PERINATAL LAMB MORTALITY — ITS INVESTIGATION, CAUSES AND CONTROL

K G HAUGHEY*

ABSTRACT

Methods of investigating perinatal loss in grazing sheep flocks are reviewed and evaluated. The "wet-dry" method is the simplest method for assessing minimal prevalence, whereas the differences between the numbers of single and twin foetuses present at ultrasonic determination of litter size during pregnancy, and the numbers of single and twin lambs present at lamb-marking, is the most precise. The veterinary investigation of field mortality involves full autopsy of a representative sample of dead lambs, a history of prenatal nutrition, disease and husbandry, as well as a qualitative estimate of weather conditions over the period of lamb collection. Pathological processes may be identified in over 95% of deaths and the specific cause determined in about 75% of deaths. The identification of the specific causes in the remainder of deaths, all classified as the starvation-mismothering-exposure (SME) complex, requires intensive, costly, on-site observation, and physiological and biochemical assessment. The probable causes of these deaths include prenatal physiological handicaps resulting from placental insufficiency, aberrant parent-offspring behaviour, management-induced mismothering, misadventure, inadequate milk supply or teat and udder abnormalities, and cold-induced starvation. The gross pathology and pathophysiology of birth stress and the SME complex, which are associated with at least 80% of mortality, are summarised. Birth injury to the foetal central nervous system, characterised by cranial and spinal meningeal haemorrhage is exclusive to parturient deaths and the SME complex. Observed flock prevalences range from 81% to 100% in parturient deaths, and 20% to 57% in the SME complex. The high total prevalence and experimental evidence, indicate the major causal role of birth stress in the pathogenesis of these entities. Lethal congenital malformations, infections (both congenital and acquired after birth), trace element deficiencies and predation are reviewed as minor causes. The new understanding of the pathogenesis of perinatal lamb mortality, recognises the heritable nature of birth mass, maternal pelvic dimensions, parent-offspring behaviour, and the resistance of neonates to cold. Control measures need to incorporate selection for maternal rearing ability, further refinement of prenatal nutritional management of twin-bearing ewes, disease control, provision of shelter for lambing flocks, and avoidance of husbandry practices which frustrate innate parent-offspring behaviour. A selection programme is summarised.

Key words: Perinatal, lambs, mortality

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*22 Stromlo Place, Campbelltown, New South Wales, 2560 Australia

INTRODUCTION

Perinatal lamb mortality, defined as deaths occurring shortly before, during or within 7 d of birth, is widely recognised as a major source of reproductive wastage among sheep[9]. It may account for 80% to 90% of preweaning mortality[33 74]. Numerous reports on loss in lambs reflect a serious and intractable, world-wide problem. Average perinatal loss in Australia, New Zealand and Great Britain ranges from 10% to 20% of lambs born[33 43 81]. Reliable data on the national level of loss under the wide range of environmental and management conditions in South Africa are scarce. Two recent studies reported that 12,6% of lambed ewes lost their lambs within one month of lambing[50], and that perinatal loss among intensively-managed flocks in the Western Cape, averaged 15,1% of lambs born[67]. Mortality ranging between 16% and 23% of lambs born, was recorded in apparently well-managed individual flocks of several breeds[25 73 127 128]. Despite a marked improvement of nutrition of ewes during pregnancy and early lactation, husbandry, and disease control in many flocks, perinatal losses remain unacceptably high. Recent evidence suggests that intrinsic maternal and/or foetal defects, including birth asphyxia and trauma, aberrant parent-offspring behaviour and neonatal susceptibility to cold, play a major role in perinatal lamb mortality. This highlights the fundamental importance of the ewe-lamb partnership to lamb survival.

Perinatal lamb mortality varies greatly within and between breeds, flocks, districts, seasons and management systems, and may reach more than 50% of lambs born in exceptional circumstances. Its complex and variable aetiology, necessitates that both professional advisers and flock-owners have a clear knowledge of the magnitude and the causes of the problem before cost-effective control programmes can be implemented successfully.

This paper reviews the investigation, causes and control of perinatal mortality.

METHODS OF ESTIMATING THE PREVALENCE OF PERINATAL MORTALITY

A. Wet-dry technique[40]

The method classifies ewes at lamb-marking (preferably within a month of lambing), by visual appraisal and udder palpation, as (1) barren (2) lambed and rearing a lamb(s) (3) lambed and lost lamb(s). Barren (not lambed) ewes show neither udder development nor "lambing stain" (the staining of the posterior udder surface and hocks with dried lambing discharges) and usually show better body condition and fleece quality than lambed ewes. Lambed ewes have enlarged udders containing secretion, usually show "lambing stain", have poorer body condition and fleece quality than dry ewes. The class can be further subdivided into: (a) Ewes rearing a lamb(s) have full, resilient udders containing milk. Teats and adjacent areas of the udder are soft, pliable and clean due to the lamb's sucking. (b) Ewes which have lost their lambs (lambed and lost ewes) have variably developed udders, often with pronounced cleavage between the 2 glands and stiff, dirty teats, with secretion ranging from milky to thin watery, or thick, viscous, honey-coloured matter, depending on the period elapsed since the death of progeny.

Heavy contamination of the teats and udder with mud or dust may make classification difficult, and hence unreliable. Skilled palpation to detect sucked teats will overcome this difficulty. The ingestion of oestrogenic pastures may cause udder development unrelated to pregnancy and parturition.

Perinatal mortality is expressed as:

$$\frac{\text{(No of ewes losing lambs)}}{\text{(No of ewes lambing)}} \times 0.9\ \%$$

as about 90% of losses to lamb-marking occur during the perinatal period. The result is a minimal estimate of perinatal mortality because it takes no account of ewes losing part or all of a set of multiple births.

An indication of the prevalence of twinning is estimated as:

$$\frac{\text{(Number of lambed marked)}}{\text{(Number of ewes lambing)}} \times 100\ \%$$

B. Pregnancy diagnosis by real-time ultrasound[51]

Pregnancy diagnosis by this technique, as early as day 45 of pregnancy, enables appropriate nutrition of non-pregnant, single- and multiple-bearing ewes respectively, in late pregnancy and lactation, and improved management of multiple pregnancies during lambing. Provided the flocks of single- and multiple bearers are maintained separately until lamb-marking, the discrepancy between foetal numbers present at scanning and the number of single and twin lambs present at marking, offers the most precise estimate of perinatal mortality relative to litter size. This is calculated as 90% of the discrepancy between the values. It is still necessary for culling purposes to "wet-dry" the lambed ewes in order to identify ewes which "lamb and lose" lambs.

C. Carcase collection

This method usually underestimates losses under extensive grazing conditions. Carcases may be removed by predators or scavengers and/or easily missed during pick-up, despite the most diligent searching.

"Wet-drying" is probably the most widely applied method on commercial farms, despite its tendency to underestimate mortality in highly fecund flocks. This method is cheap, simple to apply and is suitable for both extensively- and intensively-managed flocks. It allows the detection of teat and udder abnormalities and, given the repeatable[68] and heritable[66] nature of rearing ability, it identifies barren and "lambed and lost" ewes for culling. Age-specific classification of the failure classes may suggest causes of failure. For example, high prevalences of failing to lamb among maidens is often associated with low body mass at mating, due to inadequate weaner nutrition or disease.

Table 1 illustrates the importance of perinatal mortality measured by the "wet-dry" technique, relative to other sources of reproductive wastage in selected Australian Merino flocks. Row G highlights the apalling net reproductive efficiency of better-than-average flocks, expressed as the proportions of ewes joined, that actually were rearing a lamb(s). The superior performance of Flock 5 was associated with the implementation of policies incorporating pasture improvement, disease control, culling barren and "lambed and lost" ewes, and the use of twin-born rams. Lambs marked/ewes joined, rose from 95% to 135% over a 15-year period (Haughey, unpublished data).

METHODS OF INVESTIGATING PERINATAL MORTALITY

1. Direct observation of the lambing flock by a team of skilled observers recording relevant data, including maternal behaviour[14]. It is a labour-intensive method, suitable mainly for research of parent-offspring behaviour in individual flocks. The pathological basis of mortality is often poorly-defined as complete autopsies may not be performed.
2. Autopsy, including appropriate microbiological, serological and histopathological examination, in association with a history of flock management, nutrition and disease control, and qualitative estimates of weather conditions during lambing. Competent investigators may identify the pathological processes involved in over 95% of deaths and the precise cause in about 75% of deaths[67]. The balance invariably comprise neonatal deaths typical of the starvation-mismothe-

Table 1: **Perinatal lamb mortality relative to the reproductive performance of selected, mixed-age, spring-lambing Merino flocks with lamb-marking percentages above the Australian average (70%) [Haughey, unpublished data]**

Flock	1	2	3	4	5
Performance		**% of ewes joined**			
A. Lambs marked	106	86	76	112	135
B. Ewe deaths - joining to marking	5	9	3	3	3
C. Dry ewes	15	23	11	10	7
D. Ewes lambing	80	68	86	87	90
Lambed ewes		**% ewes lambing**			
E. Ewes rearing lamb(s)	88	84	86	77	93
F. Ewes losing all lambs born	12	16	14	23	7
Minimum fecundity of lambed ewes (lambs marked/ewe lambing)	1,33	1,27	0,88	1,29	1,50
G. Ewes rearing/ewes joined	70	57	74	67	84

ring-exposure syndrome[59], the probable causes of which include prenatal foetal physiological impairment, aberrent parent-offspring behaviour, management-induced mismothering, misadventure, inadequate milk supply, teat and udder abnormalities, and cold-induced starvation. The lack of direct observation precludes specifying the precise role of these factors in pathogenesis.

3. Physiological biochemical and pathological methods including the recording of birth mass and rectal temperature, haematocrit, plasma concentrations of lactate and fructose about 15 min after birth and age at death, were claimed to differentiate causes of neonatal death associated with placental insufficiency, acute intrapartum hypoxaemia, inadequate thermogenesis and starvation[21 89].

Although a combination of the 3 methods provides the most efficient diagnosis, the high cost precludes their widespread use. Veterinarians experienced in the ecology of sheep production, are uniquely situated to use the autopsy method supplemented by a farm history in large scale surveys and the investigation of mortality in individual flocks. The autopsy method involves the postmortem examination of a sample of about 50 carcases, collected during the first 3 weeks of lambing. Carcases should be stored in the farm coolroom until submission, usually 3 times per week. Where practicable, a tag, noting relevant data including litter size, date of birth and death, should be attached to each lamb, accompanied by a subjective assessment of prevailing weather conditions over the period of collection, for example, hot, warm, or cold, wet or dry, calm or windy. At the laboratory, all unmutilated lambs are weighed as the dead mass of parturient deaths is identical to birth mass, and the dead mass of neonatal deaths is a reflection of birth mass. As the birth mass of lambs is rarely available in commercial flocks, it may be reliably estimated from the crown-rump measurement[67].

A systematic autopsy, including examination of the central nervous system (CNS), is performed[58 84]. Each carcase is classified according to its time of death relative to birth. This is of diagnostic use because specific entities tend to occur in specific time-of-death classes (Table 3): Ante-parturient death: deaths occurring before birth commenced; Parturient death: deaths occurring during or within 3 h after birth; Post-parturient death: deaths occurring more than 3 h and less than 8 d after birth.

In the absence of specific data, the age of post-parturient deaths is estimated as follows (Haughey, unpublished data):

Less than 2 d: fat catabolism is nil, slight, moderate or marked (see section on fat catabolism); if the lamb has not fed, there is no food in the abomasum or small intestine; if the lamb has fed, food has not passed beyond the small intestine; the large intestine contains meconium.

Two to 7 d: fat catabolism is nil, slight, moderate or marked; if the lamb has not fed there is no evidence of meconium in the large intestine; when the lamb has fed, there are variable amounts of milk ingesta throughout the almimentary tract, remnants of chyle are seen in the mesenteric lymphatics and the contents of the large intestine are gritty in nature, compared to the homogeneous consistency of meconium (Haughey, unpublished data).

Table 2 summarises the main features of the time-of-death classes[58 84]. Table 3 summarises the occurrence of common causes of perinatal loss relative to time-of-death classification[34 58 67].

Examination of the CNS is mandatory because of the major role of birth injury in neonatal as well as parturient death. The brain is exposed by removing the cranial calvarium with sharp-pointed foot-paring shears. The spinal cord is exposed by cutting and removing the vertebral arches.

MAJOR CAUSES OF PERINATAL MORTALITY AND THEIR DIAGNOSIS

The aetiology of perinatal lamb mortality is complex and involves the action and interaction of many factors, including weather conditions, genetic factors, deficiency of gross and specific nutrients during pregnancy, predation, infections, maternal and neonatal behaviour, adequacy of milk supply, management, and the effects of birth asphyxia and/or trauma on the foetal CNS. Many carcases show lesions of more than one origin. The multitudinous constraints to lamb survival have been reviewed by Alexander[9].

At least 80% of deaths usually fall into 2 categories at autopsy[58 67]:

1. Deaths occurring during, or within a few hours of birth due to uncomplicated birth stress. During severely cold weather (<5°C) this class may contain very small lambs dying of peracute hypothermia.
2. Neonatal deaths, classified as the starvation-mismothering-exposure (SME) complex, characterised at autopsy by evidence of starvation and cold exposure.

Maternal prenatal nutrition

For comprehensive reviews of this topic see the appropriate literature[88 89 111]. Excess or deficiency of gross nutrients during pregnancy exerts profound effects on the proportions classified as parturient deaths or the SME complex. Perinatal survival is related to birth mass by an inverted U-shaped curve, with the highest survival of lambs between 3 and 5kg[85 93]. Birth mass is influenced by maternal prenatal nutrition, litter size, placental size and foetal genotype. The level of maternal nutrition during the third trimester affects birth mass, due to the accelerated foetal growth that occurs in that period. Excessive feeding, while substantially increasing birth mass of mainly single foetuses, predisposes them to dystocia[130].

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Table 2: **Major pathological, and other relevant features characterising ante-parturient, parturient and post-parturient time-of-death classes**[58 84]

Criteria at autopsy	Time of death relative to birth: Before	During or within 3h	After
Ante-parturient death			
Generalised, subcutaneous oedema, autolysis, blood-stained serosal fluid, haemoglobin staining) mummification.	+	-	-
Parturient death			
Subcutaneous oedema of presenting portion of the foetus	-	70-80%*	-
Abdominal haemorrhage	-	10-30%	-
Meningeal haemorrhage	rare	80-100%	35-55%
Post-parturient death			
Thrombi, umbilical arteries	-	variable	+
Breathing	-	variable	+
Walking	-	rarely	usually
Feeding	-	rarely	sometimes
Colour of fat depots			
Perirenal, Pericardial, Epicardial	pinkish cream, often haemoglobin-stained	pinkish cream	red-brown to pinkish cream depending on level of thermogenic activity

+ : present; - : absent; * : prevalence.

Underfeeding restricts the growth of litters, reduces their lipid reserves and neonatal vigour, and impairs colostral production, so necessary for thermogenesis during the first few hours of adaptation to neonatal existence[90 91]. The latter studies indicated that the current feeding recommendations[1] underestimate the nutritive requirements of twin-bearing ewes in late pregnancy by 100%. These handicaps create the potential for high mortality among litters because of low lamb vigour and predisposition to hypothermia. Ongoing penalties also accrue from undernutrition earlier in pregnancy, notably the death of fertilised ova, embryonic death during implantation and reduced placental size.

The importance of placental size to foetal growth has been underestimated, since a recent study[21] incriminated placental insufficiency in 24% of deaths. Placental weight accounted for almost two-thirds of the variation in birth mass[88] in a wide range of nutritional treatments. Although most of the within-group variation remained unexplained, moderate underfeeding in the second trimester retarded placental growth. Underfeeding during late pregnancy appears to further retard placental growth of twin foetuses but not that of singles. To an extent, improved nutrition during late pregnancy compensated for small placental size.

Increased litter size reduces birth mass because litters must share both the discrete number of maternal caruncles available for implantation, and the substrates available for foetal growth. Although birth mass is reputed to have a low to medium heritability[105], some sires are prepotent at siring very large lambs[86], suggesting that heritability may have been underestimated. Large birth mass associated with a heritable prolongation of gestation has been recorded in Merinos (Haughey, unpublished data).

Pathophysiological criteria[21 89] implicated prenatal physiological handicaps imposed by placental insufficiency, acute intrapartum hypoxaemia and inadequate thermogenesis, in 71% of perinatal deaths from a highly fecund flock, compared to 26% by conventional clinicopathological methods. The latter apparently did not include adequate pathological assessment of birth stress. This result emphasises the importance of interactive maternal and foetal factors in perinatal mortality.

Efficient sheep farmers recognise the detrimental effects of prenatal undernutrition by raising the level of nutrition of breeding flocks in late pregnancy. Indiscriminate supplementary feeding of mixed flocks of non-pregnant, single- and twin-bearing ewes, is wasteful because of their differing nutritive requirements. While it may improve the survival of twins, there is a danger that increased

Table 3: **Occurrence of major causes of death relative to time-of-death classification**[34 58 67*]

	Time of death: Before	During	After
Prevalence, %	<2	74-24	24-74
Cause of death			
Congential malformations	+	+	+
Congenital mineral deficiencies	+	+	+
Congenital infections	+	+	+
Birth injury	-	+	+
Infections acquired after birth	-	-	+
Predation	-	-	+
Starvation-mismothering-exposure complex	-	-	+

* - virutally all perinatal deaths fall into the parturient or the post-parturient time-of-death classes, except when abortion substantially raises the proportion of ante-parturient deaths.

birth mass of singles, will predispose to dystocia.

The advent of real-time ultrasound pregnancy diagnosis[51] for the detection of non-pregnant, single- and twin-bearing ewes early in pregnancy, allows selective feeding of these groups. The detrimental effects on placental growth of underfeeding twin-bearing ewes in early and mid-pregnancy is a powerful incentive to feed them preferentially throughout most of pregnancy. Blood glucose measurement[97], using a portable diabetic glucometer, shows promise for determining the nutritional status of twin-bearing ewes at 90 to 100 d of pregnancy.

Birth stress

Birth stress results from the effects of asphyxia and/or trauma on the foetal CNS during vaginal birth[113]. At autopsy its manifestations vary in the 2 time-of-death classes to which it is virtually exclusive, namely carcases classified as parturient deaths and the SME complex[58]. Birth stress is considered to be the primary cause of death only in those carcases in which there is no other complicating pathology e.g., infections, lethal malformations, or primary predation. In parturient deaths, gross evidence of birth stress includes: injury to the foetal CNS characterised by a variety of cranial subdural, cranial subarachnoid, and extradural, subdural and subarachnoid haemorrhages in and around the spinal meninges and spinal nerve roots (referred to hereafter as birth injury); subcutaneous oedema of the presenting portion of the foetus; abdominal haemorrhage due to rupture of the liver or tearing of the liver capsule; subpleural, subepicardial, subendocardial and thymic petechiae or ecchymoses. Contusions of the right myocardium are frequent. The skin and birth coat are frequently meconium-stained.

During the author's investigations, birth injury was the most sensitive index of birth stress in the parturient time-of-death class, with flock frequencies ranging between 80 and 100%, compared to 70 to 80% for oedema of the presenting portion of the foetus, and 5 to 40% for abdominal haemorrhage. Not all manifestations are necessarily present in the same carcase. In the SME complex, birth injury is the main manifestation of birth stress, invariably accompanied by varying degrees of catabolism of brown fat. Observed frequencies of birth injury among the SME complex, ranged from 20 to 57%. The overall mean prevalences of birth injury in 2 studies were 71% and 61%[59 67], highlighting the dominant role of birth stress in the pathogenesis of perinatal lamb mortality. The main sites of cranial subdural haemorrhage at decreasing frequency are: caudal fossa; middle fossa; adjacent to the fourth choroid plexus; over the dorsal cerebral and cerebellar surfaces. Cranial subarachnoid haemorrhage occurs most frequently along the course of the middle cerebral vessels. Most spinal birth injuries occur in the cervical segment of the spinal canal and spinal cord with lower frequencies in the thoracic and lumbar-sacral segments[67]. Some investigators have erred by confining their examination to the cervical segment only, thereby detecting only about 60% of occurrences of spinal birth injury[67].

Increasing duration of parturition is associated with falling foetal PO_2, rising PCO_2 and acidaemia (foetal asphyxia), damage to vital centres in the CNS and trauma to the spinal cord and spinal nerve roots[113 135]. Parturitional asphyxia may compound a pre-existing chronic foetal anoxia due to placental insufficiency. The prevalence of birth injury, and its severity as measured by the number of sites involved, were significantly correlated with duration and vigour of birth[62] and birth mass[67]. Parturient deaths were assumed to result from the effects of acute asphyxia on the vital centres of the foetal CNS. Less severe damage caused impaired sucking and locomotory activity[63], and increased susceptibility to hypothermia[11], due to temporary impairment of thermoregulation[42] in neonatal lambs. Birth-injured lambs succumbed to high ambient temperatures because impaired sucking activity precluded the maintenance of adequate hydration[63 122]. Neonatal mortality to 7 d of age among lambs surviving artificially prolonged vaginal birth, was double that of caesarean-born lambs[64].

The role of perinatal asphyxia and birth injury to the foetal CNS has been underestimated in perinatal lamb mortality. Perinatal mortalities and morbidities have been associated with these entities in human infants[113], primates[132], calves[61], foals[71], piglets[107] and guinea pigs[20]. Perinatal asphyxia is the main cause of depressed sucking activity in neonatal infants[38 133]. Foetal blood pH was lower and neonatal mortality higher among late birth order compared to early birth order piglets[107].

Foeto-pelvic disproportion, malpresentation of single lambs and less frequently of litters, and uterine load of polytocous ewes are the main causes of birth stress. Foeto-pelvic disproportion due to foetal oversize[67], small maternal pelvic size[49 68 87 103], or both, predispose to prolonged parturition. Although there is little published evidence to support the contention of an increased uterine load in polytocous ewes, the greater total foetal mass of litters compared to single births, must theoretically impose greater uterine work load during parturition, thereby increasing its duration, and the risk of pathological asphyxia and trauma to the CNS of the foetus during its extrusion through the birth canal. Support for this contention is set out in Table 4 which compares the mean birth masses, durations of Stage 2 labour[62], and maternal pelvic dimensions of uncomplicated single and twin births among fourth and fifth parity Merino ewes (Haughey, unpublished data).

The mean duration of Stage 2 labour for twin births was 2,01 times that for single births to deliver a 1,49 times greater foetal mass. Time to deliver the first twin was 1,75 times that for single births, despite the latter being 1,34 times heavier, with a mean interval of 19,3 min between first and second twin. Foeto-pelvic disproportion was an unlikely determinant of the duration of parturition of twins as their birth masses were significantly lower than those of single births and there was no difference between the pelvic dimensions of single and twin mothers.

Little information exists on the relationship between exercise during pregnancy and ease of birth. Penned, fat, pregnant Dorset Horn ewes, exercised on a tread-mill for 20 min daily for 3 weeks prior to parturition, had less dystocia than the unexercised controls (17% v. 50%; $p<0,05$) [J George 1983 CSIRO Pastoral Research Laboratory, Armidale, personal communication]. Lack of exercise and fat condition may contribute to perinatal mortality, particularly in winter rainfall environments where autumn-lambed flocks are often fed totally on supplements.

Starvation-mismothering-exposure (SME) complex

Post-parturient deaths are classified as the SME complex when there is evidence of hypothermia, manifested by varying degrees of brown fat catabolism, subcutaneous ("peripheral") oedema of the extremities, and changes in the adrenal cortex[60], accompanied by absence of, or inadequate amounts of milk ingesta in the alimentary tract. Carcases showing pathological features other than birth injury are excluded from the classification.

Foetal fat reserves in newborn lambs are composed of mitochondria-rich brown adipose tissue[7]. Below thermoneutral temperature, brown fat depots are important sites of non-shivering thermogenesis. Catabolism of brown fat occurs during cold exposure and independently of starvation[60]. At birth and at ambient tempera-

Table 4: **Mean (±SD), and range of birth masses, durations of Stage 2 of labour, maternal pelvic conjugate and transverse diameters of Merino single and twin births (Haughey, unpublished data)**

Parameter		Birth type				Significance of difference		
		Single	1st Twin	2nd Twin	Both Twins	t	df	P
Number		92	55	55	55			
Birth mass (kg)								
Single	vs 1st twin	7,4 ± 0,07 (1,9-6,6)	3,5 ± 0,07 (1,8-4,6)			11,07	145	0,001
	vs 2nd twin			3,5 ± 0,08 (1,9-4,7)		10,60	145	0,001
	vs both twins				7,0 ± 0,13 (3,7-9,2)	16,14	145	0,001
1st	vs 2nd twin		3,5 ± 0,07 (1,8-4,6)	3,5 ± 0,08 (1,9-4,7)		0,00	108	n.s.
Duration of Stage 2 labour (min)								
Single	vs 1st twin	73,9 ± 7,09 (5-400)	129,2 ± 13,29 (7-371)			2,70	145	0,01
	vs both twins				148,8 ± 14,33. (7-392)	4,02	145	0,001
Interval between 1st and 2nd twin					19,3 ± 2,31 (1-80)			
Maternal pelvic diameters (cm)								
Conjugate		11,6 ± 0,08 (10,3-14,4)			11,8 ± 0,12 (9,8-14,2)	1,45	145	n.s.
Transverse		8,2 ± 0,05 (7,0-9,3)			8,3 ± 0,05 (7,4-8,9)	0,20	145	n.s.

tures above thermoneutrality, the perirenal, pericardial and epicardial sites are cream-pink in colour. As the fat is depleted by thermogenesis, the fat depots change to a red-brown colour (hence the term "brown fat"), the extent and duration of the rise in metabolism modifying the degree of colour change. When the depots are red-brown, the lamb has exhausted its reserves of brown fat. Thus the colour of the perirenal, pericardial and epicardial fat depots is a sensitive qualitative measure of cold exposure and the level of energy reserves.

The degrees of fat catabolism are scored arbitrarily as: a) Nil (stable or uncatabolised fat) - the texture of the fat is firm, and the colour at all sites is cream-white or slightly pink; b)Slight - the prescapular, perirenal, and pericardial fat depots are distinctly pink, and the epicardial site (along the coronary grooves) is cream-white or slightly pink; c) Moderate - the prescapular, perirenal and pericardial fat depots are distinctly red-brown, similar in appearance to liver tissue. Epicardial fat is cream-white or slightly pink. Texture is less firm than the previous 2 classes; d) Marked - fat at all sites is gelatinous in texture and red-brown in colour.

Varying degrees of yellowish, subcutaneous oedema (up to 5mm thick) often occur in the distal limbs, and less frequently at the base of the tail, face, muzzle and ears ("peripheral" oedema). Duration and severity of exposure to cold weather affect the prevalence and degree of oedema. Under controlled conditions, oedema was first detected in newborn lambs with damp birth coats 4,5 h after exposure at 1°C in "still" air. Oedema extended from the coronets to above the carpus or tarsus after exposure for 8 to 10 h. After mild exposure (19°C), it was confined to the plantar and volar aspects of the pasterns. Its detection requires adequate reflection of the skin from the medial or lateral aspects of the distal limbs, tail, face and muzzle.

Changes in the adrenal cortex include cortical hypertrophy and focal petechiation, which are typical manifestations of severe systemic stress[116].

Thermo-neutral temperature in "still-air" for the newborn lamb is about 28°C. Below 28°C, thermoregulatory mechanisms are invoked to maintain homeothermy by shivering, catabolism of brown fat, and peripheral vasoconstriction to reduce heat loss[3 4 5 6 41]. The average lamb has sufficient energy reserves, mainly brown fat, to sustain maximum metabolic rate ("summit metabolism")[5 41] for about 20 min. When heat loss exceeds heat production, body temperature falls below that required for

normal metabolism and function, energy reserves are exhausted unless replenished from colostrum, and death results from primary or secondary hypothermia[65]. The extent and time of onset of thermogenesis is modified by ambient temperature[3], wind velocity[4], the thickness and wetness of the birth coat, skin thickness and birth mass[5 41]. Small lambs are vulnerable to hypothermia because of a wider surface area to mass ratio and lower energy reserves compared to those of large lambs[5 41].

During sporadic outbreaks of severe, cold, wet, windy weather (<5°C), catastrophic mortality may result from primary hypothermia. During blizzards the rate of body cooling may be so rapid in small lambs that death intervenes before brown fat can be catabolised, or before food can be digested even if the lamb has fed. In addition, severe weather depresses sucking ability[15 120]. "Sheep weather alerts", derived from physiological data[3 4 5], forecast the meteorological conditions conducive to primary hypothermia[94]. Fortunately in South Africa the severe weather conducive to primary hypothermia in newborn lambs, rarely occurs more than 2-3 times per 6-week-lambing period. Most deaths classified as the SME complex, result from secondary hypothermia. Secondary hypothermia is the result of exhaustion of substrates necessary for thermogenesis because of starvation during ambient temperatures higher than 5°C. Common causes of failure to feed, include birth injury to the foetal CNS[63], aberrant maternal[13], or neonatal behaviour or misadventure[123], udder or teat abnormalities, agalactia[77], and management-induced mismothering[102]. Invariably fat depots are moderately or markedly depleted and "peripheral" oedema is prevalent[60]. The mean percentages of the SME category, failing or ceasing to feed, approximate 50 ± 10%[67]. Pathological evidence in birth-injured lambs, suggests that cessation of feeding may be due to traumatic injury to the spinal cord and nerve roots resulting in loss of mobility and sucking dexterity, whereas failure to feed may be due to asphyxic damage to the feeding centres of the brain, resulting in suppression of sucking drive[67].

The initiation of a successful partnership between mother and offspring, involves exclusive bonding within a few hours of birth[8 10]. The visual, auditory and olfactory cues learned during this period, allow mutual recognition and acceptance so that close or proximate contact is maintained to allow frequent suckling. Maternal behavioural traits that should facilitate strong bonding, include[10]: the seeking of isolation for birth; the selection of a safe, sheltered birth site; birth of short or average duration; absence of interference with, or by, other parturient ewes; intense persistent grooming of all members of a litter; absence of aggression towards her own progeny; co-operation with the lamb's first attempts to suck; remaining on the birth site for at least 5 h; agitation at the absence of a lamb; the ability to keep the litter together after leaving the birth site; active defence of the lamb in the presence of a predator or a dog. Desirable behavioural traits of newborn lambs include[10]: standing soon after birth; sucking soon after standing; a well-defined "prone" response to handling; ability to follow the mother closely, and re-unite with her when separated; absence of separation from the mother. Breed comparisons indicate genetic diversity in some of these traits, including ease of birth[52 53], time spent on the birth site and ability to care for twins[13].

Many intrinsic and extrinsic factors adversely affect parent-offspring bonding. Maternal factors include: genotype[13]; maternal inexperience[12]; dystocia[2]; the birth of multiples[8 67]. Lamb factors include: genotype[120 123]; multiple birth type[8]; asphyxic or traumatic birth injury to the foetal CNS[63]; hypothermia[15 120]. Husbandry factors include: high stocking density of lambing ewes[14], including that induced by oestrus synchrony[78]; disturbance of lambing and recently-lambed ewes by human interference and supplementary feeding[102]; the prevalence of damaged teats and udders[77]. Nutritional factors include: prenatal undernutrition[101], low pasture availability near the birth site[14] and a poor milk supply[90]. Severe prenatal undernourishment of ewes, for example, during drought, may prevent or delay the onset of lactation. Affected ewes show poorly developed udders containing scanty, viscous, honey-coloured secretion in addition to severely-depressed maternal behaviour.

Genetic aids

The main thrust for more effective control of perinatal mortality must come from addressing the causes of birth stress and the SME complex as these entities are the largest components of mortality. These entities are currently minimised by prenatal nutrition, supervision and obstetrical assistance at lambing, and the provision of shelter, warmth and food to affected neonates. Despite their widespread and even intensive application, perinatal mortality has not been reduced below a seemingly intractable level of about 15% of lambs born[9], suggesting the involvement of unrecognised aetiological factors. For example, the mean perinatal mortality in 15 intensively-managed, including pen-lambed, South African flocks was 15,1%, ranging from 8,9% to 41,0%[67]. Intrinsic defects of the ewe-lamb partnership, and the compounding effects of some husbandry practices are now recognised as major causes of birth stress and the SME complex. Some of these defects can be manipulated genetically.

A. Selection for maternal rearing ability, including rearing of twins

The heritability of lifetime maternal rearing ability has been estimated variously between 0,1 and 0,2[32 99]. The reliability of these heritability estimates has been challenged because of possible pedigree errors[14]. More than 60% of rearing failures among ewes lambing on 4 occasions, occurred in slightly more than 25% of the flock[29 68]. In one study[29], ewes which always reared a lamb, or failed only once, weaned lambs on a mean of 90% of occasions, whereas ewes failing to rear on 2, 3 or all occasions, weaned a lamb on a mean of 59% of occasions, illustrating the wide variation in rearing ability within flocks. Ewes which reared a lamb at maiden lambing, weaned on average 8% more lambs over the next 3 years compared to ewes which lost their lamb at maiden lambing[68], suggesting that performance at maiden lambing is a useful indicator of subsequent rearing performance. The mean survival to weaning of line-bred single and twin descendants of ewes with high lifetime rearing ability, was 13% and 15% higher respectively than those descended from maternal ancestors of low rearing ability[66]. Selection for ability to rear at least one lamb also selects indirectly for fertility and fecundity. Twin-bearing ewes have a greater probability of rearing at least one lamb compared to single-bearing ewes and therefore escape culling for rearing failure.

The recommendation to select for twinning without reference to the ewe's ability to rear the additional progeny, is to be deprecated because it results in a cosmetic improvement in weaning percentage at the expense of a higher mortality among twins, compared to single lambs. Selection for ability to rear twins, improved both weaning percentage and lamb survival[18]. The reasons for the improvement were not specified, but it clearly reflects increased fitness of the ewe-lamb partnership.

Selection for rearing ability has been used by co-operative breeding schemes, commercial sheep farmers and a few progressive studs for the last 20 years[104].

A typical selection programme for improving lamb survival is summarised below:

1. Identify and eliminate obvious causes of perinatal mortality e.g. prenatal undernutrition, disease.
2. Identify and cull ewes which lose all lambs born, require obstetrical assistance, or any other intervention to

ensure the survival of their progeny. Identify the surviving progeny of these ewes at the time of the intervention and cull them at weaning. Identification of "lambed-and-lost" and "not-lambed" ewes is most accurately carried out at lamb-marking by the "wet-dry" technique. Selection for ability to rear a lamb to lamb-marking, is probably as effective as selecting for ability to wean a lamb as most lamb deaths between marking and weaning are due to extrinsic causes.

3. Cull barren ewes, after eliminating other causes of infertility, e.g. ram infertility, anoestrus. Ability to conceive and maintain pregnancy has a low heritability.
4. If possible, select for ability to rear twins. Selecting for ability to rear one lamb indirectly selects for twinning.
5. Avoid lambing husbandry practices which disrupt ewe-lamb bonding, e.g. high stocking densities, supplementary feeding during daytime, disturbance of recently-lambed or about-to-lamb ewes. Lambing flocks should be conditioned to the presence of trained shepherds moving quietly among them.
6. Ensure good nutrition of lambs, ewe hoggets, and maiden replacements to 2,5 years of age, when the pelvic centres of ossification fuse, to maximise pelvic size.
7. For selection to be successful, the efficient ewes must be joined to rams born of ewes with high rearing ability, otherwise the strategy is futile. An adequate supply of suitable rams demands that co-operative breeding schemes and studs include rearing ability in their selection programmes.
8. The programme must be backed by good nutrition, disease control and judicious husbandry including the provision of shelter and shade.

Culled ewes and their salvaged progeny should be sold preferably for slaughter. Alternatively they may be run as dry sheep for wool production or joined to black-faced mutton breed rams for easy identification of progeny. Unlike Australia and New Zealand where cull mutton is practically worthless, selection for rearing ability can be implemented in South Africa at minimal cost because of the high price of mutton. The programme may also be phased in over 4 to 5 years by implementing it in successive intakes of maiden replacements so that at the end of that period all lambed ewes, except maiden replacements, have always reared a lamb. Provided we are brave enough to implement the ruthless culling required, substantial improvement in lamb survival seems certain in the medium term. The programme is doomed unless rams selected for rearing ability are available. The stud industry, as custodians of the heritable production characteristics of the national flock, has a responsibility to include selection for rearing ability in their breeding programmes. Given their conservative attitudes, they are unlikely to do so without pressure from commercial breeders. Failure of the New Zealand studs to take up the challenge 20 years ago, resulted in the newly-established group-breeding schemes capturing 10% of the ram market in recent years - a result which has now forced the New Zealand stud industry to adopt similar selection procedures (B J McGuirk 1985 CSIRO Division of Animal Production, Prospect, personal communication). Reported results include: 10 years aggressive selection in a Romney co-operative ram breeding flock, halved lamb mortality to weaning (7% of lambs born) in a flock dropping over 80% of twins compared to the district mortality average (15%) in flocks with twinning rates up to 35%[104]; 95% survival to weaning after 15 years selection in an "easy-care" Romney flock, due in part to superior mothering[79], compared to 86% survival in a control flock with a comparable twinning rate[75]; an average of 95% survival to weaning in the Marshall Romney after 7 years natural selection on harsh hill country[17]; a 7% improvement in the survival of both singles and twin Merino lambs after 9 years selection compared to a control flock[18]; a 9% improvement in Merino lamb survival over controls in 6 years[37]. The relative contributions of culling in current generations and true genetic improvement to these results was not specified. All results were obtained in wholly pasture-fed flocks managed with minimum labour - so-called "easy-care" sheep.

B. Selection for specific components of lamb survival

Because the heritability of some specific components of lamb survival is higher than that for rearing ability, it has been suggested that selection for these traits would improve lamb survival more rapidly[32].

1. The size of the maternal pelvic conjugate diameter

At least 60% of perinatal mortality appears to be associated with birth stress[59 67]. Both elements of birth stress due to foeto-pelvic disproportion, namely birth mass and pelvic size, have a genetic basis. Effective selection for optimal birth mass would be difficult because of the wide variation in prenatal nutrition, the occurrence of twins, and the disadvantage and practical difficulties of measuring birth mass at lambing. The dimensions of the mature maternal pelvic conjugate diameter were highly correlated with lifetime rearing ability (lambs weaned/lambs born)[68], and the mean conjugate diameter of ewes with high rearing ability was larger than that of ewes with low rearing ability[29]. The genetic correlation between the size of the conjugate diameter of Merino ewes and lamb survival was 0,73, with the heritability of the dimension estimated at 0,30[19]. Thus, direct selection for the size of the mature maternal conjugate diameter, using radiography[69], appears to offer considerable scope for reducing not only parturient deaths, but also the birth-injured component of the SME complex. Little data are available on the pelvic dimensions of rams. The technique is expensive and there is a need for a cheaper technology. Attempts to exploit the high correlation between some external anatomical measurements and pelvic dimensions[49 103] and the development of a pelvimeter (Haughey, unpublished data) have been unsuccessful.

2. Selection for cold resistance in newborn

Neonatal resistance to hypothermia has a useful heritability, estimated between 0,27 and 0,44[119 121 134], with a value of 0,76 being reported in Australian Merinos (J Slee 1989 CSIRO Division of Animal Production, Prospect, New South Wales, personal communication). Although heritabilities of this magnitude offer considerable scope for improving lamb survival, the selection technique is onerous, involving measurement of the physiological response of individual lambs to cold in a progressively-cooled water-bath[124 134]. The use of rams born during, and surviving severe weather conditions may also be an option. As birth stress has a powerful depressant effect on neonatal thermogenesis[42], it is not yet clear whether the trait may be partly a reflection of ease of birth.

3. Selection for parent-offspring behaviour

This technique requires labour-intensive observations during lambing[14]. The rate of genetic progress cannot be predicted yet, as the heritability has not been estimated, but this may soon be known (G Alexander 1989 CSIRO Division of Animal Production, Prospect, New South Wales, personal communication).

Direct selection for cold resistance and parent-offspring behaviour are unlikely to find widespread application in the industry because of the costly labour-intensive selection techniques. Although it offers considerable scope for rapid genetic gain, selection for pelvic size is probably disqualified in most circumstances by its high cost. The availability of rams selected for these traits presents additional difficulties. Selection for rearing ability is the most practical technique for wide-

spread use for the reasons outlined earlier.

Nutritional aids

Provided it is cost-effective, competent ultrasonic pregnancy diagnosis of litter size facilitates more effective and less wasteful prenatal feeding of single and twin pregnancies. The risk of dystocia and birth stress is minimised in single pregnancies if the ewes are fed separately. Low-level prenatal protein supplementation of pasture-fed ewes, according to litter size, increased the birth mass and survival of single, twin and triplet lambs[82]. Lambing ewes are more likely to remain longer on the birth site in the presence of plentiful pasture. The high effective stocking densities and the inevitable stampede which accompany daytime supplementary feeding of lambing ewes increase mismothering. Lambing when plentiful pasture is available, is preferable. Where supplementary feeding cannot be avoided, Australian experience suggests that the associated problems can be minimised by feeding out at night. In the southern Cape, substantial cost-benefits resulted from winter or early spring lambing on plentiful pasture compared to autumn-lambed, supplementary-fed flocks due to increased conception, twinning, survival and growth rate of lambs, and lower feed costs[72] (I A Herbst 1989 Veterinarian, Caledon, personal communication).

Husbandry aids

Twin lambs, particularly, are prone to mismothering due to the difficulty of ewes keeping the sets of multiples together, maternal desertion, or lamb-stealing by ewes on the point of lambing. Perinatal mortality was correlated significantly with fecundity and stocking density at lambing[67], and mismothering increased disproportionately at stocking densities exceeding 18 lambing ewes/ha[14]. The prevalence of lamb-stealing was related to the number of ewes lambing at any one time[78]. Twin-bearing ewes, particularly, require low stocking densities at lambing (not more than 15 ewes/ha) to prevent mismothering. Nutritional management and husbandry which disrupt parent-offspring bonding are to be avoided. Conditioning lambing flocks to the presence of shepherds is accomplished conveniently during routine prenatal husbandry or feeding. If this is impossible, the lambing flock should experience minimal disturbance. "Drifting" unlambed ewes off the lambing camp, is successful provided the lambing and lambed ewes are not unduly disturbed. The choice of sheltered lambing camps reduces evaporative and convective heat loss from newborn lambs and therefore minimises losses from exposure. There is a lack of researched designs of lambing camps, including their orientation to adverse weather, the type and positioning of shelter, and the siting of fence lines and watering points to prevent frustration of parent-offspring behaviour. Despite the practical problems, shearing ewes within 3 to 4 weeks of lambing, improved twin survival, because shorn ewes sought the shelter provided[11]. Ewes with teat and udder abnormalities, easily identified at "wet-drying", should be culled. Obstetrical assistance, and the treatment of mismothered and hypothermic lambs with warmth, stomach-tubing, intraperitoneal dextrose and foster-mothering are traditional methods of improving lamb survival. Given a genetic basis for the major causes of perinatal lamb mortality, namely birth stress, aberrant parent-offspring behaviour and neonatal cold resistance, the retention of the affected ewes and their surviving progeny in the breeding flock is contraindicated. That practice can only ensure the continued accumulation of genetically-determined defects, leading inevitably to decreasing fitness of ewe-lamb partnerships in the evolutionary sense. Indeed, it is probable that the present unsatisfactory state of lamb survival has been compounded in part by centuries of lambing husbandry.

MINOR CAUSES OF PERINATAL MORTALITY

In general, less than 20% of perinatal mortality is due to lethal congenital malformations, infections (both congenital and acquired after birth), mineral deficiencies, predation and unknown causes[34 59 67]. Individual entities may cause sporadic heavy mortality in some flocks, seasons and districts. Because of their relative unimportance, a comprehensive review of specific entities will not be undertaken.

Lethal congenital malformations

Lethal congenital malformations occur usually at low prevalence in the ante-parturient, parturient and post-parturient time-of-death classes. They affect all body systems[36], with the highest frequency in the CNS. Multiple malformations are common. Hyperkeratinised plaques on the hooves, accessory digits and the horn buds are frequent. Pathogenesis is due mainly to environmental factors[36], including foetal viral infections[22 95 98], maternal ingestion of phytoteratogens[24], maternal hyperthermia during organogenesis[57], and less frequently, chromosomal anomalies[27]. The sporadic nature of outbreaks, low level of loss, and ignorance of causes often preclude the adoption of control measures. Avoiding vaccination for bluetongue, Rift Valley fever and Wesselsbron disease, and the grazing of teratogenic plants during pregnancy, will prevent losses due to those causes.

Congenital infections

Generally, infections occur widely at low prevalence. Nationally, they probably form a small component of total perinatal mortality[67]. Infections may be congenital or acquired after birth.

A variety of bacterial and viral agents cause ante-parturient, parturient or post-parturient death (Table 5). They are endemic to many flocks[70], but with the exception of sometimes spectacular abortion "storms", they rarely cause serious economic loss. Vertical transmission from ewe to foetus occurs during pregnancy, resulting in foetal death, abortion, or foetal growth retardation, because of placentitis, the direct effects on foetal well-being, or both. Pregnant ewes infected with *Coxiella burnetii,* the cause of the zoonosis, Q fever, pose a threat to the health of farm, laboratory and abattoir staff[80]. The ovine condition is usually inapparent and self-limiting, with localisation of the organisms mainly in the placenta and birth fluids[80]. Less frequently *Coxiella* congenital infection is characterised by placentitis, abortion and the birth of weak lambs[26 96 106]. Septicaemic infections of pregnant ewes, e.g. *Salmonella* spp, may cause secondary abortion showing non-specific foetal pathology.

Many congenital infections result in characteristic gross lesions of the placenta and/or foetus. Table 5 summarises the gross placenta and/or foetal pathology associated with specific infections. Submission of the correct specimens, as well as autopsy, are essential to efficient diagnosis. Diagnostic laboratories should be consulted as to the appropriate diagnostic material. The following are usually appropriate:

1. Placenta, including cotyledons - fresh and fixed in formo-saline.
2. Fresh foetuses, parturient time-of-death class, delivered rapidly to the laboratory in chilled insulated containers - otherwise
   (a) foetal lung and liver - fresh and fixed
   (b) foetal abomasum and contents -fresh
   (c) foetal heart blood, CSF, or effusions from serous cavities
   (d) foetal brain - fixed
   (e) serums from affected ewes

When levels of loss are low or sporadic abortion "storms" occur, no recommendations can be made, apart from observing routine hygiene. Abortion in sheep

seems to have a low repeatability, presumably because an effective immunity is acquired. Aborted ewes can be retained in the flock with impunity, comforting advice to an unfortunate sheep breeder (Haughey, unpublished data). Persistent economic loss due to infections with *Campylobacter fetus, Chlamydia* spp (Enzootic abortion), *Brucella ovis,* bluetongue, Wesselsbron disease and Rift Valley fever, can be controlled by vaccination. While the vaccination of ewes against *Coxiella burnetii* prevented placentitis and the birth of weak lambs, it did not prevent the shedding of organisms[26]. A vaccine against Akabane infection is being tested in Australia.

Infections acquired after birth

A wide variety of bacterial infections have been incriminated. Prevalence rises with intensive management systems e.g. penned lambing. Most are acquired at, or soon after birth although their pathological manifestations may extend beyond the perinatal period[67].

Common pathogens include[34 67 76]:

(a) *Clostridium septicum, Clostridium chauvoei* and *Clostridium novyi,* cause gangrene around the umbilicus and localised or generalised sero-fibrinous peritonitis.
(b) *Pasteurella haemolytica* and *Pasteurella multocida* cause pneumonia and localised or generalised sero-fibrinous peritonitis.
(c) Infection by *Staphylococcus aureus, Streptococcus* spp, *Corynebacterium* spp, *Fusobacterium necrophorum,* and other bacteria cause pyaemia with multiple purulent foci in the liver, kidneys, heart, muscles and joints.
(d) *Eschericia coli* causes syndromes characterised by enteritis, septicaemia or leptomeningitis.
(e) *Erysipelothrix insidiosa* and *Chlamydia* spp cause polysynovitis.

Diagnosis is confirmed by microbiological and histopathological examination of appropriate specimens. Vaccination of ewes in late pregnancy provides effective colostral immunity against infections with *Clostridial* spp. The similar use of *Pasturella haemolytica* vaccine is of equivocal efficacy[54]. Often losses do not warrant the cost of vaccination programmes, but they may be mandatory when penlambing is practised. Routine hygiene and the changing of bedding daily may help to minimise losses in the latter system.

Deficiencies of trace elements

Congenital swayback, congenital goitre and congenital white muscle disease associated with deficiency of copper, iodine and selenium, respectively, are usually endemic to certain soil types with sporadic outbreaks of heavy mortality[23 55 92]. Modern pasture production techniques have led to the emergence of trace element deficiencies in districts where they have not been recorded previously. As deficient and normal tissue levels of the various trace elements and the amount of supplement required for prevention of the syndromes vary between districts and countries, advice should be sought from the local authorities when deficiencies are newly-diagnosed.

Copper deficiency[48 55 125], either primary, or secondary to excess molybdenum and/or sulphate in the diet, is characterised by paralysis and other nervous signs, bone fragility, progressive emaciation any time from birth to 4 months of age and "steely wool" in adult sheep. The congenital form is associated with acute deficiency and affected lambs show nervous signs, including chorea, inco-ordination or paralysis due to extensive demyelination leading to cavitation of cerebal white matter. Histopathological examination of brain and spinal cord, and liver copper concentrations of $< 10 mg\ kg^{-1}$ dry matter, confirm the diagnosis. Levels between 10 to 90 mg kg^{-1} are definitely on the low side of normal (150-700 mg kg^{-1}). With acute deficiency, other syndromes of copper deficiency are likely to occur in all sheep and cattle grazing the same pastures. Sheep breeds vary in their susceptibility to copper deficiency[125].

Supplementation of the diet[55 125] with copper by pasture top-dressing, oral or parenteral administration, prevents copper deficiency. As the pathogenesis of copper deficiency is incompletely understood, the recommendations are guides only:

(1) Pasture topdressing, often in the form of copperised superphosphate, applied at recommended levels. Extravagant use may lead to copper toxicity.
(2) Oral administration in the form of slow-release proprietary preparations e.g. copper "needles", glass boluses, once annually to ewes at joining or early pregnancy.
(3) Parenteral administration of pro-

Table 5: **Congenital infections associated with perinatal mortality and abortion**

Agent	Gross Lesions
Bacteria	
Listeria monocytogenes[35]	placentitis, multiple focal abscesses, 1-2 mm, in
Yersinia pseudotuberculosis[70]	liver, occasionally in lungs and kidneys
Histophilus ovis[131]	placentitis
Brucella ovis[115]	placentitis, hyperkeratinised placques on horny hooves
Chlamydia spp[16]	placentitis
Campylobacter fetus var. fetus[114]	occasionally oedematous chorio-alantois, hepatomegaly, large circumscribed yellowish, necrotic liver lesions, 10-30mm, in 30 to 40% of cases
Coxiella burnetii[80 96]	usually no visible lesions, placentitis less frequently
Protozoa	
Toxoplasma gondii[39]	Yellow-white flecks, 1-2mm, in foetal cotyledons, leucoencephalomalacia
Viruses	
Akabane[98] Bluetongue, including vaccinal virus[95]	hydranencephaly, hydrocephalus, micrencephaly, scoliosis, kyphosis, arthrogryposis
Border disease[22] (Hairy Shaker disease)	hydranencephaly, hairy birth coats, some affected lambs are choreic
Rift Valley fever[30]	enlarged, yellow-brown to dark red, friable liver, with grey-white necrotic foci, 1-2mm, icterus occassionally
Wesselsbron disease[31]	infection during organogenesis - similar lesions to Akabane. Infection later in pregnancy - icterus, enlarged yellow to orange-brown liver

prietary preparations of copper, including copper edetate or glycinate, once annually at recommended dose rates. Copper glycinate sometimes causes severe local reactions (Haughey, unpublished data).

Congenital goitre[23 126] is characterised by grossly enlarged thyroids (>2g) in lambs[84]. The entity is usally endemic to soils which are variably deficient in iodine. Other factors must also be involved in the pathogenesis to explain the variation in severity and frequency of outbreaks. Feeding *Brassica* spp, heavily fertilised star grass and clovers containing goitrogens during late pregnancy, and the reduced amount of soil ingested in good seasons during pregnancy have so far been incriminated. A biochemical defect, inherited as a simple recessive, preventing the biosynthesis of thyroid hormone has been identified in Merinos[46 47]. Thyroid enlargement may be so great as to cause dystocia (>200g)[118].

Goitre due to simple iodine deficiency or the ingestion of goitrogens can be prevented by[23 44]:

(1) drenching ewes at monthly intervals during the third and fourth months of pregnancy with a solution containing 280 mg potassium iodide per dose.
(2) Providing salt licks containing 120 g potassium iodate per tonne throughout pregnancy.
(3) Oral administration to maiden ewes of a proprietary intra-ruminal device containing slow-release iodine with a claimed 3-year effective life[45].
(4) Intramuscular injection of preparations containing iodine in poppy seed oil, 2 months before lambing.

The extremely sporadic, even rare, occurrence of goitre often does not warrant the cost of preventive measures. The latter can be justified only in flocks experiencing regular outbreaks.

Congenital white muscle disease (WMD)[28 92 117] is manifested by subendocardial circumscribed, dirty-white plaques in the ventricles due to necrosis and calcification of the myocardium. Its detection requires routine opening of the ventricles during autopsy. Lambs die suddenly during or shortly after birth. The selenium status of flocks may also be ascertained by determining blood glutathione peroxidase levels in 15 to 20 young sheep. Other manifestations of selenium deficiency, including ewe infertility, delayed WMD affecting voluntary muscle, and unthriftiness of young sheep, often occur in affected flocks. The prevention of congenital WMD[92] usually has to be integrated with control of other selenium-responsive syndromes occurring in affected flocks. Selenium compounds have a relatively low therapeutic index, necessitating their prudent use. Congenital WMD may be prevented by: oral administration of 5 mg of Se as a solution of sodium selenite or sodium selenate to ewes one month before due date of lambing; oral administration of selenium "bullets" during pregnancy; parenteral administration of selenium. In some countries selenium salts have been incorporated in vaccines.

Experimentally, the application of selenium-fortified superphosphate or selenium prills to pasture has proved a safer but more expensive method of controlling selenium deficiency than sodium selenate or selenite administered orally[92].

Predation

The role of predation in perinatal mortality by carnivorous, omnivorous, and occasionally, avian species, is often overestimated because investigators fail to distinghuish between primary predation (the killing of an otherwise viable lamb), secondary predation (the killing of a lamb of low viability), and scavenging[83]. Australian and South African studies using these classifications, showed that primary predation caused low losses despite popular opinion[67 83 108 109], although sporadic catastrophes occurred in some seasons, districts, and even camps. Difficulties arise when prey are wholly consumed on site or removed from camps, as occurs with large predators, necessitating the use of indirect methods to estimate losses[100]. A substantial portion of the carcase should be skinned, as not only are external appearances misleading regarding the degree of multilation, but the site and nature of wounds may also indicate the species or genus of the predator or scavenger involved. The killing and feeding methods, and the inter-canine tooth-skin puncture distances of some Australian, South African and American sheep predators have been characterised[56 108 110 112 129]. The distinguishing pathological features of predation and scavenging are[83]:

Primary predation - carcases are characterised by lethal ante-mortem wounds showing severe haemorrhage and contusions, with no evidence of other pathological processes. When the prey has fed, there is evidence of active absorption of digested milk in mesenteric lacteals. Body fat is not catabolised.

Secondary predation - is characterised by severe ante-mortem mutilation in a carcase in which other pathological processes are evident e.g. marked fat catabolism, lethal malformation or infection.

Scavenging - is characterised by varying degrees of multilation and consumption of a carcase carried out after death, indicated by the absence of contusions and haemorrhage around the site of mutilation, and evidence of other pathological processes indicative of the cause of death.

PROSPECTS FOR IMPROVING LAMB SURVIVAL

The identification of heritable components conducive to improving fitness of the ewe-lamb partnership for survival, a more precise understanding of the prenatal handicaps imposed by undernutrition of twin-bearing ewes, and the ability to diagnose litter size early in pregnancy thereby allowing refinement of parental nutritional management, offer excellent prospects for improving lamb survival. Those prospects can be approached confidently as ruthless selection for rearing ability, along with selection for other production traits including improved fecundity, have achieved survival to weaning of 95% of lambs born in pasture-fed "easy-care" flocks in New Zealand. The conservatism of studs, in not adopting similar selection procedures to increase the availability of suitable sires, is likely to blight progress. The market forces imposed by the establishment of group-breeding schemes, eventually overcame that difficulty in New Zealand - albeit over a period of 20 years. The hard fact is, that we will not improve lamb survival beyond its present unsatisfactory level until we implement programmes aimed at minimising all the constraints imposed by genetics, nutrition, husbandry, disease and the weather.

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