Associations between placental parameters, mare and foal body condition score at birth, and foal health in the first month of life.

Research Project Institute of Veterinary, Animal and Biomedical Sciences
Massey University, New Zealand
Drs. N. Wester (3052133)
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Project Supervisors:
Dr. E.K. Gee (NZ)
Prof. Dr. P.R. Van Weeren (NLD)

Prefatory note

Within the training of Veterinary Medicine at the University of Utrecht all students have to fulfil a research project. This paper is the final report of the research project carried out by N. Wester at the Equine section of the Institute of Veterinary, Animal and Biomedical Sciences, Massey University Palmerston North, New Zealand.
Research was executed to determine the associations between mare and foal body condition score at birth, placental parameters, and health status of the foal during the first month of life.

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1. Abstract

Aim of the study: To determine the associations between mare and foal body condition score at birth, placental parameters, and health status of the foal during the first month of life

Hypothesis: Poor body condition of foals at birth is associated with abnormal placental parameters and increased risk of diseases in the first month of life.

Materials and methods: Foals and dams were condition scored using the method of Henneke *et al*. at birth and one month later. Placenta examination has been done in a same manner described by Whitwell et al (1975). Post mortem examination of the equine fetus occurred in a manner similar as described by Schlafer (2004).

The fetal membranes and health data records were collected from 2 local Thoroughbred studs. During the three months of the study placentas from 68 Thoroughbreds mares were collected.

For body condition scoring after one month, 26 mares and foals were scored

Results: Placenta dimensions found in the current study are in the same range as results found by other authors. No significant difference and no correlation was found between foal body condition score at birth and weight of the chorioallantois (p = 0.504). No significant difference could be found between the use of oxytocin or no use of oxytocin to pass the placenta (p = 0.452). Different abnormalities were found at placenta examination.

There is a significant difference for the mares body condition score (BCS) at foaling between both studs (p = 0.023). For the change in body condition score of the mare and the foal body condition score at one month of age a statistical significant difference is found (p = 0.008).

6.5 percent of the mares had a prolonged stage 2 labour. 90 percent of the mares passed their placenta in the normal time period. 51.8 percent of the foals got sternal in range. A small proportion of the foals, 14.3 percent, had a delay in standing up. At least 67.9 percent of the foals did not suckle within the first hour of life.

Conclusions: Not enough information about foal health in the first month of life was known to make a correlation between body condition score or placental parameters.

Because of the small data set and the small amount of time we cannot concluded if poor body condition of foals at birth is associated with abnormal placental parameters and increased risk of diseases in the first month of life.

2. Introduction

The placenta provides a pathway for the interchange of essential gases, nutrients and waste products between foetal and maternal vascular systems throughout gestation. Any deficiencies in placental function may be reflected in corresponding deficits in foetal growth and maturation. Foal body condition score at birth provides an indirect assessment of the adequacy of placental function during late gestation. Placental dysfunction may result in poor foal body condition at birth, and may predispose the foal to an increased disease susceptibility in the perinatal period.

The equine placenta is defined as diffuse, microcotyledenary and epitheliochorionic in character. The normal placenta consists of the chorioallantois, the amnion and the placental vasculature. The placental vasculature is incorporated in the umbilical cord. The membranes are all of fetal origin, examination of the fetal membranes is important for predicting the foals perinatal health. Information about the mare's endometrium can also be gained.

This study aims to determine the associations between mare and foal body condition score at birth, placental parameters, and health status of the foal during the first month of life. We aimed to determine if there was a strong association between poor body condition of foals at birth and abnormal placental parameters, also, if lower body condition score at birth was associated with an increased risk of diseases in the first month of life.

The hypothesis of this study:
Poor body condition of foals at birth is associated with abnormal placental parameters and increased risk of diseases in the first month of life.

2.1 Equine pregnancy

It is accepted that the mean gestation length of horses is around 340 days. Research on Thoroughbreds describes an actual range between 316 to 364 days (Whitwell & Jeffcott, 1975). During this gestation period a complex of fetal and maternal membranes develops. Evaluation of the fetal membranes at term has been done by different authors (Rossdale & Ricketts, 2002). Whitwell and Jeffcott (1975), undertook a study concerning 145 normal thoroughbred foalings with normal foal viability, and 10 pony foalings in order to define morphologically and quantitatively characteristics of normal fetal membranes, compared with those found by other authors (Whitwell & Jeffcott, 1975).

During equine pregnancy dynamic and physical interactions between uterus and conceptus occur. Essential parts of the pregnancy are embryo mobility, fixation and orientation, embryo reduction, formation of endometrial cups, fetal mobility, fetal presentation, uterine horn closures, encasement of the fetal hind limbs by a uterine horn and special mechanisms of uterine fetal rotations during parturition (Ginther, 1998).

The hormonal process at pregnancy differs from the normal cycle. When an embryo is present, the luteal response to pregnancy develops and luteolysis must be blocked in order for the embryo to develop. Progesterone, made by the corpus luteum, is vital to embryo development.

Hormones going to the ovaries are transported via a systemic pathway, instead of a local pathway like in cattle. The ovarian artery does not contact the uteroovarian vein, as a consequence hormones cannot be exchanged between these two.

The embryo

After fertilisation of the female egg an embryo will be formed in the oviduct of the mare. The embryo (till day 39, day 0 = ovulation) becomes a blastocyst by the time it enters a uterine horn on day 6 or soon after, and its development has been reviewed in detail by Allen & Stewart (2001) and Ginther (1998).

Briefly, the blastocyst consists of a central cavity and an inner cell mass which is established at one pole. This inner cell mass will form the embryonic disk, from which the embryo will later develop. The blastocyst has different layers. The capsule surrounds the whole blastocyst, and this only develops in equine species. It comprises a tough, thin, mucinous layer of glycoproteins, and develops between the trophoblast and zona pellucida. The membrane surrounding the blastocyst cavity is a single layer of ectodermal cells, the trophoblast, an absorptive placental surface in contact with the endometrium. The conversion of the single-layered wall of the blastocyst to a two-layered wall is complete around day 12, an encirclement of the blastocyst cavity by a single layer of endodermal cells occurs. This results in a primitive placental vesicle, the yolk sac. The endoderm of the yolk sac is continuous with the endoderm of the primitive gut of the embryo proper. Growth starts at the mesoderm from the embryonic disk. Eventually from the mesoderm a three-layered wall develops. The mesoderm differentiates into supportive connective tissue and blood vessels.

Mobility

Uterine mobility of the embryo occurs from the day the embryo enters the uterine horn until day 15-17. During this period the embryo can be found anywhere in the uterine lumen from the tips of either horn to the cervix. The embryo can move from one horn to another 10 to 20 times per day (Ginther, 1998). The embryo mobility is dependent on uterine contractions and is favoured by the spherical form of the vesicle, the turgidity and anti-adhesive quality of the vesicle resulting from the capsule and the longitudinal arrangement of the uterine folds (Ginther, 1998).

Fixation

Until the moment of fixation the embryo is free to move. Ginther *et al.* (1998, and earlier work) found some interesting phenomena occurring during fixation: fixation is almost always in the caudal portion of one of the uterine horns, there is a lack of agreement between side of ovulation and side of fixation, embryo fixation in postpartum mares is in the involuted horn more frequently, there is a greater incidence of unilateral than bilateral fixation in mares with twins, and the ability of a small conceptus to block the uterine luteolytic mechanism throughout a relatively large uterus (Ginther, 1998).

On the day of fixation the embryo is still spherical. But the exocoelom forms within the mesoderm near the embryo proper, dividing the mesoderm into two layers. The amnion will

arise from folds of ectoderm and mesoderm who begin to pass over the embryo proper. The chorion will consist layers of ectoderm and mesoderm. Fixation occurs usually near a flexure in the caudal portion of one of the uterine horns, the point with the greatest impair to mobility of the conceptus.

After fixation

The embryo proper will be at the ventral aspect of the yolk sac. Uterine contractions continue after fixation and this may contribute to the embryo`s orientation. The yolk sac is three layered and vascularised at the embryonic pole and two layered at the opposite pole. It does not contain stored food, but the vascularisation seems to be an efficient distributor of nutritive material from the uterus to the embryo.

By day 21, a complete amniotic cavity exists. The allantois has begun to emerge from the hindgut and it grows into the exocoelom. This becomes prominent on day 24. The growing allantois forms a sac under the amnion and the embryo proper, and the embryo will be lifted up from the floor of the vesicle. By day 24 the union of the chorion and the allantois begins to form, this is the beginning of the allantochorionic placenta. Blood vessels develop in the mesoderm of the allantoic portion and vascularise the allantochorion and the amniochorion.

The fetus

The fetus or fetal stage is defined to run from day 40 to parturition. On day 40 the beginning of fetal activity starts, the beginning of the umbilical cord formation occurs and the replacement of the yolk sac by the allantoic sac is near completion.

Fetal activity is measurable at day 40, first detected are fetal head nodes. By day 46 detectable limb movements occur. By day 48, when the fetus reaches the floor of the allantoic sac, whole body activity is sufficient to raise the fetus a few millimetres of the allantoic floor. Fetal mobility is sometimes attributable to extraneous movements, like movements of the mare or intestine, but primarily it depends of the fetus.

At term the final position of the foal is in cranial presentation in 99 % of the cases (Ginther, 1998). Both uterine horns close by month 8, this causes the placental fluids to stay around the conceptus, in the body of the uterus (Knottenbelt, 2003). At this time the fetus is confined to the uterine body and is prepared to undergo the change to the cranial presentation. Its front is toward the mare’s cervix, the hind limbs will be fitting in the cord horn and the placental membranes are close to the limbs. The hind limbs will begin to enter during months 7 and 8. This can only happen if the fetus is in dorsal recumbency, because the horn and body are at an acute angle then.

Rapid fetal growth occurs in the latest three months of gestation. The mean weight at birth is around 50 kg (Platt, 1978). Placenta weight is considered as about 11 % of the foal weight at birth. In-utero growth retardation (IUGR) can be potentially caused by twining, placentitis and in utero infections. Inefficiency of the placental function occurs, which reduces transport of nutrients and oxygen to the foal. This will cause the birth of small foals which may or may not show signs of prematurity.

2.2 Equine placentation

Placental functions

The placenta has different functions, reviewed by Rossdale and Ricketts (2002). The placenta provides nutrients and oxygen to the foal. Waste products produced by the foal can be removed from the foal's blood by the placenta. Growth factors, which control fetal growth, and regulate the intrauterine environment of the fetus, are made by and delivered from the placenta to the foal (Rossdale & Ricketts, 2002). Any factor that adversely affects the intimate relationship of the placenta with the endometrium has chance to cause serious problems.

Limitations of the placenta

Due to the complicated build up of the placenta, different consequences can be described (Knottenbelt, 2003). The transfer of nutrients is relatively poor, what causes that only one foal can be fully supported. To provide adequate nutrition and gas exchange the entire endometrial surface is necessary for one foal. Endometrial scarring or other avillous areas result in the loss of effective transfer capacity. In twin pregnancies the two placentas will be adjacent to each other and these placental surfaces will reflect avillous areas which do not correspond with endometrial damage. As a result normal fetal development is compromised.

Placental transfer of large protein molecules is not possible, this results in no transfer of immunoglobulin's before parturition. The newborn foal starts its life without immunoglobulin`s and is dependent of immunoglobulin`s in colostrum. The mare's health is critical for the delivery of a healthy foal.

Endometrial bleeding is not possible, what means that blood loss at parturition is caused by maternal trauma or early rupture of the cord. Fresh bleeding after birth is usually a result of uterine, cervical or vaginal damage. Premature separation of the umbilical cord can result in problems in the foal, because of the loss of 1-1,5 litre blood. This blood normally flows to the foal after birth in case of an intact umbilical cord and is essential in foal life (Knottenbelt, 2003).

Changes in placentation at fetal stage

The fetal stage starts at day 40 of gestation. At that moment the amnion is vascularised and the fetus is located opposite to the embryo proper. Replacement of the yolk sac placenta by the allantochorionic placenta is nearly completed. The membranes and associated vessels separating the yolk sac and allantoic sac are coming together and will form the umbilical cord. The yolk sac will be incorporated in the umbilical cord. The site of the allantochorionic attachment of the umbilical cord throughout pregnancy identifies the horn and site where embryo fixation occurred.

The filling of the uterus with allantoic fluid occurs alternately proceeding and receding. During months 2-4, the umbilical cord will increase in length and the volume-ratio of allantoic fluid to fetal mass increases remarkable.

Endometrial cups

As mentioned earlier, pregnancy and placentation have been well studied and reviewed by Ginther (1998) and Allen & Stewart (2001). Endometrial cups are formed at the beginning of the fetal stage, and it involves restructuring of interaction of placental and maternal cells. Specialized trophoblastic cells, also called the chorionic girdle, develop during days 28-35, invading the endometrium. These cells form a longitudinal ring or horseshoe-like configuration along the lateral and medial walls of the caudal umbilical cord horn. Each separate cup consists of adjacent cells with different genotypes, maternal and fetal. At day 80 the cups begin slowly to detach, and the degenerating cup lies either free between the endometrium and allantochorion or becomes enclosed in a fold of allantochorionic tissue, called an allantochorionic pouch (Ginther, 1998).

The cups produce large quantities of eCG, causing the resurgence of the primary corpus luteum. The second luteal response develops, increasing the production of progesterone and initiating the production of estradiol. Supplementary corpora lutea can also form due to circulating eCG. eCG has a temporary role to stimulate this until the fetoplacental source is adequate to take over production of progestins.

2.3 Labour

As described by Knottenbelt *et al.* (2003), most mares foal between 11.00 pm and 4.00 am. Labour can be prolonged when the mare is disturbed. Total labour is divided into three stages.

Stage 1

In this stage contractions of the uterus and cervical relaxation occurs. Total duration of this stage may range from 30 minutes to 6 hours. However in some cases this can be prolonged, possibly due to the ability of the mare to control this stage. Upsetting or disturbing the mare can result in a prolonged stage one. There are no active or obvious abdominal contractions during this stage. Stage one ends when the chorioallantois ruptures, tan-red coloured fluid will spread (breaking of the water).

Stage 2

In this stage the delivery of the foal takes place. The average duration of this stage is 20 minutes, and ranges from 10 to 60 minutes. This stage affects the mare and the foal, with both having to participate to result in final parturition. Stage 2 ends with the total expulsion of the foal.

Stage 3

This stage includes the passage of the placental membranes and the start of the uterine involution. The duration of this stage is mostly about 1-3 hours. Placentas that are not passed within 3 hours are considered retained, and potentially could lead to a septic laminitis/metritis syndrome in the mare. Uterine involution will be completed 2-3 weeks postpartum.

2.4 Placenta examination

The placenta consists of an amnion, an umbilical cord and a chorioallantois. The chorioallantois is made up of 2 horns, the body and the cervical star.

Why should the placenta be examined?

Evaluation of the fetal membranes is essential for the further life of the foal. It offers an opportunity to examine the adequacy of the prepartum milieu of the fetus. Normally the placenta will be expelled intact after birth of the foal, turned inside out with the allantoic side first seen. But for example in case of premature placental separation, the foal will be born in the fetal membranes with the chorionic surface outermost. Examination of the placenta is necessary to check for completeness, identification of anatomical structures and abnormalities when present.

Normal findings at placenta examination

Whitwell & Jeffcott (1975) described the normal findings at placenta examination. The placenta is shaped like a bag that follows the shape of the mare's uterus. Allantoic fluid surrounds the amnion and the amnion surrounds the fetus and the amniotic fluid. Normally there will be 3-7 litres of amniotic fluid and 8-18 litres of allantoic fluid. The umbilical cord is attached to the fetus, amnion and the chorioallantois. One part of the umbilicus is intra-amnionic and the other part is extra-amnionic. Normal placenta weight is between 2.2-6.4 kg (Knottenbelt, 2003).

The amnion should be translucent, without any thickening. The examiner should be able to read a newspaper through the amnion when spread out. Increased thickness may reflect scars of healed amnionitis. Sometimes dystrophic calcifications can be seen, visible as plaques. Often it contains a hippomane, a mostly light brown rubbery structure, formed of minerals and waste products (Rossdale & Ricketts, 2002).

The umbilical cord consists of one vein and 2 arteries at the foetal side. The maternal side is made up of 2 arteries and 2 veins. The length of the cord should be between 36 and 83 cm. Long cords can twist and can cause fetal death. Remnants of the yolk sac may be found in the umbilical cord (D.H. Schlafer, 2004).

Both surfaces of the chorioallantois should be examined, the allantoic side and the chorionic side. The allantoic side will be greyish with large arteries and veins for which three vascular patterns have been described (see figure 1). The chorionic side will be red, like velvet, and when autolysed more brownish. The chorionic side attaches to the endometrium via microvilli.

The nonpregnant horn is more compact, and thicker or thinner than the pregnant horn. The surface of the nonpregnant horn is held in place by vessels of the allantoic side and will be darker coloured. The pregnant horn is larger and mostly paler. It is more smoothened and has signs of oedema nearby the tip of the horn. A normal small avillous area is seen on the tip of both horns, representing the entrance of the mare's oviduct (Rossdale & Ricketts, 2002).

The body of the placenta is thinner then both horns and is the largest part. An avillous ring around the attachment of the umbilical cord represents the site of earlier endometrial cups. The cervical star, named because of its scar like tissue and white avillous areas, forms the end of the placenta and opposites the cervix. Occasionally the body of the placenta contains linear white scar-like stripes along the distribution of large blood vessels.

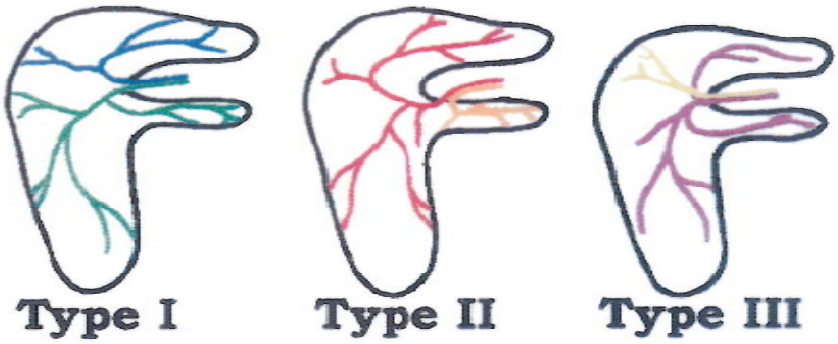

Figure 1: Placental vasculature patterns, Rossdale & Ricketts (2002).

Common abnormalities in the placenta

Large avillous areas are described and represent areas who failed to nourish the foal properly. Areas of separation will be blackened and bruised, they can develop later as scarred white, thick, avillous areas. Areas of inflammation associated with placentitis can be found at the cervical star and ascending. Typical placentitis is caused by ascending bacterial or fungal infection (fungal placentitis is not described in New Zealand). Oedema, hyperaemia and exudates can develop. Areas of thinning and separation of the villous side can occur in premature placental separation.

2.5 Foetal problems and placental problems at birth

Foetal problems

Stillbirth

Neonatal asphyxia is a cause of loss of full-term fetuses in the immediate peripartum period. Lack of oxygen in neonates can be caused by dystocia, premature placental separation, congenital abnormalities, placental infections and Equine Herpesvirus 1 or 4 infections (Knottenbelt, 2003).

For the current study foals that died during birth or immediately after birth, after a full term normal gestation, will be classified as stillbirths.

Congenital abnormalities

Hydrocephalus

The mode of inheritance in horses has not been established, but inheritance plays a part in the aetiology of hydrocephalus. Hydrocephalus is mostly seen in neonatal foals, further it is rare in horses. An increase in cerebrospinal fluid volume in the ventricular system (internal hydrocephalus) or in the subarachnoid space (external hydrocephalus) causes the bulging of the foals forehead. Causes of hydrocephalus are infection or injury of the brain parenchyma or obstruction of the cerebrospinal fluid production transport (McAuliffe S.B, 2008).

Schistosomus reflexus
This congenital abnormality is a developmental defect. The foal's abdomen remains open and the abdominal viscera are free. Together with this the vertebral column is incomplete and curved. There is no viability for the foal.

Fetal anasarca
All parts of the body of the fetus present generalized massive oedema. Most foetuses with this congenital, and hereditary defect are aborted.

Arthrogryposis
In this condition different joints of the newborn foal are fused. Muscle weakness can play a part and the foal is limited in its movements.

Prematurity and dysmaturity
Because of the difference in Thoroughbred mares gestation lengths, it is hard to distinguish if a foal is fullterm or premature (i.e. <320 days gestation). Calculating the gestation length and the clinical signs of the foal will remark if the foal is premature, dysmature (full gestation, with signs of prematurity) or fullterm. The clinical signs of both conditions, premature/dysmature, are similar and only the gestation length differs. These conditions can both be named unreadiness for birth.

Clinical signs of prematurity or dysmaturity are reviewed by Knottenbelt *et al. (*2003). Signs to notice are: underweight for type, bulging prominent forehead and eyes, weak musculature and tendons, collapsed tarsal or carpal bones due to incomplete ossification, tongue often prominent red-orange instead of normal colour, silky coat, soft and floppy nose/ears, dehydration, reduced tolerance to oral feedings, synechium present (slippery hooves, not dry out and separated normally), uncoordinated limb movements, colic directly after birth, slow or abnormal respiratory pattern, progressive worsening, diarrhoea, hypothermia and different from normal biochemical conditions (Knottenbelt, 2003; McAuliffe S.B, 2008).

Meconium staining
In response to severe distress in utero, meconium staining will occur. This can develop in cases of fetal and placental infections and in cord compressions or placental separation (D.H. Schlafer, 2004).

Umbilical cord torsion
Torsion of the umbilical cord causes restriction of blood flow from the placenta to the foal and vice versa. Torsion can cause acute fetal compromise, sometimes with fetal death and abortion, or sub acute to chronic cord compression, which results in chronic placental insufficiency. When torsion in the amniotic segment of the cord develops, urine flow can be constricted, resulting in urachal and/ or bladder distension (D.H. Schlafer, 2004).

Placental problems that may result in fetal disease and death

Placentitis

Placentitis can cause abortion, stillbirth and perinatal death in horses. Etiologic agents are viruses, bacteria and fungi (fungal placentitis is not described in New Zealand). The placenta gets infected, starting at the cervical star and ascends further. Inflammation and detachment of the chorioallantois at the level of the cervical star occurs. Other ways of infection are the hematogenous spread of placentitis caused by Leptospira spp. and the Nocardioform placentitis which gives characteristic lesions localised at the body of the uterus. Bacterial ascending placentitis is most common in New Zealand. Nocardioform placentitis has never been reported.

Premature placental separation

This phenomenon occurs when the placenta separates from the endometrium before the fetus can break through the allantochorion at birth. This is also called red bag delivery. The placenta does not rupture at the cervical star, and the foal will be born totally covered in membranes. Causes of premature placental separation are described by Knottenbelt (2003). Most common seen are: fescue toxicosis (not in New Zealand), stress in late gestation, perchance excessive nutrition in late gestation, ascending infection or other pathology and induced parturition. At placenta examination a typically thickened and oedematous chorioallantois can be found.

Retained placenta

Within 3 hours after birth normal placenta release will occur. Retained placenta can occur in the mare. Most of the time the tip of the nonpregnant horn will be still in the mare's uterus. If not removed, autolysis will develop and the placenta may become secondary infected, this results in illness of the mare (Rossdale & Ricketts, 2002). Retained placenta is probably the most common complication in the postpartum mare, it occurs in 2-10% of all the deliveries (reviewed by Knottenbelt). Retained placenta has a multifactorial etiology including uterine inertia and fatigue, calcium/phosphorus imbalances, selenium deficiency (evidence lacking), induced delivery, dystocia and caesarean section.

2.6 Foal diseases in the first month of life

Cohen (1994) determined the causes of and farm management factors associated with disease and death in foals. The most common cause of incident disease was respiratory disease, followed by diarrhoea. However most common cause of death was reported as pneumonia, followed by septicaemia. As a group the most common cause of death were musculoskeletal disorders. This includes traumatic, infectious or deforming problems. Risk of death was greatest during the first week of foal life. This will decrease in the later foal life (Cohen, 1994).

Morley & Townsend (1997) reported the morbidity and mortality in foals, this was higher than previously reported. 25% of foals had health problems and 5% died during the first 2 weeks after birth. 27 % of foals surviving the first 2 weeks were affected by some health problem between age 15 days and one year, and 6% died during this period. Health problems up to age 2 weeks, or between age 15 days and one year were 5 to 7 times more likely to result in physically unacceptable foals for athletic use (Morley & Townsend, 1997).

3. Materials and methods

3.1 Study site

The fetal membranes and health data records were collected from 2 local Thoroughbred studs within 20 km of Massey University, Palmerston North (Goodwood studfarm and Wellfield studfarm). The placentae were collected daily, and carefully examined, weighed, measured and sampled. Foals and dams were condition scored using the method of Henneke *et al*. at birth and one month later (Henneke, Potter, Kreider, & Yeates, 1983). Data on health status and routine management of foals during the first month of life were obtained from stud managers. The total time for this research was three months.

3.2 Thoroughbreds

During three months placentae from 68 Thoroughbreds mares were collected (35 at Goodwood, 33 at Wellfield). During this time period 62 foals were born alive, five foals were stillbirth or died in a few minutes after birth, and one foal was euthanized directly after birth (five at Goodwood and one at Wellfield). Details of foaling, behaviour of the foal and the used drugs were obtained from the grooms/ owners. Details about parity and age of mares was found on the website of the Thoroughbred Studbook.
For body condition scoring after one month, 26 mares and foals were scored (13 mares with foals at Goodwood and 13 mares with foals at Wellfield).

3.3 Experimental design

3.3.1 Placenta examination

The placentae examinations were planned on the same day as they were collected to reduce autolysis. A total of 28 variables were investigated from each placenta. To start with, the total weight was measured. The amnion membrane was inspected, and in case of aberrations/ defects, samples were taken. The allantochorion, allantoic side, was inspected and suspicious details were written down. The vascular pattern and the site of attachment of the umbilical cord were noted. In case necessary the variant vascular patterns were drawn on the form. In case of suspicious areas, samples were taken immediately, before any extra contamination could occur.

Length of the total umbilicus and the amniotic portion of the umbilicus (from the fetal site to the attachment of the amniotic membrane) were measured, after any twists had been unwound. Then the umbilicus and the amnion were weighed separately. The umbilicus was cut of the allantochorion to its attachment.
Linear measurements and morphological variations on the allantochorion were noted. The allantochorion was smoothened flat, but not over stretched at the allantoic side. The total surface was spread like a reverted F-shape as seen in earlier work done by Whitwell & Jeffcott, 1975. And a total of twelve lengths were measured, A to K (see figure 1; table 1).

Before turning the allantochorion inside out, the total weight was measured and noted. After inspection of the villous site of the allantochorion, samples were taken for PCR for equine herpes viruses and samples in formalin for later histological examination. If necessary photos were taken of suspicious or remarkable spots.

The cervical star was inspected and in case of placentitis or other striking details photos were taken, sampling occured and details were written down.

Other details, like extraordinary hippomanes and allantochorionic pouches were noted and photographed.

Total water displacement of the allantochorion was measured through cutting it in two almost identical pieces of allantochorion, along the F shaped edges, and putting these pieces separately in a measuring jug of 5.0 L with 2.0 L of water. The two separately numbers were added.

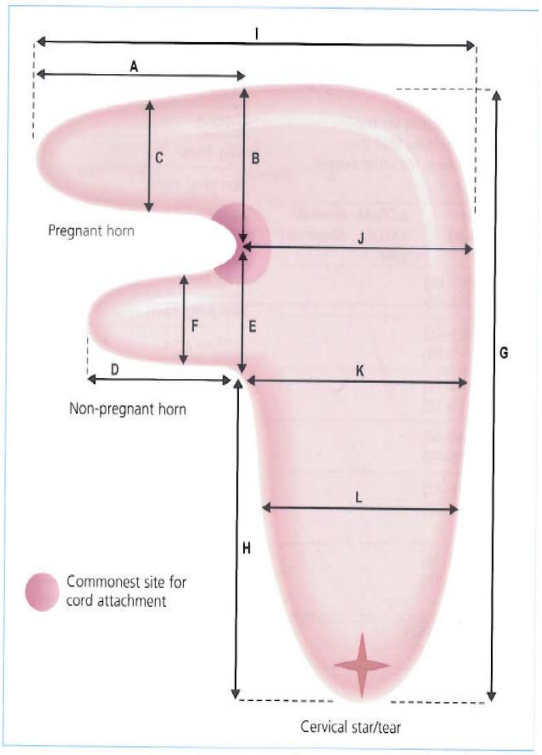

Figure 2: Placenta sites for measurement and blood vessel distribution (Knottenbelt, 2004, after Whitwell and Jeffcott 1975).

Site	Structure	Breed	Mean (Range) (cm)
A	Length PH	TB	66 (55–80)
B	Diameter PH entrance	TB	32 (25–45)
C	Diameter PH middle	TB	21 (14–34)
D	Diameter NPH	TB	60 (46–80)
E	Diameter NPH entrance	TB	24 (16–32)
F	Diameter NPH (middle)	TB	20 (12–29)
G	Total body length	TB	130 (110–152)
H	Posterior body length	TB	67 (53–90)
I	Anterior body (PH)	TB	109 (94–142)
J	Body width (anterior)	TB	45 (30–55)
K	Body width (middle)	TB	45 (37–55)
L	Body width posterior	TB	44 (37–60)
Total cord length		TB	55 (32–90)
Amniotic cord length		TB	26 (16–46)

Table 1: Dimensions of the equine placenta, means and range by Whitwell and Jeffcott 1975 (Knottenbelt 2004).

3.3.2 Post mortem examination of the equine fetus

Local veterinarians and studs were asked to donate any aborted or dead foals (up to one month of age) to post mortem for free. This contributed to a study examining the major causes of foetal and foal losses in the local region. Veterinarians were encouraged to send in the placenta if available. Post mortem examination of the equine fetus occurred in a manner similar as described by Schlafer (D.H. Schlafer, 2004).

Case files of ten post mortem examinations of the equine fetus are added to this report (see appendix 9.3).

3.4 Statistical analysis

Data of placentae examination, body condition score and foaling were entered into MS Excel. Data analyses was to determine relationships between mare and foal body condition score at birth and at one month of foal age, placental measurements and health status in the first month of life. Data examination was completed by using parametric and non-parametric analysis procedures in SPSS v16 (SPSS Chicago, Il, USA) with a significance level set at $p<0.05$.

4. Results

4.1 Placenta results

During the period of this study a total of 67 placentas were collected, as seen in figure 3 (calculated until the 20th of November). In total 31 colt foals were born and 36 filly foals: 16 colts and 19 fillies were born at Goodwood, and 15 colts and 17 fillies at Wellfield. There is no significant difference in foal sex born at both studs (n = 67, p = 0.926).

The foals were born during day and night time. As described by Knottenbelt *et al.* most mares foal during the period from 11.00 pm till 4.00 am. However this is described for mares who foal inside, instead of foaling in the paddock like the New Zealand standard (Knottenbelt, 2003). Visible in figure 4 is that there is no clear pattern of foaling during this time period, but a higher percentage foals during night-time. There is no significant difference between both studs in foaling during day or night time.

Statistical analysis shows that there is no significant difference between both studs for parity or mare age (p = 0.472 and p = 0.241). Figure 5 shows the distribution of parity for both studs contributing to the study.

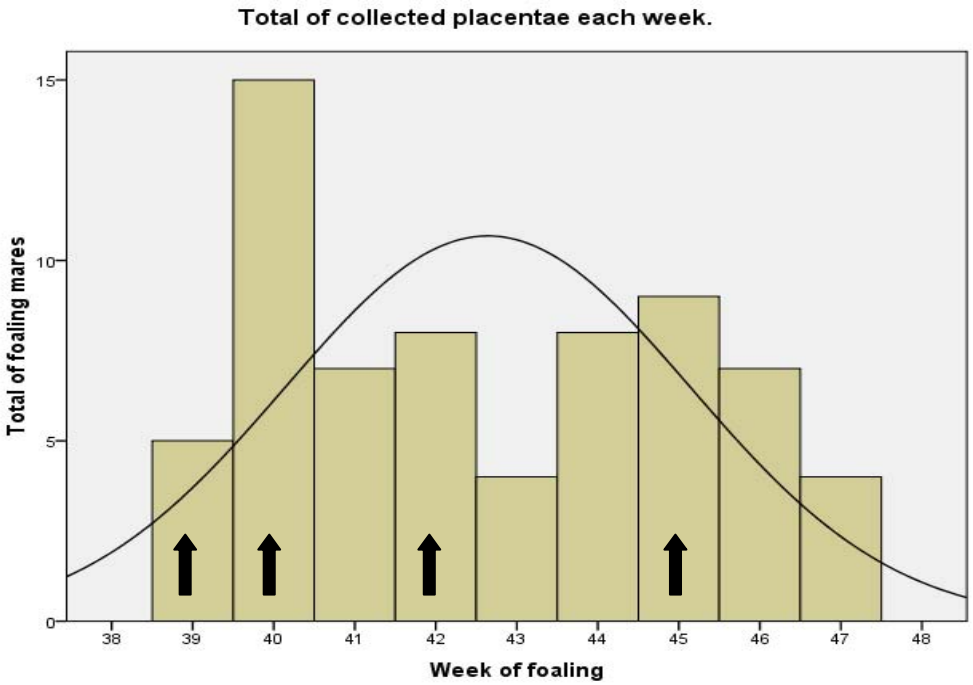

Figure 3: Total of collected placentas each week of the year during this study (*n* = 67). The arrows indicate the incidence of stillbirths. In week 39 there were two stillbirths, in week 40 two, week 42 one, and in week 45 one foal was euthanized.

Figure 4: Time of birth during the day/night of the foals contributing to the study, *n* = 66.

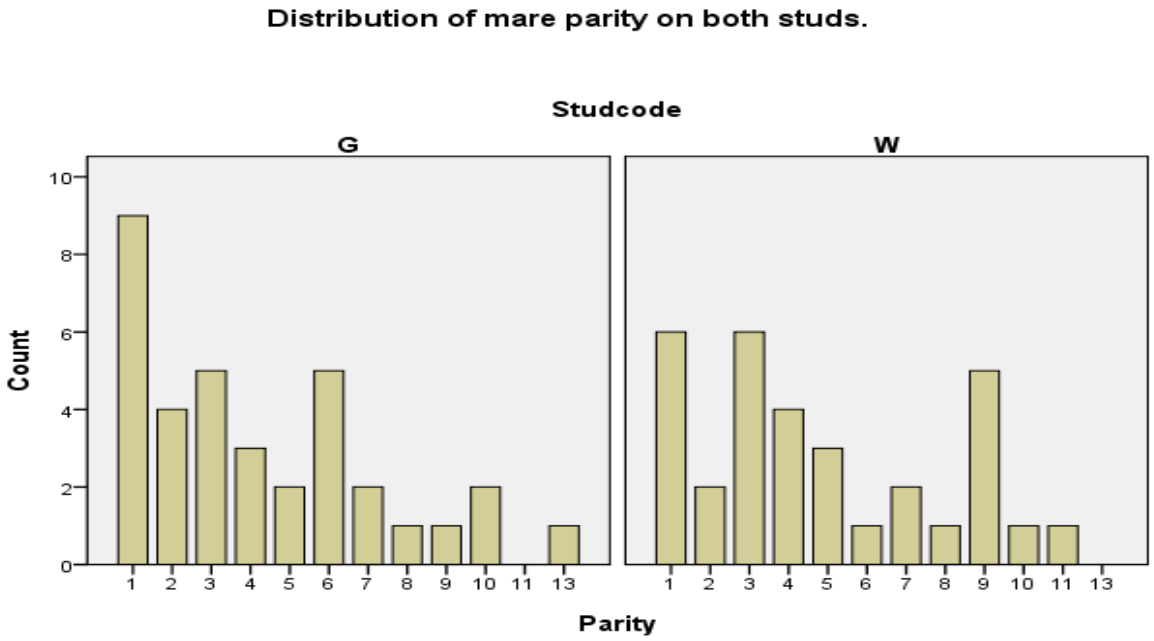

Figure 5: Distribution of mare parity at both studs (G = Goodwood *n* = 35, W = Wellfield *n* = 32).

As showed by table 2 and 3, placenta dimensions found in our study are in the same range as results found by other authors. Table 2 and 3 show the actual range, mean and standard deviation found in our study on the left and the mean and actual range found by Whitwell & Jeffcott at the right. Only the means found in our study of the linear measurements K an L do not fit in the range found by Whitwell and Jeffcott. Table 4 shows the different abnormalities found at placenta examination.

Table 2: Placenta dimensions. Results of our study on the left, on the right results found by Whitwell & Jeffcott (1975).

	Current study				**Whitwell *et al.***	
	n	Actual range	Mean	Std. Deviation	Mean	Actual range
A Length pregnant horn	59	35-86	59	9.1	66	54-85
B Diameter PH, entrance	59	18-57	30	5.9	32	25-45
C Diameter PH, middle	59	14-27	20	2.8	21	14-34
D Length non-pregnant horn	58	27-86	54	11.0	60	46-80
E Diameter NPH, entrance	58	15-33	23	4.0	24	16-32
F Diameter NPH, middle	58	12-22	17	2.5	20	12-29
G Total body length	59	100-160	125	12.2	130	110-152
H Posterior body length	59	35-100	65	12.1	67	53-90
I Anterior body + PH	59	62-126	96	11.8	109	94-142
J Width of body, anterior	59	25-47	37	4.7	45	30-55
K Width of body, middle	59	30-45	36	4.1	45	37-55
L Width of body posterior	59	25-50	35	5.5	44	37-60

Table 3: Weight and lengths of different placental parts. Results of our study on the left, on the right results found by Whitwell & Jeffcott (1975).

	Current study				**Whitwell *et al.***	
	n	Actual range	Mean	Std. Deviation	Mean	Actual range
Amnion (g)	59	485-5278	2038	810	1850	1000-3140
Placenta (g)	60	2300-10774	6526	1797	5700	4000-8400
Chorioallantois (g)	58	2125-6025	3796	888	3600	2200-5300
Umbilicus (g)	58	157-831	284	124	247	120-411
Umbilicus (cm)	59	33-98	53	12	55	32-90
Amniotic portion (cm)	58	20-46	28	6	26	16-46

Table 4: Different abnormalities found at placenta examination, *n* = 68.

Different abnormalities found at placenta examination	**Frequency (***n* = 68)
Meconium staining amnion	1
Oedema Amnion	12
Amniotic plaques	7
Mineralization tip pregnant horn	16
Missing non pregnant horn/ torn	8
Vasculature pattern I	54
Vasculature pattern II	10
Vasculature pattern III	2
Vasculature pattern undecided	2
Different umbilical attachment	18
Evidence placentitis	4

4.2 Body condition scoring results

Foals and mares were scored at birth/ foaling and one month later. The scorings show how the foal develops its body condition score. Figure 6 shows the body condition score of the foals at birth, varying from score 2.0 to 6.0. Figure 7 shows the body condition score of the foals at one month of age, varying from 5.5 to 8.0. There was no significant difference found between the body condition score of the foals for both studs, further analyses are done by arranging one group. Shown in figure 8 are the body condition score at birth of the foals at the x-axis and the change in body condition score of the foals at one month of age at the y-axis. The change was calculated by decreasing the BCS at one month of age with the BCS at birth of the foals. Foals born with a high body condition score show a less change in body condition at one month of age. Foals born with a low BCS at birth show a higher change in body condition at one month of age.

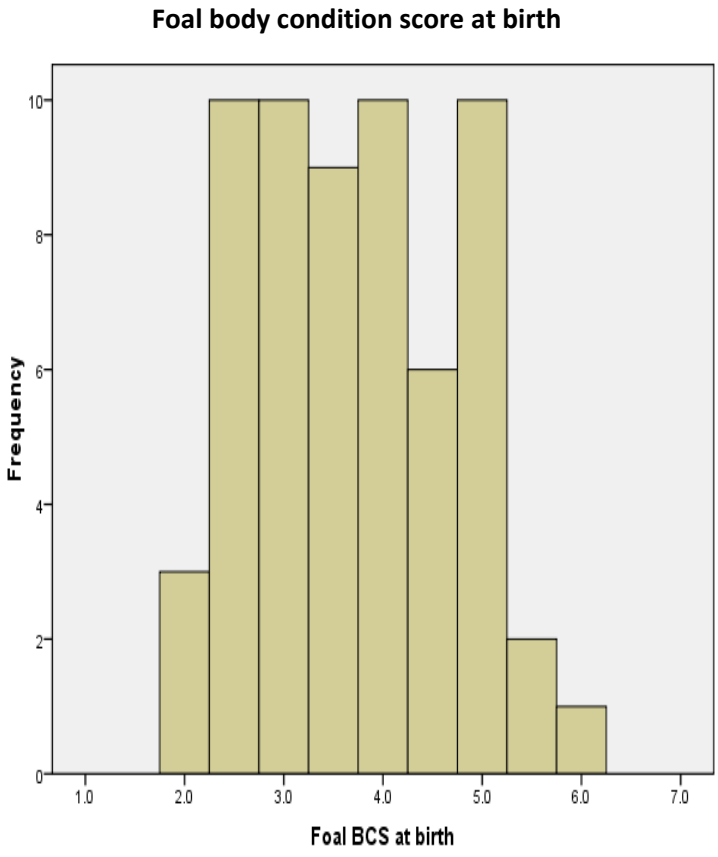

Figure 6: Foal BCS at birth for both studs, *n* = 61.

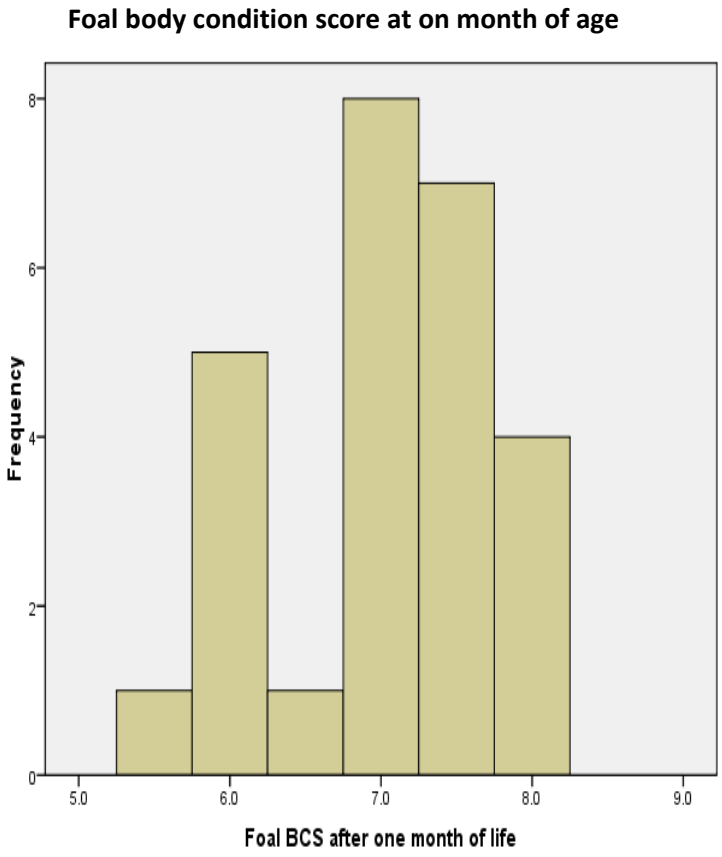

Figure 7: Foal BCS at one month of age for both studs, *n* = 26.

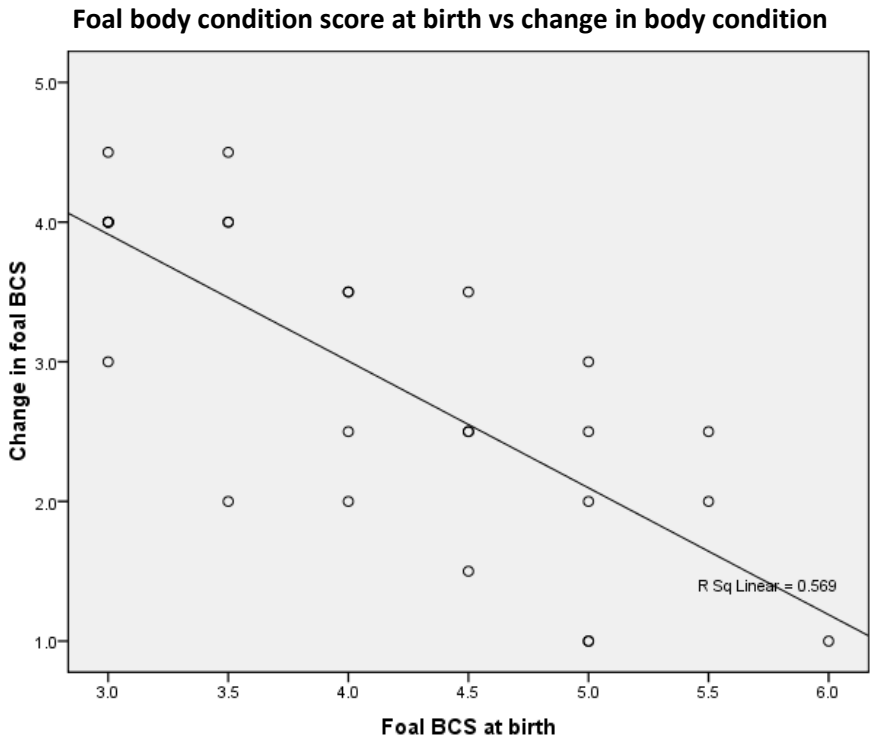

Figure 8: Change in BCS for the foals. The change was calculated by decreasing the BCS at one month of age with the BCS at birth of the foals. Foal BCS at birth at the x-axis and the change in foal BCS at one month of age at the Y-axis, *n* = 26.

Statistical analysis shows that there is a significant difference for the mares BCS at foaling between both studs (p = 0.023). At Goodwood stud mares have a slightly higher body condition score at foaling and is the distribution along the x-axis spread at higher start values.

The histogram of Wellfield shows more mares with a lower body condition score (see figure 9). There is no significant difference between parity and body condition score of the mare at foaling (p = 0.273). As shown in figure 10 the body condition score of the mares one month post partum changes almost for all the mares. There was no significant difference found between body condition score of the mares one month post partum and stud of foaling (p = 0.532).

Figure 11 shows the correlation between mare body condition score at foaling and the change of body condition one month post partum. The change was calculated by decreasing the BCS one month post partum with the BCS at foaling of the mares.

There is a weak correlation between the change of body condition in the mare and the foals body condition at one month of age R^2 = 0.429 (see figure 12). A statistical significant difference is found (p = 0.008).

No significant difference between foal sex and mare body condition score at foaling is found (p = 0.907). Also, no significant difference has been found for the parity of the mare and the body condition score of the foal at birth, and the correlation is low (p = 0.788). So the condition of the foals at birth is not dependent of the parity of the mare.

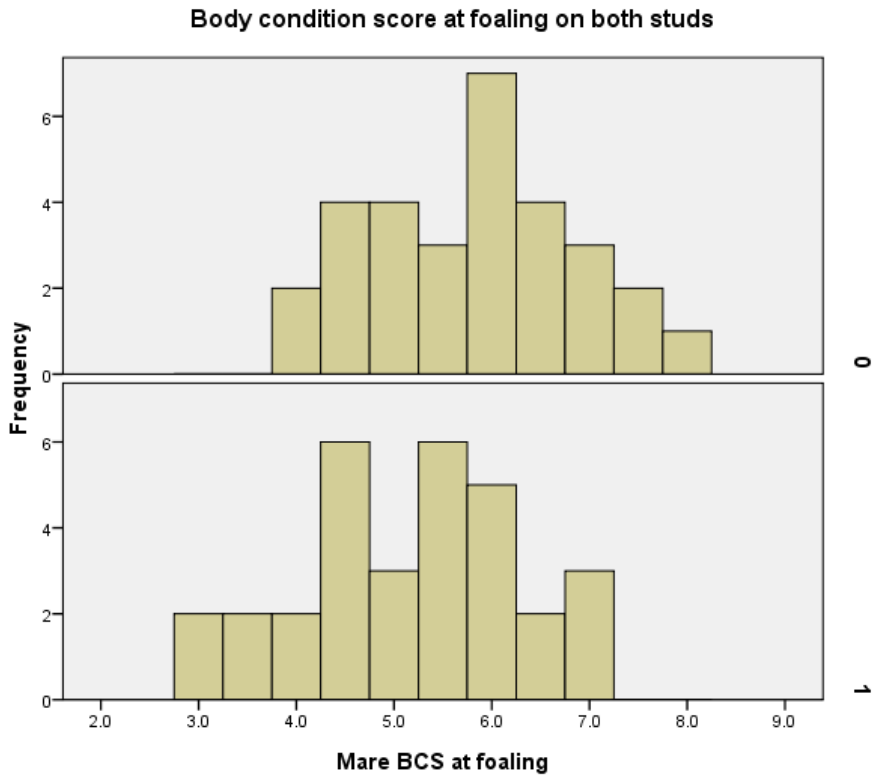

Figure 9: Mare body condition score at foaling on both studs. A significant difference was found between both studs in BCS at foaling, for each stud, *n* = 30 at Goodwood (0), *n* = 31 at Wellfield (1).

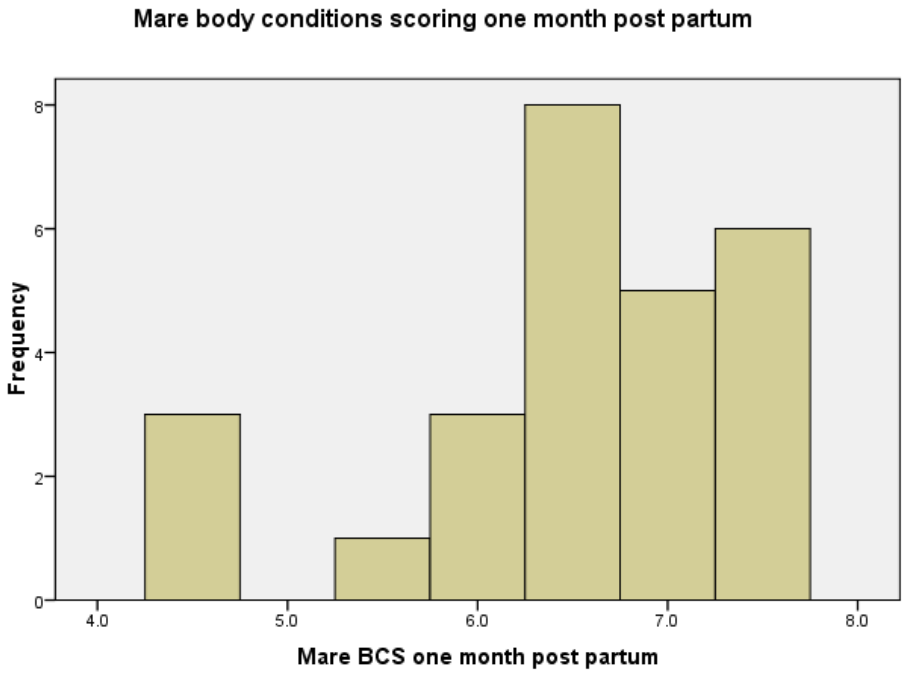

Figure 10: Mare body condition score one month post partum. No significant difference was found between both studs, *n* = 26.

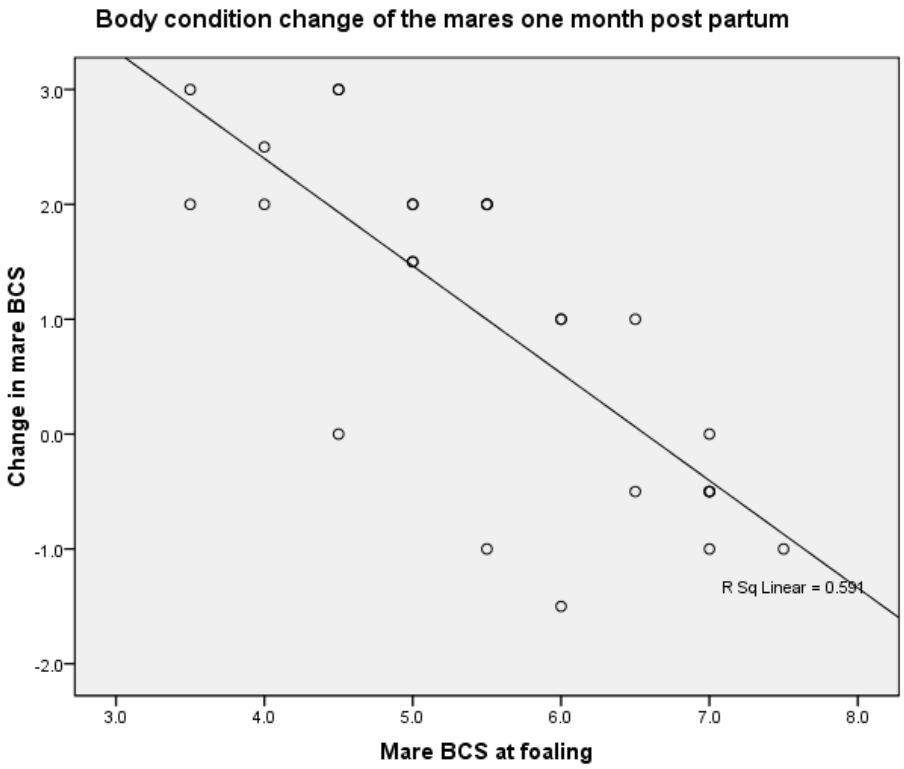

Figure 11: Change in body condition measured one month post partum for both studs, *n* = 26.

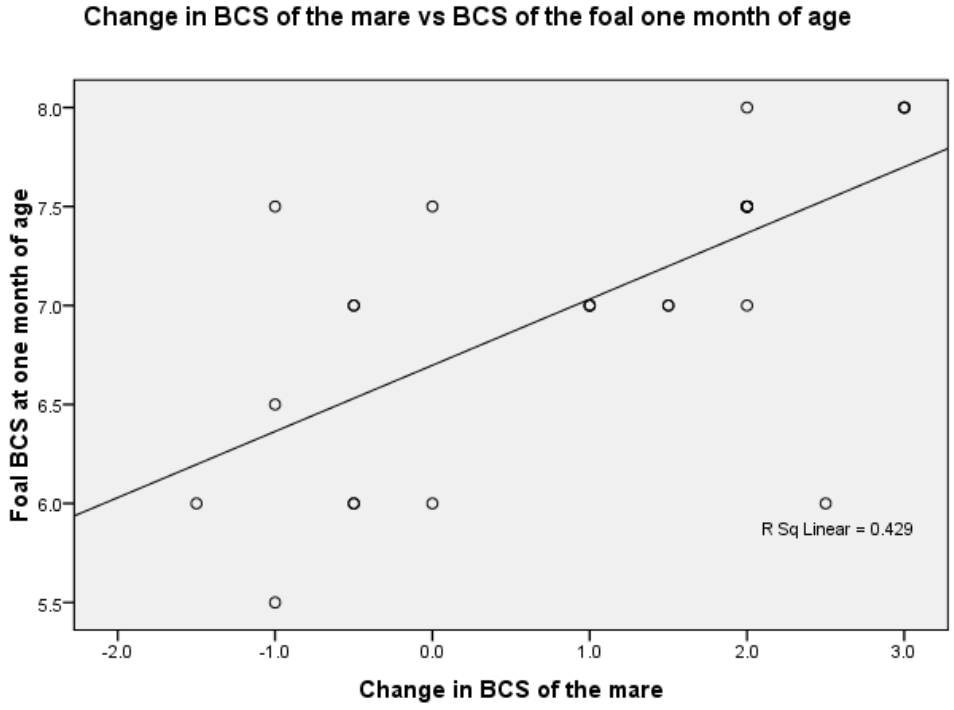

Figure 12: Change in body condition of the mare vs. BCS of the foal at one month of age, *n* = 26.

4.3 Foaling

Data were pooled from the two studs. The mean gestation length, as described in the introduction would be 340 days, and could range from 316-364 days. As shown in figure 13 the mean gestation length of the total mare group is 353 days, this is in range with data found by Whitwell and Jeffcott. No gestations shorter than 330 days are registered. Only 11.9 percent of the total pregnancies had a longer gestation length as 364 days (n = 8).

The duration of labour, stage 2 labour, was measured by the foal watch/ stud manager. Normal stage 2 labour takes 20 minutes, but can range from 10-60 minutes. As figure 14 shows, 6.5 percent of the mares had a prolonged stage 2 labour (n = 4).

The normal interval to pass the placenta, stage 3 labour, is less than 180 minutes. Within three hours the total placenta needs to be passed if normal. As shown by figure 15 almost 90 percent of the mares passed their placenta in the normal time period (n = 49). The other ten percent did not pass their placenta within 180 minutes and will be considered as abnormal (n = 6).

Data collected about the foal's development the first few hours of life were also obtained from both studs. Figure 16 shows the interval from birth till sternal of the foal. The normal interval should be within 5-7 minutes. As seen in the figure only 51.8 percent of the foals got sternal in range (n = 29).

Figure 17 shows the interval from birth till standing of the foals. Normally a foal will get up till standing and suckling within 60 minutes post partum. A small proportion of the foals, 14.3 percent, had a delay in standing up (n = 8). As seen in figure 18 at least 67.9 percent of the foals did not suckle within the first hour of life (n = 36). The one foal with a 180 minutes interval, was bottle feed before suckling at the mare.

4.4 Comparisons

No statistic correlation was found between the whole placenta weight and the foals body condition score at birth (p = 0.447). Also there is no correlation found between mare age and whole placenta weight (R^2 = 0.058; p = 0.243). Furthermore no significant difference and no correlation was found between foal body condition score at birth and weight of the chorioallantois (p = 0.504).

As regards to the use of oxytocin on one stud as standard and not used at the other stud as standard, no significant difference could be found between the use of oxytocin or no use of oxytocin to pass the placenta (p = 0.452). Visible in figure 20 is the bigger spread of the placenta passing time for the use of oxytocin. The numbers 10, 50 and 51 are mares at Goodwood stud. The numbers 37, 57 and 61 are mares at Wellfield stud.

No significant difference can be found between mare age/ parity and colostrum quality (p = 0.394; p = 0.696). Also there is almost no correlation (R^2 = 0.008; and R^2 = 0.007). There is no significant difference in colostrum quality between both studs (p = 0.365). See figure 19.

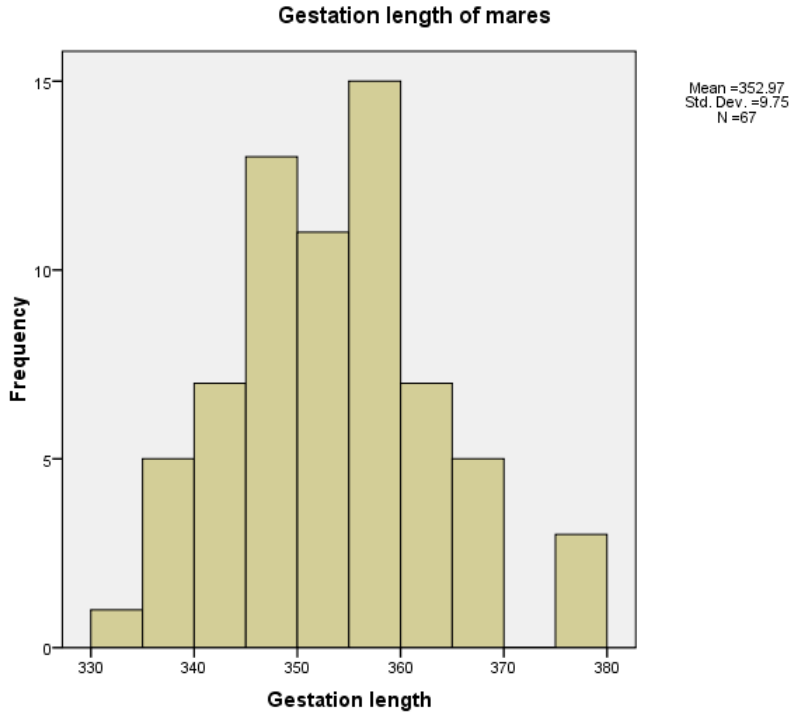

Figure 13: Gestation length in days of the mares contributing to the research, both studs together, *n* = 67.

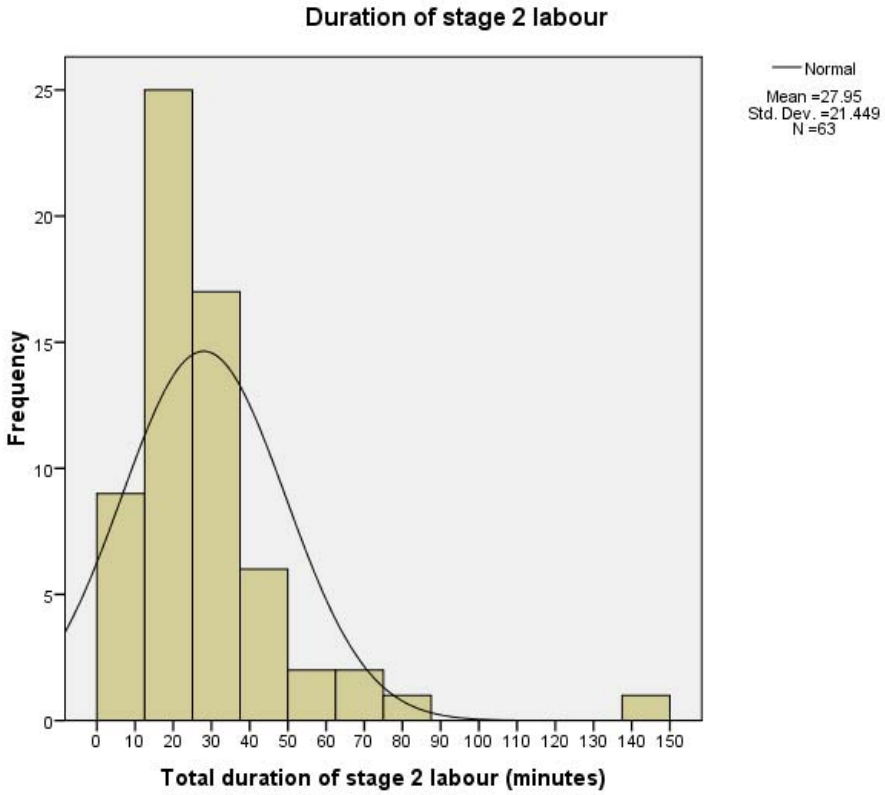

Figure 14: The total duration of stage 2 labour for both studs together (*n* = 63).

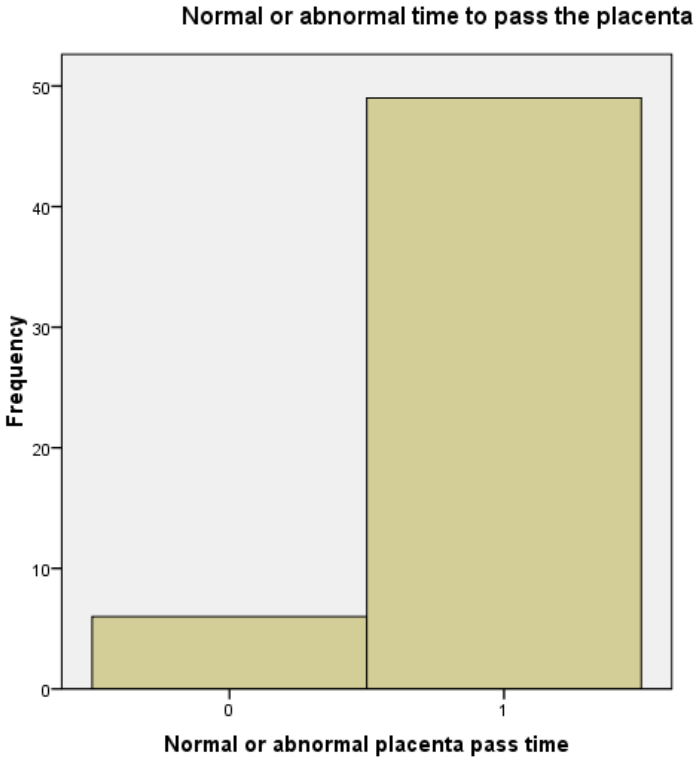

Figure 15: Normal or abnormal time to pass the placenta. Accepted as normal is <180 minutes, *n* = 55 (0 = abnormal, 1 = normal).

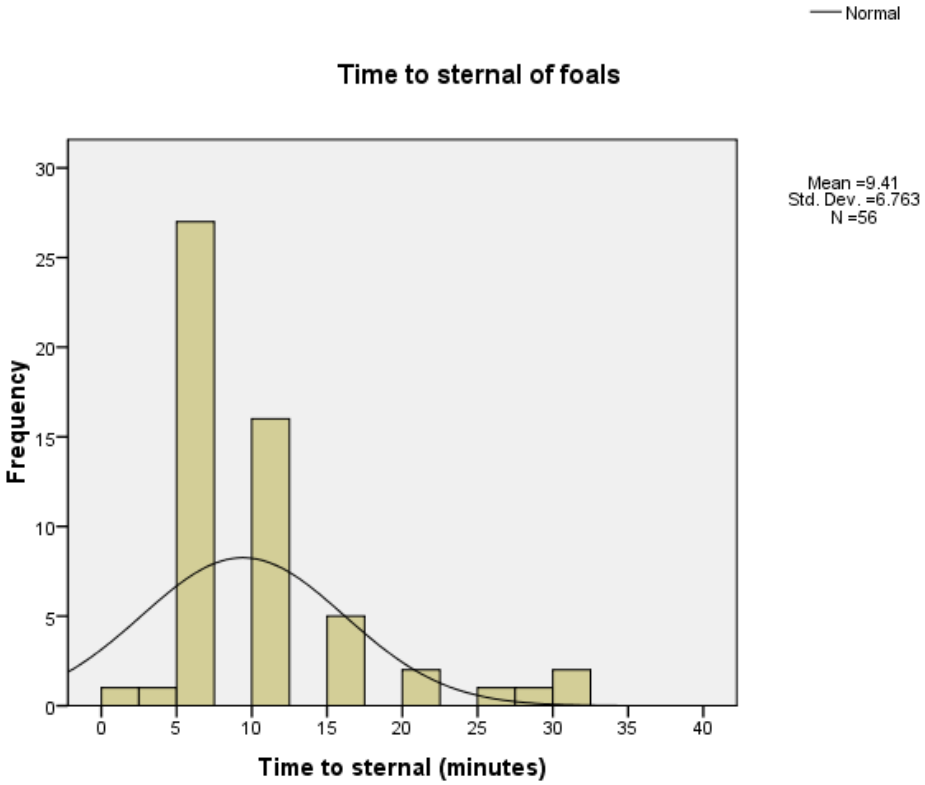

Figure 16: The total duration of the interval birth to sternal of the foal (*n* = 56).

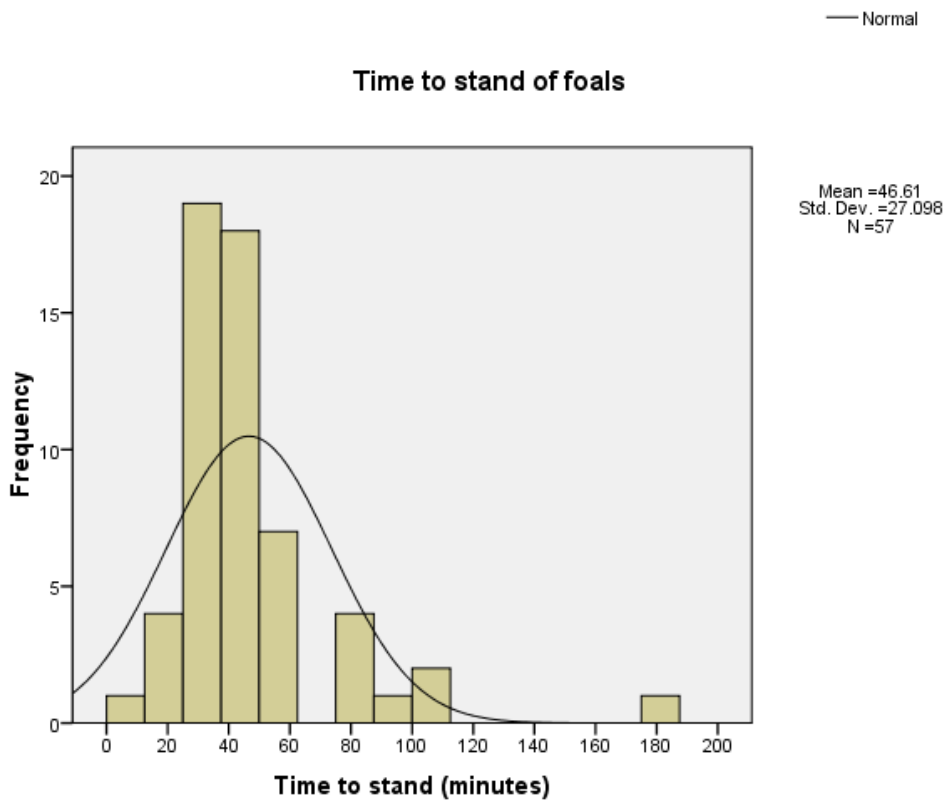

Figure 17: The total duration of the interval birth to stand of the foal (*n* = 57).

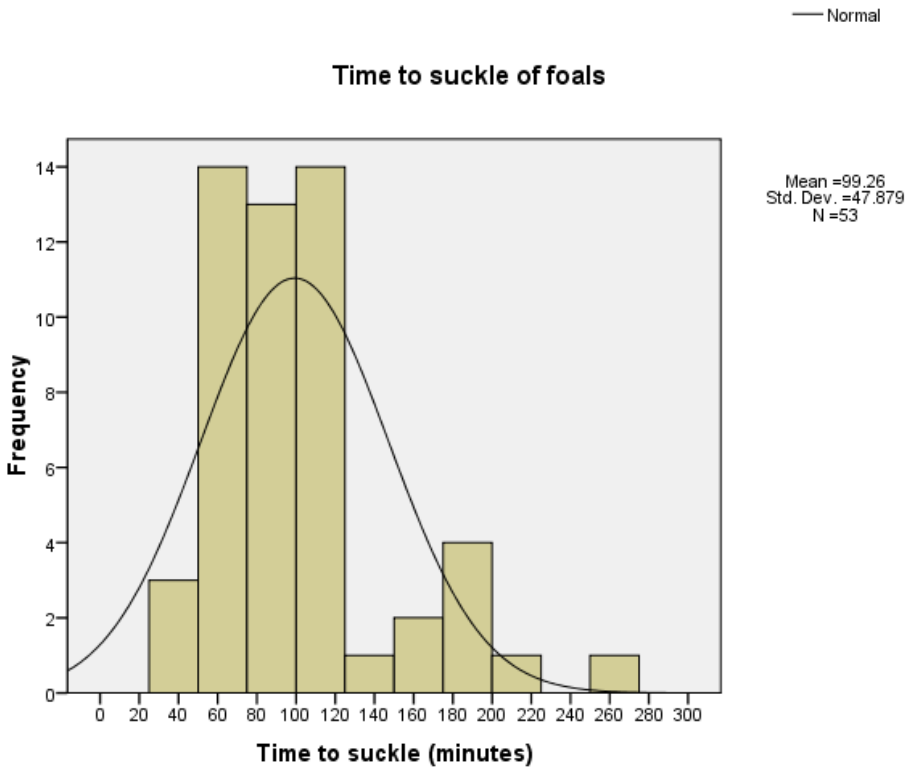

Figure 18: The total duration of the interval birth to suckle of the foal (*n* = 53).

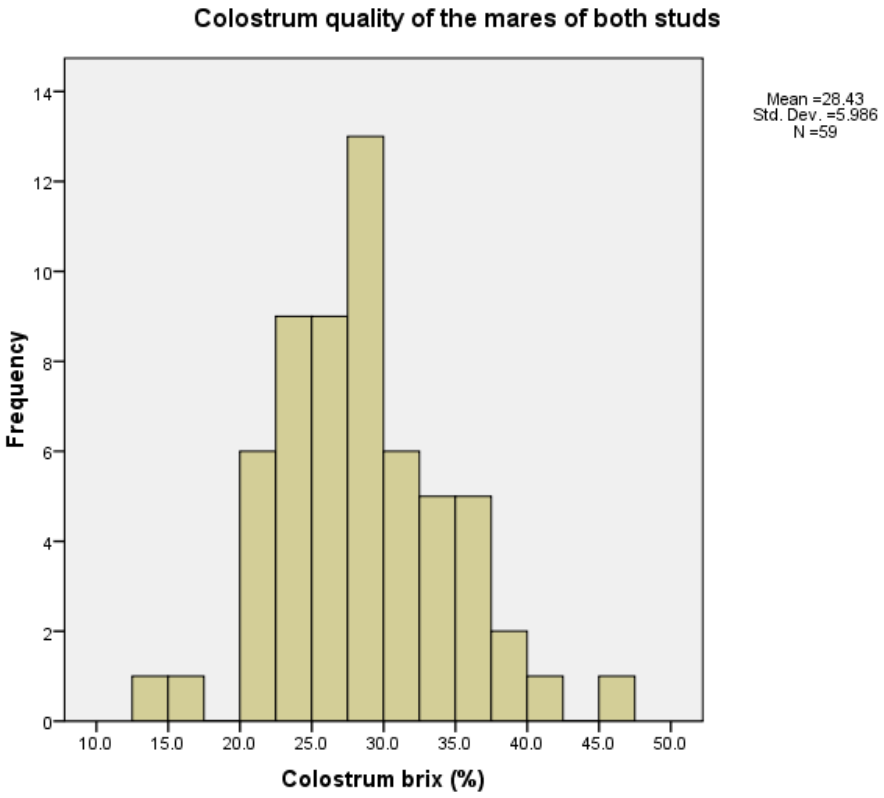

Figure 19: Colostrum quality of mares, both studs together (*n* = 59).

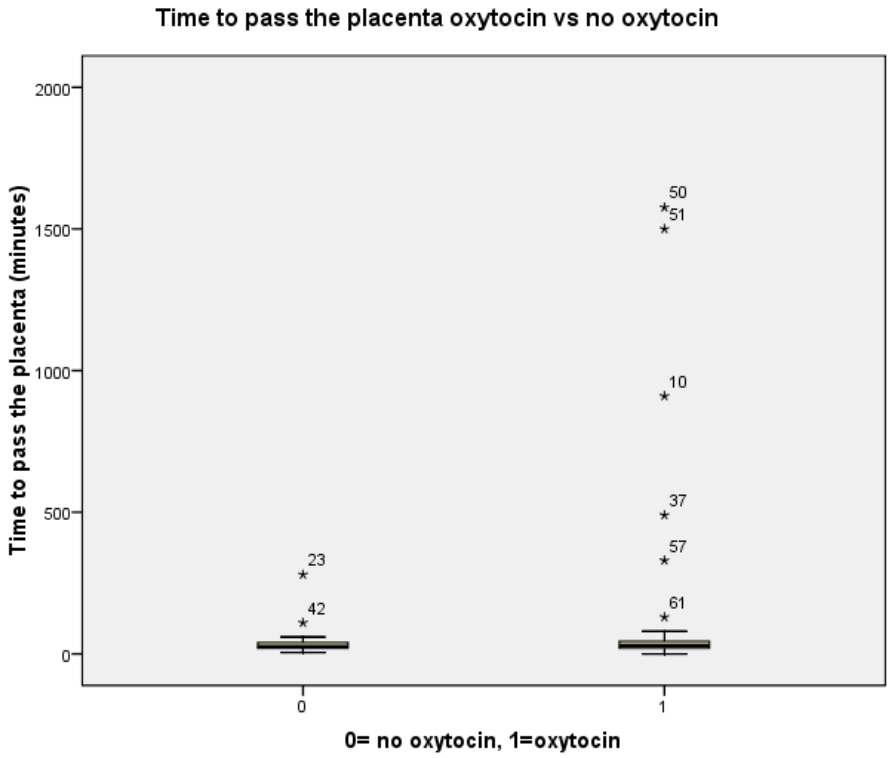

Figure 20: Use of oxytocin or no use of oxytocin versus the time to pass the placenta (*n* = 66). The box plot indicates the mean and the standard error of the statistical analysis.

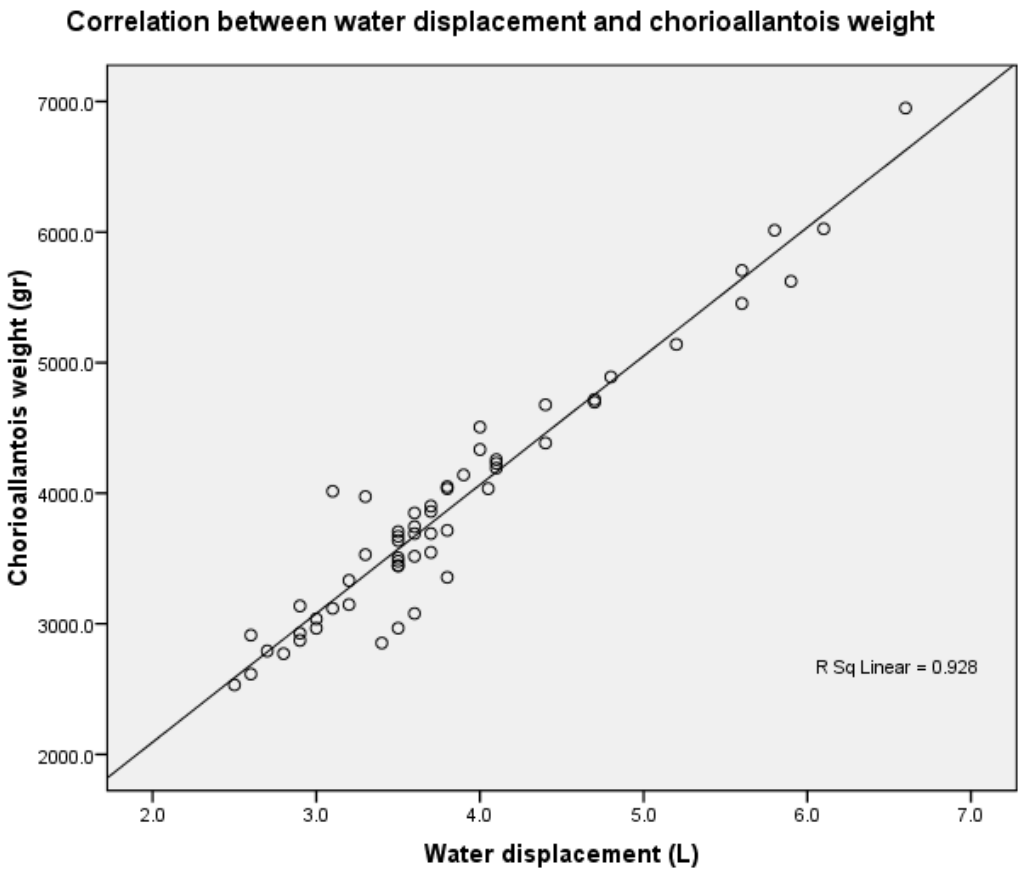

Figure 21: Correlation between water displacement and chorioallantois weight, R^2 = 0.928 (*n* = 59).

The water displacement measured in our study is strongly correlated to the chorioallantois weight (R^2 = 0.928). This is not so surprising, but it could be a good measure point.

4.5 Foal health after one month

During week 11 of the project both studs were visited and questioned about their foal health. During these meetings studmanagers were asked to give a view of their foal health in the first month of life of the foals. In this part of the study 25 foals at Goodwood stud and 16 foals at Wellfield stud were participating.

At Goodwood stud nine foals were treated for the diarrhoea with antibiotics (Peptosyl ®). Antibiotics were given for 3-4 days. Two foals were treated with antibiotics directly after birth, because of placentitis developed by the mare. One foal was born with low IgG and has been given three plasma infusions after birth. Until now only one foal evolved bowed knees and had extensions. All foals developed foal heat diarrhoea. No pulmonary diseases were seen.

At Wellfield stud none of the foals were treated for diarrhoea. Foal health on this stud has been good at present, as far as known.

5. Conclusion

There has been found a significant difference between both studs in mare body condition score at foaling, but not in foal body condition score at birth. Further there is no significant difference found between studs for the mare body condition score one month post partum and also not for the foal body condition score at one month of age.

The body condition score of foals changed substantially for foals born with a poor body condition. Foals born with a higher body condition score end up with a lower change in body condition. All foals end up almost at the same body condition score at one month of life.

Not enough information about foal health in the first month of life was known to make a correlation between body condition score or placental parameters.

The placenta parameters measured in our study are almost the same as found by earlier studies. This validates our method to work with. Different abnormalities were found at placenta examination as showed in table 4.

A significant difference was found between mare body conditions score change and foal body condition score after one month of life.

Because of the small data set and the small amount of time we cannot concluded if poor body condition of foals at birth is associated with abnormal placental parameters and increased risk of diseases in the first month of life.

6. Discussion

One of the conclusions of this study is that the mare body condition scores were significant different between both studs. This may be caused by the pasture availability on both studs. Goodwood stud commands a large area with big paddocks and high amounts of good pasture at the start of the foaling season and during the season. Wellfield on the other hand started with good pasture in every paddock, but because of the smaller paddock surface the pasture amount decreases. It is also possible that Wellfield accepts more mares from other owners, instead of more mares owned by them self. Mares owned by people out of the stud, could be not as good to keep in shape as mares were they take care of the whole year.

To make sure about our findings a bigger data set should have been used. Also not all the information asked for has been given, the stud managers were not always able to get all the information. The research done by Whitwell started with 211 foalings, 145 satisfied to their criteria. The meaning of this current study could be bigger if a larger dataset had been used.

The time foaling started may be seen as the start of stage 2 labour. This is the actual stage of delivery of the foal. Unknown is what time it took to get the mares through stage 1. No data could be collected about this stage because of lack of knowledge and observation time.

According to dr. D.H. Schlafer mineralization of the tip of the pregnant horn may be caused by pressure of the fetal hoofs. This causes necrotic structures which may mineralize. There are no other articles found with explanations of this phenomenon.

Determination of the vascular pattern was not always easy to do. If the umbilical attach was on for example the non pregnant horn, instead of the normal attach, the vascular structures were also a bit strange. This may have influenced our capability to distinguish the patterns.

The first few placentas of this study were measured on a large scale, which was less exact. The later placentas were all weighted on a small, more exact scale. In first the weight of the buckets were not correct measured. This has been corrected further in the study.

BCS from a distant is done by Cameron et al.(Cameron, Linklater, Stafford, & Veltman, 1999). The body condition scores were estimated by visual body fat distribution, based on a 11-point scale (1-5). Earlier done by other authors (Carroll and Huntingdon, 1998; Huntingdon and Cleland, 1992; Rudman and Keiper, 1991). Inter observer reliability was high in this study, in our study we practiced before starting and only a difference of 0.5 was allowed. In horses body condition scores correlate with body fat percentage (r-0.81, Henneke et al., 1983).

Body condition scoring on foals at birth has never been done before to our knowledge. We used the same method as common used in adult horses. In foals it is not possible to give a strong correlation about real foal fat and weight. Foals will start first in their early life with growing muscle instead of fat. In a few other studies correlations are found between placenta parameters and foal weight. Before we started this study we had to search if there was a strong correlation between foal weight and body condition score. On this stage of the study conclusions about possible correlation cannot be made.

7. Acknowledgements

I would like to thank Prof. Dr. P.R. Van Weeren for his effort and the opportunity to fulfil my research project in New Zealand and for him as my supervisor in the Netherlands. Thanks to his good contacts at Massey University it was possible to arrange the project at the Veterinary Teaching Clinic in Palmerston North.

I want to thank dr. E.K. Gee as my supervisor at Massey University. I really enjoyed the project and hope it will be a good trial for a bigger project. I also would like to thank dr. C.W. Roger for his knowledge about statistics and his patience.

Further I would like to thank the studs, Goodwoodstud and Wellfieldstud. Without their cooperation the project would not be fulfilled and successful.

Thanks to Post Mortem at Massey University for the opportunity to use their space for the placenta examination and the post mortem examination of the neonatal foals.

Anne Marks and Ngaire Larsen were really helpful during the research project period and when I was writing my report, thanks for this. Thanks to Marielouise Bijnen for sharing the weekly days and weekends picking up placentas. At last I want to thank everyone else who helped with the project and came with good ideas.

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9. Appendix

9.1 Blastocyst development

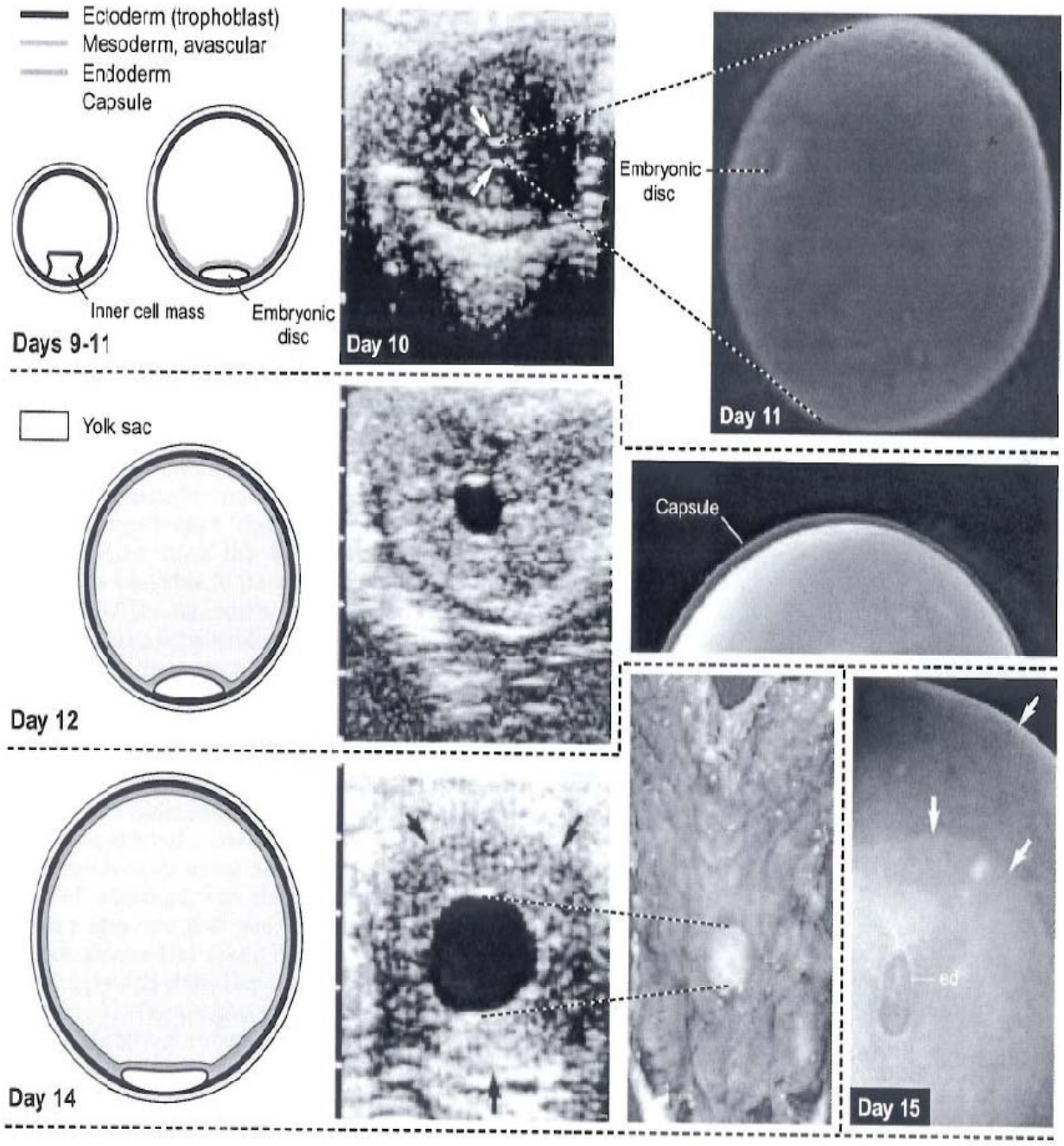

Figure 22: Blastocyst development. Diagrams, sonograms and photographs of the embryo for days 9-15 (Ginther, 1998).

9.2 Photos placenta examination

The following photos have been taken during several placenta examinations. Written down at the caption are the cause of the phenomena and what they are. The casefiles are available at Massey University, searched by P09 number.

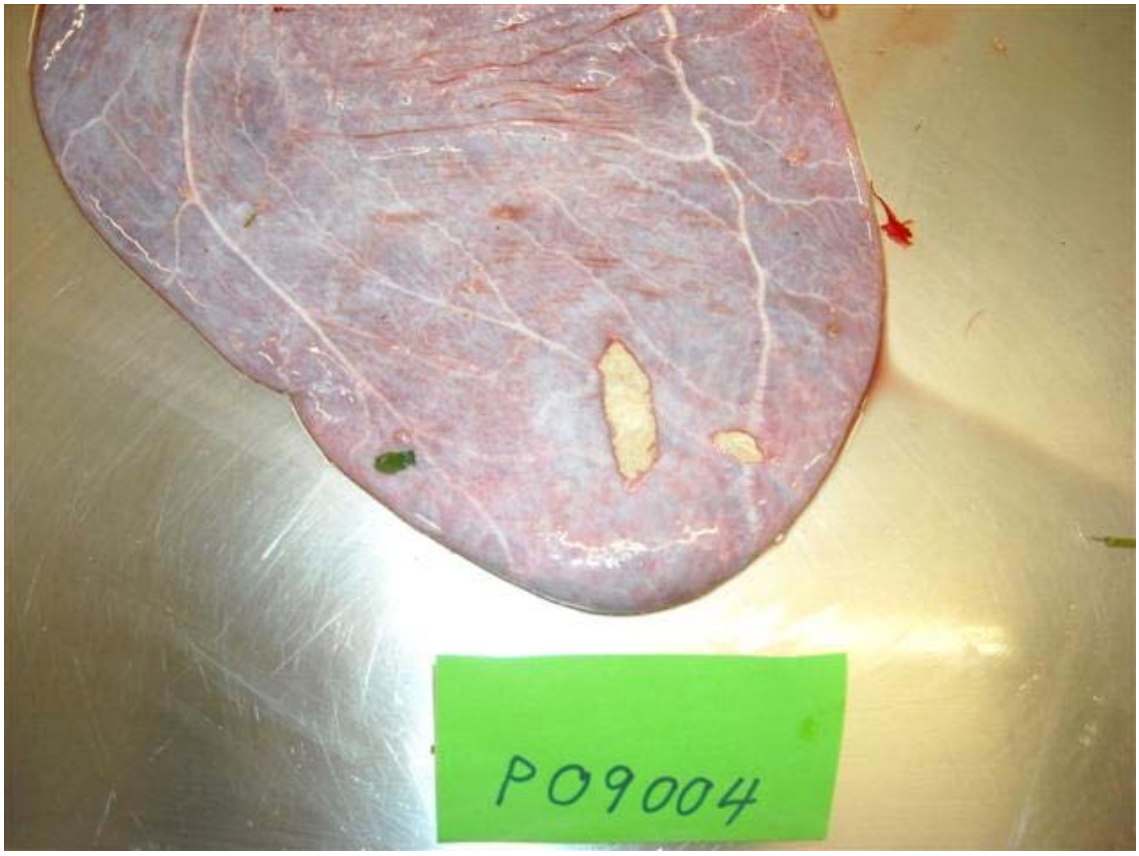

Figure 23: Hippomane attached to the pregnant horn, allantoic surface of the placenta. Cause is not really known, hippomanes can be found over the total placenta surface (P09004).

Figure 24: Tip pregnant horn, allantoic side of the placenta. Mineralization of the tip of the pregnant horn. Cause is not really known (P09030).

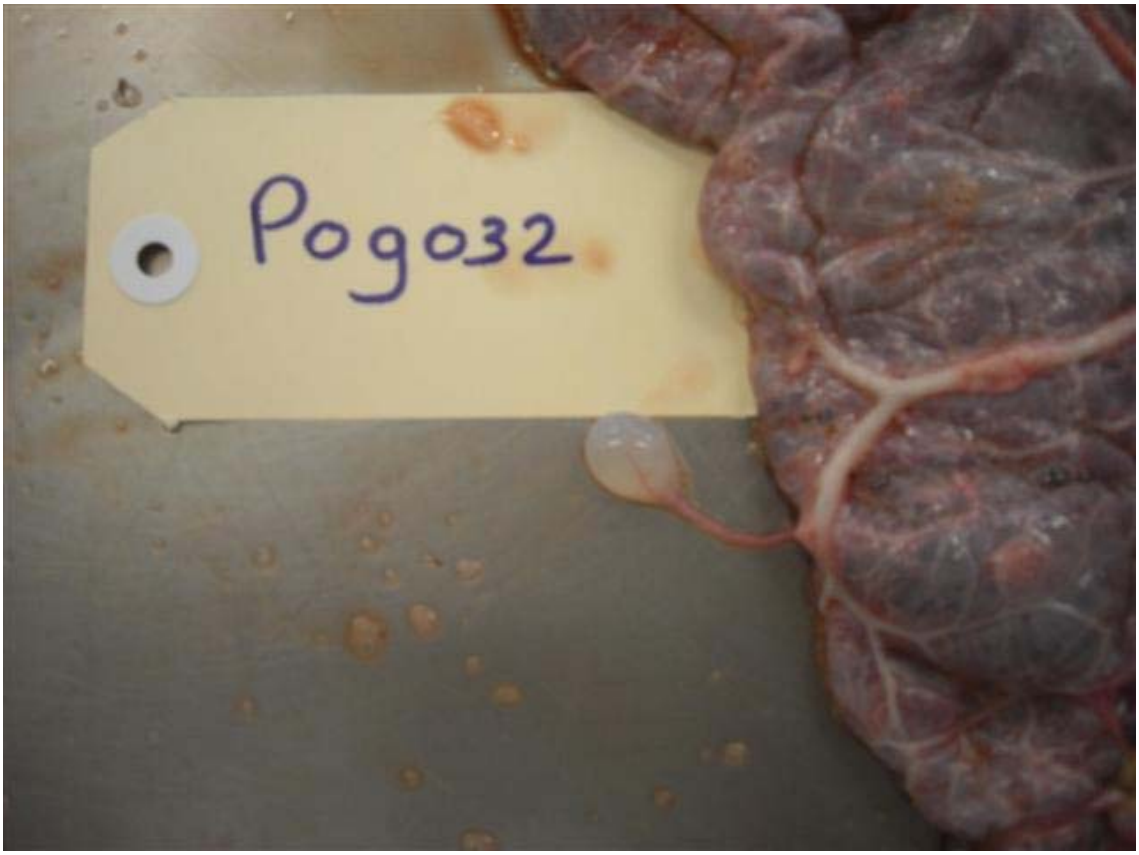

Figure 25: Allantoic side of the placenta, allantochorionic pouch found along the non pregnant horn, can be found on total placenta site. Caused by degenerating endometrial cups at day 80 of pregnancy. The cups can be enclosed by folds of allantochorionic tissue or can stay free (P09032).

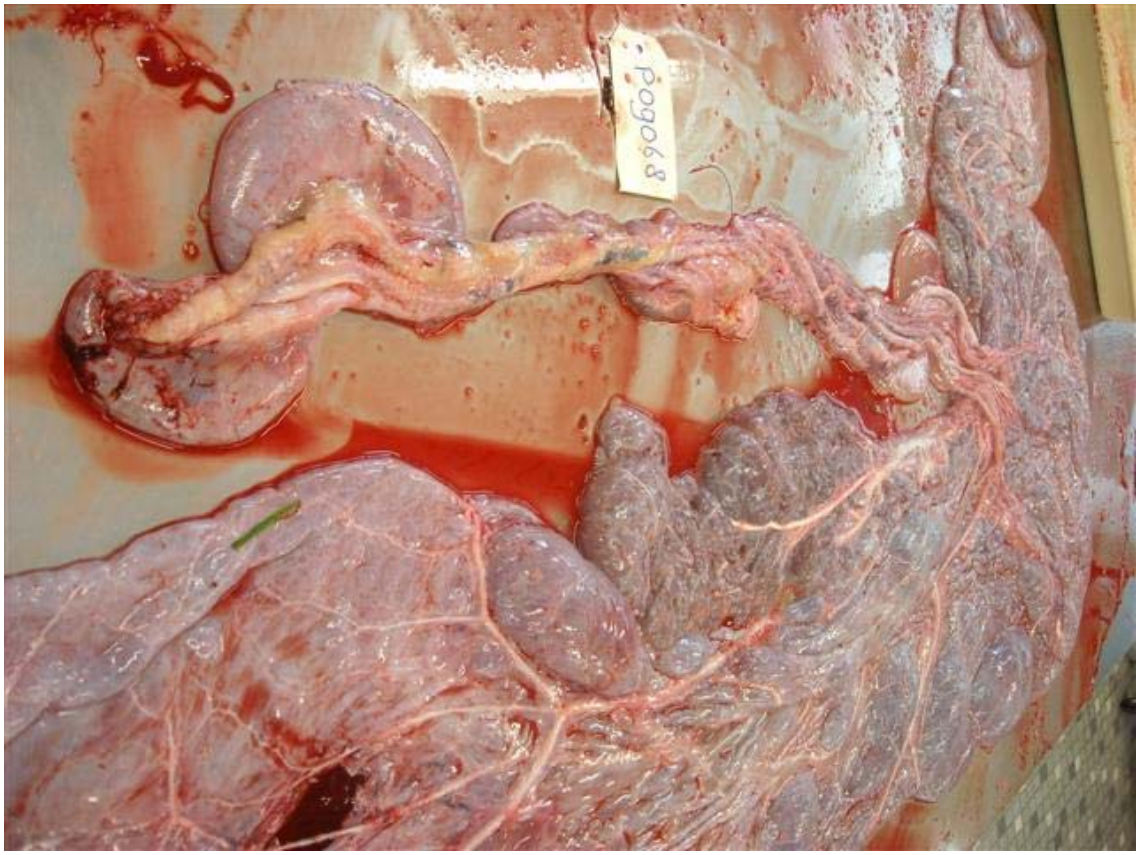

Figure 26: Dilations along the umbilical cord, allantoic side of the placenta. Caused by pressure in de vessels (P09068).

Figure 27: Allantoic side of the placenta, close up of the attachment of the umbilical cord. Dilations along the blood vessels, may be caused by pressure (P09068).

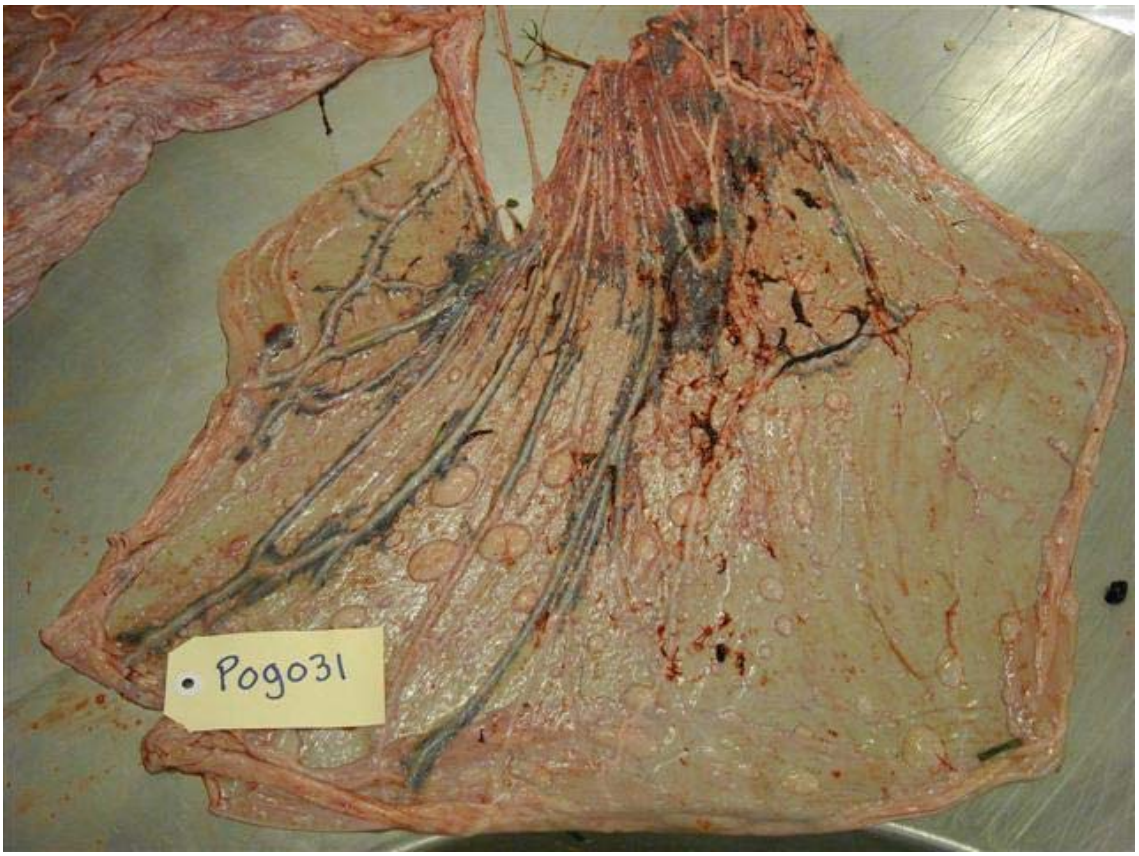

Figure 28: Amniotic membrane, engorged amniotic vessels, probably caused by compressed vessels due to pressure (P09031).

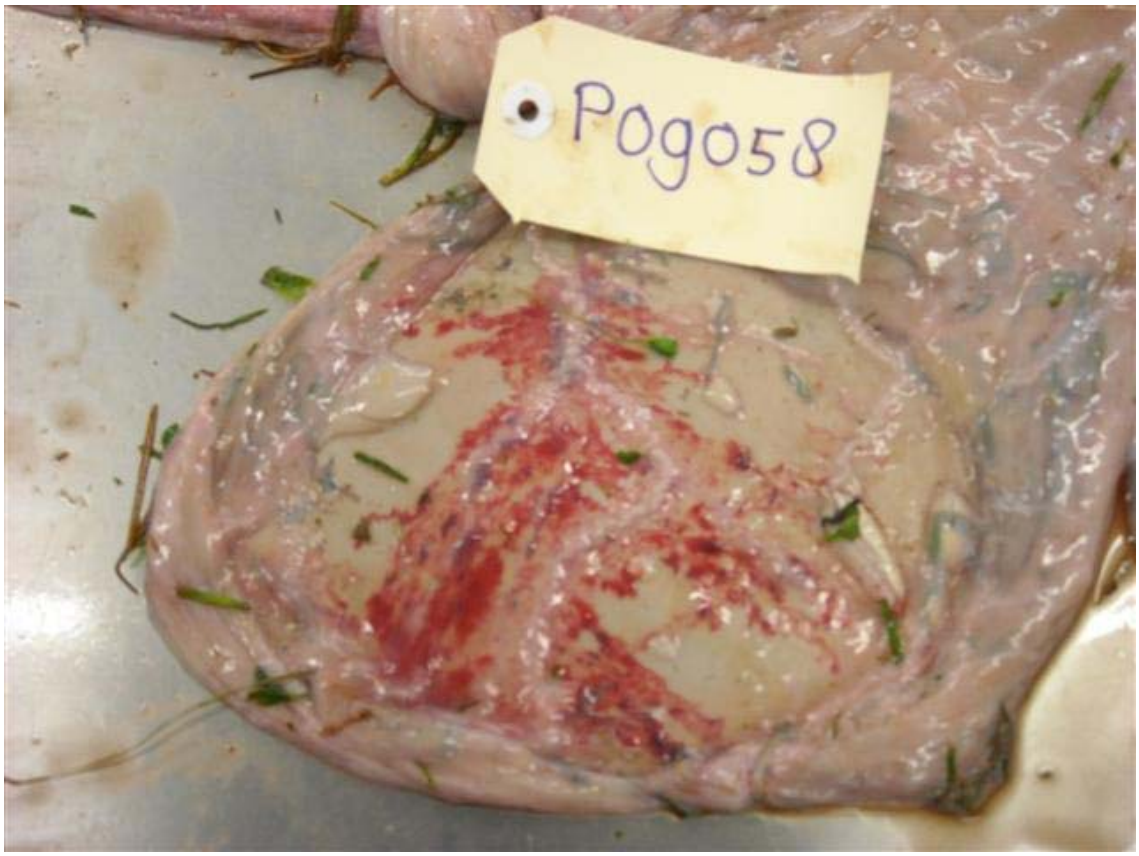

Figure 29: Blood staining on the amniotic membrane. May be caused by compressed vessels due to pressure (P09058).

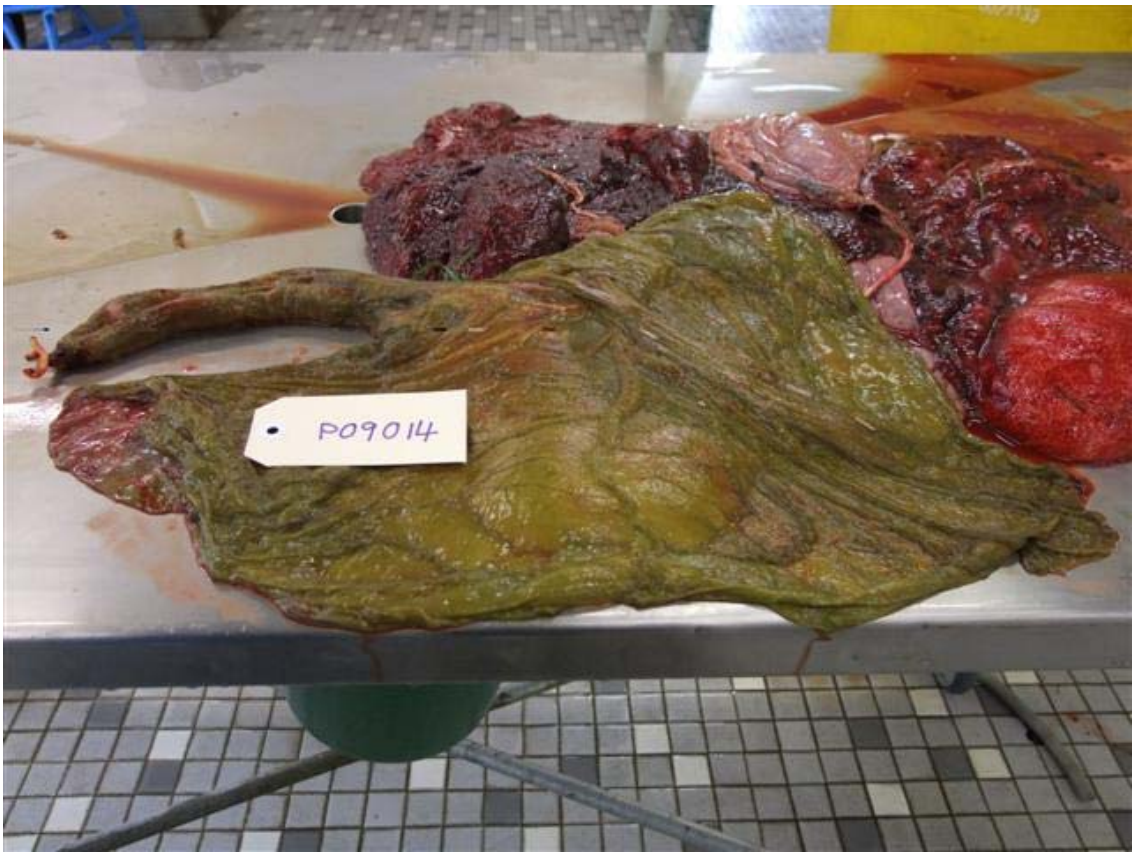

Figure 30: Meconium staining amnion and umbilical cord. In response to severe stress in utero , caused by fetal or placental infection, cord compressions or placental separation, meconium staining develops (P09014).

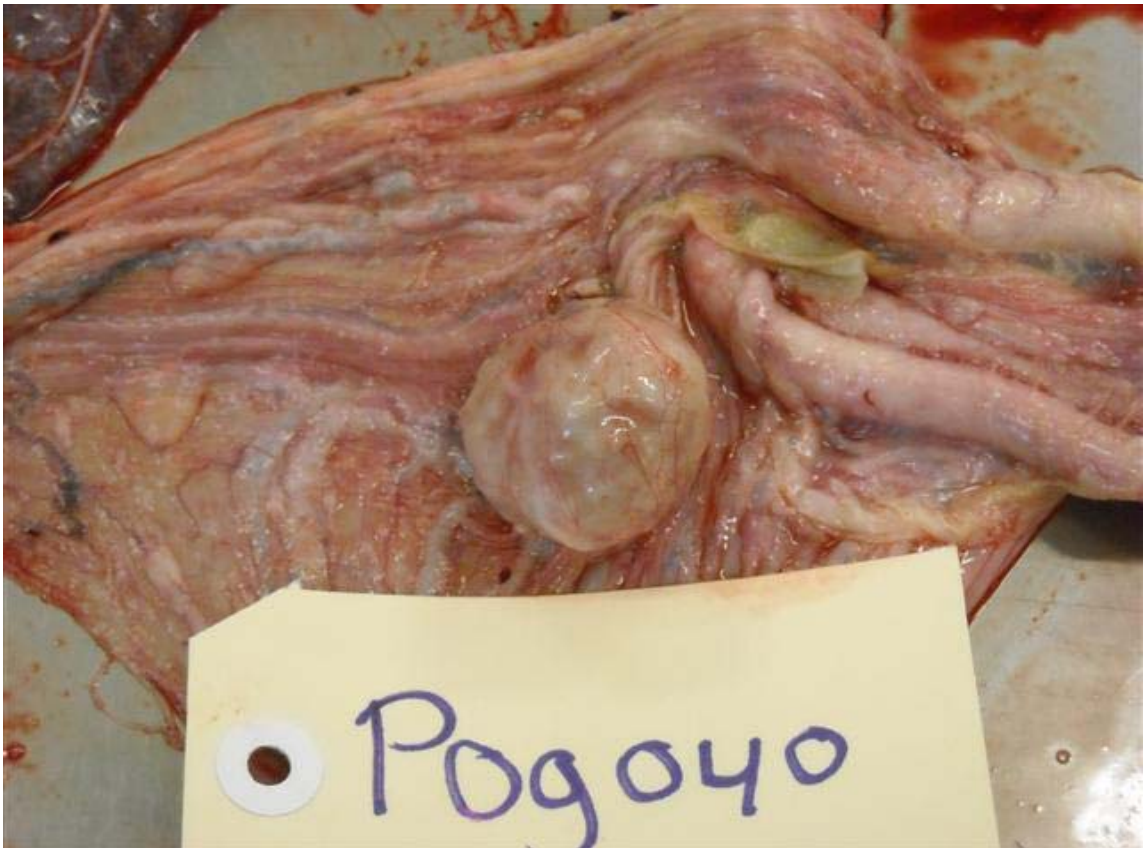

Figure 31: Remnant of the yolk sac found in the amniotic membrane. The remnant develops when the yolk sac regresses and gets incorporated in the umbilical cord (P09040).

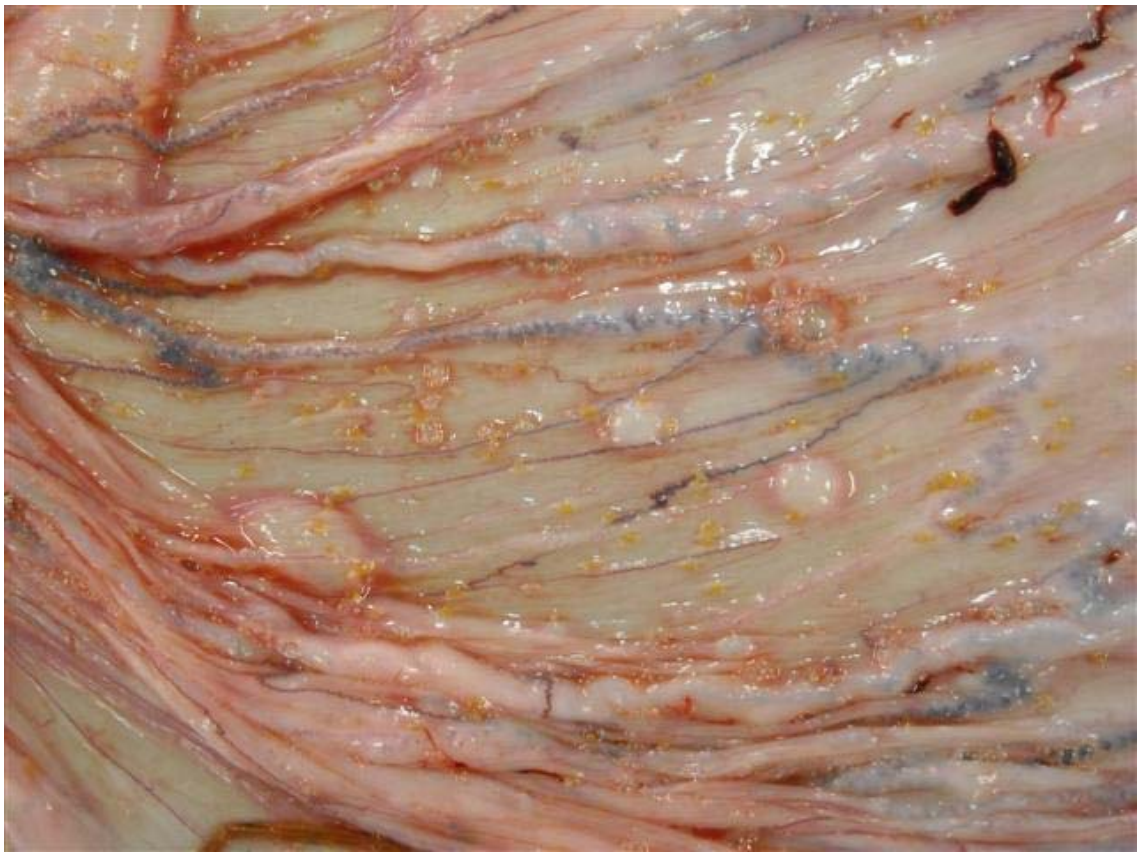

Figure 32: Amniotic plaques on the amniotic membrane. Caused by dystrophic calcifications on the amniotic membrane (P09027).

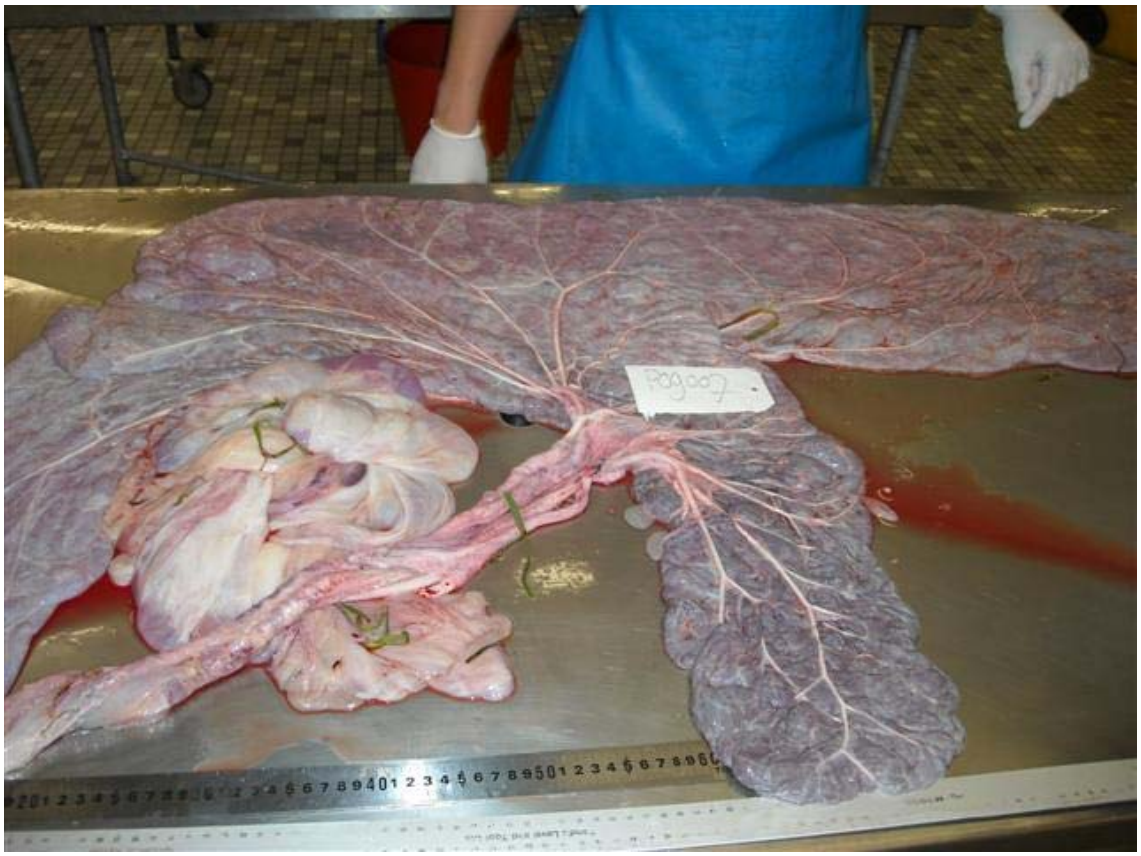

Figure 33: Different umbilical attachment than normal. The attachment of the umbilicus is dependent on where the embryo fixates in fixation stage (P09007).

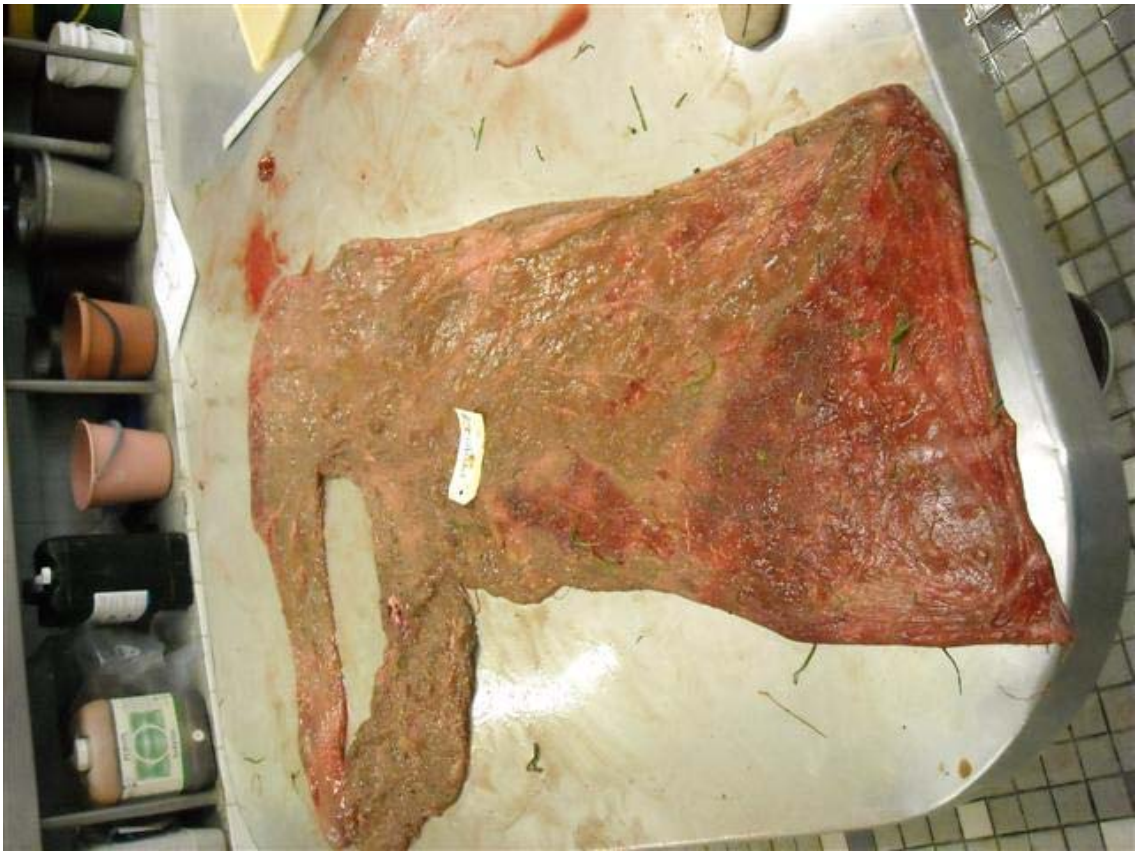

Figure 34: Severe placentitis, chorionic site of the placenta. In New Zealand mostly caused by an ascending bacterial infection. Clear area of discolouration visible around the cervical star (P09027).

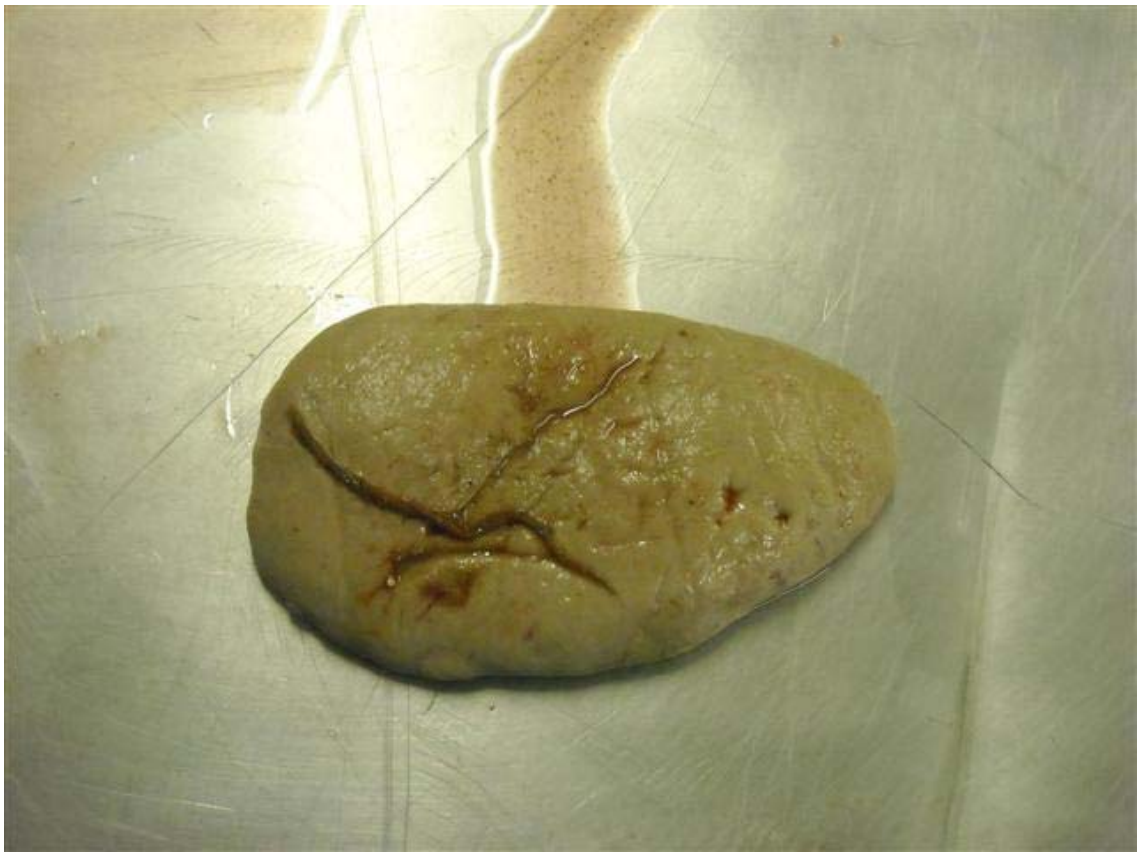

Figure 35: An hippomane, mostly found by inspecting the amnion, normally free in the allantoic cavity. Mostly light brown, rubbery structure. This structure is formed by minerals and waste products.

9.3 Post mortem case files

Case PM 44029 (17-09-2009)
History:
Foal one day old was kicked by the mare and died overnight.
Foal:
The 51 kg filly foal was in good body condition score. Hair was missing in a possible-hoof shaped print over the left. There was severe subcutaneous oedema and extensive haemorrhage in the upper left limb extending to the caudal abdomen. There was a displaced mid-shaft femoral fracture present at the time of examination.
Placenta:
Not presented.
Probable diagnosis:
Death due to blood loss resulting from rupture of the femoral artery associated with a displaced femoral fracture.

Case PM 44030 (17-09-2009)
History:
Close to full-term gestation, presented as dystocia with head at vulva only.
Foal:
The foal showed signs consistent with dwarfism with overall disproportionate short stature. The head was dome shaped with a severe prognathism, abnormally lax joints of all lower limbs, in good body condition. The trachea was collapsed along the entire length.
Placenta:
Intact with gross contamination. Yolk sac remnant present.
Diagnosis:
Dystocia due to severe congenital abnormalities/ dwarfism.

Case PM 44053 (23-09-2009)
History:
Full-term gestation, stillbirth.
Foal:
65 kg filly foal in good body condition. Swelling was presented around the ventral mandibular and sub laryngeal region. Grossly haemorrhages were presented in part of the cortex of the brain and heart muscle. Only a small portion of the lungs were aerated.
Placenta:
Torn with gross contamination.
Diagnosis:
Findings support the tentative diagnosis of terminal hypoxia/ anoxia due to dystocia.

Case PM 44060 (24-09-2009)
History
Full-term gestation, stillbirth.
Foal:
50 kg filly foal in good body condition. There was evidence of bleeding on the gums, and grossly haemorrhages were present in the brain and the heart muscle.
Placenta:
No significant bacteria cultured. No gross abnormalities.

Diagnosis:
Most likely to be terminal foetal hypoxia/anoxia associated with difficult delivery.

Case PM 44085 (29-09-2009)
History:
Full-term gestation, stillbirth.
Foal:
51 kg filly foal in good body condition. Haemorrhages were grossly apparent around the brain, in the heart and on serosal surfaces of the small intestine.
Placenta:
Retained fetal membranes, autolysed.
Diagnosis:
Terminal fetal hypoxia/anoxia associated with difficult delivery.

Case PM 44090 (30-09-2009)
History:
Full-term gestation, stillbirth, dystocia.
Foal:
65 kg filly foal. Multiple haemorrhages grossly visible in the brain, heart muscle, adrenals and kidneys.
Placenta:
Not presented.
Diagnosis:
Findings support a tentative diagnosis of terminal fetal hypoxia/anoxia associated with dystocia.

Case PM 44094 (1-10-2009)
History:
Birth was attended by a veterinarian. The foal was born alive, but died within minutes following birth.
Foal:
Colt foal in good condition and hydration. Mild degree of meconium staining of the perineum and gaskin. Slippers were presented at all four feet. Meconium was noted around the nares and muzzle, and in the oral cavity and larynx.
Placenta:
Meconium staining on the placenta, faeces present, plaques on amnion and tip pregnant horn.
Diagnosis:
Perinatal stress/ hypoxemia caused by dystocia.

Case PM 44120 (7-10-2009)
History:
Full-term gestation, head back/ dogsitter.
Foal:
63 kg colt foal in good condition. Contracted tendons presented. Fluid in abdomen and in pericardium. Haemorrhages in the brain. Cystic structure attached to left kidney.
Placenta:
Not presented.
Diagnosis:
Dystocia.

Case PM 44164 (17-10-2009)
History:
Mare didn`t bag up. Red bag delivery. Mare had been eating macrocaipa. Treated with tmps first 5 days of the ninth month. Previously had neomycin. Regumate on day 120. History of pregnancy loss.
Foal:
43.6 kg filly foal with moderate fat stores. Not a lot of bleeding. Thoracic and abdominal organs within normal limits.
Placenta:
Torn, thickened chorionallantois. Umbilicus engorged and reddened.
Diagnosis:
Premature placental separation due to unknown cause.

Case PM 44215 (2-11-2009)
History:
Born with polydactyly, euthanized after birth. Full-term gestation.
Foal:
51 kg filly foal in good body condition. Carpal flexion and abnormal flexion of the phalanges.
Placenta: Not presented.
Diagnosis:
Carpal flexion/ polydactyly, congenital abnormality.

9.4 Photos post mortem examinations

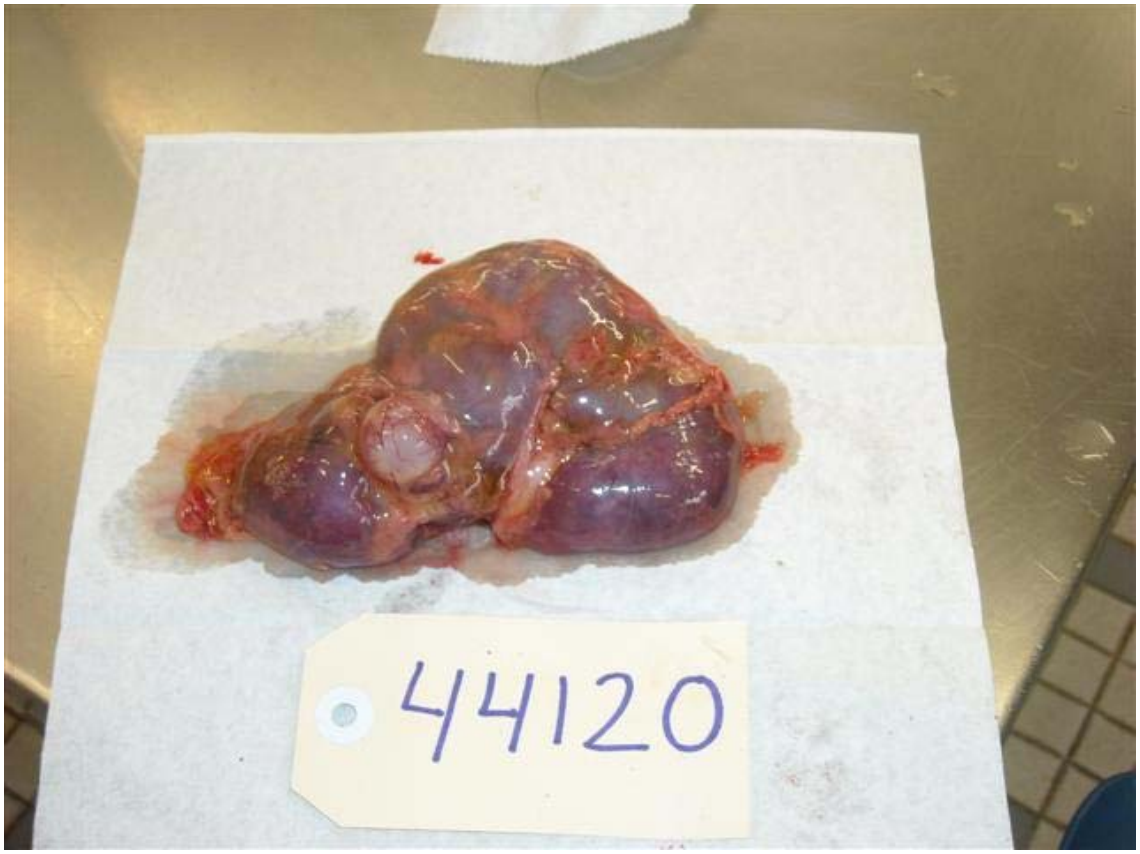

Figure 36: Apparent cyst on a kidney (PM44120).

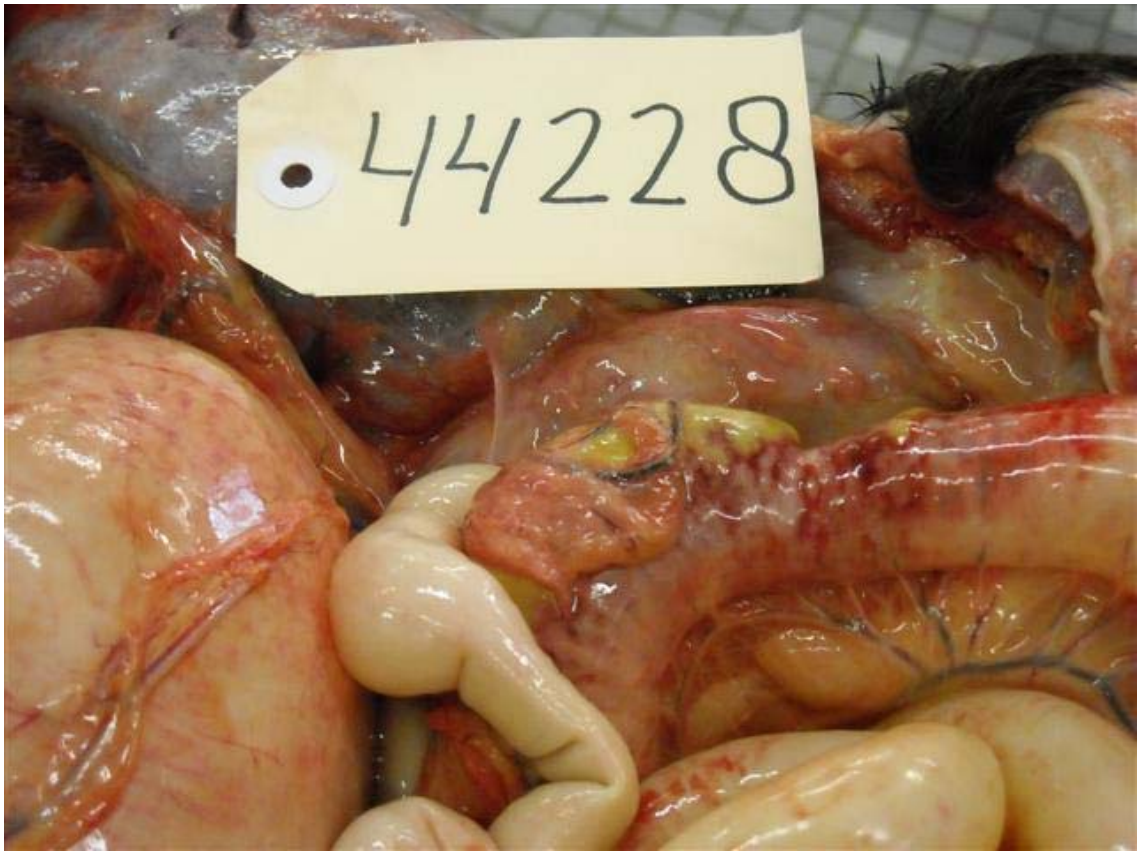

Figure 37: Severe mucosal damage of the intestine, almost opened. The foal was euthanized (PM44228).

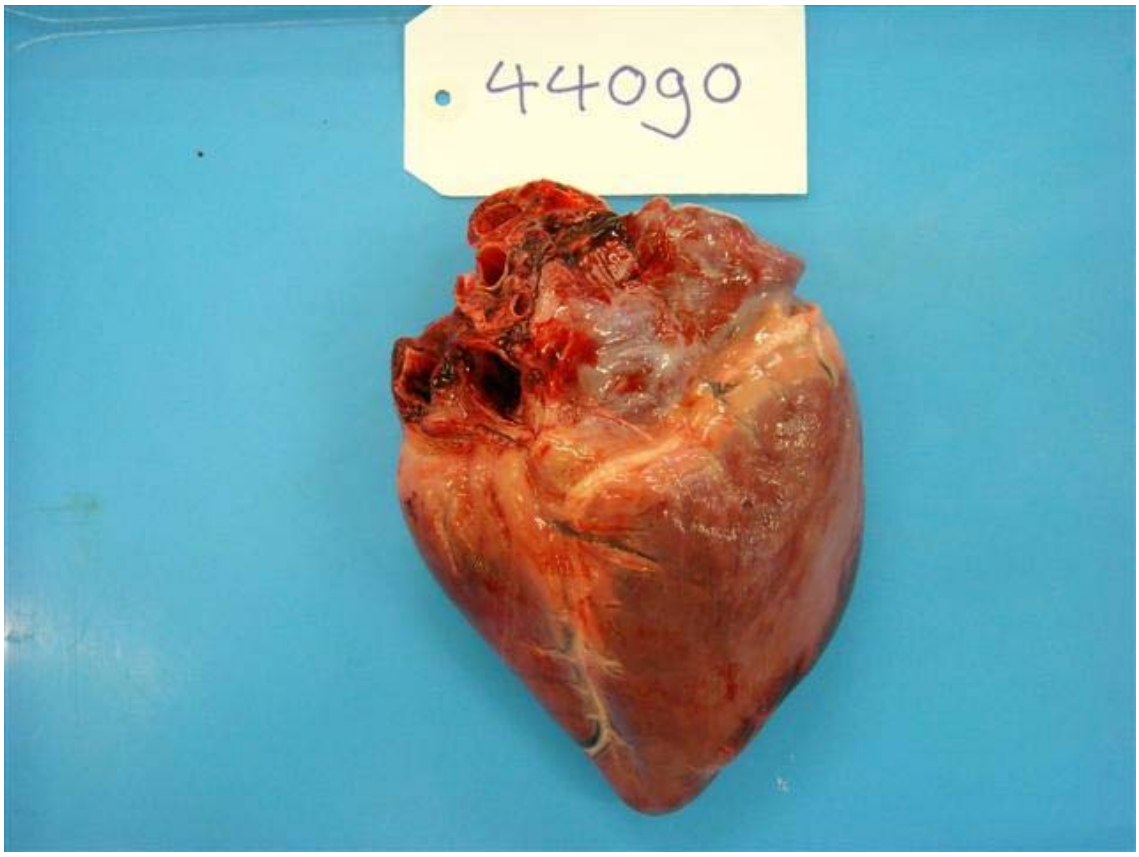

Figure 38: Haemorrhagic areas at the heart due to dystocia (PM44090).

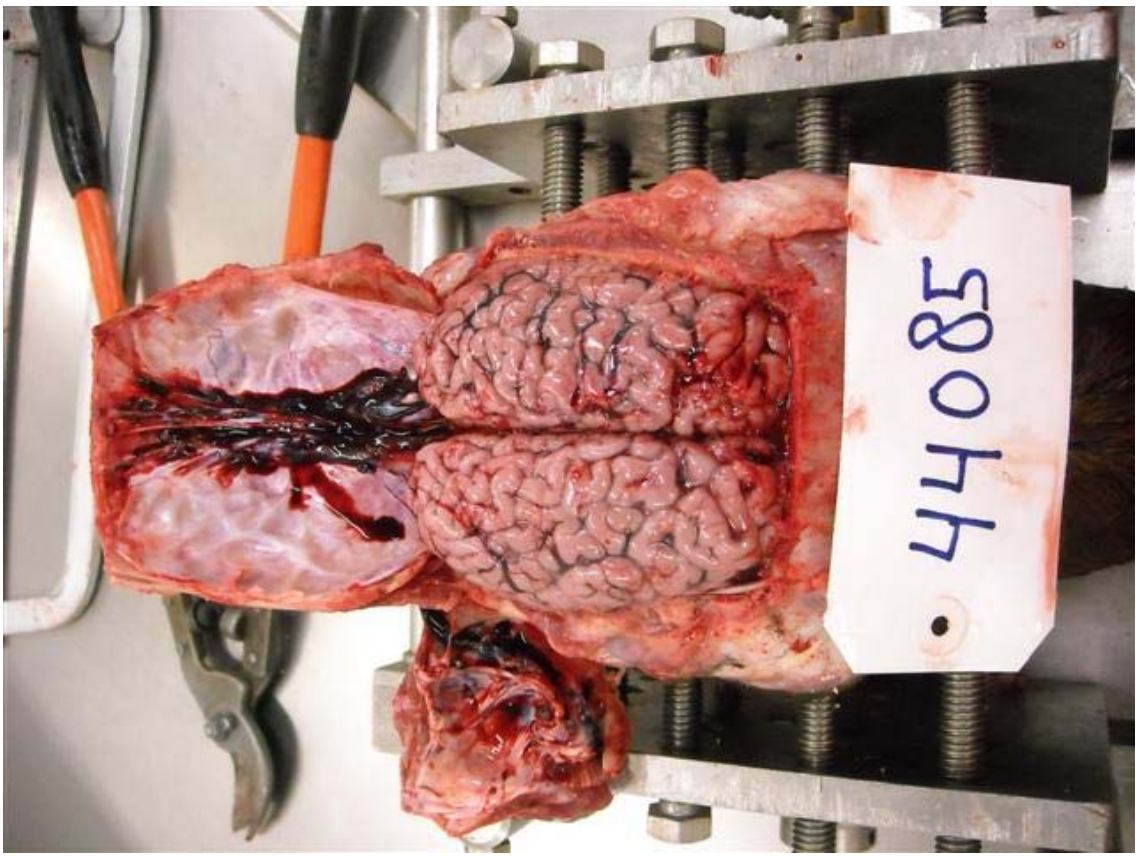

Figure 39: Severe haemorrhagic areas in the brain of a foal, caused by lack of oxygen due to dystocia (PM44085).

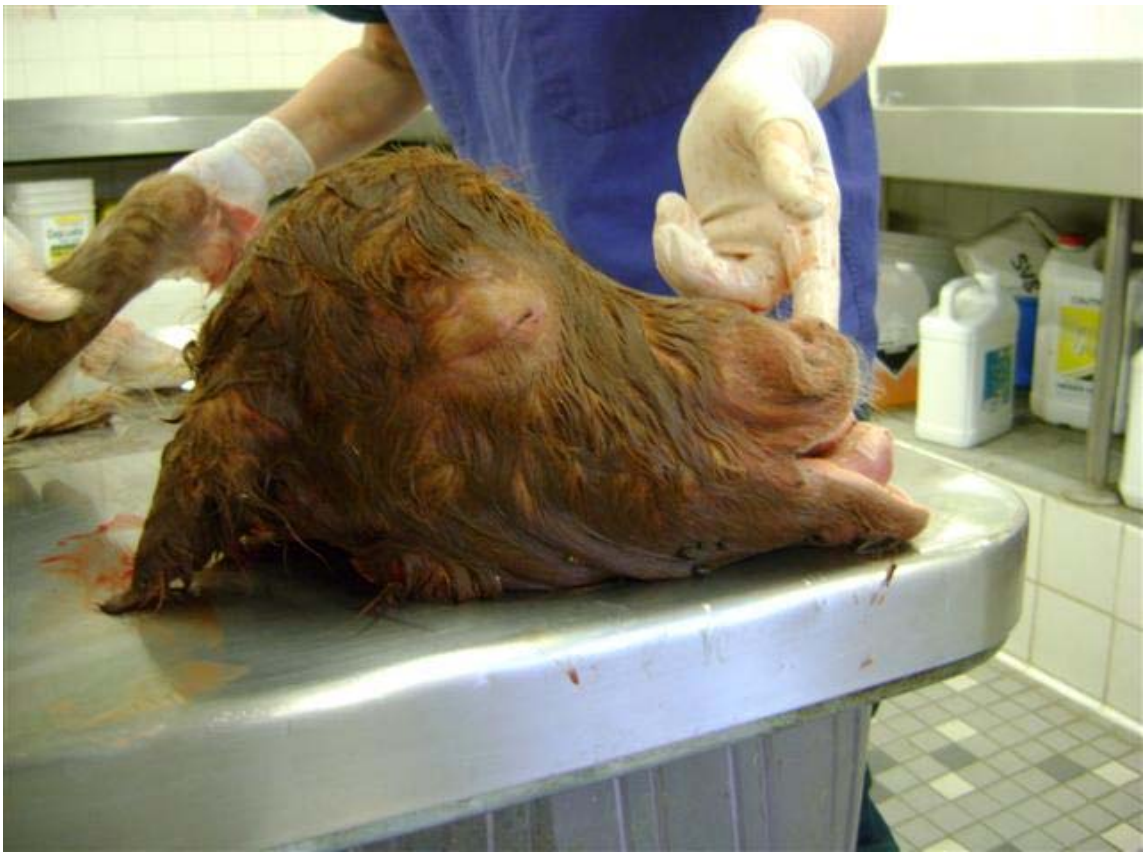

Figure 40: Dome shaped head with severe prognathism and dwarfism of a miniature horse foal.

Figure 41: Front limbs of the miniature horse foal, congenital abnormality, dwarfism.

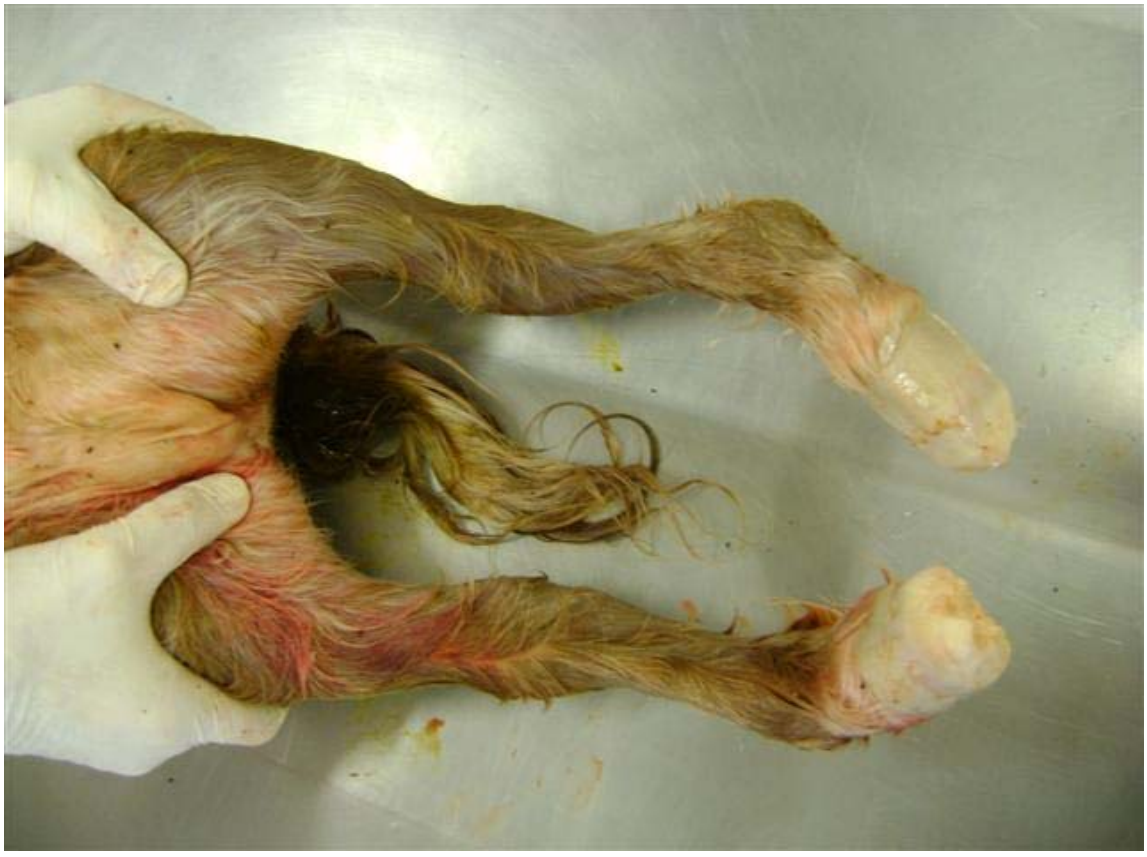

Figure 42: Hind limbs of the miniature horse foal. Congenital abnormality, dwarfism.

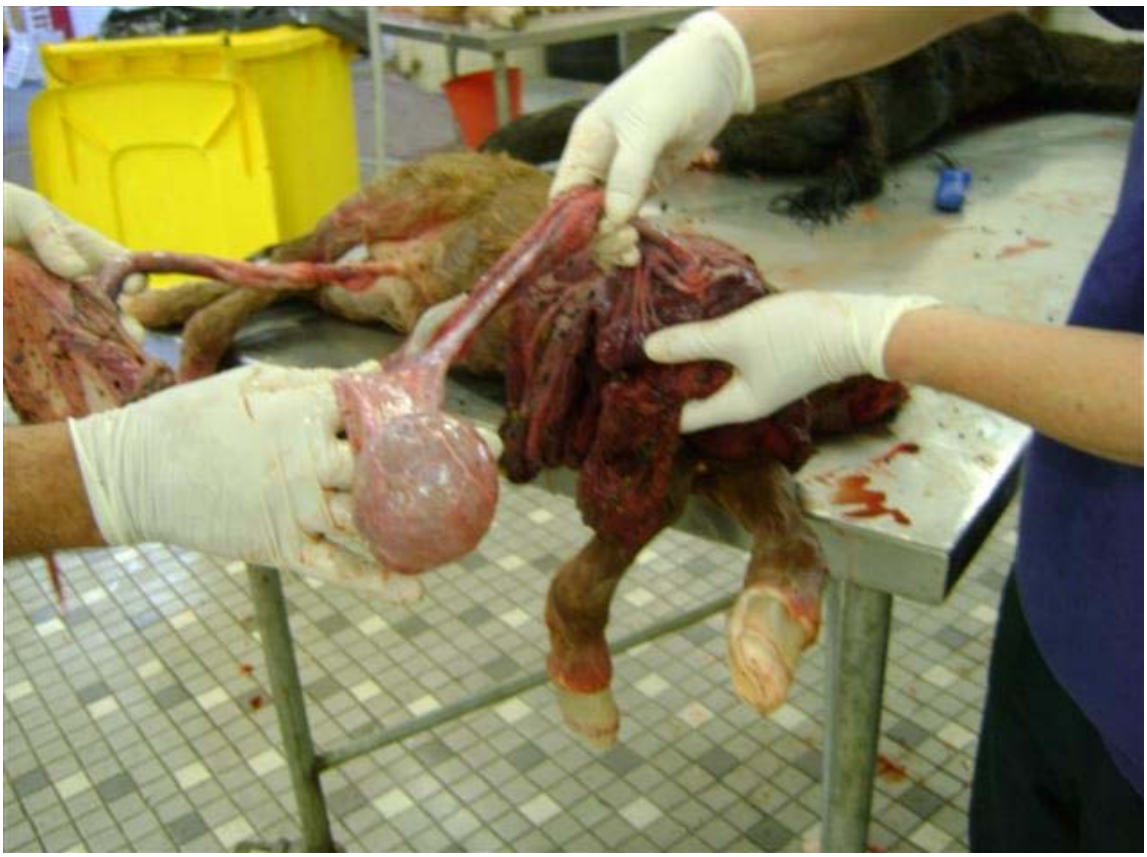

Figure 43: Remnant of the yolk sac in a miniature horse foal.

Figure 44: Carpal flexion/ polydactyly in a Thoroughbred foal. Congenital abnormality, foal was euthanized (PM44215).