid to these animals and to the higher number of them which were subjected to caesarian section or lambed immediately.

It is obviously worth recognising the earliest forms of CVP and treating them effectively. Any progression of CVP to the more severe forms would immediately result in a reduced financial return to the farmer at the end of the season.

6.5 : RETAINED EWE LAMBS :

Following questions from farmers and numerous veterinary surgeons concerning the inheritable nature of CVP, several of the ewe lambs born to ewes affected by CVP were purchased and maintained in their respective flocks to be treated exactly as normal breeding ewe lambs. The intention was to attempt to verify or otherwise the suggestion by Koch and Haisch (1943), Laing (1945), Edgar (1952), Clarke, quoted in McLean and Claxton, (1959), Jones (1958), Roberts (1971), Fielden (1980) and others, that predisposition to CVP might be inherited. Koch and Haisch (1943) had suggested that it
may be due to an inherited hormone imbalance in the hypophysis, whilst most other authors have suggested that anatomical features such as the breadth of the pelvis may be involved. Watson (1959) stated that 100 ewes, all progeny of CVP affected animals, were being run together at the Ruakura Research Centre in New Zealand, to test the heritability of the condition. No results appear to have been published, although McLean and Claxton (1959) quote Clarke as having found positive evidence of an hereditary component at the same Research Station.

It was decided to leave the ewe lambs on their original farms so that management could be maintained. It was also impossible for me to support such a flock of ewes without land! As a result, 16 ewe lambs were identified and retained for up to four years, when the work had to cease. Amongst these 16 ewes three developed CVP during this time. The incidence of CVP was, therefore, about 19%, which is far higher than the overall population. One ewe prolapsed twice in consecutive years, the first occasion being at one year of age. In the group of progeny, the lambing rate was noted to be higher than in the control animals of the same age. This would seem to confirm that there is an increased lambing rate amongst ewes which are prone to CVP irrespective of their age. The numbers of animals is inadequate to support conclusively the heritable nature of the disorder but the results are at least indicative, and this should stimulate further research into this important aspect. The effort is all the more important as the work also shows that those ewes affected by CVP tend to carry more lambs, and thus, having been specifically bred for improved fecundity, there may be an increasing trend in the number of cases of CVP in the population as a whole. This may be one possible explanation for the greater overall incidence found in this work as opposed to Edgar's (1952) survey, (1.34% and 0.53% respectively).

There seems little doubt that the aetiology is a complex of factors including management, nutrition, heredity and fecundity.
6.6: PATHOLOGY AND PATHOGENESIS:

A post mortem examination was performed on all the cases of CVP which were found dead or in extremis (all being cases of the ruptured vagina syndrome). Particular attention was directed at the contents and disposition of the urogenital organs. Bassett and Phillips (1955b) published the results of autopsies in ewes affected by CVP and similar to this study, found that the abnormalities were largely confined to the pelvic organs. Bayly et al. (1936) noted that congestion and focal haemorrhages were found at the bladder neck and commented that there was no evidence of constipation. They noted that the major problem appeared to be urinary obstruction. In the present study, however, it was noticeable that no case affected by the ruptured vagina syndrome, which, according to McLean (1956), Roberts (1971), Clarkson and Faull (1983) and Knottenbelt (1988), is a potential complication of CVP, showed the bladder to be dangerously enlarged or ruptured. The repetition of the extensive investigative pathology carried out by Bassett and Phillips (1955b) was not possible in this study but, as far as could be ascertained, the lesions involving the displacements of the pelvic peritoneal pouches were similar. Haemorrhages in the pelvic cavity and into the vagina were most apparent.

Ewes which died between treatment and lambing showed marked pelvic inflammation and varying degrees of vaginal, rectal and bladder damage. It was noticeable that such deaths generally occurred from 1 day to 9 days following treatment with the majority of deaths just before lambing. The possibility of stress of impending parturition, superimposed on a toxaemia, may have been responsible as necrosis was often apparent in the urogenital tract. The post-mortem examinations carried out in this work revealed intra-pelvic haemorrhages and distortions of the pelvic anatomy, particularly with respect to the peritoneal pouches. It appeared likely that these animals died either from toxaemia/septicaemia or secondary acetonemia. This is in general agreement with the observations of Bayly et al. (1936). It would seem prudent, therefore, to ensure that ewes treated for
CVP are isolated and allowed access to familiar palatable food of high nutritional value as soon as possible.

Straiton (1973) found that post-mortem examinations of normal pregnant ewes showed the rumen to be severely compressed and small. In the post-mortem examinations of ewes affected by CVP which died subsequent to treatment, the rumen was also found to be small but enquiry of the shepherds generally indicated that the ewes had been inappetent for variable periods and this may be an alternative explanation for the rumen contraction. None of the control ewes died between examination and lambing and no comparison between the relative sizes of the rumen was possible.

Biopsy specimens from the external cervical os showed that there were significant changes in the epithelium and in the stroma. The epithelial integrity was invariably compromised in the more severe forms of CVP. These changes were proportional to the severity of the CVP in most cases but it was apparent that where straining was not severe oedema within the stroma was reduced.

An inflammatory response was present in almost all cases (98%) and reflected the severity of the physical appearance of the prolapsed tissue and the duration of the disorder. Mild degrees of CVP were accompanied by some inflammatory changes and as straining progressed oedema and inflammation become more apparent. Haemorrhage into the stroma was an occasional complication (13% of cases) and may have been a result of straining and ischaemia and/or local trauma.

Consideration of the pathogenesis of CVP has been a frustrating factor for many researchers. Anatomically, it would seem that the condition seen in this study is identical to that described by Bassett and Phillips (1955a, 1955b). From the results of post-mortem and biopsy examinations the pathogenesis seemed to follow a pattern. In very mild CVP the displacement of the vagina appeared to cause little irritation but inflammation was invariably present possibly as a result of vaginal contamination. This suggested that any minor contamination of the vagina or any local circulatory compromise may result in
oedema and inflammation. These occurred in even the mildest of cases and may partially explain the onset of straining. Displacement of the bladder or other abdominal organ into the pelvis could result in dysuria. This may be worst if the bladder becomes trapped within the pelvic cavity. In this position it would be unable to dilate and urine could not in any case be voided. This may induce severe tenesmus which may then result in the bladder (or other organ) being pushed beyond the pelvic limits. Once in this position it may then continue to fill. Tenesmus may be reduced but urination becomes increasingly difficult. Further enlargement of the bladder may result in a tighter flexion of the urethra and progressively greater difficulty with urination. Continued straining and exposure of the vagina and cervix may induce serious inflammation and oedema. Drying, infection, local ischaemia and hypoxia could result in tissue damage and eventually necrosis. The presence of such a large protruding mass of tissue means inevitably that some local trauma would also be induced either by the other sheep in the group or by the ewes continued recumbency and expulsive efforts.

The scheme of pathogenesis derived from this work does not explain, however, the initial onset of the displacement nor the finding of some prolapses in which no straining was apparent and no abdominal organ could be identified in the prolapse. It seems unlikely that this is the total explanation for CVP and it may be that the condition occurs in two forms, one of which results from organ displacement and the other from some other cause. McLean (1956) was of the opinion that some tract displacement was normal in both pregnant and non-pregnant ewes and this may be an area for further research.

6.7: HAEMATOLOGY:

The results of the haematological examinations produced few features of note. Comparison of the erythron between the diseased and the control ewes showed no
significant changes in any parameter. This would seem to indicate that the haemorrhages seen in post mortem examinations might be insignificant, although of course no examinations were carried out of the pelvis of the affected live ewes except the few subjected to caesarian section and no blood samples were obtained from animals already dead. The ewe's homeostatic mechanisms apparently compensated adequately for the loss of blood within the limits shown in the cases. No previous author appears to have published results relating to this aspect of the metabolic status of CVP-affected sheep.

As might be expected there was, frequently, though not invariably, a leucocytosis which was due to a neutrophilia. The difference between the control and the affected animals was statistically significant \((p = 0.004)\). The response of ruminants to disease in respect of the leucogram is notoriously low and slow (Schalm et al., 1975; Doxey, 1983) and the earliest response \((4 - 6\) hours) to an insult is shown as a neutrophilia with left-shift. The normal response which follows this after about 8 hours is a depression of the both total and neutrophil counts, and thereafter the counts may rise above the normal resting level significantly but not often dramatically. The prompt attention given to most cases in the course of this study, might therefore explain the frequent finding of a leucocytosis with a marked neutrophilia (with left-shift).

Occasionally the response was more marked, and although the prolapses were all very recent when examined, it is possible that these represent those animals in which the condition had been present, perhaps intermittently, for more than eight to ten hours before examination. The early recognition of CVP may not always be easy, even in the particular circumstances of this investigation.

Conditions associated with endotoxaemia in the ruminant, result, initially, in a dramatic panleucopaenia within a few hours (Schalm et al., 1975), a response which was seen in a few of the cases. The two extremes of leucocyte response were probably responsible for the wide range which was encountered.
6.8: BIOCHEMISTRY:

Assessment of the metabolic parameters (including serum total protein, albumin, globulin, hydroxybutyrate, urea and plasma glucose) showed several important changes when compared to the control ewes. The major problem, highlighted by Sobiraj et al. (1986), was that the blood samples were taken from ewes already affected with CVP and it is difficult to be certain to what extent the changes were the result of the CVP or involved in its pathogenesis.

6.8.1: Protein parameters:

The protein analyses showed little difference between the control and affected groups although the albumin showed a minor variation from year to year which was of uncertain significance. The total protein and albumin values did tend to be lower in ewes affected by the more severe degrees of CVP. This may reflect the loss of serum protein or sequestration of significant amounts as a result of straining and circulatory compromise. It may also be possible that the protein was inherently lower in these animals as a result of an increased demand by the lambs. If an animal were suffering from a long term nutritional deficiency of protein, the total albumin may be expected to be low. If, however, ewes had suffered from long term inflammatory changes, then raised globulin levels may have been expected. The lack of any significant overall change in the protein levels indicated that, as a group, the affected ewes were not dehydrated, were in reasonable long term dietary protein balance and were not suffering from any sub-clinical intercurrent disease. These results corresponded to the clinical findings of reasonable bodily condition, a satisfactory level of nutrition in respect of protein, adequate internal parasite control and the absence of any major intercurrent disease. The latter aspect would seem to confirm Fowler and Evan’s (1957) opinion that, although epidemic forms of the
disease are occasionally seen, there is no metabolic evidence of an infectious cause for CVP.

6.8.2: Beta-hydroxybutyrate (BHBA):

One of the most interesting findings in the biochemistry of CVP cases was an apparent elevation in the serum BHBA levels in the affected ewes when compared to the controls. The extent of CVP showed a detectable effect on the values obtained; the more severe the prolapse the higher the levels of BHBA. There was a small but significant overall elevation in the diseased animals when compared to the controls. This metabolic parameter has been used as an index of the nutritional status of sheep (Russell, 1985). Russell (1985) suggested that a mean level of 0.8 mmoles/l in normal ewes approaching term and carrying multiple lambs indicated an adequate nutritional status. Russell, Maxwell, Sibbald and McDonald (1977), however, considered that ewes with plasma levels of 0.7 mmoles/l were moderately undernourished and while individual affected ewes in this study showed higher levels, the mean for the affected ewes was below this. A mean value of 0.63 mmoles/l was obtained from ewes affected by CVP. This is within the limit set by Russell (1985) as an indication of adequate dietary energy status. Affected ewes were generally in good lambing condition clinically, which might correspond to the adequate levels of BHBA, but the control ewes showed a lower mean value (0.42 mmoles/l). The reason for the difference is questionable but it is possible that the affected ewes were not eating adequately prior to the onset as a result of appetite suppression, as was suggested by Straiton (1973). This would seem to be likely as changes in the metabolic status of the rumen are notoriously slow and may not have been expected to result in a raised BHBA level within the limited time between onset and sampling. However, this presupposes that CVP is an acute condition and this may not invariably be the case (Bassett and Phillips, 1955b).

In a study on the effect of the feeding of sodium monensin to pregnant ewes Austin and Wilde (1985) found that normal ewes (carrying multiple lambs) in the last week of gestation, showed concentrations of BHBA between 0.55 and 0.86 mmoles/l. This
corresponds closely to the levels found in affected ewes in this investigation. No case in
the present work had a concentration over 0.93 mmoles/l. Only if elevated levels of BHBA
were demonstrable prior to the onset of CVP could an aetiological relationship be alluded
between the condition and the metabolic status of the ewe.

The significance of the raised BHBA requires further investigation, particularly as the
more severe CVP cases showed higher levels.

6.8.3: Urea:

The urea status of ruminants has been used effectively by Blowey (1975) as an index of
rumen protein digestion and short-term protein status. While this author has confirmed its
significance in normal, healthy cattle, disorders of the urogenital system introduce other
factors which may be responsible for influencing blood urea levels. Most authors,
including Bayly et al. (1936), Fethers (1939), Laing (1945, 1949), McLean (1956), Watson
(1959), Straiton (1973) and others, have concluded that clinically, and at post-mortem, the
bladder was invariably obstructed to some degree. Such obstruction may result in a
progressive rise in blood urea levels. The complete obstruction of the urethra, such as is
seen for example in urolithiasis, may induce a two-fold increase in urea within two to
three hours and then a progressive rise over several days. Death does not follow quickly
from such a situation. In this series of cases, which represent the only extensive group of
CVP cases studied in this respect, the mean urea level was found to be high (7.63
mmoles/l) when compared to the control ewes (4.51 mmoles/l). The range of values found
in the control ewes was relatively narrow (2.15 – 9.06 mmoles/l) while in the affected
ewes the range was 1.17 – 47.9 mmoles/l. It is unlikely that the high levels of urea in the
affected ewes could have arisen as a result of the dietary protein status and are not found
in normal animals. It is far more likely that these values arise from post-renal urinary
obstruction. In the absence of permanent renal damage, the values may fall quickly once
an obstruction has been relieved. This may explain why, in spite of some moderately
elevated urea levels, the ewes generally survived once urine flow had been re-established. Animals in which the levels were very high (over 20 mmoles/l), seldom survived (80% mortality), and in cases of long-standing CVP the urea may, therefore, be used as a prognostic indicator.

It was also noticeable that ewes affected by third and fourth degree CVP showed higher levels of urea than the milder types (first and second degree). This difference was statistically significant (p = 0.003) and indicates that, in the more severe CVP cases (which would, generally, have been present for a longer time and might result in a more complete obstruction), more obstructive uraemia is likely, and this in turn might result in poorer lamb and ewe survival. As stated by Watson (1959), early bladder relief is obviously an important part of the therapy. In the course of this work the reduction of the prolapse was often accompanied by a flood of urine passed under considerable pressure. This urine was often dark and contained obvious evidence of blood. All these factors point strongly to the fact that bladder obstruction is an important complication of CVP but, at post-mortem of the dead ewes, no bladder was found to be grossly enlarged. This may, possibly, be explained by the agonal passage of urine once tenesmus ceased and the bladder presumably fell back into the abdomen.

Unfortunately the creatinine levels were not obtained. High concentrations of creatinine may have supported the opinion that the high urea levels were the result of urinary obstruction or renal disease, whereas low levels may have indicated a pre-renal uraemia.

6.8.4 : Glucose :

Plasma glucose concentrations showed that the control group and the affected ewes were largely similar, but there was a noticeable increase in the more severely affected ewes. Although it was not significant, there was an impression that there was a reduced plasma glucose in those ewes with a first degree prolapse. It is possible that in these ewes the appetite was depressed at an early stage so depressing the available energy. The marked
rise in plasma glucose seen in the more severely affected ewes may be the result of a toxaemia induced stress response. Very high levels of plasma glucose were found in the ewes in extremis which may have been an agonal response. Further research into this aspect may clarify the reason for the elevation of plasma glucose in the more severe forms of CVP.

6.8.5: Calcium:

Cases of CVP showed significantly reduced serum calcium levels which were in agreement with the findings of Stubbings (1971) and Sobiraj et al. (1986). The levels did not appear to be sufficiently low to cause clinical hypocalcaemia (Doxey, 1983) but may be the result of stress (Moseley and Axford, 1973). This may resolve the doubts registered by Hindson and Turner (1972) who disputed the clinical significance of Stubbings' (1971) findings. In the more severe forms of CVP the serum calcium was found to be significantly lower than in the milder forms which may indicate an increased stress response.

The possibility that calcium is involved in the aetiology of CVP requires further investigation. This was impracticable from the practice situation in which this work was undertaken, but in view of the possible relationship between CVP and ringwomb and the belief that certain ringwomb cases respond to calcium therapy active research should be encouraged.

6.8.6: Hormones:

Koch and Haisch (1943) suggested the possibility of CVP being the result of an inherited hormonal deficiency and Edgar (1952) explored this by administering high levels of stilboestrol to pregnant sheep in an attempt to induce CVP. Sobiraj et al. (1986) followed the hormonal changes as parturition approached, in ewes affected by CVP and found that there were significant but subtle changes in the hormone status of affected
sheep. The finding by these latter workers of an increased level of 17-oestradiol in CVP cases appeared to support the belief that the condition was due to hormonal imbalances related more to the oestrogen status than to the progesterone. The normal changes in hormones in late pregnancy have been the subject of considerable work and in the present investigation some significant changes were, indeed, apparent.

The most obvious feature was the wide range of values obtained. This may be expected if the hormone changes in the terminal stages of pregnancy follow a set pattern and the timing of CVP relative to parturition was variable. Most cases occurred in the last 5-9 days of pregnancy when both hormones would be expected to be changing most rapidly.

The work by Challis (1971), Challis et al. (1971), Robertson and Smeaton (1973) and Tsang (1974, 1978) have shown that the most significant rise in oestrogen levels occurred in the last 24-48 hours of gestation. Levels of up to 411 pg/ml were found by Tsang (1974). In the present work the oestrogen levels were found to vary between 40 pg/ml and 860 pg/ml. These values were obtained from ewes between lambing and up to 29 days prepartum. The median value for the affected ewes was 180 pg/ml, compared with 320 pg/ml in control ewes. This showed that, contrary to the findings of Sobiraj et al. (1986), the concentrations were lower than in unaffected sheep and this difference was statistically significant.

The condition may, therefore, not be primarily related to an oestrogen-induced tissue slackness and oedema, as was proposed by Sobiraj et al. (1986). Edgar's (1952) attempts to reproduce the disease through the administration of very high doses of oestrogen (far higher than would ever occur naturally) failed to produce anything like the typical disorder. Roberts (1971) interpreted the investigations of Bennetts (1944) and Bennetts et al. (1946) into the occurrence of prolapse of the uterus following high levels of phyt-oestrogens in the diet, as indicating that CVP occurred under such conditions. Bennetts (1944), Bennetts et al. (1946), McLean (1956) and Cox (1981) emphasised, however, that CVP (as described in this thesis) was certainly not caused by such substances. Cox (1981)
noted consequently, that oestrogens cannot be the sole cause of CVP, and the findings of this work would seem to agree with this statement.

The levels of serum progesterone were found, in the present work, to be significantly elevated, when compared to those in control ewes. This differs from the findings of Sobiraj et al. (1986), who were unable to show any differences between normal and affected ewes. The work by Bassett and Phillips (1955a) and McLean and Claxton (1958) indicated that the dilatability of the vagina increased through pregnancy; it is suggested that this may correspond with the progressively rising progesterone concentrations described by Bassett et al. (1969) and it would seem unlikely that the very low levels of oestrogens present up to the final 48 hours or less before lambing could cause significant changes in the distendability of the birth canal.

The continued influence of progesterone may explain far better the tract changes recorded by Bassett and Phillips (1955a), particularly the greater vaginal dilatability and displacement capacity in CVP cases. Indeed, in this work, the relative rise in progesterone levels was marked and it would seem to be a more rational explanation for the aetiology than an oestrogen excess, although it is most unlikely that this is the only factor involved. The steady levels of progesterone found by Bassett et al. (1969), and others, after the 120th. day of gestation is not consistent with the wide range of values found here in affected ewes. The control ewes showed a much closer distribution of values, but the samples included some which were within hours or even minutes of lambing. These may normally be expected to have reduced progesterone levels (Tsang, 1978). Significantly, Tsang (1978) demonstrated a dramatic fall in circulating progesterone in the last two days of normal pregnancy. Only 8% of cases were seen in the last two days of gestation (excluding those subjected to elective caesarian section).

It is also of interest that progesterone concentrations are apparently elevated in ewes carrying twin lambs when compared with those carrying singletons (Stabenfeldt et al., 1972; Challis et al., 1971). During this work the greater uterine burden of ewes with CVP
compared to control ewes was quite apparent, and could explain the higher median value for progesterone concentrations in the affected animals. The progressive increase in the tract dilatability and displacement capacity in CVP cases found by Bassett and Phillips (1955a) indicated that progesterone has a long-term effect. This may also explain the high rate of recurrence found in this and other studies (Gerring, quoted in McLean, 1959).

The limitations following the collection of single samples in this way inevitably restricted the validity of the results and this aspect of the condition should be further explored, possibly through serial sampling of ewes approaching parturition. Indeed, the sampling of peripheral blood may not necessarily accurately reflect the effects on the genital tract or the concentrations in the myometrium or the vagina of either oestrogen or progesterone.

The concentrations of progesterone and oestrogen in ewes suffering from recurrence of CVP were not measured. This may warrant further investigation to determine whether ewes affected by CVP, or with a tendency towards the condition, have higher circulating progesterone concentrations than normal ewes. No investigations into the role of progesterone by the experimental administration of the hormone are evident. Koch and Haisch (1943), however, advocated the treatment of CVP in pregnant and non-pregnant cattle with chorionic gonadotrophin which might be expected to increase the progesterone levels. This may indicate that the disease seen in cattle is significantly different from that in the sheep.
Figure 120: SUCCESS!....it's not all bad!
6.9: CONCLUSIONS:

The initial objectives of this study, as set out in the introduction, were largely achieved and, indeed, the project turned into a far wider investigation than was at first envisaged. The limitations placed upon the investigation through being in practice are apparent throughout the work. There were omissions and errors of sampling, lost records and some dubious results as a consequence of the well meaning enthusiasm of some farmers and exhaustion on the part of the author, but overall this represents a considerable contribution to the knowledge of CVP and its limitations are accepted!

There is little doubt that CVP is a significant and distinct clinical entity. Its cost to the British sheep industry, with an incidence of 1.34% of pregnant ewes, in terms of mortality alone, can be calculated at about 100,000 animals per annum (assuming a total breeding population of 16 million ewes) (Annual Agricultural Returns, MAFF, 1984). Today this could mean a loss of at least 5 million pounds per year! Obviously this is an approximate figure, but the extent of the loss is significant.

The most likely factors in the aetiology of the disorder which emerged from this study are the significantly increased number of lambs carried by ewes affected by CVP (which may be either a result of managemental or hereditary factors or both), the elevated serum progesterone (which may in turn be related to the uterine burden) and the possible inherited tendency towards the condition.

The quality and quantity of the diet are likely to be important factors, particularly with respect to palatability. The length of the tail may also be important either through a mechanical effect or possibly other factors.
Diarrhoea or constipation were not regularly encountered and are unlikely to be directly or indirectly involved.

The condition occurs in both over- and under-conditioned sheep, generally up to 9 days before parturition. This is the stage of maximal lamb growth but there is little evidence to suppose that it is only the size and number of the lambs or the body condition of the ewe which are involved in the pathogenesis.

A major factor in the pathogenesis appears to be bladder obstruction and the resultant straining. If tenesmus is relieved effectively before severe systemic effects have developed, the physiological and physical state of affected ewes can be improved significantly.

The understanding of the pathogenesis was not greatly aided by this work but several important factors may be added to the already considerable list of features proposed by other workers. There is little doubt that the disorder may progress rapidly from the mild forms to the more severe. Such progression does not invariably follow and depends largely upon the extent and persistence of tenesmus.

Significant metabolic changes occurred in affected ewes. These were leucocytosis, hypocalcaemia, elevated concentrations of beta-hydroxybutyrate, uraemia and, terminally, hyperglycaemia. It is uncertain whether any or all of these were the result or the cause of CVP.

Complications as a result of CVP include the ruptured vagina syndrome, secondary ketosis, toxaemia, septicaemia and local infarction or necrosis of the exposed tissues. Ewes do not appear to tolerate the condition well, rapidly succumbing to the effects of the disorder upon the metabolism and physical status. The lambs in utero are adversely affected relatively early in the process and this was reflected in a high lamb mortality.

The consequences of CVP are severe and result in an inordinate number of ewes which either die, carry dead lambs or require various forms of treatment but in the early stages there do not appear to be any metabolic differences between affected ewes and normal
control animals. As the disorder progresses significant metabolic changes occur, any of which might in turn affect the prognosis for the ewe and her lambs.

The effectiveness of the treatment regimens employed for the correction and/or retention of CVP are largely dependent on the efficiency with which they are applied and the duration of the CVP. Provided that careful cleansing and lubrication of the exposed tissues are carried out at an early stage in the development of CVP, and that tenesmus is effectively controlled, the various retention methods described showed no great individual advantages. The use of elective caesarian section seemed to be an effective method of preserving the life of the ewe, but at the expense of the lambs. Any delay in resorting to surgery improved the lamb survival rate but reduced that of the dams.

If technically possible, the Buhner method appeared to be the best treatment. The Dalton's Spoon may be effective but had significant disadvantages. However, any method, provided it is applied correctly with anatomical understanding, should be reasonably effective.

While in many published studies limited investigations have been made into CVP in ewes, the study described in this thesis has attempted to highlight some of the gaps and to emphasise areas which require further research effort. Several young veterinary surgeons have already been inspired by the work and it is hoped that this will lead ultimately to its complete understanding, so that it will no longer be necessary to devote time, effort and financial resources to the hitherto disappointing treatment and control of CVP.
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9.0 : APPENDIX OF PUBLISHED PAPERS:

Permission to obtain a photocopy of the article published in the Veterinary Annual has been obtained and the article from the Veterinary Record is an official offprint obtained from the publisher.
Cervico-vaginal prolapse in the ewe

CERVICO-VAGINAL PROLAPSE (C-VP) occurs in a variety of animals but its greatest significance is in sheep in the last two weeks of pregnancy. In spite of its clinical and economic significance there have been few investigations into its aetiology, pathogenesis, treatment and prognosis. Edgar (1952), McLean (1956, 1957, 1959) and McLean and Claxton (1958, 1959, 1960) have reviewed the literature and carried out the only in-depth and controlled studies into C-VP. There have been several limited investigations into individual aspects of the disorder, by Jones (1958), Mayor (1958), Hughes-Ellis (1958), Hardy (1959), Watson (1959), Blackmore (1960), Fowler (1962), Hindson and Turner (1962), Eagelman et al. (1967), Stubbings (1971), Rusch and Berchtold (1978).

The consensus of opinion is that there is a persistent and unpredictable incidence varying between 0 and 11 per cent (Edgar, 1952) in the UK and in exceptional cases, up to 20 per cent or more. There is a general feeling that the aetiology is a complex of hereditary, hormonal, dietary, physical and environmental factors and no worker has substantiated any single factor.

There is little doubt that veterinary surgeons in practice will encounter cases of C-VP even though shepherds have become accustomed to undertaking treatment themselves in many cases. It would seem that the profession has abdicated its responsibility over this distressing disorder, possibly as a result of its uncertain aetiology, its poor prognosis and the frustrations of its occurrence.

TREATMENT

Many different approaches have been advocated but there are consistent statements made outlining the basic principles (Bayly et al., 1936; Fethers, 1939; Laing, 1945). These are:

i. Clean and disinfect the prolapsed tissue.
ii. Reduce the prolapse safely and completely.
iii. Prevent any recurrence.
iv. Control straining which might follow irritation or inflammation.
v. Control infection locally and systematically.
Thorough cleansing of the prolapse is often difficult due to the almost inevitable contamination and laceration. However, the use of warm, dilute disinfecting agents (e.g., Hibitane or Cetavlon) or soft soap may aid in this. A final flush with warm sterile saline is beneficial. The ewe should then be restrained upon her back by an assistant so that the hind legs are raised. Using gentle pressure and manipulation it is generally possible to reduce the prolapse.

Occasionally the bladder creates an obstruction and it is then necessary partially to empty it using a sharp, sterile hypodermic needle (Watson, 1959).

Other organs that may occupy the prolapse include the pregnant uterine horns in which the lambs may sometime be felt, or variable amounts of intestine. Both of these return to the abdomen readily once gravity and pressure have an effect. The raising of the hindquarters is most important and Laing (1945) found that tying a ewe to a fence in such a position was a crude but effective treatment on its own.

Having reduced the organ the patient often strains vigorously and the prolapse may reappear. If the ewe successfully passes urine, which may be intensely haemorrhagic, straining often subsides quickly.

A wide variety of methods have been used to retain the vagina including:

i. Tying perineal wool strands across the vulva. This is seldom sufficient except in the mildest of cases where no treatment might be expected to be as effective.

ii. Fitting a string, canvas or sacking truss (Fowler, 1982). This is effective in mild cases where tenesmus is not a problem.

iii. The ‘Arnolds’ type plastic or wire intravaginal device as described by Laing (1949), Fowler and Evans (1957) and Jones (1958). These have been used widely all over the world but have a tendency to penetrate the fornix in ewes that strain heavily. In the author’s experience they have little to commend them apart from allowing parturition without interference.

iv. Vulval closure. These are the most popular methods amongst shepherds and veterinary surgeons. It is common practice to use safety pins, particularly the ‘nappy’ locking type. These are, however, too small to avoid the lips of the vulva, penetration of which often induces severe straining and consequent damage both locally and to the prolapsed tissue. This often results in re-prolapse. Two or three ‘blanket’ or ‘kilt’ type pins are more suitable.

The use of vulval sutures follows the same principle and has the advantage that it is possible to place the sutures at the wool line and carefully adjust the tension. Umbilical tape, cat-gut or nylon suture material are suitable. Two or three such sutures are generally adequate. Horizontal mattress sutures should only be used with hollow quills as advocated by Rusch and Berchtold (1978). Tension is difficult to gauge
and it is easy to over-tighten or under-tighten the suture, with undesirable consequences. Modifications of the suture methods using buttons or 10–12 cm lengths of motor fan belt to spread the pressure are effective and sympathetic to the tissues. The 'fan belt' method is particularly useful as the belt has an inbuilt curvature and is often serrated, allowing locking of the sutures without undue tension.

vi. 'Buhner's' technique (Buhner, 1958). The method is described by Arthur et al. (1982) and is advocated strongly. A length of umbilical tape or heavy suture material is laid subcutaneously around the vulva with the aid of a long half-curved suture needle after small incisions have been made above and below the vulva. The 'purse-string' effect so obtained produces little irritation and maximal retention without excessive pressure. There is little risk of infection gaining access to the vulval tissues. Removal of this suture is, however, not always simple.

vii. Trans-pelvic retention (Eagelman et al., 1967). This method employs a flat button introduced against the dorsolateral vaginal wall and sutured directly to another button on the skin over the gluteal muscles. One such button is inserted on each side of the vagina. There are considerable advantages at parturition and by inducing fibrous adhesions, this technique may prevent recurrence at subsequent lambing.

viii. Caesarean section. This approach is advocated in the last 2–3 days of pregnancy when milk is present or when fetal membranes are evident. Earlier interference may be warranted, as suggested by Arthur et al. (1982), but it should be remembered that this may result in a poor prognosis for the lambs.

ix. Other methods advocated include: (a) postural restraint or elevation of the hind quarters by other means (Palsson, 1950; Laing, 1945); (b) exercise (Bayly et al., 1936); (c) vitamin D, calcium and phosphate supplementation (Chavance, 1944); (d) parenteral stilboestrol (Watson, 1959); (e) parenteral chorionic gonadotrophin (Koch and Haisch, 1943).

COMPLICATIONS

STRAINING

The use of local analgesics is often beneficial or essential. Roberts (1971) advocated surface analgesia while Hughes-Ellis (1958) used local infiltration. Epidural anaesthesia (Eagelman et al., 1967) or various regional blocks upon sacral or pelvic nerve routes have been suggested.
Lignocaine 2 per cent solution is commonly used but 25 per cent ethyl alcohol may be used to provide prolonged analgesia in regional or epidural methods (Hertrampf et al., 1978). Surface methods have a short duration of action and are often inadequate on their own. Prolonged tenesmus may be controlled by using an intra-epidural catheter through which 2 per cent lignocaine can be introduced as necessary. Complications arising from epidural infection or excessive analgesia are paralysis, meningitis or spinal bleeding.

**Repeated Prolapse**
There is little doubt that repeat episodes result in a progressively poorer prognosis.

**Lamming**
Methods of treatment involving vulval obstruction require supervision at lamming. Every effort should be made to ensure that straining is associated with labour before opening the vulva. Dystocia as a result of ‘non-dilatation of the cervix’ is a common sequel (Edgar, 1952; Kenworthy, 1957; Hindson and Turner (1962).

**Split Vagina**
Descent of abdominal organs through a vaginal tear is invariably fatal, though salvage of the lambs is occasionally possible.

**Infection and Laceration**
This may be the result of an incorrect retention technique or delayed treatment and is complicated still further by weakness and often by pregnancy toxaemia and carries a high mortality.

**Post-parturient C-VP or Uterine Prolapse**
Ewes with damaged birth canals may strain and produce either a persistent C-VP or a uterine prolapse. In either case treatment may be possible using standard methods.

**Prognosis**
C-VP may occur in all classes of sheep of the susceptible breeds (Edgar, 1952) but the prognosis depends upon the severity and upon the efficiency of treatment. Young ewes carry a worse prognosis, even where the prolapse is mild. Any delay in treatment results in a worse outlook and every effort must be made to avoid delay. Several workers have suggested an hereditary predisposition and a tendency to recurrence and advocate culling of the affected ewes and their progeny (Laing, 1949; Edgar, 1952). Watson (1959) could offer no support for this theory.
CONTROL

Any control measures must rely upon knowledge of the aetiology, and as little is known, control advice is likely to be uncertain. The incidence of C-VP may be reduced by encouraging exercise, reducing the amount of bulk forage of low nutritional value and by avoiding environmental conditions that tend to produce the disorder. This may not always be feasible, of course, but until more is known, we are left with this somewhat vague and unsatisfactory answer.

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Vaginal rupture associated with herniation of abdominal viscera in pregnant ewes

D. C. Knottenbelt

Seventeen cases of vaginal rupture with herniation of abdominal organs were examined. The injury consisted of a dorsal tear in the vagina and, most frequently, eversion of the bowel. The tear was accompanied by extensive vaginal bleeding. Serum calcium concentrations were low in the affected cases and the concentrations of β-hydroxybutyric acid and urea were high. The average age of the affected ewes was four years (range 3 to 6). They were generally in poor condition and carrying more than one fetus (2-7 lambs per ewe) although younger ewes and ewes in good condition were also affected. No primiparous ewes were affected. Ten of the 17 ewes were found dead and six were humanely destroyed; in one, the injury was repaired surgically but the ewe died 48 hours later.

AN outbreak of an 'unexplained' fatal disorder of heavily pregnant ewes characterised by prolapse of variable lengths of intestine through a vaginal tear was described by White (1961). He gave no explanation for the condition and having at first concluded that it was the result of sadistic interference came to recognise it as a distinct clinical entity. The fourteen of the intestines, bladder or fat through a vaginal, cervical or uterine rupture, was recorded by Roberts (1971). He described the condition as having a poor prognosis and recommended euthanasia, although he suggested a possible method of repair by laparotomy, McLean (1950) described a similar condition arising as a complication of acute vaginal prolapse accompanied by continuous tenesmus; death usually following from the concomitant haemorrhage. Fox (1962) recorded a single case of vaginal rupture, apparently unrelated to vaginal prolapse or hypocalcaemia. Arthur and others (1982) pointed out that vaginal rupture, vaginal prolapse and 'ring-womb' (retarded cervical dilation) occur under similar seasonal conditions.

As part of a study of vaginal prolapse in pregnant ewes, carried out in the United Kingdom over a four year period, 17 cases of vaginal rupture were encountered and the opportunity was taken to clarify some of the features of the disorder.

Materials and methods

Case material

Seventeen cases of vaginal rupture were seen during four years in general practice in Warwickshire. They were examined as soon as practicable after being found, the breed of the ewe was recorded and its age estimated according to Miller and Robertson (1947). Details of parity, management and feeding were obtained from the shepherd. The body condition was determined according to Hughes (1981).

In 10 cases the ewe was found dead and of the remaining cases one was treated surgically. Six cases were euthanized immediately and a laparotomy was performed in an attempt to save the lambs.

Surgical repair of the vaginal tear was undertaken in one case. After scrupulous cleansing and reduction of the herniated length of small intestine the vaginal tear was closed using absorbable suture material (Dexon; Davis and Geck) in a continuous Lambert pattern. The ewe was treated with amoxycillin (Clamoxyl; Beecham Animal Health) at 7 mg/kg daily, trenbolone acetate (Finajet; Hoechst UK) at 5 mg/kg and multivitamins (Vitatonic; Form Laboratorys). The ewe died approximately 48 hours later. A post mortem examination did not reveal any specific cause of death.

Blood samples were taken from five of the seven live ewes into lithium heparin, fluoride oxalate and plain 'vacutainers' (Becton-Dickinson). Samples were also obtained from 'normal' ewes of the same breed, age and gestational stage from the same flocks as the five affected animals. Rectal faeces samples were obtained for helminth egg counts using saturated sodium chloride solution as flotation medium and a MacMaster counting chamber.

Post mortem examinations

Post mortem examinations were performed on 16 ewes. The herniated tissues were identified and the extent of the vaginal and intra-abdominal bleeding recorded. The site and extent of the vaginal tearing, and the presence of any vaginal contamination, were also recorded. A rough estimate of the gestational stage and maturity of the lamb was made by using the size, coat maturity and dentition of the lambs and the vulval and mammary development of the ewe. The weight of some of the lambs was measured after gross liquid had been wiped off.

Particular attention was given to the presence of vaginal prolapse at the time of examination or any history of vaginal prolapse before vaginal rupture and to the presence of any vaginal contamination. Several ewes had been seen to show prolapse before rupture occurred but were not found to have a vaginal prolapse at the time of examination.

Laboratory measurements

Haemoglobin, haematocrit and total leucocyte counts were estimated using a Coulter S cell counter with auto-diluter. Differential leucocyte counts were obtained from thin smears stained with Wright-Giemsa (DIF OIK; Dade, Harleco). Total protein, albumin, urea, glucose, calcium, inorganic phosphate, magnesium and creatine kinase estimations were carried out within 12 hours using commercially available kits (BCL) and a Cecil 303 diffraction-grating spectrophotometer (Cecil Instruments). The methods were standardised with Precinorm and Precinorm S quality control solutions (BCL) for
each sample. Beta-hydroxybutyric acid levels were determined using the method described by Williamson and Melanby (1970). Selenium levels were derived from glutathione peroxidase estimations, carried out at the Worcester Veterinary Investigation Centre.

Plasma progesterone and oestrogens were measured by radioimmune assay by the methods of Challis and others (1973) and Challis (1971) respectively. The antiserum for oestrogen was raised in goats (Specific Antisera). The intra- and inter-assay coefficients of variation were 9-1 per cent and 21-6 per cent at 113-7 pg per tube, respectively. The progesterone antiserum was raised in goats (Specific Antisera) and the intra- and inter-assay coefficients of variation were 17-4 per cent and 18-8 per cent at 1-4 ng per tube, respectively.

Results

Clinical findings

Seven of the 17 cases (41 per cent) were mules (Table 1) but as this breed was very commonly encountered in the survey, this high proportion was probably not significant. The average age of the affected ewes was four years (ranging from three to over six years) (Table 1). Body condition varied from poor to very good (score 2 to 4); 10 ewes were in poor condition, six were in a condition suitable for lambing and one was grossly fat (Table 1).

Seven ewes were examined while they were alive and surgical treatment was attempted in one case. The remaining 10 cases were examined after death.

Six ewes were destroyed and a laparotomy performed immediately. Only one viable lamb was obtained (case C50) from a possible 16 lambs. Eight of the remaining 10 ewes, found dead, carried a total of 22 lambs. The average number of lambs carried by the affected ewes was 2-7 lambs per ewe.

Three ewes were at full term pregnancy, as judged by the presence of colostrum in the udder and cervical dilatation. Nine other ewes were estimated to be up to 14 days prepartum.

Fourteen cases showed prolapse of variable lengths of large and small intestine (Fig 1).

In one case (C136) the entire genital tract from the ovarian ligaments to the cervix had herniated through the vaginal tear and had been physically separated from the ewe (Fig 2). There was extensive haemorrhage in the abdomen; the uterus contained three normal lambs and colostrum was present in the mammary glands. Case C90 had a prolapse of small intestine and a single gravid uterine horn as described by Fox (1962).

In those ewes examined the vaginal tear was apparently longitudinal in the dorsal wall, and varied in length from approximately 5 cm to 15 cm. Extensive bruising, laceration and haemorrhage made it difficult to assess the tear accurately. Case V43 showed vaginal tearing and haemorrhage but no prolapsed viscera. Pelvic and subperitoneal haemorrhages were seen in all cases and 12 showed extensive vaginal haemorrhage.

Vaginal prolapse had occurred before the vaginal rupture in five cases. Tenesmus had been marked in these cases. Of the remaining 12 cases, no information was available on the presence of vaginal prolapse, but five of them showed marked vaginal contamination (Table 1). Case C49b had a history of a mild vaginal prolapse the previous year and a slightly more severe prolapse preceded the rupture. It was estimated to be close to term and no treatment had therefore been undertaken for the prolapse; the ewe was found dead 24 hours later with extensive evagination of the bowel.

Ten ewes were found to have grossly fatty livers post mortem and those in poor condition showed little abdominal fat.

Management systems

Fifteen of the cases were from housed or open yarded flocks and only two from extensively managed systems. Feeding methods varied widely, but all the ewes had received whole or crushed barley as part of their diet. All the affected ewes showed evidence of loose faeces.

The ewes had been vaccinated against enterotoxaemia and tetanus and they had all received anthelmintic therapy within the previous two months.

Laboratory results

Worm egg counts showed that only four ewes had faecal evidence of parasitism.

The mean haemoglobin and haematocrit levels were 125 g/litre and 34 per cent respectively, compared with 109 g/litre and 33 per cent in the control ewes. These values are within the normal range (Doxey 1983).

The total leucocyte count of the affected ewes were generally high (mean 10-46 × 10⁶/litre) and showed a marked neutrophilia with a 'shift to the left'. The mean leucocyte count for the control ewes was 6-76 × 10⁶/litre and that for normal ewes is between 4-0 and 10-0 × 10⁶/litre (Doxey 1983). The absolute neutrophil count was 6-44 × 10⁶/litre in the affected ewes and 2-32 × 10⁶/litre in the control ewes; Doxey (1983) quotes a range of 0-4 to 5-0 × 10⁶/litre.

Serum urea and plasma glucose levels were high. Beta-hydroxybutyric acid concentrations were higher than in the control ewes and serum calcium concentrations were lower (Table 2). Other measurements were not significantly different (Table 2). Values quoted by Doxey (1983) and Kelly (1984) are shown for comparative purposes in view of the small number of control ewes sampled.

There were no significant differences between the normal

![FIG 1: A typical case (C153) of vaginal rupture with herniation of small and large bowel showing the vaginal tear and haemorrhage](image-url)
TABLE 1: Clinical descriptions of ewes with vaginal rupture

<table>
<thead>
<tr>
<th>Case</th>
<th>Breed</th>
<th>Age</th>
<th>Parity</th>
<th>Body condition</th>
<th>Live or dead</th>
<th>Number of lambs</th>
<th>Weight of lambs (kg)</th>
<th>Prena-</th>
<th>Pro-</th>
<th>Vagina-</th>
<th>Cervico-</th>
<th>Vaginal</th>
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<tr>
<td>C14</td>
<td>Masham</td>
<td>6</td>
<td>5</td>
<td>2</td>
<td>Dead</td>
<td>3</td>
<td>2.5</td>
<td>14</td>
<td>Gut</td>
<td>+</td>
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<td>NK</td>
</tr>
<tr>
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<td>Leicester</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>Live (T)</td>
<td>2</td>
<td>NK</td>
<td>0</td>
<td>Gut</td>
<td>-</td>
<td>-</td>
<td>NK</td>
</tr>
<tr>
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<td>Suffolk X</td>
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<td>5</td>
<td>3</td>
<td>Dead</td>
<td>10-4</td>
<td>0</td>
<td>Gut</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>C50</td>
<td>Suffolk</td>
<td>6</td>
<td>nk</td>
<td>2</td>
<td>Live (E)</td>
<td>3 (live)</td>
<td>NK</td>
<td>2-3</td>
<td>Gut</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>C54</td>
<td>Mule</td>
<td>4</td>
<td>3</td>
<td>2</td>
<td>Live (E)</td>
<td>1</td>
<td>5-1</td>
<td>0</td>
<td>Gut</td>
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<td>+</td>
<td>+</td>
</tr>
<tr>
<td>C59a</td>
<td>Mule</td>
<td>4</td>
<td>3</td>
<td>3</td>
<td>Dead</td>
<td>7</td>
<td>Gut + one</td>
<td>ulcerine horn</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>C116</td>
<td>Mule</td>
<td>3</td>
<td>2</td>
<td>3</td>
<td>Live (E)</td>
<td>2</td>
<td>NK</td>
<td>2-4</td>
<td>Gut</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>C134</td>
<td>Masham</td>
<td>5</td>
<td>5</td>
<td>2</td>
<td>Dead</td>
<td>3</td>
<td>NK</td>
<td>7</td>
<td>Gut</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>C136</td>
<td>Masham</td>
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<td>2</td>
<td>2</td>
<td>Dead</td>
<td>3</td>
<td>NK</td>
<td>1</td>
<td>Gut +</td>
<td>one uterus</td>
<td>-</td>
<td>+</td>
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<tr>
<td>C148</td>
<td>Mule</td>
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<td>4</td>
<td>2</td>
<td>Live (E)</td>
<td>3</td>
<td>NK</td>
<td>7</td>
<td>Gut</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>C153</td>
<td>Suffolk X</td>
<td>3</td>
<td>2</td>
<td>3</td>
<td>Dead</td>
<td>3</td>
<td>NK</td>
<td>7</td>
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<td>+</td>
</tr>
<tr>
<td>C154</td>
<td>Mule</td>
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<td>2</td>
<td>2</td>
<td>Dead</td>
<td>nk</td>
<td>NK</td>
<td>2</td>
<td>Gut</td>
<td>+</td>
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</tr>
<tr>
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<td>Mule</td>
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<td>3</td>
<td>4</td>
<td>Live (E)</td>
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<td>11.6</td>
<td>1</td>
<td>Gut</td>
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<td>+</td>
<td>+</td>
</tr>
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<td>V20</td>
<td>Leicester X</td>
<td>6</td>
<td>nk</td>
<td>2</td>
<td>Live (E)</td>
<td>3</td>
<td>NK</td>
<td>Gut</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>V43</td>
<td>Suffolk X</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>Death</td>
<td>3</td>
<td>NK</td>
<td>Nil</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<td>nk</td>
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<td>Dead</td>
<td>nk</td>
<td>NK</td>
<td>Gut</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>V65</td>
<td>Dorset X</td>
<td>4</td>
<td>4</td>
<td>2</td>
<td>Dead</td>
<td>3</td>
<td>NK</td>
<td>Gut</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
</tbody>
</table>

E Euthanasia  
T Treated  
NK Not known

TABLE 2: Mean (± sd) of biochemical parameters of affected and control ewes with values given by Doxey (1971) and Kelly (1984)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Total protein (g/litre)</th>
<th>Albumin (g/litre)</th>
<th>Urea (mmol/litre)</th>
<th>Glucose (mmol/litre)</th>
<th>β-hydroxybutyrate (μmol/litre)</th>
<th>Selenium (μmol/litre)</th>
<th>Creatine kinase (μl/litre)</th>
<th>Calcium (mmol/litre)</th>
<th>Inorganic phosphate (mmol/litre)</th>
<th>Magnesium (mmol/litre)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Affected ewes (5)</td>
<td>62.0 (5.3)</td>
<td>30.2 (3.6)</td>
<td>7.19 (1.39)</td>
<td>3.08 (1.69)</td>
<td>1.09 (0.18)</td>
<td>3.90 (0.25)</td>
<td>58.35 (5.2)</td>
<td>2.06 (0.13)</td>
<td>1.43 (0.34)</td>
<td>0.29 (0.10)</td>
</tr>
<tr>
<td>Control ewes (5)</td>
<td>66.4 (6.2)</td>
<td>32.0 (7.5)</td>
<td>4.27 (1.08)</td>
<td>2.31 (0.66)</td>
<td>0.81 (0.16)</td>
<td>1.45 (0.38)</td>
<td>30.9 (5)</td>
<td>2.40 (0.23)</td>
<td>1.36 (0.12)</td>
<td>0.87 (0.09)</td>
</tr>
<tr>
<td>Doxey (1983)</td>
<td>81.4 (4.9)</td>
<td>30.8 (2.6)</td>
<td>2.65 (7.6)</td>
<td>3.32 (3.3)</td>
<td>0.25 (0.08)</td>
<td>1.89 (0.25)</td>
<td>50.27 (7)</td>
<td>2.50 (0.07)</td>
<td>2.03 (0.64)</td>
<td>0.90 (0.00)</td>
</tr>
<tr>
<td>Kelly (1984)</td>
<td>60.79</td>
<td>29</td>
<td>1.66 (6.6)</td>
<td>2.11 (3.33)</td>
<td>-</td>
<td>-</td>
<td>0.9 (9)</td>
<td>2.23 (2.0)</td>
<td>1.23 (1.4)</td>
<td>1.0 (1.4)</td>
</tr>
</tbody>
</table>

Discussion

Little is known about vaginal rupture although shepherds have recognised its existence for many years (D. Slatter personal communication) and have come to accept it as an inevitable, if unexplained, loss. White (1961) described an episode in which four cases occurred in one flock on successive days and it is possible therefore that 'epidemic forms' of the disease may occur. Of the eight cases he described seven were Border Leicester ewes.


The results of this investigation would seem to confirm this observation although the condition may apparently be unrelated to vaginal prolapses. It is possible that vaginal rupture results from excessive tenesmus which may precede or follow a vaginal prolapse. The extent of the prolapse need not be severe and it is apparent that even slight or intermittent prolapse may precede a rupture. Bassett and Phillips (1955a, 1955b) recorded the intrapelvic anatomical changes that occur during normal pregnancy and in cases of vaginal prolapse. They observed that the peritoneum of ewes with a prolapse was severely stretched and that it showed 'lines of stress'. They also observed blood clots in the peritoneum and under the peritoneal lining of the dorsal wall of the pelvic cavity. In every case investigated in this survey, the tear was found in the dorsal vaginal wall and vaginal haemorrhage was generally heavy. Once the tear in the vaginal wall has occurred, the eversion of abdominal viscera occurs rapidly and together with haemorrhage, results in shock and subsequent death.

Serum calcium concentrations were low but the number of cases precludes a statistical analysis. Stubbings (1971) and Sobrare and others (1986) found low calcium levels in ewes with vaginal prolapse, and in their cases, as in the present series the blood samples were obtained after the ewes were affected. It is therefore uncertain whether the reduced levels were a result of the condition or preceded it. Moseley and Axford (1973) have shown that the serum calcium level may fall significantly under conditions of stress. It is not possible to conclude therefore that hypocalcaemia is an aetiologically significant feature of vaginal rupture. No case was considered to be affected by clinical hypocalcaemia although the values published by Kelly (1984) (Table 2) would indicate that the values were outside the normal range. The concentrations of serum β-hydroxybutyrate appeared to be slightly increased and fell within the range which Russell (1985) considers indicative of slight underfeeding. The selenium levels were indicative of a low marginal status but there was no evidence of a significant rise in the creatine kinase levels. Urea levels were above the higher end of the normal range quoted by Kelly (1984). This may have been a consequence either of catalysis or urine retention.

Other blood biochemical values showed little change from

TABLE 3: Mean (± sd) concentrations of progesterone and oestrogen in serum of affected and control ewes

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Progesterone (ng/ml)</th>
<th>Oestrogen (pg/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Affected ewes (5)</td>
<td>3.2 (2.13)</td>
<td>252 (185)</td>
</tr>
<tr>
<td>Control ewes (5)</td>
<td>3.36 (1.89)</td>
<td>460 (262)</td>
</tr>
</tbody>
</table>
the normal. The number of cases from which blood samples could be obtained was too low to allow statistical analysis.

Vaginal rupture appears to occur more frequently in ewes carrying more than one lamb and in ewes after the second pregnancy. The average number of lambs carried by the affected ewes was 2-7 per ewe, considerably higher than the 1-5 lambs per ewe in the unaffected ewes and the 2-2 lambs per ewe found in ewes affected with volapalpse (unpublished data). The additional uterine burden would increase the intra-abdominal mass. However, two cases carried only single lambs and another carried three lambs with a total litter weight of only 2-5 kg, suggesting that the litter size and weight may be only a minor factor in the aetiology of the condition.

No case was seen in primiparous ewes and older ewes in poor condition featured prominently among the 17 cases. Generally when vaginal rupture occurred in younger ewes they were in good lambing condition. That the majority of the cases affected came from housed flocks suggests that this may be a factor in the occurrence of the condition.

It is not clear why some ewes suffer from vaginal rupture after a relatively mild or even inapparent prolapse while others sustain a severe prolapse without vaginal rupture. Ten cases sustained both conditions but the extent of strain is not related to the apparent severity of either condition. The significance of diarrhoea is similarly not clear but it may be possible that it is in some way linked to tissue weakness in the vaginal musculature or the supporting structures of the genital tract, or to the onset of tenesmus.

Vaginal prolapse may apparently resolve spontaneously once the bowel is herniated which suggests that intra-abdominal pressure may be as important in the aetiology of vaginal rupture as is suggested by McLean and Claxton (1960) for vaginal prolapse.

Serum progesterone and 17-β-oestradiol levels in ewes affected by vaginal prolapse have been recorded by Sobiraj and others (1986). They claimed that cases of prolapse had higher levels of 17-β-oestradiol and progesterone than controls, and that progesterone showed a delayed fall toward parturition in affected ewes. In the present series, however, the serum oestrogen and progesterone concentrations were not significantly different from normal. Sobiraj and others (1986) suggested that a raised oestrogen level may cause oedema and, possibly, weakness of the parietal canal. The number of samples obtained was too low for statistical analysis and this aspect requires further investigation.

The apparent slight underfeeding, as indicated by the increased β-hydroxybutyrate concentrations (Russell 1985), may have been related to the ewes' poorer body condition or to a subclinical tissue weakness even in those ewes with an acceptable body score.

The poor prognosis, suggested by Roberts (1971), for both the ewes and the lambs is supported by this study. The one treated case died after 48 hours. Ewes found alive generally failed to sustain live lambs even for limited periods; immediate lambing does not, therefore, appear to be a useful approach. Until more information is available concerning vaginal prolapse and rupture it is difficult to see how they can be controlled. Dietary changes may help but any dramatic changes may induce a more serious metabolic disease and increase in roughage intake may merely induce an increased incidence of vaginal prolapse (Bayly and others 1936, Laing 1949, Edgar 1952, McLean and Claxton 1960) and thereby an increase in the incidence of vaginal rupture. The results of this investigation suggest that two of the factors which may be involved in the aetiology of the disorder are an increased litter size and inadequate or poor nutrition in housed flocks. The availability of ultrasound pregnancy diagnosis may help to identify ewes with more than one lamb, which may be considered to be at risk.

Every effort should be made to prevent and treat diarrhoea in heavily pregnant ewes and, by careful shepherding, to reduce the incidence of vaginal prolapse which appears to be a precursor to most cases of vaginal rupture. Gentle exercise, particularly for housed ewes, may help to reduce the incidence of vaginal prolapse and improve the general health of the breeding flock.

Acknowledgements.—I thank Mr B. Needham and Mr P. W. Moody, the staff of the MAFF veterinary investigation centre at Worcester and the farmers of Warwickshire for their help and co-operation. Hormone analyses were performed by the department of obstetrics, Royal Veterinary College, Professor D. E. Noakes, Professor F. W. G. Hill and Dr D. D. Duxey gave constant encouragement and the project was supported through the Mackellar Award.

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Liver glycogen in slaughtered pigs

GLYCOGEN was assayed in the livers of 1873 pigs within 20 to 30 minutes of death. From previous data, the relationship between liver glycogen concentration and fasting time was used to predict how long the pigs had been without food. Of the 1873 pigs, three-quarters had been fasted for more than 8 h, half for more than 18-7 h and one quarter for over 30 h before slaughter. Long fasting times are associated with a reduction in carcass yield and an increase in the incidence of dark, firm, dry (DFD) meat. In addition, lasts of 12 h or more have been found to be associated with increased aggression between pigs.