Abomasal Diseases

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1. Functional Disorders

Functional disorders of the abomasum can be divided into those resulting in any kind of displacement of the abomasum and those only associated with a decreased motility or emptying. The latter are often associated with the term "Hoflund syndrome" or "vagal indigestion". In fact the Hoflund syndrome is not one single syndrome, but it consists of several different syndromes. However, the term "Hoflund Syndrome" is generally used to indicate abomasal functional disorders with decreased motility or emptying and without displacement.

1. 1 Abomasal displacement

1.1.1. Introduction

Abomasal displacement has been recognized since the 1950's in dairy cattle in increasing incidence. In beef cattle it is observed rarely. The incidence is reported to be higher in high producing dairy herds as well as in high producing individual cows, although herds are known with high productions and without any significant problem of displaced abomasums.

Most cases of abomasal displacement are seen during the first 4 weeks after parturition, although occasionally dry cows, calves and even bulls have been reported to have abomasal displacement. Cows aged from 4 up to 7 years show the highest incidence (8) but also heifers are often affected. Although the etiology is still not completely understood, general agreement exists that an atony or hypotony and hence a decreased abomasal emptying rate results in the accumulation of gas and fluid in the abomasum. This gas accumulation consequently results in the abomasum enlarging and floating, either to the left or the right-hand side. If it moves to the left, upward between the rumen and the left abdominal wall, this is called a left-sided displacement of the abomasum (LDA). If the abomasum moves to the right, three main possibilities are distinguished (35):

- The abomasum is dilated without any real rotating movement around an axis and extends caudally and dorsally in the right flank. This is called a simple dilatation of the abomasum.
- If it moves to the right, around a horizontal axis in the median plane, the terms right-sided displacement of the abomasum (RDA) or flexion to the right (in short flexion) are used.
- In part of the cases flexion is complicated by a rotation in an anticlockwise direction around a vertical axis in a sagittal plane through the abomasum. This condition is called flexion-rotation, abomasal torsion or abomasal volvulus.

1.1.2 Etiology and pathogenesis

A large number of factors have been shown to play a role in the etiology and pathogenesis of abomasal displacement. Using different hypotheses as a starting point, studies mentioned in the literature sometimes resulted in different conclusions. On the other hand, some of the etiological factors are obviously related, so that it is often difficult to establish which factor is the real cause.

1.1.2.1. Diet related factors

High concentrate rations in the early post partum period have been shown to play an etiological role (31). These rations result in increased concentrations of VFA in the ruminal fluid. After sudden ration changes, ruminal mucosal papillae need an eight week adaption period to proliferate and develop full absorptive capacity. When high concentrate rations are fed to cows in which the ruminal mucosa is not adapted to these rations, the ruminal absorption of VFA will be insufficient (16). When this ruminal fluid passes on to the abomasum and abomasal concentrations of VFA increases as well, this results in a decrease of abomasal motility and an increase of abomasal gas production (31). However, other investigations falled to confirm that high concentrate rations result in increased abomasal VFA concentrations (4,32), so there is no general agreement at this point.

Lack of crude fiber or structure in the ration has been mentioned (10). Obviously in cows fed for high productions, this factor is hard to distinguish from the above mentioned high concentrate rations. Rations containing less than 16% crude fiber have been shown to be associated with a higher incidence of abomasal displacements. As a possible mechanism for this relationship, it is suggested that lack of crude fiber causes a decreased saliva production, thus a ruminal acidosis.

In recent years, the ration during the dry period has received more attention. High energy rations during the dry period ("steaming up" or "lead feeding"), seem to predispose to abomasal displacement, although some other investigations gave opposing results (5,16).

1.1.2.2. *Environmental factors*

Although abomasal displacement has been reported to occur also in calves, bulls and pregnant cows, by far the

majority of cases occur shortly after calving with a peak of incidence one to three weeks after parturition (8). The relation of abomasal displacement to parturition is subject to a number of hypotheses and investigations.

Independently of parturition, the incidence of abomasal displacement increases towards the end of the stable season and the beginning of the pasture season. The incidence of abomasal displacement is assocaited with high milk yields. Higher incidences are found in herds and individuals with high productions, although herds with high productions and without problems of abomasal displacement are found.

Some investigations have shown that the incidence of abomasal displacement is associated with the incidence of concurrent disorders, mostly inflammatory processes such as milk fever, emdometritis, retained placenta, sole ulcers, arthritis or mastitis. However, other investigators found that the "concurrent disorders" had no increased incidence in cows with abomasal displacement when compared with normal cows in the postparturient period. Assuming a relation between abomasal displacement and concurrent disorders, several investigators have done research into the nature of this supposed relation, however, without success.

1.1.2.3. *Mechanical factors*

Some investigators have approached the etiology of abomasal displacement from a mechanical, rather than a functional point of view. During late pregnancy, the abomasum is pushed forward by the uterus, thus getting more or less S-shaped. Although it is incorrect to speak of "room" in the abdomen, the anatomic relations within the abdomen after parturition permit more movements than normal. Besides, the rumen is often moderately filled soon after parturition. So, depending on the position of the abomasum and food intake before and after parturition, the abomasum can be "trapped" by the bigger and heavier rumen.

In this situation an accumulation of gas in the abomasum will easily result in an upward displacement to the left of the proximal corpus or an upward displacement to the right of the distal abomasal corpus.

1.1.2.4. Genetic factors

In many of the above mentioned etiological factors, the anatomical and physiological characteristics of the individual cow will play a role. These characteristics are at least partly genetically determined. A genetic predisposition to abomasal displacement has been shown in the progeny of certain bulls as compared with other bulls of the same breed (29).

1.1.2.5. Endotoxins

Ruminal motility has been shown to be decreased in endotoxaemia.

As some of the concurrent disases might result in an endotoxaemia, an inhibition of abomasal motility by endotoxins has been suggested as a possible cause. Experimental endotoxaemia did decrease abomasal emptying (37). However, no decrease in plasma Zn concentration, a known consequence of endotoxaemia, was found in DA patients.

1.1.2.6. Histamine, serotonine and prostaglandins

Intramuscular prostaglandin administration causes a slight decrease in abomasal emptying (36). However, administration of prostaglandin inhibitors did not significantly improve the condition of cows with LDA (33, 34).

Polymorphonuclear leukocyte function has been studied in cows with LDA. Phagocytic function was depressed. In cows with LDA and some concurrent infection, phagocytic activity was lower than in those without concurrent infection which showed a depressed intracellular killing in their polymorphonuclear leukocytes. However, this might be a consequence as well as a causative factor. The significance of these findings for the etiology of either abomasal displacement or concurrent infection is not clear.

1.1.2.7. Metabolic factors

Cows with abomasal displacement often show some degree of hypocalcemia (27) which can decrease ruminal and abomasal motility (6) and abomasal emptying (25). This effect is partly due to a pH dependent decrease in ionized calcium concentration but partly independent of pH. Therefore hypocalcemia has been suggested to play a significant role in the etiology of abomasal displacement (25). Other investigation, however, showed that within the range of calcium concentrations, involved abomasal mechanical and myoelectric activity did not decrease. Only with Ca concentrations below ± 1,2 mmol/l a decrease in abomasal activity is found (18).

An influence of extracellular pH directly on abomasal motility and emptying rate has been suggested by Poulsen (24). He found a slight seasonal variation in the acid base balance of dairy cows, resulting in a slight metabolic alkalosis toward the end of the stable season, the period where most abomasal displacements occur. As in experimentally induced metabolic alkalosis, abomasal emptying rate decreases (26). This seasonal variation might contribute to the pathogenesis of abomasal displacement.

1.1.2.8. Fatty liver and insulin resistance

Fatty infiltration of the liver to some extent is often seen in highly productive dairy cows. Cows with abomasal displacement had more severe fatty infiltration than comparable cows without abomasal displacement. A causative ralationship between these two findings is not established and both might be the result of a common factor (13). High energy-low protein rations during the dry period result in fatty livers. The same feeding practice has been recognized

as a factor in the etiology of abomasal displacement.

An interesting theory has been proposed by investigators in Gand (20). They suggested insulin resistance as an important etiologic factor. Cows with abomasal displacement often have increased blood glucose concentrations in the face of high insulin levels. Experimentally, insulin decreased abomasal emptying.

Butyrate in the rumen is metabolised by the rumen epithelium to 3-hydroxybutyrate. In lactating cows, this metabolism is much faster than in dry cows possibly because of the adaptation of ruminal papillae. In cows with abomasal displacement, this metabolism appeared to be slower than the normal postparturient cows, possibly because the rumen is not yet adapted to the changing ration and ruminal papillae are less developed. This could result in an overflow of butyrate to the blood, which leads to an insulin response. Intraruminal infusions of butyrate gave increased insulin levels in dry cows and not in lactating (adapted) cows. The increased insulin levels in cows with abomasal displacement did not result however in hypoglycemia. On the contrary, glucose concentrations were often increased and intravenous glucose tolerance tests showed a decreased glucose clearance in spite of the hyperinsulinaemia; so blood glucose did not respond to insulin any more.

The same authors (20) investigated also the energy status of the liver by means of determination of triglycerides and glycogen in liver biopsies. Compared to the normal peri-parturient cow, cows with abomasal displacement showed sometimes glycogen depletion, often increased triglyceride contents and most often both. Triglyceride contents often were very high, some of them being over 200 mg fat per gram liver. However, a considerable number (25%) showed no abnormalities in the liver biopsies. Acetonaemia was only found in animals with glycogen depletion in their livers.

1.1.3. Consequences of abomasal displacement

Abomasal displacement can cause a variety of clinical symptoms.

Left displacement of the abomasum can occur without any symptoms at all. Normally, left displacement of the abomasum causes only slight discomfort, resulting in decreased appetite. This sometimes intermittent decrease in appetite is the result of abomasal distension, which inhibits reticuloruminal motility, thus causing forestomach distension (8). Animals affected often partly refuse concentrates and only eat hay. Passage of food is only rarely inhibited, although mostly somewhat delayed. Rumen function can be slightly to severely disturbed sometimes with atony and even some tympany.

A typical distention may be seen in the upper left flank. The amount of faeces passed may be decreased and often appear to be dark and oily.

As the animal shows a loss of appetite, food uptake does not meet the requirements for optimal milk production. So, depending on the severity of stomach function disturbance, the milk yield will decrease. The negative energy balance will result in a secondary acetonaemia.

On clinical examination, the displaced gas and fluid filled abomasum causes very typical symptoms, especially when auscultated and percussed.

The consequences of right-sided abomasal displacement (RDA) depend much more on the actual position of the abomasum and on the severity of the disturbed passage of food. In the case of a simple dilatation or a flexion, the consequences may be the same as in LDA: only a slightly decreased appetite and decreased milk yield. The clinical symptoms will then correspond to those in LDA: slight discomfort, a delayed passage of faeces and on auscultation and percussion typical sounds connected with the distended abomasum in the upper right abdomen.

If the abomasal flexion to the right is complicated by a stenosing kink, this will result in an acid base balance disturbance, as seen in the posterior stenosis of the Hoflund syndrome. A hypokalaemic, hypochloraemic, metabolic alkalosis will develop, accompanied by hypovolaemia and even hypovolaemic shock.

In the case of an abomasal displacement to the right with flexion rotation, the consequences may be even more serious. Apart from the developing posterior stenosis signs, the twisting of the abomasum will cause circulatory congestion in the abomasal wall, resulting in necrosis and toxaemia. In advanced cases, blood acid-base balance will show a metabolic acidosis instead of a metabolic alkalosis, probably because of lactic acid formation. These cases usually have a very poor prognosis.

Depending on the severity of the obstruction of abomasal emptying and on the severity of the circulatory consequences, the prognosis of abomasal displacement can vary.

Abomasal displacements to the left and flexions to the right are known occasionally to recover spontaneously. Many recurrencies are also seen. The majority of cases however, will not recover spontaneously and prolonged inappetence or anorexia can result in weight loss and ill-thrift. If abomasal emptying or circulation is impaired, immediate fluid therapy and surgical correction are necessary and any delay will aggravate the prognosis.

1.2. Posterior stenosis of the Hoflund syndrome

The most improtant disturbance of abomasal motility is pyloric stenosis or posterior stenosis of the Hoflund syndrome. As has been mentioned above, the Hoflund syndrome consists of several different syndromes. In contrast with the conclusions of Hoflund in 1940 (12), general agreement nowadays exist that vagal nerve damage does not play an important role in the aetiology of the Hoflund

syndrome. As mechanical obstructions or morphological abnormalities are mostly the cause of posterior stenosis, the term "vagal indigestion" is not very applicable in most of the cases. However, in some cases a vagally mediated inactivity of the abomasum cannot be excluded.

A number of causes are known to result in a decrease or inhibition of the passage of food through the pylorus. Mechanical obstruction can be caused by local peritonitis on the reticulum or on the abomasum by liver abscesses, leucosis, small intestinal strangulation or intussusception, foreign bodies (hair balls, stones) in the pyloric region and by abomasal impaction. LDA rarely causes obstruction, RDA with flexion can often cause obstruction, and RDA with flexion and rotation will always cause obstruction. In 7 to 8 months pregnant cows, occasionally the uterus presses on the abomasum or small intestine, thus causing occlusion. In generalized peritonitis and secondary indigestion due to septicaemia or toxaemia (mastitis, pneumonia, etc...) posterior stenosis can occur as a complicating disorder. Possibly this may be considered as a "paralytic ileus". Finally, large intestinal disorders, such as caecal volvulus, can result to a lesser extent in a posterior stenosis (9, 15).

In posterior stenosis the hydrochloric acid secreted in the abomasum cannot pass on to the duodenum and will accumulate in the abomasum. The overfilling of the abomasum results in abomasal reflux, either by passive backflow or by internal vomiting (15). Eventually chloride will be sequestrated in the abomasum and in the rumen, whereas the loss of chloride from the extracellular fluid results in hypochloraemia, metabolic alkalosis and hypokalaemia. The accompanying fluid loss causes hypovolaemia, prerenal uraemia and eventually hypovolaemic shock. Impairment of the passage of food thus has serious life threatening consequences.

The cow with posterior stenosis shows a very distended abdomen, due to the overfilled rumen and abomasum, both being overfilled with fluid material. The distension of the rumen and abomasum gives the abdomen a typical shape, often referred to as "papple", apple shaped on the left and pear shaped on the right. Splashing sounds are heard on ballottment of the rumen. No faeces are being passed and the cow is dull, weak and dehydrated.

The diversity of causes of posterior stenosis demonstrate that it can hardly be considered as a disease entity and even less as a functional disorder of the abomasum. However, several of the listed causes of posterior stenosis are not easily established on physical examination. Therefore the term "posterior stenosis" is used to indicate the ensemble of clinical signs and events connected with an impairment of the passage of food through the pylorus, irrespective of its actual cause.

1.3 Abomasal Impaction

Abomasal impaction is characterized by a dilatation of the abomasum and an overfilling with rough, structural material like ruminal contents.

Sometimes this is associated with peritoneal adhesions around the abomasum. However, cases without any morphological abnormality at necropsy occur. The etiology in these cases is unknown (19). Sometimes abomasal impaction occurs shortly after surgical correction of RDA with flexion and rotation. This suggests that lesions, possibly of the vagal nerve (12), in the omasal canal or in the abomasum play a role in the etiology. As a consequence, either too much coarse material is transported through the omasal canal to the abomasum or too little coarse material is transported from the abomasum to the duodenum. The result is an enlargement of the abomasum and a further inhibition of abomasal motility.

Depending on the amount of coarse material and its position within the abomasum, the passage of food through the abomasum can be obstructed and a posterior stenosis can occur (15). In that case the disease is acute and life threatening. If the passage of food through the abomasum is not seriously impaired, the result will only be weight loss and emaciation. The lower right hand flank may be distended. Sometimes the solid mass of the abomasum can be palpated in the lower right hand flank or on rectal examination.

2. Inflammatory Diseases of the Abomasum

Two different kinds of inflammatory diseases can be distinguished: abomasitis and abomasal ulcers.

2.1 Abomasitis

Inflammation of the abomasal wall occurs mostly as a result of parasitic or viral infection. In parasitic abomasitis, Haemonchus, Ostertagia and Trichostrongylus species are the causative agents. Viral abomasitis mostly is caused by the Bovine Virus Diarrhoea (BVD) virus. In both parasitic and viral abomasitis, other parts of the gastrointestinal tract will also be affected. The relative significance of Haemonchus, Ostertagia and Trichostrongylus varies regionally.

Abomasal Ostertagia infections are divided into two types. In type 1, the glands of the mucosa in the abomasal corpus are affected. This causes inflammation of the abomasal mucosa and oedematous swelling of the submucosa, visible as nodules throughout the mucosa. Type I Ostertagiosis occurs in the summer and fall. Type II Ostertagiosis, which is often much more serious, occurs in early spring. In type II, the larvae of the fourth stage have been inhibited in the abomasal glands for up to several months and emerge from the mucosa in spring. They then cause severe lesions in the abomasal mucosa and inhibit normal digestion. The pH of the abomasal contents increases from about 2 up to 6 as the result of destruction of mucosa. Con-

sequently pepsinogen is not converted to pepsin. Moreover, more bacteria than normal are found in the abomasum. Thus, abomasal digestion is inhibited, which results in diarrhoea and loss of protein. An additional cause of loss of protein is the damaged abomasal mucosa, where leakage of protein occurs (1).

Haemonchus in many countries is more important in the sheep and the goat than it is in the cow. Haemochus lives in the abomasum and feeds on blood and protein. Consequently the most striking symptom is anaemia. In primary Haemonchus infection, there is no diarrhoea.

BVD affects in a variable degree the whole gastrointestinal tract. The most important clinical manifestation of this disease is Mucosal Disease, mostly found in older calves and yearlings. However, the abomasum is not a very typical, and often not a very severely affected organ. As in parasitic abomasitis, the consequences and clinical symptoms are the result of not only abomasitis, but generalized gastro-enteritis. Chronic weight loss, emaciation, diarrhoea, and hypoproteinaemia are observed, eventually leading to dehydration and acid-base balance disturbances which may be fatal. Whereas in parasitism treatment with anthelminthics, if administered in time, can be successful, the prognosis of Mucosal Disease is unfavorable.

2.2 Abomasal Ulcers

2.21 Introduction

Gastric peptic ulcers occur in many different species. The abomasal ulcers found in ruminants show many similarities with peptic ulcers of monogastric animals.

The aetiology of both gastric and abomasal ulcers has not yet been solved, although several hypotheses have been proposed.

The prevalence of abomasal ulcers of various stages in slaughtered dairy cows varies regionally and can amount to over 5% (3).

The lesions found in the abomasal wall may vary considerably. Slight superficial mucosal necrosis may occur and may result in erosion. Erosions do not penetrate the muscularis mucosae and heal by epithelial regeneration without scar formation. They are usually small, circular and multiple.

In contrast, ulcers penetrate the entire thickness of the mucosa and may extend through the submucosa, muscularis externa and serosa (30). Ulcers vary in size, but they are generally larger than erosions and more irregular in shape. A healing ulcer results in scar formation (28,30). Depending on the tissues penetrated by an ulcer, it may result in abomasal hemorrhage, when an artery is affected, or in perforative peritonitis, which either may remain localized due to the formation of adhesions or may expand and result in diffuse peritonitis.

In cattle, abomasal ulcers are found in adult dairy cattle (3, 22, 23), in veal calves (7) and in feedlot beef cattle (14).

The localization of the lesions within the abomasum in adult cattle is different from that in calves. In adult dairy cattle, most ulcers are located along the greater curvature of the abomasal wall and along the smaller curvature of the pyloric part. Predominantly, those along the greater curvature of the abomasum result in bleeding ulcers. In the veal calf, most ulcers are located in the pyloric part of the abomasum and even in the pylorus itself.

2.2.2 Etiology and pathogenesis

Although the etiology of abomasal ulcers is not clarified, a number of hypotheses have been proposed, some of them based upon observations in monogasatric animals.

Various stress factors have been reported to be associated with the development of abomasal ulcers. Stress related to parturition, such as advanced pregnancy, early lactation and attainment of peak milk production, or recent parturition itself have been mentioned (14, 23, 28). However, contradictory results have been reported and season or weather related incidence, independent of parturition, have been found (3). Concurrent diseaes, mostly common periparturient diseases such as metritis, mastitis, ketosis, fatty liver or abomasal displacement, have been reported and might be an additional stress factor.

Normally, the gastric mucosa is a barrier against digestion of the gastric wall by the secreted acid and pepsin. Hypersecretion of acid, pepsin or both, or locally impaired mucosal resistance can result in the protein of the gastric wall being digested. This is called a peptic ulcer.

Abomasal hypersecretion can be caused by increased levels of corticosteroids due to stress (21). Increased abomasal volume and VFA content also stimulate secretion (2,11). So diet related factors can influence secretion and cause eventual hypersecretion.

Locally impaired mucosal resistance possibly is more important than hypersecretion, but it is more difficult to establish and to explain. Several factors have been suggested to cause mucosal damage. Ruminal lactic acidosis may damage the abomasal mucosa. Focal circulatory disturbances or ischaemia of the mucosa, possibly due to stress, have been suggested. The role of fungi and bacteria, especially spirilla, in this respect is still not known.

The localization of the majority of ulcers in the adult cow along the greater curvature of the abomasum suggests that small traumatic lesions by food particles, straw, etc...may play a role (3). In veal calves, the rapid consumption of large volumes of milk replacer causes excessive contractions of the pyloric part during a rather long period of time, thus resulting in local ischaemia and focal erosions (7), possibly aggravated by straw and rough material if the calves have access to this.

The mucosa of the abomasum is covered by a layer of mucin. In this mucous layer the acid from the abomasal secretions is buffered, thus creating a gradient of decreas-

ing acidity from the lumen to the mucosa and protecting the mucosa. The amount of epithelial mucins on the mucosa of normal abomasums varies. In abomasums of calves with ulcers, a similar variation was found outside areas showing lesions. The epithelium bordering on mucosal lesions was frequently completely depleted of mucins, with peripherally a gradual increase. This suggests an inadequate mucous barrier (17).

Consequences:

Depending on the localization and depth of the ulcer the consequences may vary. Four different situations are distinguished (28):

- Non-perforating ulcer, with incomplete penetration of the abomasal wall, resulting in a minimal degree of intraluminal haemorrhage, focal abomasal thickening or local serositis. Mostly these animals do not show any clinical sign at all and their general condition is only slightly affected. If there is some blood loss, this may cause melena and anaemia. Sometimes there is some abdominal pain, but this is a very aspecific symptom. In most cases, this diagnosis is made only by chance during surgery (for instance because of a concurrent abomasal displacement) or at necropsy.

- Ulcer causing severe blood loss. If the wall of a major abomasal blood vessel is penetrated, usually in the submucosa, this will result in severe intraluminal haemorrhage. Mortality rate in these patients is high and even after recovery, many recurrencies occur. In acute cases, a cow shows some discomfort and abdominal pain. Pale mucous membranes and a pale udder may be noticed. After some hours, melena will appear. The pulse rate will increase, due to circulatory disturbances and hypoxic or hypovolaemic shock will often be fatal.

- Perforating ulcer with acute localized peritonitis. The full thickness of the abomasal wall is perforated, resulting in leakage of abomasal contents. The resulting peritonitis is localized to the region of the perforation by adhesion of the involved portion of abomasum to adjacent viscera, omentum or peritoneum.

- Perforating ulcer with diffuse peritonitis. In these cases the peritonitis is not localized to the region of the perforation, thus digesta are spread diffusely throughout the peritoneal cavity.

The difference between 3 and 4 is in the peritoneal response to the leakage of abomasal contents, this response in type 4 ulcers being inadequate to contain the peritonitis. The mortality rate in cases of localized peritonitis is about 50%. If the peritonitis is diffuse, this is always fatal.

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Abstracts:

Prophylactic medication of feedlot calves with tilmicosin

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Veterinary Record (1991) **128,** 278-280

The parenteral administration to calves of the antibiotic tilmicosin either on arrival at a feedlot or 72 hours later was evaluated in a group of 308 steer calves. The calves were allotted to 24 pens so that there were eight replicates of the two medicated groups and eight replicates of the control group. The need for veterinary treatment was reduced significantly ($P < 0.05$) during the first month of the feeding period in the two medicated groups. The medicated groups had an improved average daily weight gain ($P < 0.01$) over the trial period compared with the non-medicated animals. This improved average daily gain by the medicated groups was not reduced when animals with respiratory disease were excluded from the calculations. The medicated groups also had an improved feed conversion efficiency ($P < 0.01$) over the first 60 days of the feeding period compared with the non-medicated animals.

Resection of the distal interphalangeal joint in cattle: an alternative to amputation

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A technique for the resection of the distal interphalangeal joint of cattle with infectious arthritis of the joint is described. The procedure has advantages over amputation of the digit because a weight-bearing claw is retained so that the animal can move on slatted floors and at pasture, and its productive life can be extended.