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Farm Pets: The 10 Minute Consult Mini Series

Session One: Farm Ruminants

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Notes for Session 1 Farm Ruminants

Farm animal equipment

1. Rubber boots.
2. Waterproof trousers.
3. Waterproof parlour top or parturition gown.
4. Stiff boot brush.
5. A bottle of farm disinfectant.
6. A bucket
7. Stethoscope.
8. Thermometer.
9. Syringes 2ml – 60ml.
10. Needles 14 – 21 Gauge. Length 5/8ths – 2 inches.
11. Rope Halter.
12. A long rope at least 10 metres.
13. One pair of straight scissors.
14. One pair of artery forceps.
15. Thick suture material.
16. Adequate size needle for the suture material.
17. Long Seton needle.
18. Uterine tape.
19. Scalpel blades with handle which fits blades.
20. Arm length sleeves.
21. Flutter valve, which is compatible with Calcium solutions shown below.
22. Umbilical clip.
23. Hoof knife.
24. Aggers Pump

Extra Equipment

A Caesarean box containing

a. A sterile pack of instruments
b. A sterile scrubbing brush
c. A sterile tray cloth
d. Two packets of sterile large swabs
e. A sterile embryotomy knife or a disposable embryotomy knife (Kruuse Ltd)
f. Scalpel blades (Must fit scalpel blade holder)
g. Syringes and needles

h. Pieces of cotton wool in a bag for cleaning the skin.
i. Bottle of surgical spirit
j. Bottle of Hibiscrub
k. Bottle of Savlon
l. Oxytocin injection (Temperature controlled)
m. 3 X 100ml Local Anaesthetic (Must be licensed for food producing animals)
n. 100ml Penicillin/streptomycin
o. Dopram drops (Temperature controlled)
p. NSAIDs (Must be licensed for food producing animals)

Extra Equipment for calving cows.

1. Embryotome.
2. Embryotomy wire.
3. Three 12-inch long pieces of rigid plastic pipe. .
4. Two calving ropes of different colours.
5. One thin calving rope (lambing rope size).
6. Calving Jack.
7. Two very thick ropes for Calving Jack.
8. Calf feeder bag.
9. Resuscitation equipment.
10. GynStick
11. 5 Litre container with 0.5 of a litre containing 20 grams of sodium citrate.
12. A small trocar and cannular.
13. A large plastic bloodline.

Extra equipment for choke cases.

1 Trocar and Cannular (Red Devil Type).
2 Drinkwater Gag.
3 Probang. (Very contentious)
4 Bull dog clip.

Large animal medicines

1. Local anaesthetic licensed for use in farm animals. 5% Procaine hydrochloride. E.g. Adrenocaine.
2. 2% Xylazine. E.g. Sedaxylan CEVA Animal Health (This is licensed for both intravenous and intramuscular routes)
3. NSAIDs licensed for use in farm animals.
4. Injectable 2% Dexamethazone.
5. Various injectable antibiotics.
6. Oxytocin injection. Should be in the fridge in the summer
7. 400ml 20% Calcium.
8. 400ml 40% Calcium with added 5% Magnesium and Phosphorus.
9. 400ml 20% Calcium with added 5% Magnesium, Phosphorus and Glucose.
10. 400ml 25% Magnesium Sulphate.
11. 2 x 100ml Barbiturate euthanasia solution.
12. 2 x 50ml Somulose solution. 400mg/ml Quinalbarbitone, 25mg/ml Cinchocaine hydrochloride. Arnolds Veterinary Products.
13. One bottle Birp
14. Antibiotic aerosol.
15. Obstetric lubricant. (J Lube can also be useful.)
16. Red, Purple, Green and Grey topped vacutainer, with needles and holder.
17. Dopram Drops. 20mg/ml Doxapram Hydrochloride. Should be in the fridge in the summer
18. Oral electrolytes.
19. Foston. 20% Sodium salt of Toldimphos. Intervet UK Ltd.
20. Aggers Solutions

Parturition

Relative foetal over size Except for hobby farmers and very inexperienced under cowmen, the days of the easy calving are over. I have been calving cows for fithty years and still the decision on whether to carry out a caesarean section worries me. Obviously a live calf inside a healthy cow on a farm with good facilities will push me into a caesarean section if there is any doubt on getting a live calf naturally per vagina. This is particularly so if there is any Belgium Blue blood in the parents. However with bad facilities or a dead calf the pendulum will swing the opposite direction, as cow survival rates will decrease. Practitioners should be aware of the problems caused to calves by Schallemburg virus which may necessitate an embryotomy or a caesarean section.

The very worst scenario is for an embryotomy to be unsuccessful and a caesarean is undertaken as a last resort.

Various formulae have been put forward to guide veterinary surgeons on the decision as to whether to perform a caesarean or not. I will often carry out a trial of labour by getting the calf into the birth canal with moderate traction (e.g. one man pulling). If there is room to get a hand between the head and the pelvis, I will carry on with a normal delivery. Often it is soft tissue obstructing the calf. I will then perform an episiotomy, after an epidural anaesthetic.

However if there is any doubt in my mind I will consult with the owner and carry out a caesarean section.

Caesarean section

The welfare of the cow and calf should always be paramount when deciding if a caesarean section is required. If welfare is going to be compromised by lack of facilities and personnel then euthanasia should be considered. I feel it is quite acceptable to euthanase the cow and retrieve a live calf. What is not acceptable is to attempt a caesarean only to let the cow die a slow death from peritonitis caused by unsuccessful suturing of the uterus.

There is good evidence that cow survival rates drop if there are not two experienced operators available to carry out the caesarean. I am not saying they both have to be veterinary surgeons, although that is the ideal. However the main surgeon needs to be experienced and the assistant should have helped with caesareans before, e.g. a veterinary nurse, a final year student, an experienced farmer.

Uterine torsion This occurs at parturition (unlike in the mare), except in exceedingly rare circumstances (when the torsion is in the neck of the uterus like in the mare and on those occasions the cow will show signs of colic and not be parturient). The history will be that of a calving cow “not getting on with it”. Diagnosis is straightforward on vaginal examination as the condition is wrongly named. It is in fact a torsion of the vagina. I have never managed to rotate the calf per vagina with the cow standing. However it has been reported. In my hands, or rather with the help of three people, rolling the cow is very efficient. If she is standing I cast her with Roeffs method and then roll her IN THE DIRECTION OF THE TWIST.

Usually when the torsion is resolved the cervix will only be partly dilated. Many textbooks advise waiting to allow cervical dilation to occur. In my experience (Duncanson 1984) and others (Pearson 1971) this DOES NOT OCCUR. You are left with a ring-womb like condition. Therefore I advise dilating the cervix with slow traction without any delay. Caesarean section should in my opinion be the very last resort in uterine torsion cases. If the torsion is not that severe and the surgeon is able to put on leg ropes, then a GynStick can be used (See Utube video by Vetsonic)

The hung calf

Often a calf, which has stuck at the hips, can be born by slight rotation with renewed traction. However prolonged severe traction (equivalent to three men) will result in damage to either the sciatic or the obturator nerve and should be avoided. Caesarean section is obviously not an option. Thus foetotomy is the only answer. Sadly if the calf is not dead, euthanasia of the calf should be carried out. However although I have performed over 200 such foetotomies I never have had a live calf, which I have been unable to draw with rotation and lubrication. Foetotomy is not a real problem provided certain tips are followed.

1. Place the loop of wire round the trunk as far CAUDALLY as possible. This is helped by some traction on the calf and pushing the embryotome into the cow's pelvis. The reason for this is that the next cut will be easier.
2. Cut through the trunk with steady long sawing strokes. The cranial part of the calf up to the lower lumbar area can be removed.
3. Having replaced the caudal end of the calf into the cow's uterus, place a heavy introducer or a closed bullring attached to a thin calving rope over the dorsal aspect of the calf and drop it between the calf's legs. Reach in ventrally and grasp the bullring.
4. Tie the embryotomy wire SECURELY to the calving rope and pull it into the cow and out again. Before starting the second cut MAKE SURE the wire is exactly in the middle of the calf's pelvis. The danger is that if it is to the side, the second cut will only cut off one hind leg and the calf's pelvis will be as wide as ever.
5. The two parts of the hindquarters are now easily removed separately.

Hydrops uteri Two forms of the condition are recognised, hydrops amnion when excessive fluid is present within the amniotic sac and hydrops allantois when the allantoic sac is involved. At term in the normal cow approximately 20 litres of foetal fluids are present in the uterus, three quarters is in the allantois. In hydrops uteri over ten times that may be present. If the cow is in good condition, she can be left to come to calve normally. However if she is in poor condition parturition should be induced with a prostaglandin injection. Uterine drainage has been advocated prior to parturition to lessen the shock of such a large amount of fluid being lost at one time. However I feel the danger of causing a uterine infection is too great.

Prolapsed Uterus

Old clinicians will give you advice on the use of large quantities of sugar to reduce the size of the organ and meat skewers to hold it back in place. However there are more modern remedies which you may find more useful.

There are two causes of this condition. The primary cause in high producing dairy cows is hypocalcaemia and the primary cause in beef cows is relative foetal oversize causing trauma at parturition. If there is any danger of the cow dying of hypocalcaemia then treat this condition first. Cows may die from internal haemorrage after rupture of one of the very large uterine arteries. I advise you to sedate any fractious cows with Xylazine. I feel an epidural anaesthetic is vital.

If the cow is standing I prefer to have two helpers holding the uterus in a clean parturition gown, which I keep in the car for the purpose. If she is recumbent I still require two helpers to arrange the back legs to stick out straight behind her. When this has been accomplished, one of these helpers sits astride the cow facing backwards holding the tail up like a flag.

I clean the uterus as best I can, leaving any attached membranes in situ. I then work the uterus back into the cow using a variety of techniques making sure that it is not twisted, The smooth dorsal endometrium having no cotyledons is a good guide. Some practitioners try to put to put the smaller inverted horn of the uterus in first; others push in the body of the uterus in from the sides. One thing is for certain initially you imagine that it will never go back. However I have always eventually been successful except for one poor cow, which had been left over a weekend in Kenya.

It is very important to insure that the uterus is totally back in the correct position. Rarely is the human arm quite long enough so a bottle can be useful. There is a special instrument (like a rubber boxing glove on a long rod) made by Kruuse Ltd, if you are feeling wealthy. A large dose of oxytocin should be given intravenously immediately after replacement.

In theory there is no need to stitch the vulva but I feel it is a good insurance policy. I use a single “Buhner suture” of uterine tape. Along Seton needle is inserted into the skin at the ventral end of the vulva. It is pushed carefully in a dorsal direction subcutaneously and slightly laterally to emerge ventral to the anus.

The tape is treaded and withdrawn. This repeated for the other side of the vulva. The two ends are tied tightly so only two fingers can be inserted into the vulva. The owner is advised to remove the suture in 48 hours.

I give the cow 50 ius of Oxytocin and a dose of a suitable NSAID intravenously. I also give antibiotics and advise the owner to repeat them for to further days. I do not routinely revisit but instruct the owner to report if the cow is off her food. Some authorities claim that this condition greatly reduces subsequent fertility and in the unlikely event of a subsequent pregnancy the condition will reoccur. This has not been my experience.

Prolapsed Cervix

I am well aware that except in extremely rare circumstances this is a pre-parturient non-emergent condition. However it seems logical to deal with this condition at this stage.

The cow is best retained in a crush. I give an epidural but rarely do I find sedation necessary. The trick is to place the “Buhner suture” before replacement. Then it can be tightened immediately after replacement. I always tie the suture in a bow so that if parturition is suspected the suture can be left in place while an examination is carried out. If the cow is not parturient it can be retied without replacement of the suture.

Normally antibiotics and NSAIDs are not required. This condition will ALWAYS reoccur so further pregnancies are not recommended.

Post-partum arterial haemorrhage

This is usually the uterine artery and massive blood loss can be seen lying behind the cow. The pulsating vessel can be felt per vagina, normally only a forearm length from the vulva. My experience with trying to ligature this vessel is bad. My standard procedure therefore is to grasp the vessel with a pair of artery forceps and leave them in the cow. I return to the cow in 48 hours to retrieve them. If they fall off I remember to charge the client! Recently I managed to get a plastic disposable umbilical clamp on to the artery. This is obviously preferable.

Sadly haemorrhage can occur into the abdomen. The cow will appear to die before the cowman’s eyes.

Treatment of post-partum haemorrhage is very important. Antibiotic cover must be continued for a minimum of 10 days to prevent the danger of secondary haemorrhage, which in my experience is always fatal.

The cows with massive haemorrhage are obviously in shock. Normal or hypertonic saline is not worthwhile. Whole blood is required. This is not easy. There is no danger of miss matching with blood, unless repeated transfusions are carried out. Therefore the donor needs to be selected for temperament and lack of pregnancy. If there are decent crush facilities available you may not need to sedate the donor as local infiltration over the mid jugular region will be sufficient. Try to prepare the site aseptically but in my very limited experience the collection procedure is very crude. Cut through the skin with the vein NOT RAISED. Push a small trocar and cannular through into the raised jugular. (The trocar used for the toggling procedure for anchoring the abomasum is ideal). The 5 litres of blood will require 5 minutes to drain out. It should be collected in a container, containing half a litre of sterile water containing 20 grams of sodium citrate (Do not use heparin as any excess will delay clotting time for the recipient and make the situation worse). 5 litre plastic containers are available with a top to allow a large bloodline to be inserted. The blood should be infused into the recipient over a 20 minute period with a 14 gauge catheter

Notes on the recumbent cow

My definition of a recumbent cow is one, which can not get to its feet. The causes are many. I have tried to list the common causes below. However you are unlikely to make a diagnosis in every case, so often the treatment is symptomatic.

On the other hand “downer cow” is also a cow which can not get up but by definition the cause of the recumbency is unknown. A “downer cow” appears bright and has a normal temperature. She eats and drinks. Once again by definition her calcium, phosphorous and Magnesium levels are normal. She does not appear to be hurt or ill. The main problem for the clinician is the prognosis. If she is never going to get up, then the sooner she is destroyed the better. Equally the owner will be hoping for miracles. If she is on concrete it is vital that she is moved outside on to firm ground or if the weather is bad, on to a well-strawed deep bed of muck.

Provided the cow is being properly nursed i.e. regularly turned on to her other flank and sat up, if she is unable to get into sternal recumbency, then I feel it is quite justifiable to give her some time.

Some years ago the BCVA carried out a survey of “downer cows”. They recorded a wide range of parameters to see whether there was any statistical evidence for any of them to be used as prognostic indicators. Interestingly the most useful was the judgement of an experienced cattle practitioner! The only other worthwhile indicator was CK level. If this continued to rise this was a very poor prognostic sign.

Therefore my advice when faced with a “downer cow” is to take a blood sample in a red topped tube on day one. Store the serum. If the cow is not up in two to three days then take a second sample. If CK levels are higher in the second sample, I feel euthanasia should be recommended.

Hypocalcaemia

I feel this is not the place for a complicated scientific discussion. Every recumbent cow should be suspected of having low calcium levels. The case maybe a true “milk fever” around parturition or in Jersey cows it may be brought on by oestrus. Equally too many sugarbeet, particularly the tops will cause hypocalcaemia.

One 400ml bottle of 40% Calcium into the vein should be sufficient except in a very large cow. There is no evidence to confirm that the solution is better to be given at blood heat slowly. However I think these are wise precautions.

If the cow is staggering but not recumbent, I strongly advise you not to try to give large volumes of fluid intravenously. Obviously you do not want the cow in a crush. Therefore trying to inject a staggering cow is going to be difficult and dangerous. The worst scenario is that you will get some fluid in the vein but then some perivascular. This will make the cow move her head violently and you are likely to get hurt. You will have to admit failure and put the rest subcutaneously. When the cow falls two days later and fractures her femur you will get the blame! It is much more prudent to say at the beginning “As she is not down we will give her a full dose of calcium subcutaneously, which will give her the same blood levels in half an hour as an intravenous dose.

Hypomagnesaemia

This condition may have the highest mortality of all conditions in beef suckler cows. Mercifully it is rare in dairy cows. Magnesium levels in most sucklers are on a knife-edge. Any upset to the absorption of sufficient magnesium by the small intestine will bring on the condition. The laxative effect of lush grass is the normal cause. However 24 hours in oestrus or weaning of a calf is sufficient to trigger the condition.

I always advise at the very onset that the clinician warns the owner that the cow can die AT ANY MOMMENT and that any treatment will be hazardous. I also stress that although the level of magnesium in the blood will be raised by the treatment, irreparable brain damage may already have occurred. Historically my first task with a cow having convulsions, which are almost pathognomic for the condition, was to inject 10mls of a small animal euthanasia barbiturate solution intravenously to calm her so that treatment was not so hazardous for the cow or the handlers. However with the current legislation on veterinary medicines, I do not advise this. So my first treatment is to try to inject a 400ml bottle of 25% Magnesium sulphate (normally has a black top) **subcutaneously.** Rub this well in and then try to inject a 400ml bottle of 20% Calcium borogluconate with 5% Magnesium Hypophosphite (normally has a blue top).

I think it is worth remembering that it is unwise to use calcium bottles for storing other medicines. In the dark it would be unfortunate if a cow was injected with anything harmful.

Nursing is important in hypomagnesaemia cases. Trying to maintain the cow in sternal recumbency is difficult but worthwhile.

Sadly many cases are found dead. Aqueous fluid sucked into a red topped vacutainer from the eye gives a good estimation of the magnesium status of the cow at death.

Hypophosphataemia

I suspect I over diagnose this condition. Usually farmers have already given a calcium solution with added phosphorous before we visit a recumbent cow. Therefore blood levels are not very helpful. I have always been told that cows lacking in phosphorous show pain in the loin area and may even show haemoglobinuria. If I am in any doubt I give 25ml of “Foston” daily for two days. However I suspect most of these so called “loin drop” cases are really traumatic injuries.

Traumatic injury

I find recumbency cases, which are of traumatic origin very distressing. Not only is the cow in pain but also moving her to a better situation is going to be painful. Most of the problems are through bad management and could have been avoided,

A thorough examination including a rectal is very important. NSAIDs are helpful but often the kindest action is euthanasia. The only major condition, which is a surgical option, is a dislocation of the hip. If this injury is caught early enough a replacement is possible with a favourable prognosis. Sadly this has not been my experience. A South African colleague has had better results. This is his procedure.

5 strong people as well as the surgeon are required, together with 2 lengths of 4metre rope, a halter and a surgical stitch up kit. For the surgery you require a clean dry draught-free area which has a firmly-fixed pole to which the cow can be tied. Inject antibiotics and NSAIDs before the operation. Sedate cow with xylazine 0.1mg/kg i/v) then clip and scrub from spine to stifle, hip bone to pin bone. Induce general anaesthetic with ketamine 2mg/kg i/v. Tie a rope to bottom of good leg - pull on this so that she falls with the affected leg uppermost. You also want her to fall with the fixed pole at her back - so you can tie another rope from the pole, between the upper-most leg and the udder, then back to the pole. This provides a sling against which to apply traction to the dislocated leg.
Make an incision 30cm long from the major trochanter, distally along the cranial edge of the femur. This line is blocked with local anaesthetic after the GA. Cut through the fascia lata, through the gluteobiceps muscle and blunt-dissect down to get a hand around the cranial surface of the proximal femur, and then also around the caudal edge of the proximal femur. One has dissected far enough when one can feel the head of the femur cranially, and the acetabulum from caudally. Apply traction on distal limb, exerting steady increasing pressure in a distal direction, as well as rotating the leg internally by putting weight on the stifle joint (downward pressure with surgeon's knee). With a hand in the incision wound, the surgeon can feel how close the head of the femur is to the acetabulum, and direct more traction accordingly. Once the leg has been pulled enough, it will fall into joint quite easily. The most difficult part is applying sufficient traction. As long as the dorsal rim of the acetabulum hasn't been fractured, it should stay reduced. If it doesn't stay in place, it either wasn't properly reduced initially, or the acetabulum has been fractured, or there is a blood clot/muscle piece in between femur head and acetabulum. Feel for this and remove it before attempting reduction. Closure of incision - apply surgical principles: dead space, tissue apposition etc. Hobble back legs of cow before she stands up. Leave hobbles on for 2 days if she tolerates it. Assist her to stand once she is awake enough from the anaesthetic. Don't allow her on concrete for 2 days after the procedure, keep her close to dairy for 10 days. Up to 5 people may be needed to apply sufficient traction.

Toxic Mastitis This may pose a diagnostic challenge if it is peracute and recumbency occurs before there are changes in the udder. The signs of dehydration as a result of the diarrhoea and shock will help differentiate this condition from hypocalcaemia. Body temperature in my experience is rarely useful as a diagnostic indicator. It is usually sub normal in both conditions. The pulse is always raised. E coli is the normal organism involved but Klebsiella will give similar symptoms. The cow has only a one in three chance of surviving. A blood sample taken at first examination if tested for Creatinine, Urea and a PCV can be a useful prognostic indicator. Treatment regimes are numerous but Evidence Based Medicine (EBM) is lacking. I now no longer give fluids i/v but favour fluids per os. I use antibiotic parentrally as well as in the udder after stripping out. However I think they are of doubtful value. Large doses of NSAIDs are in my opinion the key to success. However in advanced cases humane destruction is the kindest for the cow and the farmer’s pocket. The old adage is very true “They can do worse than die”.

Toxic Metritis

There are no problems with diagnosis of this condition. The cow will have a raised temperature and a uterine discharge. Parentral penicillin is the antibiotic of choice with prostaglandins as an aid in cases, which have been calved for some days.

Salmonellosis The organism is S. dublin and classically occurs at calving. The signs are a very high temperature with acute diarrhoea. Sometimes the cows are found dead. Diagnosis is straightforward with a faeces sample for culture. (It is worthwhile advising the laboratory that you ONLY require salmonella culture or you will be faced with a large bill for tests, which were not relevant). There is a vaccine available Bovivac S (Intervet UK Ltd).

Acute Acidosis

This condition also causes acute diarrhoea. However the temperature will be sub normal and the cow will be very dehydrated. Treatment must be to correct this with suitable fluids intravenously and by mouth. NSAIDs and vitamin B injections are also helpful.

Abdominal Catastrophy

1. Uterine rupture.
2. Right sided displacement of the abomasum (RDA).
3. Caecal torsion
4. Small intestinal torsion
5. Peritonitis
6. Colic

Acute Babesiosis This will only be seen when adult cattle are moved from an area free of the disease to an endemic area. Animals show the classical “pipe-stem diarrhoea” with very loud tachycardia. Haemoglobinuria may not be apparent initially. The temperature will be raised.

The cow with respiratory problems

Acute pneumonia The list of differentials is not long, however diagnosis maybe a challenge, if the animal is outside. Parasitic bronchitis, Fog fever and Atopic rhinitis are possibilities. If indoors IBR must be top of the list which then only contains some very rare conditions. I used to think haemoptysis was pathognomic for thrombosis of the caudal vena cava until I saw a case which had been horned in the chest. A ruptured pulmonary or mediastinal abscess, is a possibility. Endocarditis, which my experience often manifests itself at parturition, may mislead you. However the corded jugular if the left hand side of the heart is affected will help you with a diagnosis. I haven’t seen Contagious Bovine Pleural Pneumonia (CBPP) for nearly forty years, when I worked in Kenya, but I think you can relax in the UK.

Fog Fever

There are many theories on the aetiology of this condition of interstitial pneumonia. The theory with the most credence is that of a toxicosis from ingestion of large quantities of L-tryptophan in lush autumn grass. The disease occurs only in adults. The main sign is respiratory distress. The temperature is normal. It is a disease of beef suckler cows, which tend to be quieter than normal. Animals maybe found dead or die following exercise. Care should be used in rounding up animals for treatment, which is far from evidenced based. Apart from more normal therapies e.g. antibiotics, NSAIDs, steroids etc, atropine and methylene blue have been advocated. In reality my advice is to stick to antibiotics and NSAIDs.

Lung worm

The oral vaccination against this condition using irradiated larvae is sadly being neglected with the use of pour-on ivomectin type anthelmintics. The animals at risk are two year old breeding heifers and of course adult cows. Lung worm causes devastating effects in naive adults. Coughing is violent and very marked. Mortality may be as high as 10%. Treatment with anthelmintics, antibiotics and NSAIDs is often not effective. Many survivors are left with irreversible lung damage.

Bovine Viral Diarrhoea (BVD)

Normally this condition in adult cattle is associated with reproduction problems. However there is a virus BVD type 2, which can cause acutely ill cows and is usually fatal. The cows feel ice cold to the touch and have a low rectal temperature. They have profuse watery diarrhoea and death follows in a few hours. Old African vets will see the signs as reminiscent of Rinderpest. However the muscle and eye lesions are more marked in Rinderpest. The Zebra marking are horizontal in the rectum in Rinderpest but longitudinal in BVD. There is no treatment in either disease but there excellent vaccines for both conditions.

Bovine Malignant Catarrh

This viral disease is rare in the UK. Sheep appear to be the secondary host. The animal will have a high fever and acute ocular discharge with corneal opacity and erosions. Mortality is high. Treatment is hopeless.

Acute Toxicity

Plant Poisons

1. Acorn poisoning

This poisoning is usually seen in early autumn after a wind. Cattle have to consume quite a large quantity. The main sign is constipation. Liquid paraffin should be used as a treatment. Rolling the pasture is useful as a preventative measure.

2. Bracken poisoning.

Not actually an acute problem as it is a cumulative poison. However the signs of haematuria and bright blindness may be perceived by the owner as acute.

3. Mycotoxicosis.

The toxins normally come from stale food. Acute cases are rare with diarrhoea the main sign. Normally cases are sub-acute.

4. Ragwort

This poisoning usually occurs from dried ragwort in hay or silage. The poisoning can cause acute illness from liver toxicity. However the condition is normally of a chronic nature. There is no antidote.

5. Water drop-wort.

The roots are called ‘dead man’s fingers’. They are usually eaten after ditches or ponds have been dredged. Cattle can be found dead or with dilated pupils and convulsing. There is no treatment.

6. Yew

Animals are found dead. Normally cattle will not eat the growing plant only when it is cut and thrown into a place where cattle have access. There is no antidote. Coffee, which is the traditional remedy in my hand’s is useless.

Chemical Poisons

1. Ammonia

This is caused by large amounts of urea in the diet from broken down feeding blocks. It is often called the ‘Bonkers syndrome’. Cattle stampede, froth at the mouth and have convulsions. Treatment is with cider vinegar.

2. Arsenic

The signs are of acute abdominal pain, diarrhoea and cyanosis. There are specific treatments recorded e.g. British antilewisite and sodium thiosulphate. However the actual recorded success is limited and the advice must be to use pain relief with NSAIDs and intestinal absorbents

3. Lead

This condition is now very rare with modern lead free paints. However calves will show nervous signs from licking old gates. The specific antidote Calcium edentate is very effective. There used to be a chronic form on animals graizing near old lead mines.

4. Nitrates and nitrites

The normal cause is ingestion of fertiliser. This causes the formation of methaemogloblin, which is a vasodilator and causes a sudden usually fatal lowering of the blood pressure. Methaemoglobinuria may be seen. Treatment is specific. 1% methylene blue solution at 9mg/kg should be given by slow intravenous injection.

5. Selenium

The signs are profound depression, salivation, blindness and severe dyspnoea. There is no specific treatment only symptomatic remedies

Choke

Normally diagnosis will not be a problem and the farmer will have assessed that his cow is choked, because of the increased salivation and signs of bloat. I always ask if he has tried to move the obstruction, which is normally a potato or piece of another root feeding stuff. If he has my golden rule is to say, “I am sure I will do no better”. I then proceed to place a rumen cannular. The reason I do not try to remove the obstruction is that if the farmer has perforated the oesophagus, I do not want to be blamed. Sadly in such an occurrence the animal will develop a fatal mediastinitis and will need to be euthanased. Remember, however blown the animal is to place the cannular on the LEFT-HAND paralumbar fossa. If in doubt always place it lower than the middle of the equilateral triangle to make allowances for the bloat. Unless the animal is flat out and on the point of death, I trim a 2-inch diameter circle of hair and inject 5 ml of local anaesthetic under the skin and into the muscle. After five minutes I cut a 1-inch incision with a scalpel blade through the skin. Then I stab the muscle layers with the trocar and cannular (Red Devil Type) and twist in a clock wise direction until the flange of the cannular is flush with the skin. It is important not to remove the trocar prematurely releasing the gas or the rumen wall will fall off the end of the cannular. Having removed the trocar I give it to the farmer so that he can use it to clean out the cannular should it become blocked. I am then confident that the obstruction will soften and be swallowed within 48 hours. Care should be taken not to remove the cannular until one is 100% certain there is no obstruction remaining. If there is concern that the animal is becoming dehydrated, then oral electrolytes can be given via the cannular. Antibiotic cover and NSAIDs should be given appropriately.

If the farmer has not tried to remove the obstruction, I am left three options;

- Place a cannular as described above.
- Pass a probang and try to dislodge the obstruction. There is an inherent danger of oesophageal perforation as described above. However with careful firm pressure with a probang success if a potato is the culprit is 80% achievable.
- If the obstruction can be felt in the neck, in adults it is possible to remove it per Os. The animal is given Xylazine at 20mg per 100 Kg Bwt intramuscularly and restrained in a crush. A running noose is applied to the neck caudal to the obstruction and kept tight by an assistant. The head is restrained by a bull dog clip not a halter. A Drinkwater gag is placed in between the cheek teeth and held in position by the tongue with one hand. The other hand is then pushed into the mouth down into the oesophagus and the obstruction grasped by the nails (Sadly these invariably get damaged) and withdrawn to the outside. It is vital that the obstruction is not released into the pharynx or it will be reswallowed.

There are a few differentials, which you should be aware of;

- Frothy bloat. This is normally caused by over eating of clover.
- Bloat can be seen as the end stage of chronic bracken poisoning. The actual cause of the bloat is a tumour in the oesophagus.

- Equally actinobacillosis of the oesophagus may cause bloat.
- An abscess in the medastinum
- Tetanus
- Rumen acidosis
- Traumatic reticulitis
- Fore stomach obstruction

In all these cases massive salivation is not seen and a stomach tube or a probang can be passed into the rumen.

The acutely ill sheep

The pregnant ewe

Hypocalcaemia

This is normally seen as a flock problem following a stressful incident like Clostridial vaccination (This is best carried out 6 weeks prior to lambing).[It is prudent to vaccinate rams and teasers at the same time]. Several ewes will be found recumbent and dull. Blood sampling before treatment is useful. However treatment with Calcium borogluconate will give a rapid response negating the need for incurring the expense of laboratory testing, if the diagnosis is correct. Cases after parturition are rare.

Hypomagnesaemia

This condition maybe seen in all adult sheep regardless of Pregnancy/lactation State. It will also occur in rams. Affected animals will be recumbent and show nervous signs. Prompt treatment with a mixture containing Calcium borogluconate and Magnesium sulphate is vital. If nervous signs are allowed to persist the brain will be damaged irreparably. Then the nervous signs will continue regardless of the magnesium status, resulting in euthanasia. New lush grass causing increased bowel movement is the normal cause. Prevention with some dry food over the risk period is worthwhile. Molassed blocks containing extra magnesium are rarely useful, as a third of the flock will not touch them.

Animals may be found dead. Testing the vitreous humour for Magnesium levels is a useful diagnostic tool.

Pregnancy toxaemia

This condition is commonly called ‘twin lamb disease’ but in fact can occur in all pregnant ewes if the feeding is wrong. Ewes in the last six weeks of pregnancy need a rising plain of nutrition. This can be provided by spring grass in very late lambing flocks but normally hard feed has to be provided as well as ad lib forage. A good rule of thumb is to give quarter of a pound of hard food a day per ewe six weeks before lambing. This should be increased by quarter of a pound a day every week so that at lambing the ewes are receiving one and three quarter pounds per day.

It is vital that adequate trough space is provided so every ewe gets a chance for its share of the food. Hard ewe rolls, which can be spread out on the pasture should be avoided. The ewes, which are less active or have poor teeth then, miss out.

Treatment of pregnancy toxaemia is often not successful and is controversial. I treat the ewes with oral concentrated electrolytes three times daily. I also inject them with a solution containing Calcium, Magnesium, Phosphorous and Glucose (usually labelled Number 6). Injections of NSAIDs maybe useful. I do not abort the ewe by giving her either Dexamethasone or Prostaglandins. However I know many clinicians, who do. My experience is that you end up loosing the ewe and also the lambs, which are born premature.

Vaginal Prolapse

The normal gestation length of the ewe is put at 148 days. Except for certain horned breeds e.g. Dorset Horn, ewes are stimulated to start cycling as the day length gets shorter. Therefore the majority of lambings will be in the spring. Management of a vaginal prolapse, which occurs before parturition can be accomplished in many ways. Epidural using a 1 inch 20G needle in the first intercoccygeal space is always helpful for replacement. 2ml of 2% Lignocaine and 0.25 ml of 2% Xylazine is ideal for most ewes. It is easiest to have the ewe standing. I favour a single, so-called ‘Buhner’ suture, of uterine tape. A long Seton needle is inserted into the skin at the ventral end of the vulva. It is pushed carefully in a dorsal direction subcutaneously and slightly laterally to emerge ventral to the anus.

The tape is treaded and withdrawn. This repeated for the other side of the vulva. The two ends are tied tightly so only two fingers can be inserted into the vulva.

I tie this in a bow so that if parturition is suspected it can be untied but not cut. If the ewe is not actually parturient it can be retied. There are vaginal spoons and trusses available but in my experience these lead to complications if parturition is delayed. This condition invariably is worse in subsequent years, so culling at the end of the season is to be recommended. There are various spoons and trusses available.

This condition can seem to occur as an outbreak. The reason often given is the feeding of root crops. However I cannot find any substantiating evidence.

<u>The parturient ewe</u>

The problems associated with malpresentations are covered by other authors. However the ewe suffers from a condition not seen in other farm species called ‘ring womb’. In this condition the cervix not only fails to dilate but also cannot be dilated. If the lambs are thought to be alive, then caesarean section is indicated.

My approach is to have the ewe in lateral recumbency with her left flank uppermost. After clipping off a large area of the left flank, I give approximately 40 ml of local anaesthetic in the shape of an inverted L (The procedure is the same in the goat but the total amount of local anaesthetic should be 20ml in pygmy goats. I dilute the local with sterile water). This is followed by an injection of antibiotics and NSAIDs.

I also give tetanus antitoxin if the ewe is not vaccinated. After thoroughly cleansing the area I make a seven-inch incision through the skin, muscle layers and peritoneum in a dorsal ventral direction starting three inches below the lumbar transverse processes. I exteriorise the uterus and make a six-inch incision on the greater curvature of the organ. I remove **ALL** the lambs and close the uterus with a single uninterrupted layer of Lembert sutures. I then close the peritoneum and the muscle layers in two rows of uninterrupted vertical mattress sutures. For all these closures I use number 5 Dexon on a non-cutting needle. I finally close the skin with interrupted vertical mattress sutures using monofilament nylon on a cutting needle. I finish by giving the ewe 20 international units of Oxytocin intramuscularly.

If the lambs are dead then humane destruction should be carried out without delay. Normally in a commercial flock this is carried out with either a humane killer or a suitable firearm. However increasingly sheep are kept as pets and this manner of euthanasia is unacceptable to the owners. I therefore advise in this situation that the sheep be given a very large dose of Xylazine (e.g. 2ml of a 2% solution) intramuscularly. Normally they become laterally recumbent in five minutes. Euthanasia can then be accomplished with 20ml of triple strength barbiturate intravenously into the jugular. My reasons for this approach are:

- I find it difficult to give large volumes of barbiturate intravenously to standing sheep.
- I am reluctant to keep part bottles of Somulose.

Uterine prolapse is much easier to reduce than in the cow as the sheep can be lifted by her hind legs, which allows gravity to assist. As with vaginal prolapse I favour an epidural and a 'Buhner suture'. In theory if the uterus is fully replaced and 20 international units of Oxytocin are given the prolapse should not reoccur. However I am off a nervous disposition! I always stitch the vulva. I advise antibiotic cover and removal of the suture in 48 hours. This condition does not always reoccur at subsequent lambings so with much loved pet sheep I do not insist on not breeding again, unlike animals, which have had a vaginal prolapse.

<u>The lactating ewe</u>

Metritis is the likely sequel with uterine prolapse or any another per vaginal interference. I still feel that Penicillin/ Streptomycin is the antibiotic of choice by intramuscular injection, supported by NSAIDs and Oxytocin. I feel intrauterine pessaries, as advised in older manuals are a thing of the past.

Mastitis in the ewe can be peracute and gangrenous caused by *Staphylococcus aureus.* However more commonly it is of a more chronic nature caused by *E coli* or *Mannheimia haemolytica.* The main danger then is malnutrition of the lambs. It is important to cull effected ewes before service. Treatment by injection of antibiotics and NSAIDs is helpful coupled with stripping of the affected mammary gland. Intramammary treatment is possible but difficult in a flock situation.

<u>The adult sheep</u>

1. Acute Fasciolosis

This condition occurs between October and January. The main signs are anaemia, dyspnoea, ascites and abdominal pain. Diagnosis is by PM or blood tests. The flukes are immature so faeces examination is not useful. Fascinex is the only flukicidal drug, which is effective.

2. Acute Haemonchosis

This mainly occurs in growing lambs but can occur in adults. Anaemia and even sudden death are the signs. A massive visible worm burden is seen on PM. Obviously early treatment with an appropriate anthelmintic is vital.

3. Acute Pneumonia

This condition is normally caused by *Pasteurella* species (Recently the bacteria has been renamed *Mannheimia haemolytica*). The sheep will show acute respiratory distress and a raised temperature. Antibiotics and NSAIDs are the normal treatment. Although Pasteurella is a short gram negative rod, Penicillin and Streptomycin combinations are often effective. 'Micotil' used to be our Rolls Royce treatment. However it is now restricted to professional injection only. (Micotil is toxic to goats)_Ovine Pulmonary Adenocarcinoma (OPA) is a chronic condition seen only in adult sheep and would not normally be seen as an acute problem. However the terminal stages will show as respiratory distress but the temperature is unlikely to be raised. The 'wheel barrow test' is pathoganomic. This test is carried out by lifting the hind legs of the sheep. In OPA there is an outpouring of watery nasal discharge. The prognosis in these cases is hopeless.

4. Acute toxicity

- Acidosis

This is quite a common condition resulting from an excess of corn or sugar beet tops. Treatment is rather unrewarding. Rehydration with fluids by mouth, NSAIDs and Vitamin B preparations are recommended.

- Adder bite. I have only seen one confirmed case. The shepherd saw the ewe struck.

It was not fatal. My treatment consisted of Dexamethazone and Pen/Strep. The ewe was bitten on the face and the whole head swelled.

- Plant Poisons

These are rare in commercial sheep. They may occur in pet sheep.

i) Acorn Poisoning

There is normally rumen atony and constipation. This causes pain which can be shown as bruxism. The course of the poisoning maybe several days, death being preceded by melena and diarrhoea. There is no specific treatment but fluids are obviously beneficial.

Rolling acorns into the ground is a good preventative.

ii) Brassica Poisoning

This normally shows as acute anaemia with cardiac signs, but nervous signs namely blindness may also be seen. Removal from the plants will allow some to then self-cure. However in pet sheep a more aggressive type of treatment may be required. Blood transfusions have been suggested but I can find no actual reported usage.

iii) Rhododendron poisoning

Abdominal pain with vomiting is pathognomic for this condition. Treatment by injection of Atropin sulphate and Morphine is usually successful.

vi) Ryegrass staggers

I have only seen this condition once in the dry summer of 1975. It occurred, when eventually we had rain. No sheep died but they showed a strange kangaroo gait. It is caused by a mycotoxin on the grass

- Chemical Poisons

i) Copper poisoning

Sheep are very sensitive to copper. Poisoning now is rare, as copper is no longer added to pig food as a growth promoter. The main sign is jaundice. In more chronic cases you may see photosensitisation. Treatment in the acute cases is unlikely to be successful.

ii) Nitrate poisoning

The signs are acute diarrhoea, respiratory distress and collapse. If they live long enough you may see methaemoglobinurea. The treatment is specific. 1% methylene blue solution at 9mg/kg should be given by slow intravenous injection.

5. Babesiosis

The signs of this disease are fever, jaundice and haemoglobinuria. It occurs when naïve sheep are moved into a tick-infested area. There is no licensed treatment but Imidocarb diproprionate is effective at the cattle dose on a weight basis

6. Blue tongue

This viral disease spread by *Culicoides* has not been seen in UK but is becoming more common on mainland Europe. Practitioners should be vigilant for outbreaks of acute respiratory disease. Individual sheep when examined will show petichial haemorrhages on the gums.

7. Cerebro-Cortical Necrosis (CCN)

This is mainly a neurological disease of growing lambs but it can occur in adults. The affected animals circle and appear blind. If treated at this stage with Vitamin B1 there is a good recovery rate.

8. Clostridial Disease

This condition is rare in adults. However Clostridium sordellii may cause disease to ewes and rams on a steep rising plane of nutrition. Covexin 10 is the only clostridial vaccine available at the present time, which includes cover against this organism. However at present supplies are difficult.

9. Coenuriasis

This parasitic condition of sheep, which is manifest by neurological signs, is usually known as ‘Gid’. It is caused by the invasion of the brain by the intermediate stage of the tapeworm *Taenia multiceps.* Surgical decompression has been reported as being successful.

10. Fly strike

This is an extremely important condition of all ages of sheep, both from a welfare and an economic point of view. Preventative treatment is paramount and is easy with modern pour on products, however good husbandry methods to prevent fleece soiling are still important. The products vary in the stage, at which they prevent fly strike, so the directions should be strictly adhered to.

In the event of a strike welfare must be considered. If over a quarter of the body area is affected they euthanasia should be immediate. If treatment, in less serious cases, is under taken, antibiotics and NSAIDs should be given immediately. The fleece will need to be removed over all the affected area and a two-inch margin round the area. Then the maggots should be removed or killed and the whole skin area affected should be covered with an oily cream. There are many insectival preparations, which are licensed for the treatment. These should be used, not strong disinfectants e.g. Lysol etc. A hair drier is useful to bring all the larvae to the surface so they can be removed. Battles 'Summer fly cream' which contains Acriflavin and BHC is useful to apply to the affected area.

11. Frothy bloat

This is seen from ingestion of large amounts of clover. Treatment with birp or trocarisation is often not very rewarding.

12. Listeriosis

This disease is associated with feeding of poor quality silage. The sheep show nervous signs and a fever, which helps with the diagnosis. There is no licensed treatment but Florfenicol in my hands appears to be curative. Provided the ewe is not pregnant, I often give an injection of Dexamethazone to cut down the inflammation. However some authorities consider this treatment delays the antibiotic penetration through the blood/brain barrier.

13. Orf

This viral skin disease found near the mucosal skin junctions is not an acute disease. However it may lead to secondary infections in all ages of sheep. The usual pathogens are *Dermatophilus species, corynebacterium pseudotuberculosis* or *actinobacillus lignieresii*. These may cause severe mastitis if round the teat in lactating ewes or starvation if round the mouth in young lambs. Although it is a virus, comparison studies have shown that injections of Penicillin/Streptomycin are very beneficial.

14. Looping ill

This viral disease is invariably fatal. It is mainly spread by ticks. There is a biphasic fever with nervous signs appearing on the second rise. There is a vaccine available.

15. Pneumonia

Pasteurella now called *Mannheimia haemolytica* is the main causal organism. Vaccination is normally carried out with a combined Pasteurella and Clostridial disease vaccine. The organism is sensitive to large doses of Penicillin or Tetracyclines. "Micotil" might have been considered the drug of choice but it is now no longer allowed for farmer's use.

16. Sheep Scab

This acutely irritant skin disease with welfare considerations is no longer notifiable. It is caused by the mite *Psoroptes ovis.* Dipping used to be the accepted method of treatment. However now a single injection of 'Dectomax' is sufficient.

17. Urethral Obstruction

This normally occurs in young males either entire or castrated, kept inside and fed on dry food. The prepuce will be dry. Pulsation will be felt under the anus but no urine will be passed. The obstruction may be only in the vermiform appendix. If so this can be cut off with a pair of scissors. However if the obstruction is higher a urethrostomy can be performed for salvage of the lamb for slaughter but obviously not for breeding. I am worried about the welfare considerations of this type of surgery. The remainder of the group should be fed on food containing more water. Ammonium chloride should be added to the diet at the rate of 200mg per kg per day.

This list is by no means exhaustive but covers the normal acute diseases affecting adult sheep in the UK.

A fresh approach to skin examination in large animals

There is a welfare aspect to skin disease in large animals which must not be forgotten by practitioners. Whether the animals are kept in large flocks or herds by commercial farmers or as small flocks or herds by smallholders or as pets by members of the public, animals with skin disease are suffering. The practitioner needs to examine the skin with all the means at his or her disposal in order to make an accurate diagnosis and institute the best treatment. These notes are written with the best interests of the large animals at heart without forgetting any zoonotic implications.

The fresh approach relies on a 'key point' code

These codes will provide basic information only, but they should allow a degree of disease recognition and give effective and rapid guidance on basic therapy. Thus, if the disease is seasonal and restricted to the summer months (e.g. 'fly strike' in sheep), the code suggests that in winter this disease is very unlikely.

Code number	Descriptor	Description
[1]	Geographical restriction	Geographical restrictions may be absolute but care must be taken on account of movement by vehicle.
[2]	Hereditary	Diseases/disorders with a genetic origin that can be passed on from parent to offspring.
[3]	Congenital	Congenital disorders and malformations are not necessarily hereditary. Most are present at or shortly after birth.
[4(0+)] [4(1+)]	Age-restricted	0+ = 0-1 month 1+ = 1-6 months etc.
[5]	Seasonality	Winter, spring, summer & autumn.
[6]	Contagion	Spread from an animal to another of that species.
[7] C, S, G,	Other species	Transmissible to another animal of a different species.
[8]	Zoonosis	Transmissible to man
[9]	Nodular	Focal skin thickening larger than 0.5mm
[10]	Alopecia	Hair loss from the disease not from self trauma.
[11]	Crusting	Dried exudate
[12]	Scaling	Excessive exfoliation
[13]	Pigmentary changes	Either to hair or skin
[14]	Single focus	A single isolated or occasional few isolated lesions.
[15]	Multifocus	Several often many similar foci.
[16]	Generalized	Covers a wide area of the body.
[17]	Pain	Mild to extreme
[18]	Pruritus	Itching
[19]	Systemic effects	Cutaneous disease can have systemic effects and systemic disease may have cutaneous effects. Both of these circumstances are covered by this code.
[20]	Persistent	Does not resolve but may vary in severity.
[21]	Intermittent	Unpredictable recurrence at very irregular intervals.
[22]	Recurrent	When reoccurrence is not linked with seasonality.
[23]	Pathognomic pathology	A true diagnosis from tests.
[24]	Prognosis	Good, guarded, poor & hopeless.
[25]	Reportable	Notifiable (now differences in countries within the UK i.e. Sheep Scab)

Table 1 Key Codes

General History

Before any physical examination the practitioner needs to prepare a relevant history. It is vital that this is taken on site. Many skin diseases, particularly after there has been treatment given or self trauma by the animal has occurred, look the same. However practitioners will have an eye for relevant small details which may well be missed by the client. There is no harm in using a magnifying glass. Photographs taken by the client are useful and with the easy of email will be more utilised in future. However it would be very difficult to get the 'feel' of the whole farm and group with photographs. Videos certainly will also help. How many years before dermatologists can sit in a warm laboratory and down load good pictures and videos, and then instruct the owner to take good samples and wait for their arrival.

Practitioners will want to record the details for the practice computer. Ideally this should be done on farm, like in the small animal consulting room, however such sophistication may not be available but it is coming. The key codes shown in table 1 will save time.

The general geographical location code 1 can be taken as read. There may be difficulties for a farmer on the border between Scotland and England for the notification to DEFRA with an outbreak of sheep scab. The reportable code 25 will create problems in these incidences.

Code 5, seasonality will be recorded with the date. However careful questioning maybe required ascertaining whether the disease is really seasonal. Did those lice actually appear on housing?

Code 4, age, will be recorded at this stage. Juvenile warts caused by a Bovine papilloma virus are unlikely to affect very young calves or adult cattle. On the other hand newly born or very young animals are going to attract codes 2 and 3. Congenital conditions such as imperfecta (Picture) will be readily apparent. Practitioners will need to take care when hereditary conditions are suspected. Luckily many e.g. the sticky kid syndrome seen in Golden Guernsey's are clearly inherited.

Group History

Code 6, contagion will be readily apparent. Practitioners will need to know what species for code 7 are affected at this stage, together with an inquiry whether any humans are affected to rule out code 8. Obviously if a diagnosis is made e.g. *Trichophyton verrucosum* infection in a group of cattle this code has to be reinstated. Clients can also be warned at this stage that other species e.g. sheep and goats are at risk. Code 19, systemic disease, will be covered at this stage although it will be developed further when an individual animal is examined.

Individual History and Examination

Codes 20, 21 and 22, persistent, intermittent and recurrent will need to be decided. Skin disease is the same as any other contagious disease affecting a group of animals. Clinicians need to select individuals carefully before examination. Often the newly affected individuals will yield not only clearer clinical signs, before self trauma and secondary infection, but also will provide better affected areas for sample taking. In this age of accountability and traceability it is important for practitioners to record the identity of individuals examined. Ear tags will need to be read. When examining the individual animal the clinician needs to decide if there any systemic effects resulting from this cutaneous disease, equally whether the cutaneous signs seen are a result of a systemic disease. In either case code 19 will apply.

Unlike companion animals, farm animals may be nervous of strange humans and not show pruritus readily. Careful observation may be required to decide on code 18. Pruritus is a very useful sign. Always with pruritis 'think parasites'. Parasites e.g. lice will cause pruritus just from their physical presence. Equally parasites will cause irritation with a hypersensitivity reaction e.g. chorioptic mange can be extremely irritating to goats. The itching by the goat will cause further histamine release making the condition worse.

As stated earlier many skin conditions are a welfare issue. Pain is caused by sheep scab but because of the stoicism of sheep this may not be readily apparent. The psoroptic mite initially will cause intense pruritis, which will cause the sheep to inflict self trauma. These open sores will be painful particularly if there is a secondary staphylococcal infection. Code 17 will certainly help with diagnosis as streptococcal and dermatophilus infections are not normally as painful as staphylococcal infections. Obviously the clinician will want to treat the initial cause of the problem, the psoroptic mite. However treating the inflammation and the pain with NSAIDs will be useful. Antibiotics will also help to reduce the secondary infection. All three of these treatments will be required to lessen the suffering of the sheep.

Codes 14, 15 and 16 are going to require some detective work. With an individual that the clinician is examining it is fairly straight forward to decide whether the skin disease is general, multifocal or just a single focus. The clinician has to decide if this individual is typical of the group and also if this number of lesions is typical of the disease. Staphylococcal dermatitis on a goat's udder may well start with a single focus but rapidly in a few days it will progress to being multifocal. It rarely infects any other area of the skin and therefore will never be a generalized code 16. On the other hand sarcoptic manage in an alpaca may well start as a single focus then progress to a multifocal disease and then unless there is aggressive treatment will become a generalised skin condition.

Dermatologists have a large number of ways to describe the histopathology of skin lesions, when they are sent a biopsy. However the clinician needs to describe the visible appearance of the lesion. This is covered in codes 9, 10, 11 & 12. It is also useful to record where a single lesion is located and also where a more generalised area of skin is affected. Parasites certainly focus on specific areas of the body e.g. chorioptic mange is most frequently found around the tail head in cows.

Further simple diagnostic tests

These are certainly less used in farm animal medicine. There are many reasons for this. I hope the most important reason is that practitioners are more familiar with the causes of farm animal skin disease and therefore rapid pattern recognition is carried out. With the advent of small cameras, particularly those linked to mobile telephone, photographs are very helpful for recording skin disease e.g. A Norfolk Wenn in a goat. They allow the practitioner to confer and refer cases to colleagues almost instantaneously.

They allow accurate recording of not only the visual appearance of the condition but also the extent. Obviously if taken on subsequent occasions the progression of the condition can be recorded. However practitioners should always remember the value of further diagnostic tests so that code 23 can be fulfilled. It is only by a confirmed diagnosis that an accurate prognosis, code 24 can be given. Ringworm is a good example to study. First of all it is very common in cattle and is very easy to diagnose. The clinician can give an exact prognosis in the individual animal i.e. there will be self cure in 16 weeks. The organism is well known. It is *Trichophyton verrucosum.* However this same organism can infect not only man but other farm species. I suggest that pattern recognition of this organism in sheep or goats is not easy. Without an exact diagnosis the clinician will not be able to give an accurate prognosis. Hair sampling as a further diagnostic test will need to be carried out. The hair and associated scabs should be plucked out from the margins of the lesions. These samples can be collected into envelopes or sterile bottles and transported to the laboratory. If sterile bottles are used these should not be sealed but closed loosely and secured with some zinc oxide tape for transportation. Airtight closure will encourage the growth of saprophytic fungi. Obviously these samples can be sent away. However many practitioners will choose to process the samples themselves. The specimen hairs should be placed on a microscope slide, a few drops of 10% potassium hydroxide should be added and the hairs teased out. The slide should then be warmed over a spirit lamp. A couple of drops of a fungal stain such as cotton blue should then be added before a cover slip is placed in position. Initially the hair should be identified under the low power. Then it should be examined under the high power. *Trichophyton verrucosum* spores will be present as chains on the hair shaft. *Microsporum canis* spores will be seen actually inside the hair shaft. In sheep, goats, pigs and alpacas *Trichophyton verrucosum* is a very serious condition and will not clear up without very aggressive antifungal treatment. It is also very contagious to humans. On the other hand *Microsporum canis* is self limiting in all species other than in dogs and is not very infectious to humans. Therefore this simple diagnostic test which can easily performed by practitioners with no extra equipment can clarify the diagnosis and prognosis of a common condition in farm animal practice.

Biopsy

The taking of a biopsy is the ultimate diagnostic tool. It is likely to answer code 23 with a pathognomic finding provided certain conditions are adhered to. A full description of the case must be sent with the biopsy to the pathologist. The biopsy must include both healthy and diseased tissue. The pathologist MUST BE fully familiar with skin disease in farm animals. There are many excellent small animal dermatologists available. There are a few excellent equine dermatologists available. The number of farm animal dermatologists in the UK can be counted on the fingers of one hand. Make sure you send your sample to one of those. Good advice is to ask your local Veterinary Laboratory Agency (VLA). They have always given the author excellent advice in the past. They have some extremely good dermatologists within their ranks and therefore they will refer your samples to the best in the country.

Common things commonly occur

The title of this article is ‘A fresh approach to skin examination in large animals’. However although the author has tried a fresh approach, sadly it is to the same old skin diseases. Therefore they are summarised in table form by species See tables 2 - 6. The conditions are restricted to those found in the UK. The tables list actual skin diseases not systemic diseases which have skin manifestations.

Disease	Cause	Codes
Ringworm	*Trichophyton verrucosum*	6,7 S G,A&P,8,9,10,15,18,23,24 Good
Lice	*Damalinia (Bovicola) bovis* *Haematopinus eurysternus* *Linognathus vituli*	5 Winter,6,7,10,16,18,20,23,24 Good
Mange	*Chorioptes bovis* (Very rare *Psoroptic manage*)	6,7A&G,9,10,15,18,23,24 Good
Abscess	*Arcanobacterium pyogenes*	14,17,23,24 Good
Wooden tongue	Actinomycosis	14,17,19,24 Good
Lumpy jaw	Actinobacillosis	14,19,20,24 Poor
Furunculosis	*Staphylococcus aureus*	11,15,17,23,24 Good
Ticks	*Ixodex ricinus*	7S,G & A,14,23,24 Good
Cellulitis (Malignant oedema)	*Clostridium spp* (Normally *Clostridium septicum*)	16,17,19,20,23,24 Poor
Blackleg	*Clostridium chauvoei*	14,17,19,20,23,24 hopeless
Fly strike	*Calliphora spp*	5 Summer,11,14,17,19,23,24 Good
Herpes Mammilitis	*Bovine herpesvirus-2*	6,9,15,17,23,24 Guarded
Dermatitis, Pyrexia and Haemorrhagic Syndrome	Multifactorial	10,11,16,17,19,20,23 Poor
Photosensitisation	Live toxin e.g. St John's Wort	5Summer,9,10,11,13,15,17,19,21,23,24 Guarded
Urticaria	Immunological	5Summer,9,15,19,22,24 Guarded
Pseudocowpox	*Parapoxvirus bovis-2*	6,9,15,17,23,24 Guarded
Snake bite	Adder	5 Spring & Summer,9,13,14,17,19,24 Good
Burns	Infrared Lamps	11,14,17,24 Good
Cow pox	*Orthopoxvirus*	6,8,9,15,17,23,24 Guarded
Epidermolysis bullosa	Autosomal recessive inheritance	4(0+),10,11,13,15,17,19,20,24 Hopeless

Table 2 Skin diseases seen in cattle in Norfolk in order of prevalence as seen by the author

Disease	Cause	Codes
Orf	Contagious pustular dermatitis *Parapox virus*	6,7 G & A,8,9,11,15,17,23,24 Good
Sheep scab	*Psoroptes ovis*	6,7,G & A,10,15,17,18,23,24 Good,25
Caseous lymphadinitis	*Corynebacterium pseudotuberculosis*	6,7,G ,9,15,20,23,24 Hopeless
Myasis (Fly strike)	*Calliphora spp & Musca spp*	5 Summer,11,14,17,19,23,24 Good
Ticks	*Ixodes ricinus*	7C,G & A,14,23,24 Good
Keds	*Melophagus ovinus*	7,G,14,23,24 Good
Lice	*Damalinia (Bovicola) bovis* & *Linognathus ovillis*	5 Winter,6,7C,10,16,18,20,23,24 Good
Mycotic dermatitis	*Trichophyton verrucosum*	6,7C,G &A,8,10,12,13,15,18,23,24 Good
Wool-slip	Stress	4(2+),10,16,19,21,24 Good
Photosensitisation	Liver toxin e.g. St John's Wort	5Summer,9,10,11,13,15,17,19,21,23,24 Guarded
Demodectic mange	*Demodex*	6,9,10,11,15,18,20,23,24 Poor
A Norfolk Wenn	*Actinobacillosis*	9,11,14,23,24 Good

Table 3 Skin diseases seen in sheep in Norfolk in order of prevalence as seen by the author

Disease	Cause	Codes
Furunuculitis	*Staphylococcus aureus*	6,9,11,15,19,23,24 Good
Orf	Contagious pustular dermatitis *Parapox virus*	6,7 S & A,8,9,11,15,17,23,24 Good
Sheep scab	*Psoroptes ovis*	6,7,S & A,10,15,17,18,23,24 Good,25
Psoriasiform perivascular dermatitis	Unknown often called Pygmy goat syndrome	5Summer,9,10,11,12,15,20,23,24 Poor
Warts	*Papavovirus* or *Papilloma virus*	4(1+),6,9,15,23,24 Good
Caseous lymphadinitis	*Corynebacterium pseudotuberculosis*	6,7,S,9,15,20,23,24 Hopeless
Myasis	*Calliphora spp & Musca spp*	5 Summer,11,14,17,19,23,24
Chorioptic mange	*Chorioptes caprae* (*Chorioptes equi*)	6,11,15,18,23,24 Good
Ticks	*Ixodes ricinus*	7S,C & A,14,23,24 Good
Fleas	*Pulex irritants*	5 Summer,6,8,18,23,24 Good
Ringworm	*Trichophyton mentagrophytes*	6,7 A,8,10,12,13,15,18,23,24 Good
Lice	*Damalinia caprae & Linognathus stenopsis*	5Winter,6,,10,16,18,20,23,24 Good
Sarcoptic mange	*Sarcptes scabiei*	6,11,15,18,23,24 Guarded
Psoroptic mange	*Psoroptes cuniculi*	6,7 S,11,15,18,23,24 Good
Harvest mites	*Trombicula atumnalis*	5 Autumn,18,23,24 Good
Poultry mites	*Dermanyssus gallinae*	11,18,23,24 Good
Sheep keds	*Melophagus ovinus*	7S,14,23,24 Good
A Norfolk Wenn	*Actinobacillosis*	9,11,14,23,24 Good
Demodectic mange	*Demodex caprae*	6,9,10,11,15,18,20,23,24 Poor
Sticky kid syndrome	Only in Golden Guernsey goats	4(0+),16,19,20,23,24 Hopeless

Table 4 Skin diseases seen in goats in Norfolk in order of prevalence as seen by the author

Disease	Cause	Codes
Chorioptic mange	*Chorioptes bovis*	6,10,11,12,15,18,23,24 Good
Sarcoptic mange	*Sarcoptes scabiei*	6,8,10,11,12,13,16,17,18,2o,23,24 Poor
Myasis (Fly strike)	*Calliphora spp & Musca spp*	5 Summer,11,14,17,19,23,24 Good
Orf	Contagious pustular dermatitis *Parapox virus*	6,7,S & G,8,9,11,15,17,23,24 Good
Lice	*Damalinia breviceps & Microthoracius spp*	5Winter,6,18,23,24 Good
Psoroptic mange	*Psoroptes cuniculi & P. ovis*	6,7S,11,12,15,18,23,24 Good
Lumpy jaw	Normally tooth related. *Fusobacterium necrophorum*	4(2+),9,14,19,20,23,24 Guarded
Ringworm	*Trichophyton mentagrophytes & T. verrucosum*	6,7CSG,8,10,12,13,15,18,23,24 Good
Ticks	*Ixodes ricinus*	5Summer&Autumn,7S,13,14,23,24 Good
A Norfolk Wenn	*Actinobacillosis*	9,11,14,23,24 Good

Skin nodules in goats

Definition
A relatively hard roughly spherical abnormal structure associated with the skin. This includes papules, which are raised skin lesions with distinct borders of different shapes and sometimes associated with other features such as crusts and scales.

Classification
Nodules may be classified in a variety of ways e.g. causes, appearance, position on the animal and even age of animal affected. The cause of nodules may be sub divided into infectious e.g. viruses, bacteria, fungi and parasitic, or non-infectious e.g. trauma, neoplasia and auto-immune conditions.

An approach to diagnosis
A good history is vital. The clinician needs to know the age of the goat and how long it is thought to have had the nodule. It is helpful to know any changes that have occurred and any treatment, which has been administered. Naturally the clinician needs to know if other goats are affected, the size of the herd and whether other species are in contact with the goats. I think it is prudent for the practitioner to ask if any humans are affected with lesions. The clinicians should beware as many quite minor looking nodules are a disaster waiting to happen. They may enlarge rapidly, infect other goats and may infected humans. It is also useful to find out if the herd is closed or if there have been any new goats brought on to the holding. It is tactful to find out if any vaccines have been given to the goats and helpful to find out if there is pressure for a swift resolution to the problem e.g. is the goat due to go to a show or to be sold. It is very important that the clinician takes every case seriously. The lesion may look trivial but it is important to the owner.

The ultimate diagnostic tool is the biopsy. However less invasive procedures can be carried out first. A photograph is always worthwhile. A skin-scrape to obtain some of the crusts or scales maybe helpful. This can be looked at under low power to diagnose mites, or stained and examined under oil emersion to study any bacteria. It must be remembered that these may be secondary. Swabs are rarely helpful but fine needle aspirates or tru-cut biopsies are often diagnostic. It is important to send any samples to a laboratory, which deals with goats. In my experience the VLA is always helpful. A full history should be included together with a description of the lesion. Biopsy material should include an area of normal skin adjoining the lesion. Great care should be taken to separate any fresh material from histological samples as formalin in the smallest amount will kill bacteria and viruses. Samples for these should be taken first in special transport medium and sent separately to the laboratory.

Remember that a second opinion in a refractory case is always useful. This can be a colleague from your own practice or email with a photograph and a history to a more remote colleague. On the whole I do not advise physical referral to a specialist dermatologist as these are more familiar with small animal cases. One has to consider disease control and the fact that goat owners are not usually impressed with experts in white coats!

Viral causes of skin nodules
In the UK the most common viral skin disease in goats is Contagious Viral Pustular Dermatitis, which is normally acquired from sheep. It is called ‘Orf’ in sheep. The disease is caused by a *Parapoxvirus.* Practitioners should be aware that it is a zoonotic disease and so normal hygiene precautions should be carried out. The use of rubber gloves is recommended. The condition is normally diagnosed clinically. However the virus is readily isolated from fresh scabs. The nodules are found mainly around the mouth but can occur on the udder. There is a live vaccine available for sheep but its use in goats is not recommended.

Goat pox caused by a *Capripoxvirus* is not found in the UK but is seen in Central Asia. Although it causes nodules around the mouth like ‘Orf’, it is also a serious systemic disease. Diagnosis is either by virus isolation or histopathology.

There are warts, cutaneous papillomas, found in goats, which may be of viral origin but they are normally either self limiting or remain as a benign hard growths. There occurrence on the udders of white goats can be annoying for the milker but they do not seem to spread.

Bacterial causes of skin nodules

In my experience the most common nodules caused by bacteria in goats are iatrogenic from clostridial disease vaccination. These are embarrassing if the vaccination has been carried out by a veterinary surgeon. They are not serious, but will take time to ulcerate and disappear.

On the other hand nodules caused by Corynebacterium. P*seudotuberculosis* are very serious. The bacteria will spread to the lymph nodes and will spread to other goats. Immediate humane destruction must be recommended as soon as a positive culture has been made. There is no effective treatment available.

Staphylococococcus. aureus is a common cause of nodules on and in the udders of goats. If they are in the mammary tissue, they may remain dormant for years. The body walls them off. However if they burst internally, they may well cause a septicaemia and severe illness. They may occur on the skin. They burst and then heal leaving a scab and eventually a scar.

Dermatophilus. congolensis will cause skin lesions in goats. These are rarely nodules but are manifest as scaling and crusting.

Nocardia. spp will caused skin nodules but they are rarely reported.

Actinobacillus. lignieresii has been reported as causing a skin nodule in Norfolk.

Fungal causes of skin nodules

Fungi are rare in goats. It must be remembered that they are a zoonotic condition. Therefore the practitioner should warn the owner to take normal hygienic precautions. Washing carefully with soap and water or dilute chloro-hexidine is worthwhile. Owners should avoid rigorous scrubbing or strong disinfectants as the skin barrier will be breached allowing the fungi to penetrate. The most common infections are caught from other species, namely *Trichophyton verrucosum* from cattle and *Microsporum canis* from dogs. The species actually linked to goats is *Trichophyton mentagrophytes*. The clinical picture of round crusting lesions is the same for all three species. Pruritis is more marked with *T. verrucosum* infection. Obviously several animals are likely to be infected. The organism is readily grown on proprietary plates and will be identified by a red colour change within ten days. Often there will be secondary bacterial infection. Only debilitated goats will get a bad infection. In normal goats the infection is self limiting in a few months.

Parasitic causes of skin nodules

Parasites will cause scaling and crusting with secondary bacterial involvement. However the true nodule is only seen in Asia. *Przhevalskiana. silenus* is the goat warble fly and will cause nodules on the dorsum in goats in the spring. There will be a small hole on the top of the noodle. The pupa will emerge as the weather gets warm and pupate into fly, which lays eggs on the legs of other goats. These hatch and bore through the skin of the goat. During the winter they migrate through the goat to emerge in the spring.

As with fungi mite infestation normally occurs from other species. Although it is called *Chorioptes. caprae,* there is considerable argument that it is a separate species from *Chorioptes. equi* which is found on cattle, around the tail head and horses on the lower limbs. This is the most common mite found on goats. *Sarcoptes. scabiei* is extremely rare. *Psoroptes. ovis*, the mite responsible for sheep scab will occur on goats in close contact with sheep. *Psoroptes.* cuniculi, which is normally associated with rabbits ears has been isolated from goats. All four of these species will cause pruritis and can be found on a skin scraping examined under low power. Goats will get *Demodex. caprae*. This is very hard to demonstrate except with a very deep skin scraping. I have only managed to get a positive diagnosis with a skin biopsy.

Goats will become infested with lice, *Damalinia. caprae.* The resulting pruritis will cause considerable skin thickening. In the UK *Ixodes. ricinus* is found on goats. There are many ticks found on goats in other arts of the world.

The final parasite to cause skin thickening and skin nodules in goats is a *Culicoides. spp* sensitivity.

Traumatic causes of skin nodules

Never forget common place trauma. I have removed two air-gun pellets from goats. One of these came out unexpectedly when I lanced an abscess on the flank of a ‘Billy’ goat. The other was suspected by an owner. We confirmed this quite simply with the rectal linear scanner. However actual location and removal was not so easy. Tethered goats will develop tether galls. This is a welfare issue and owners should be counselled carefully. Chronic foot lameness in the front legs will result in excessive kneeling and the formation of hygromas on the carpi. Burn cases will often result in keeloids, crusty nodules. Sunburn will cause crusting in photosensitised animals.

Neoplastic skin nodules

These are rare in goats but are more often reported than in sheep, mainly because goats tend to be kept to greater ages. The more common neoplastic skin nodules tend to be benign. The thyroid gland can become enlarged and will appear as a benign growth. This is not goitre and has no relationship to iodine deficiency. Further down the neck the thymus can become enlarged in young goats. This is a thymoma and is also normally benign. Benign cysts may occur in the wattles or in the salivary glands. Tetragenic dental cysts may be seen on the faces of goats.

The nasty tumours which may occur in the skin anywhere on the body are Histiocytomas, Lymphosarcomas, Malignant melanomas and Squamous cell carcinomas. The later in my experience occur in the conjunctiva, on the third eyelid, on the penis and on the vulva. Melanomas are not restricted to white goats.

Skin nodules caused by auto-immune disease.

Pemphigus Foliaceus causes a diagnostic challenge for the clinician. Bacteria, fungi and even parasites may well be found as secondary invaders. If the skin condition persists after treatment for these conditions the practitioner should suspect pemphigus. It can be confirmed by a biopsy. In my experience pemphigus is very common in pygmy goats.