Calf Feeding Practices in Relation to Health

R.W. Blowey, *BSc, BVSc, MRCVS*
124 Stroud Road, Gloucester, England

Key Factors

- Stimulation of oesophageal groove closure is achieved from the delight of feeding, not just thirst.
- Many management features lead to poor groove closure, which in turn causes colic, bloat and ill-thrift.
- Formation of the abomasal milk clot is an important first stage in digestion.
- Most milk substitute problems are associated with incorrect mixing.
- Abomasal dilation and ulceration may be caused by irregular feeding.
- The causes of chronic post-weaning scour are often difficult to identify.

Although infectious diseases are common in young calves, often their appearance is a consequence of some failure of management. This paper considers some of the factors which may be involved.

The Oesophageal Groove

The calf is a ruminant and in common with other ruminants it has four stomachs, namely the reticulum, rumen, omasum and abomasum. The very young calf only uses its fourth stomach, the abomasum, and functions essentially as a monogastric animal. Milk has to flow directly from the oesophagus into the abomasum, by-passing the rumen and reticulum via the oesophageal groove. When the groove is in the open position, milk passing from the oesophagus would fall into the rumen, become sour and cause a digestive upset. When the groove closes, a "pipe" is formed which transports milk directly through to the omasum and into the abomasum.

The oesophageal groove is a continuation of the lower oesophagus, passing across the medial wall of the reticulum and terminating in the reticulo-omasal orifice. In the young calf, where the abomasum comprises 60% of the capacity of all the four stomachs, the abomasum lies ventrally and slightly to the left of the abdomen. The groove now runs almost vertically. As rumen development progresses, the abomasum is pushed towards the right flank (abomasum now equals only 10 - 15% of total stomach capacity) and the groove is deflected to run in a ventro-medio-caudal direction, in other words, diagonally downwards, towards the right.

Closure Mechanisms

The muscles of the groove are controlled by the vagus nerve. There are three steps in groove closure:

1. Contraction of the pillars, pulling the lips of the groove together.
2. Contraction of longitudinal muscles, leading to shortening of the groove.
3. Twisting of the right lip, so that it curls over and conceals the left lip, thereby totally enclosing the channel.

The stimuli which produce groove closure also induce a simultaneous dilation of the reticulo-omasal orifice and an opening of the omasal canal. Milk can now flow directly into the abomasum.

Sucking produces quite high vacuums, e.g.

40mm Hg below atmospheric in teat
200mm Hg below atmospheric in mouth
(cf. milking machine vacuum = 380mm Hg)

Suckled milk is therefore projected rapidly into and through the cervical oesophagus. A separate series of pulsatile movements of the caudal oesophagus propels liquid through the reticular groove and quite forcefully into the abomasum. During sucking, ruminal activity virtually ceases.

Even in the adult, the groove continues to contract, some 3000-4000 times per day, when it acts to draw fluid from the reticulum into the omasum with each contraction.

Factors stimulating groove closure

Home (1806): postulated the role of the oesophageal groove to prevent milk entering the rumen, Webster (1926): assessed oesophageal groove closure manually through a ruminal fistula. He showed that:

1. In the very young calf, the groove closed when sucking milk or water.
2. However, milk was much more effective than wa-

Paper presented at the BCVA Summer Meeting, July 1994 and published in Cattle Practice JBCVA Vol. 2, Part 3, July 1994

ter after 2-3-weeks old.

3. The groove did not close when
   - milk was fed through a tube directly into the upper cervical oesophagus
   - the buccal and pharyngeal mucosa were anesthetized by swabbing with a local anesthetic.
   - atropine was given (partially inhibits closure).
4. Groove closure occurs in sham-fed calves (i.e. suckled milk passes back out through an oesophagostomy fistula and does not enter the abomasum).

This work has since been repeated by others and shows that oesophageal groove closure requires a complex series of stimuli. It is not sufficient for the calf simply to suckle from a teat. It needs to build up a behavioral pattern, associated with suckling and it is the activation of the whole behavioral pattern, through a range of visual, olfactory, thermal and chemical stimuli, which affects full groove closure. The mechanism can be suppressed significantly by prior administration of intravenous adrenalin and hence stress inhibits closure. It is the delight from suckling which activates groove closure, not just the desire of thirst.

Practical Considerations

Groove closure is stimulated by sight, sound, taste and other stimuli associated with feeding. It is therefore very important to build up a specific feeding pattern so that the calves know what to expect. They need to be "wooed into the mood" and ideally should be able to experience some of the activity of feed preparation prior to its allocation. Poor groove closure results from:

1. Irregular feeding times and/or feeding pattern. For example:
   feed at the same time of day.
   feed housed calves in the same order.
   remove the same bucket (e.g. concentrates?) each time and replace it with milk
   let the calf see and hear the milk being prepared.
2. Incorrect/variable milk temperatures.
3. Feeding after stress -
   moving
   arrival from Market
   dehorning
   castration
4. Incorrect position of the feeding point, e.g. the base of the feeding bucket should be at least 30cm above floor level. This means that the bucket needs raising as straw accumulates in the pen.
5. Sudden changes of flow rate from the teats, especially from slow to extremely rapid. This can overload the groove and lead to spillage.
6. Lack of "mothering" in the general sense. Drenching clearly does not produce groove closure.
7. Slow drinkers present a problem. Milk left in front of them cools down and if drunk later may lead to groove spillage.

Milk spilling into the reticulo-rumen ferments to produce colic, bloat and a chronic "pasty" diarrhea. Affected calves often become slow drinkers and this further exacerbates the problem. Bloat may be continuous or appear only 1-2 hours after a feed. Treatment of affected calves includes:

1. Releasing gas with a stomach tube, if badly bloated.
2. Remove milk and feed with electrolytes only for 3-4 days.
3. Oral antibiotics to suppress ruminal fermentation.
4. Remove concentrates for 1-2 weeks.
5. Slowly reintroduce milk, preferably through a teat, to encourage suckling and groove closure.
6. Severe or recurrent cases are best surgically prepared with a permanent ruminal fistula. Such calves usually go ahead quite rapidly. The fistula can be removed surgically 6-12 months later.

Closure in the Adult

Transfer of nutrients and especially pharmaceuticals (e.g. anthelmintics) directly into the abomasum of the adult ruminant can have pronounced advantages. Nutrients may be more efficiently utilized and pharmaceuticals more effective.

Copper sulphate solutions produce groove closures in sheep, but are not particularly effective in cattle. Probably the most effective is prior drenching with 60ml of 10% sodium bicarbonate, which achieves groove closure with a 93% efficiency (Reilk 1954). Less concentrated solutions (e.g. 5%) are less effective. Both the anion and cation must be important, since sodium sulphate and sodium acetate achieve only poor groove closure. Sodium chloride is also effective, but less so than sodium bicarbonate (Reilk 1954).

Scholz (1990) reviewed the use of lysine vasopressin to produce groove closure. Intravenous administration of 40iu immediately prior to drenching improved the clinical response of diarrhoeic cows given oral absorbents and saline and of ketotic cows given oral glucose, compared with controls.

The effectiveness of different benzimidazole anthelmintics depends on their ability to bypass the rumino-reticulum via the oesophageal groove and pass directly into the rumen (Pritchard & Hennessy 1981).

The Abomasal Milk Clot

For both whole milk and the majority of milk substitutes, formation of a milk clot in the abomasum is an

essential first step in the process of digestion. Under the influence of the enzyme rennin and in an acid environment, the protein in the milk clot solidifies and then contracts, squeezing out the liquid whey fraction (containing non-casein whey proteins and sugars such as lactose) which then passes down into the small intestine. The clot in the abomasum is slowly digested by the enzymes pepsin (digesting casein) and lipase (digesting the fat) and any remaining material forms a focus for the next milk clot. The small intestine is alkaline, digestion being carried out by enzymes produced by the pancreas. Lactose is split into glucose and galactose, two simple sugars which can be absorbed and used by the calf for energy. Protein is split into amino acids which can also be absorbed.

If abomasal milk clot formation is poor, then whole milk passes into the small intestine, where casein cannot be digested. This provides an excellent medium for bacterial fermentation and scouring results. Some of the adverse factors associated with poor clot formation are:

- irregular feeding times
- nervous or stressed calves
- overfeeding, especially in the young calf
- milk fed at the wrong temperature
- milk substitute fed at the incorrect strength
- inflammation of the abomasum.

Some of the newer milk substitute powders, especially those associated with *ad libitum* acidified cold milk feeding, do not need to form an abomasal clot. Overfeeding, leading to improperly digested food passing into the intestine, is still important however, even though the acidified milk helps to prevent excessive bacterial growth.

Problems with Milk Substitutes

There is a wide variety of milk substitutes on the market. Many are based on a high level of skim milk powder and form a clot in the abomasum, although some of the newer products, so-called "zero" replacers, because skim milk powder is absent, do not form a clot. If milk powder is overheated during manufacture, then clot formation is poor and scouring may result. However, most of the problems associated with milk substitutes are "on farm" in their origin.

The first golden rule must be to read the manufacturer's instructions. Many manufacturers recommend that milk is first mixed at a higher temperature (45-50°C) before feeding. To do this a thermometer is needed. You cannot accurately judge the temperature of the milk using your hand. On a cold day you will overestimate the milk temperature and feed it too cold and vice versa on a hot day. Trials have shown that if the milk is too hot, a calf simply will not drink it and no harm is done. However, if it is mixed and fed too cold a variety of problems can arise:

- the fat may be poorly dispersed. It rises to the top of the milk and leaves a ring around the calf's nose, often leading to hair loss. If your calves develop bald noses, check your milk substitute mixing routine.
- proteins and minerals may form a sediment at the bottom of the bucket and be wasted
- oesophageal groove closure is poor
- milk clotting time in the abomasum is retarded. A reduction of only 6° C may double the time for the abomasal milk clot to form. There is then an increased risk of undigested milk spilling over into the small intestine.

If a long row of calves has to be fed from a single container, the milk for the last calf in the row can be appreciably cooler - again, watch for bald noses!

Inefficient mixing is probably the biggest problem. Mixing with the hand is simply not adequate - a whisk is essential. Carelessly mixed powders leave lumps, poorly dispersed fat and a protein sediment in the bottom of the bucket. Trials have shown that up to 60% of the oils in the replacer may be wasted in this way, in addition to problems arising from poor abomasal clot formation and subsequent scouring. Ensure that the milk is mixed at the correct strength. This is usually 125g per liter, but may be increased to 150g per liter if fed once daily. Do not dilute milk powder, for example, for a scouring calf. If fed diluted, abomasal milk clot formation will be retarded. Similarly, do not allow a calf to drink large quantities of water immediately after it has finished its milk, as this will have an effect equivalent to diluting the milk. When the milk bucket is empty, it is best to put in a handful of coarse mix or calf pencils, or immediately replace with the concentrate bucket. Some people remove the water bucket for 30-40 minutes after feeding milk.

Certain brands of electrolytes can be mixed with whole milk and actually improve clotting time, but the clot formed may be less stable. In general, therefore, it is best to avoid diluting milk. If electrolytes are needed, feed four times daily, twice with milk or milk substitute, and twice with electrolytes.

Finally, do not feed excessive quantities. Most feeding schedules are designed for a 40kg calf and suggest starting at one liter per feed of the first 4-5 days, increasing by 0.25 liter every second day, up to two liters per feed by 10-12 days old. If you have a smaller calf, feed less. Overloading the abomasum can lead to "spillage" and scouring. In summary, the golden rules for feeding calves with milk substitute are:

- correct temperature
- correct strength
- properly mixed
- fed in the correct amount.

Hair loss around the muzzle is likely to be a sign of problems.

Abomasal Disorders

Abomasal Dilation

This is a condition of sudden onset also seen in beef suckler calves. The calf is presented as being dull, dehydrated and often having colic. Large quantities of fluid are audible on auscultation. This prognosis is poor and treatment includes:

1. *Abomasal flushing.* Place the calf in lateral recumbency on a bale of straw. Pass a gastric feeding tube into the abomasum, run in an electrolyte solution and allow it to flush out under gravity.
2. *Metaclopramide ("Emequell").* Used as an antinausea agent in man, promotes ruminal and abomasal activity in cattle. Give 4-6cc (3 vials) intravenously and repeat in 12 hours.
3. *Intravenous sodium bicarbonate therapy.* Using a Harleco bicarbonate measuring system, Dai Grove-White has found that the majority of calves with severe abomasal dilation and bloat are acidotic and respond well to intravenous bicarbonate therapy (e.g. plasma bicarbonate at 4-10 mmol/liter, compared with a normal value of approximately 25 mmol/liter. Total "bicarbonate space" is assumed to be 0.4 liters per kilogram bodyweight, i.e. 24 liters for a 60kg calf).

Abomasal Ulceration

Studies have shown that a surprisingly high proportion of otherwise clinically healthy calves raised for veal have evidence of chronic ulceration at slaughter. In a few herds there may be significant mortality due to abomasal ulceration in calves 2-4 weeks old. Suggested causes include:

1. *Irregular feeding times,* leading to excessively hungry pre-ruminant calves eating excessive quantities of hay or straw. Undigested fibrous material then passes into the abomasum and acts as an irritant.
2. *Abomasal overload.* Excessive quantities of milk or milk substitute entering the abomasum overloads the digestive enzymes, leading to poor clot formation and "souring" with resulting inflammation. I have seen one such problem resolved following the fitting of teats with slower flow rates. In another recent case, gross overfeeding (2-2.5 liters twice daily, given to 4-day old calves) led to death due to abomasal dilation and ulceration. The problem was resolved by changing to feeding one liter three times daily.
3. *Stress,* leading to changes similar to gastric ulceration in man.

Chronic Diarrhea

There is a well-recognized syndrome of a chronic, pasty diarrhea, which may occur pre- or post-weaning and from which no specific pathogens (e.g. cocci, cryptosporidia etc.) can be identified. Some cases are idiopathic, others may be related to a feed or management change. Clinically the syndrome appears to be a ruminal disturbance and hence may be associated with a number of factors, for example:

- *managemental and other problems* leading to abomasal groove failure, "souring" of the rumen and subsequent poor development.
- *sudden feed changes*
- *excess starch and inadequate fiber in the ration,* leading to poor development of the rumen wall. Many proprietary calf feeds contain nutritionally improved straw (NIS) to stimulate rumen development. I have experienced one case where feeding a home-mix of rolled/ground barley and wheat led to chronic bloat and post-weaning scour for several years. This resolved following the feeding of a proprietary calf ration.
- *unsuitable feeds*, e.g. high intakes of maize gluten to young calves. Remember that the calf is not a full ruminant until it is at least 12 weeks old. Prior to this the rumen is much more acid than in the adult and feeds which further increase ruminal acidity are contra-indicated. The syndrome is sometimes seen on farms where adult dairy concentrates are fed to young calves.

Treatment is often difficult. Badly affected calves should be returned to a whole milk diet, with almost total removal of solid food, and maintained on this for 3-5 weeks, depending on the severity of the condition. The use of a coarse calf ration in such cases is often beneficial. While I accept that trials have shown no significant difference between calf pellets and coarse mix in healthy calves, I have seen occasions where the use of coarse mix appears to have alleviated instances of ruminal bloat or chronic diarrhea. On one farm, where intakes were carefully monitored, calves were found to eat coarse mix rations more slowly than pellets and there was more vigorous chewing. This would promote the flow of saliva, reduce ruminal acidosis and improve overall rumen function.

References

Pritchard RK & Hennessy DR (1981) *Res. Vet. Sci.* 30 22 Reilk RF (1954) *Aust. Vet. J.* 20 29 Scholz H (1990) *Vet. Annual* 3049 Webster J (1926) *"Die Physiologie und Pathologie der Vormagen der Rinde"*, Berlin p. 37.