

# Surgery of the Bovine Large Intestine

Mireille Meylan, Dr med vet, FVH, MS, PhD, Dr habil

## KEYWORDS

- Cecal dilation • Atresia • Colon • Malformation
- Field surgery • Cattle

This article consists of a short review of the anatomy of the bovine large intestine, and a more thorough description of the two most relevant diseases of the large intestine amenable to field surgery: cecal dilatation and dislocation (CDD) in cows and intestinal atresia in calves. Rectal prolapse is addressed in another article in this issue.

In disorders affecting the large intestine, as in any other disease, a complete history and careful physical examination are prerequisites for successful treatment, which includes not only surgical intervention but also pre- and postoperative medical treatment. The clinical examination of cattle that have abdominal disease has been described in detail.<sup>1-3</sup>

## ANATOMY OF THE BOVINE LARGE INTESTINE

The bovine large intestine consists of the cecum, the ascending, transverse, and descending colon, and the rectum (**Fig. 1**). The cecum usually lies in the supraomental recess with its blind end directed caudally toward the pelvis. It is connected ventrally to the ileum by the ileocecal fold, which contains blood vessels and nerves, and dorsally to the proximal loop of the ascending colon (PLAC) through the short cecocolic band. At the ileocecolic junction, the cecum continues as the PLAC without visible transition. After extending cranially to the level of the 12th rib, the PLAC turns back laterally until it reaches the caudal flexure of the duodenum. There, it turns around cranially and continues as the spiral colon (spiral loop of the ascending colon), which is embedded in its mesentery in the form of a flat oval. The spiral colon consists of centripetal and centrifugal coils (with 1.5 to 2 revolutions each). The distal loop of the ascending colon runs first in a caudal, then in a cranial direction, and leads to the descending colon over the short transverse colon, which crosses from the right to the left side of the abdomen in front of the cranial mesenteric artery. The rectum lies mostly in the peritoneal part of the pelvic cavity.<sup>4-7</sup>

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Clinic for Ruminants, Vetsuisse Faculty of the University of Berne, Bremgartenstrasse 109A, 3012 Berne, Switzerland

E-mail address: [mireille.meylan@knp.unibe.ch](mailto:mireille.meylan@knp.unibe.ch)

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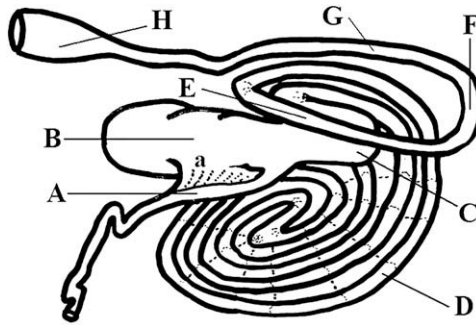


Fig. 1. The bovine large intestine. A, ileum; B, cecum; C, proximal loop of the ascending colon; D, spiral loop of the ascending colon; E, distal loop of the ascending colon; F, transverse colon; G, descending colon; H, rectum; a, ileocecal fold. The cecocolic fold between the cecum and the proximal loop of the ascending colon is not visible under the distal loop of the ascending colon.

## CECAL DILATATION AND DISLOCATION

### *Clinical Presentation and Diagnosis*

CDD is a common and economically important abdominal disorder that affects mainly dairy cattle. In an epidemiologic study in Switzerland, similar prevalences (0.05% at individual cow level) were found for CDD and abomasal displacement.<sup>8</sup> The pathologic features of the condition include distention, displacement, and, in some cases, retroflexion or torsion of the cecum and proximal portion of the colon, leading to partial or complete obstruction of the passage of digesta. A retroflexion of the cecum is defined as a displacement of the cecal apex from its normal position toward the pelvic inlet in a cranial direction. A cecal torsion is present if a twist of the cecum occurs along its longitudinal axis, leading to increased tension on the ileocecal fold, thus jeopardizing blood circulation and innervation of the organ. Cecal torsion occurs less frequently than CDD with or without retroflexion.<sup>7</sup>

The clinical presentation of the disease has been described in numerous clinical reports.<sup>3,7,9-16</sup> Typical clinical signs include acute onset of mild colic, a normal to moderately elevated heart rate, decreased appetite, reduced rumen motility, decreased or absent fecal output, distention of the right flank, and positive succussion and percussion auscultation of the right flank (in the dorsal part of the paralumbar fossa, over an area extending caudally to the tuber coxae). In a retrospective study of 111 cases, rectal examination was considered the most reliable diagnostic investigation for CDD, with swinging and percussion auscultation of the right flank.<sup>16</sup> In that report, the distended, displaced, or twisted cecum could be palpated at rectal examination in 95% of the affected animals. In the case of a simple dilatation, the cecal apex is easily recognizable as a rounded, dome-shaped structure, with a diameter of approximately 15 cm, extending into the pelvic inlet. If a retroflexion is present, the body of the cecum, but not the apex, can be palpated as a large, rounded, tense structure in front of the pelvis; rectal palpation alone does not always allow for differentiation of cecal dilatation with retroflexion from right abomasal displacement or volvulus. Cecal torsion can often be identified on rectal examination: in addition to the typical dilated cecal apex, the ileocecal fold is palpable as a taut, twisted, and painful band. Dilated loops of small intestine and of the spiral colon are often encountered on rectal examination of animals that have CDD.<sup>15-17</sup> The appearance of the dilated cecum at ultrasonography from the right flank has been described.<sup>18,19</sup> Blood

chemistry values and acid-base status are mostly normal in cows that have CDD. Changes similar to those seen with obstruction of the small intestine (hypokalemic, hypochloremic metabolic alkalosis) can be seen in protracted cases, or if retroflexion or torsion of the cecum is present.<sup>15,20</sup> In one study, hypocalcemia was observed in 48% of 79 affected animals.<sup>20</sup> In the same study, elevated packed cell volume values indicating circulatory compromise were present in only 13% of 86 patients. The concentration of chloride ions in rumen fluid was normal (<25 mmol/L) in 83% of 93 animals that had CDD, whereas it was elevated in the remaining 17%.<sup>20</sup>

Although an association between the occurrence of CDD and season, age of the animals, stage of pregnancy, or time after parturition has not been demonstrated, some investigators report a trend to more frequent occurrence of the disease during the fall and winter months.<sup>11,14–16</sup>

The main differential diagnoses for CDD include right displacement of the abomasum or abomasal volvulus, small intestinal ileus, complete or partial volvulus of the mesentery, or other causes of abdominal pain (eg, pyelonephritis).<sup>11,16,21</sup>

### ***Etiopathogenesis***

Despite numerous clinical descriptions of CDD, the pathogenesis of the disease remains poorly understood. In the past, atony or dysmotility of the cecum and PLAC, leading to accumulation of gas and digesta, and followed by dilatation and secondary displacement of the cecum, has been hypothesized to be of primary relevance in the pathogenesis of CDD.<sup>11,12,21,22</sup> However, the patterns of myoelectric activity observed in the cecum and PLAC of cows that showed delayed recovery or recurrence of CDD after surgical correction presented a striking similarity to those recorded immediately orad to a mechanical obstruction in the distal PLAC of an experimental cow. These observations led to the hypothesis that decreased motility preventing normal transit of ingesta might be present not in the cecum itself but in the spiral colon.<sup>23,24</sup>

Alterations in mRNA expression of receptors involved in the regulation of gastrointestinal motility, such as adrenergic,<sup>25</sup> serotonergic,<sup>26</sup> and muscarinic<sup>27</sup> receptors, have been reported in the ileum and large intestine, especially the spiral colon, of cows that have CDD, but it remained unclear whether the observed changes indicate that these receptors are involved in the pathogenesis of the disease or if they are a bare consequence of cecal dilatation.

Elevated concentrations of volatile fatty acids (VFA) in intestinal contents, as observed in cattle fed carbohydrate-rich diets, have been suspected of inhibiting gastrointestinal motility. Total VFA concentrations similar to those in the rumen have been reported for the bovine large intestine.<sup>22,28</sup> Cecal-colonic fermentation accounts for 8.6% to 16.8% of total VFA production in sheep,<sup>29</sup> and the efficiency of the large intestine for VFA production is similar to that of the rumen.<sup>30</sup> Starch resistant to microbial digestion in the rumen (bypass starch) is partially digested enzymatically in the small intestine and partially fermented in the large intestine, the rest being excreted in the feces. Corn starch, especially, is fermented much more slowly than starch from other cereals, thus resulting in large proportions of bypass starch and increased postruminal fermentation.<sup>22,30</sup> When increased amounts of fermentable carbohydrates reach the large intestine, more VFA are produced there by microbial fermentation.<sup>30,31</sup> Several investigators have reported the effects of increased VFA concentrations or high-concentrate diets on ruminant gastrointestinal motility. However, their results are contradictory, with five studies<sup>22,32–35</sup> reporting inhibition of motility with increased VFA levels and three investigators<sup>36–38</sup> being unable to detect an effect on motility parameters. Nevertheless, VFA concentrations were found to be

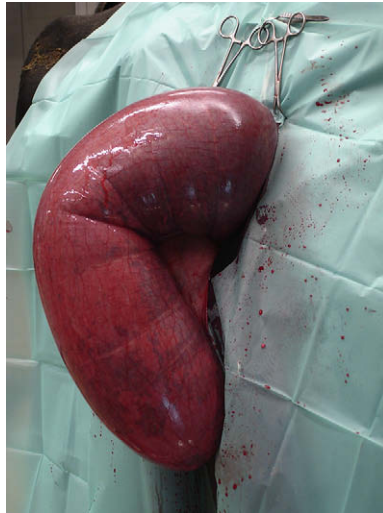
elevated in the contents of cecum, PLAC, and rectum in cows that had spontaneous CDD, as compared with healthy control cows, but it remained unclear if this elevation of VFA was the cause or the consequence of reduced motility and stasis of digesta.<sup>39</sup> In contrast, comparison of the contractility of ex-vivo intestinal wall specimens from the cecum and spiral colon of healthy cows with or without preincubation with VFA did not reveal significant differences.<sup>40</sup> An abrupt increase of concentrates in the diet of healthy dairy cows (from 0% to 50% of the ration over 60 hours) did cause a significant increase of VFA concentrations in large intestinal ingesta, but no relevant inhibition of the myoelectric activity of the spiral colon was observed.<sup>41</sup> In conclusion, elevated VFA concentrations do not appear to play a predominant role in the regulation of bovine intestinal motility or in the pathogenesis of CDD.

Several cows with CDD have been referred to the author's clinic for treatment during outbreaks occurring in herds after introduction of rumen buffers, but these cases could not be documented in detail and a possible role of rumen buffers in the pathogenesis of CDD remains speculative.

### **Therapy of Cecal Dilatation/Dislocation**

Various approaches to the treatment of CDD have been reported. According to earlier reports, attempts at conservative therapy of CDD were first considered useless or even contraindicated.<sup>10,11,21</sup> It is now accepted that in the early stages of the disease, when the general condition of the affected animal remains good, when some feces are still passed, and in cases where a torsion or retroflexion of the cecum can be ruled out by physical examination, conservative treatment with prokinetic drugs, purgatives, or intravenous infusions may be attempted.<sup>14,42</sup> Administration of neostigmine (87.5 mg in 5 or 10 L of isotonic saline solution supplemented with 5% dextrose as a continuous intravenous infusion at a rate of 1–2 drops per second)<sup>42,43</sup> or of bethanechol (0.07 mg/kg subcutaneously, every 8 hours for 2 days)<sup>44</sup> have been advocated. Paraffin oil (3 L) or sodium sulfate (300 g in 10 L water) administered per stomach tube have been described as adequate purgatives for conservative or postoperative treatment of CDD.<sup>14,42</sup>

In more advanced cases, if no improvement occurs on medical treatment, or when a retroflexion or torsion of the organ is suspected, surgical correction of the condition by means of a right flank laparotomy, allowing drainage of the cecum and PLAC contents, is the therapy of choice (see Refs.<sup>7,11,13,15,21,42</sup>). The intervention is best performed under local anesthesia of the right flank (eg, using a proximal or distal paravertebral block) with the animal standing. Positioning the animal in left recumbency with or without general anesthesia, while preventing the risk of the patient lying down during surgery, renders manipulation of the dilated intestine more difficult because the weight of the filled cecum pulls it away from the incision to the left (down) side of the abdomen. After surgical preparation and local anesthesia, a 25- to 30-cm incision is made either vertically in the middle of the right paralumbar fossa or slightly more caudally in a caudodorsal to cranioventral direction. The abdomen is opened in a routine manner. After exploration of the abdominal cavity, an additional plastic drape is added to minimize contamination of the sterile draping around the incision, and the apex of the cecum is exteriorized (**Fig. 2**), which is best achieved by gently pushing the cecum from behind toward the body wall with the palm of the hand. Grasping the cecal wall and pulling toward the incision should be avoided because it bears an increased risk for rupture of the distended cecum. The apex is fixed on both sides by the assistant surgeon with two sterile gauzes, incised over approximately 4 cm, and the contents of the cecum are drained. The ingesta in the exteriorized part of the cecum normally flow freely out of the incision (unless



**Fig. 2.** The dilated cecum is exteriorized through a right flank incision for drainage of its contents.

they are especially dry) and additional material is massaged outside of the abdominal cavity from the rest of the cecal body and the PLAC (**Fig. 3**). When no additional material can be removed, the incision is closed with an inverting pattern (Cushing or Lembert) using absorbable material (eg, 2-0 polyglyconate or polyglactin 910). The cecal apex is copiously flushed with warm saline solution and replaced in the abdomen. In some cases, intestinal contents flow into the cecum from the distal small intestine or from the colon after reposition. If the cecum fills again with a large volume of ingesta, the apex is exteriorized and the suture is reopened to allow for further



**Fig. 3.** A stab incision of the cecal apex allows for drainage of its contents. Contents from the cecal body and from the PLAC are massaged in the direction of the cecal apex.

removal of cecal contents. It may be necessary to repeat this procedure several times, but usually not more than once or twice. Contents of the spiral colon often do not flow back into the cecum and are eliminated as feces when large intestinal motility returns after decompression of the cecum. When the cecum remains empty after reposition in the abdomen, a second layer of suture is added using the same pattern to close the cecal apex definitively (**Fig. 4**). After extensive flushing, the cecum is repositioned in the abdomen in its normal anatomic position with the apex directed caudally. The abdominal cavity is closed routinely.<sup>45</sup>

Postoperative care includes antibiotics for 5 days (eg, 30'000 IU Na-penicillin intravenously three times a day), anti-inflammatory drugs as needed (eg, flunixin meglumine, 2 mg/kg intravenously once daily for 1 or 2 days), and infusions of saline or polyionic solutions according to the results of blood chemistry analysis or to the expected changes (hypochloremia, hypokalemia, alkalosis). Under field conditions, the use of a penicillin product with depot effect (eg, procaine penicillin) requiring only one daily application may be preferred, and fluid replacement can be performed through drenching of 20 to 30 L of salt solution into the rumen. Restrictive feeding as part of the therapy for CDD has been described.<sup>14,42</sup> At the author's clinic, no feed is offered on the day of surgery for correction of CDD (day 1), 25% of the calculated ration is offered on the second day, 75% on the third day, and 100% from the fourth day onwards. The animals should be monitored clinically for signs of recurrence for several days after surgery, with normalization of fecal output and rumen activity together with the absence of dilated intestines at rectal palpation indicating normal recovery from CDD.<sup>14,42</sup>

Partial typhlectomy is recommended if the cecal wall is devitalized (eg, after cecal torsion) or if CDD recurs after a first surgical intervention.<sup>7,12,15,43</sup> In such cases, drainage of cecal contents is performed as described earlier, but when the cecum is empty, it is exteriorized as far as possible through the flank incision and an amputation is performed. First, the ileocecal fold is infiltrated with 2% lidocaine to minimize pain at manipulation and thus the risk of the animal trying to lie down. Mass ligatures (eg, with overlapping simple interrupted sutures, using USP 2 chromic catgut or USP 1 polydioxanone) are used to prevent hemorrhage from the vessels in the ileocecal fold. The ligatures are placed near the cecum in order to preserve the vessels supplying the ileum. The ileocecal fold is dissected from distal (cecal apex) to as far



**Fig. 4.** The cecal apex has been rinsed and closed with a double Cushing suture using 2-0 polyglyconate.

proximal as would be safe to remove the cecal apex without causing abdominal contamination. In the chosen location, two intestinal clamps are placed from the mesenteric and the antimesenteric side of the cecum, through the entire width of the cecal body. The distal part of the cecum is resected 2 to 3 cm distally from the clamps, and the incision is closed in two inverting layers (Cushing or Lembert, with 2-0 polyglyconate or polyglactin 910). The stump is flushed thoroughly with warm saline solution and replaced in the abdominal cavity, which is closed in a routine manner.<sup>7,43</sup> A method with staple closure of the stump was compared with double inverting suture, but the results were not significantly different,<sup>46</sup> and the use of stapling devices in food animals is mostly cost prohibitive. Postoperative care for typhlectomy is the same as that described for typhlotomy.<sup>42</sup> In one study,<sup>43</sup> the long-term prognosis after partial typhlectomy was not better than after simple typhlotomy. Thus, the technically more complicated cecal amputation, resulting in prolonged surgery time and increased degree of contamination, is not recommended as a routine treatment of CDD.<sup>15,43</sup>

Total typhlectomy including removal of the ileoceceocolic junction has been performed in experimental<sup>17</sup> and clinical settings,<sup>47</sup> and partial resection of the PLAC has been described;<sup>48</sup> however, these complicated procedures normally require general anesthesia and are unsuitable for treatment of CDD under field conditions.<sup>7</sup>

### **Prognosis**

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Except for the risk for recurrence and for peritonitis due to devitalization of the intestinal wall or abdominal contamination at surgery, the prognosis of CDD is considered to be reasonably good, with reports of approximately 90% of referred cases being discharged in good general condition after treatment.<sup>11,42,43</sup> Up to 10% of treated animals will show recurrence of the disease within the first week after typhlotomy and another 12.5% within the first year after surgery.<sup>15,43</sup>

### **SURGERY OF THE BOVINE COLON**

Difficult mobilization and exteriorization of the bovine colon preclude extensive surgical interventions under field conditions. Intussusceptions involving the large intestine (colon or cecum) have been reported predominantly in young animals.<sup>49-52</sup> This condition is presumed to be more common in calves than in adult animals because of the thin colonic mesentery of young animals that allows for more extensive movement of adjacent intestinal segments, in contrast to more extensive mesenteric fat deposits, which stabilize the large intestine in adults.<sup>49,51,53</sup>

Trichobezoars, phytobezoars, and enteroliths, although they mostly lead to disease in the rumen, abomasum, or small intestine, can also cause obstruction of the bovine spiral colon. Clinical signs are similar to those of CDD and a definitive diagnosis is made on abdominal exploration through right flank celiotomy. In such cases, the affected intestinal segment is exteriorized and opened, and the bezoars removed. The enterotomy site is closed with two inverting continuous sutures (Lembert or Cushing [eg, with 3-0 or 2-0 polyglyconate]) apposed transversally if necessary to maximize lumen and prevent stricture formation.<sup>54</sup> Colonic obstruction may further be caused by extraluminal compression (eg, due to adhesions from prior surgery or to mesenteric fat necrosis) (**Fig. 5**). Bypass of the obstruction by side-to-side anastomosis of the bowel proximal to the obstruction and the outer loop of the spiral colon has been described.<sup>7</sup> Two cases of obstruction of the descending colon caused by cecal incarceration in a mesocolic defect in cows have also been described.<sup>55,56</sup>

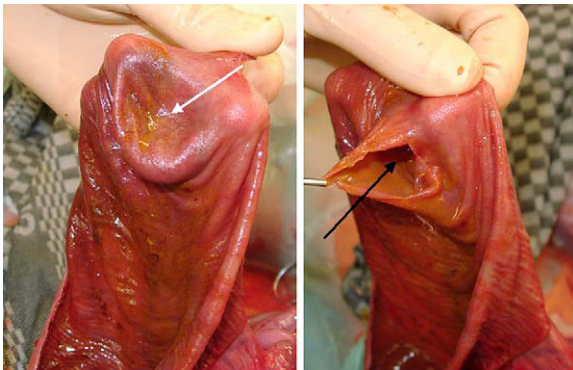


**Fig. 5.** Loops of the spiral colon embedded in a mass of necrotic fat, which had caused large intestinal obstruction in an Angus cow.

### ATRESIA OF THE LARGE INTESTINE

#### *Clinical Presentation and Diagnosis*

Atresia of the large intestine (ie, complete occlusion of the intestinal lumen due to abnormal development of the intestinal wall) is a common congenital defect in calves, and numerous descriptions can be found in the literature. This malformation is most commonly seen in the colon of calves, but atresia recti and ani are also of clinical relevance. Atresia of the small intestine, especially of the jejunum, has also been described but is distinctly less common than large intestinal atresia.<sup>57–64</sup> Colonic atresia, mostly encountered in the spiral part of the colon,<sup>53,58–60</sup> has been classified into four types in humans. In type I, the ingesta passage is only hindered by a fold of mucosa occluding the lumen of an otherwise normally developed intestinal canal (**Fig. 6**). In contrast, in type II, the proximal part of the colon ends blind and is connected to the distal segment by a fibrous band devoid of lumen. Type III is similar to type II, but the proximal and the distal intestinal segments are completely separated and a mesenteric defect is present at the level of the atretic segment. Type IV, with



**Fig. 6.** Type I atresia in the intestine of a 3-day-old Holstein calf. The lumen is completely obstructed by a membrane, the proximal part of the intestine is greatly dilated (*left, white arrow*). After the membrane has been cut open, the lumen of the distal part of the intestine is visible beyond the membrane (*right, black arrow*).



several sites of atresia, is described by some investigators as occurring only in humans.<sup>53,60,63,65-67</sup>

Calves that have large intestinal atresia are typically presented to a veterinarian at the age of 2 or 3 days (although presentation of calves as old as 11 days has been reported), with a history of having been alert and nursing well immediately after birth, but showing decreasing appetite, increasing abdominal discomfort and distention, and progressive weakness after 24 to 36 hours of life. The calves' owners often report that defecation has not been observed since birth. At physical examination, these animals show signs of colic (restlessness, saw-back stance, kicking toward the abdomen), a distended abdomen, an elevated heart rate, positive percussion and succussion auscultation of the right flank, and neither spontaneous nor provoked defecation. Whereas the absence of an anal opening is immediately visible at physical examination of calves that have atresia ani (**Fig. 7**), the rectum is empty at palpation or contains only mucus in colonic atresia, or can be obstructed at a variable distance from the anus if rectal atresia is present.<sup>57,59-64,68</sup> The dilated rectum protrudes in the perineal region at abdominal palpation in the case of anal atresia alone.<sup>57,61,64</sup> Ultrasonographic examination of the right flank, showing dilated loops of small and large intestine without contractions or movement of ingesta, confirms the diagnosis of ileus (**Fig. 8**). In protracted cases, dilated loops of intestine can be detected by adspersion and manual palpation of the right flank.<sup>62</sup> Attempts to confirm a diagnosis of colonic atresia by introducing a catheter from the rectum into the colon to localize the obstruction site should be discouraged because of the risk for perforating the fragile rectal wall of neonates.<sup>2,62,63</sup> Abdominal radiography confirms gas distention of the intestine but even contrast radiography usually does not allow one to identify the atretic area.<sup>61,62,68</sup> Definitive confirmation of the diagnosis occurs at exploratory celiotomy (**Fig. 9**).<sup>59,61,64,68</sup> Because atresia ani is often observed in combination with a malformation or malformations of other parts of the large intestine, a careful, complete physical examination must be performed. Furthermore, if multiple defects are suspected (eg, if abdominal palpation does not induce perineal bulging, suggesting atresia recti in addition to atresia ani), detailed examination of the intestine through celiotomy is indicated before surgery for anal reconstruction.<sup>57,61,64</sup> In some cases of anal atresia in female animals, a rectovaginal fistula develops that allows for defecation through the vagina. In these cases, the animals may be presented much later to the veterinarian because the malformation does not become evident immediately after birth.<sup>57,61</sup>

Hematologic and blood chemistry panels may be normal or may indicate dehydration (elevated packed cell volume or, in advanced cases, signs of prerenal uremia),



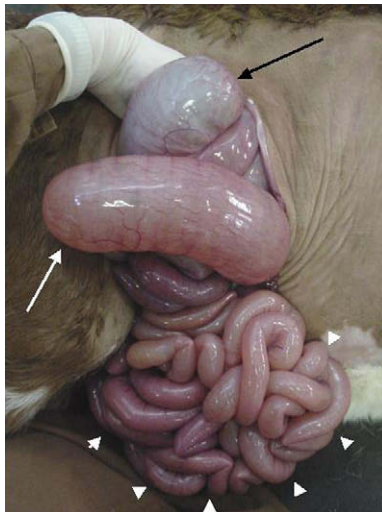
**Fig. 7.** Atresia ani in a Red Holstein calf.



**Fig. 8.** Typical ultrasonographic findings in the right flank of a calf that has atresia coli, showing dilated small intestines and increased abdominal fluid ventrally (*left*), and dilated loops of large intestine with a cuboidal shape dorsally (*right*). (Courtesy of A. Steiner, Dr med vet, FVH, MS, Dr habil, Berne, Switzerland.)

intestinal obstruction (as manifested by hypochloremic, hypokalemic metabolic alkalosis), or in the case of cardiovascular shock or intestinal devitalization, signs of toxemia and metabolic acidosis. Leukocytosis with neutrophilia and left-shift is commonly observed in calves that have atresia coli, but no correlation is evident between white cell count and outcome.<sup>62</sup> Hypogammaglobulinemia is a common finding in calves that have atresia coli, because colostrum ingestion does not necessarily result in absorption of immunoglobulins in calves that have intestinal obstruction.<sup>2,53,59–62</sup>

Differential diagnoses for large intestinal atresia include atresia of the small intestine and, at least in theory, other causes of ileus. However, when the typical combination of anamnesis and clinical presentation described above is present, intestinal malformation must be considered the most likely diagnosis until proved otherwise. A case for



**Fig. 9.** Atresia coli in a 4-day-old Red Holstein calf with the dilated cecum (*white arrow*), the blind end of the dilated proximal part of the spiral colon (*black arrow*), and dilated small intestine loops (*white arrowheads*). (Courtesy of A. Steiner, Dr med vet, FVH, MS, Dr habil, Berne, Switzerland.)

which another cause of ileus would have been found on explorative surgery has not been documented at the Clinic for Ruminants of the Vetsuisse Faculty of Berne during the last 20 years.

### ***Etiopathogenesis***

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In humans, hereditary factors leading to failure in fetal intestinal development and disruption of the blood supply to the affected portion of the intestine have been discussed as potential causes of intestinal atresia. Intestinal atresia has been reproduced experimentally in dogs and rabbits through in utero ligation of mesenteric vessels.<sup>65</sup> In calves, however, the pathogenesis of intestinal atresia is unknown.<sup>61,64,69,70</sup> The proximal blind end of the colon was examined histologically after surgical resection in nine calves, but no consistent alteration of the intestinal wall was observed.<sup>60</sup> Genetic and traumatic causes have been discussed for intestinal atresia in calves.<sup>58,63,69,71</sup> A genetic component appears to play a role in certain breeds,<sup>71–73</sup> but a report of atresia coli in only one of two identical Simmental twins does not support the hypothesis of a genetic cause, at least in that case.<sup>74</sup> Planned mating experiments with putative carrier dams and sires were suggestive of an autosomal recessive mode of inheritance in the Holstein breed.<sup>72</sup> On the other hand, early pregnancy diagnosis after 35 to 42 days of gestation through palpation of the amniotic vesicle has been suspected of causing disruption of the embryonic vasculature, leading to atresia of intestinal segments.<sup>61,62,69,71,72</sup> Several investigators have observed higher frequency rates of intestinal atresia in calves if amniotic palpation for pregnancy diagnosis had been performed on their dams;<sup>58,71,75</sup> however, in one case, palpation had taken place not before 40 days but between 55 and 90 days of gestation.<sup>62</sup>

### ***Treatment***

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Anal reconstruction for atresia ani can be performed under epidural anesthesia, with or without sedation. After surgical preparation of the area, a 2.5- to 3-cm-diameter circular piece of skin is removed 4 to 6 cm ventral to the tail, where bulging of the perineum is observed at abdominal palpation. Subcutaneous tissues are prepared bluntly until the meconium-filled rectum can be identified and mobilized. The rectal submucosa is first sutured to subcutaneous perineal tissue in a simple interrupted pattern (2-0 polyglactin 910 or polydioxanone), starting with 4 sutures at 180° and 90° to ensure accurate circumferential alignment. Further adaptation sutures are set subcutaneously as needed and finally, a circular piece of the rectal blind end is excised approximately 1 cm central from the sutures. Skin and full-thickness rectum wall are then apposed in a simple interrupted pattern (0 or 2-0 polypropylene).<sup>53,61,64</sup> Skin incision using a cruciate pattern (instead of resection of a circular piece of skin) leads to anal stricture and is not recommended.<sup>61</sup>

A tentative diagnosis of atresia coli must be confirmed through explorative celiotomy. If the owner refuses surgery, affected calves must be euthanized immediately, before the condition leads to death through autointoxication, circulatory failure, or fecal peritonitis following intestinal rupture. Conservative treatment should not be attempted.

For surgery, the calves are positioned in left lateral recumbency and the right flank is prepared surgically. Depending on the planned intervention (exploratory laparotomy only for diagnosis confirmation before euthanasia, colostomy, or anastomosis of the intestinal blind end to the descending colon), the right paralumbar fossa is anesthetized (eg, with an inverted L-block) or general anesthesia is performed. In the author's opinion, sedation or general anesthesia are not necessary and not recommended for exploratory celiotomy in neonates that have pre-existing cardiovascular compromise,

but inhalation anesthesia has been described as the method of choice if anastomosis is to be attempted.<sup>61–63</sup> Preoperative care includes the administration of antibiotics (eg, Na-penicillin, 30,000 IU/kg or a broad-spectrum antibiotic) and intravenous fluids to correct acid-base and electrolyte abnormalities, and non-steroidal anti-inflammatory drugs (eg, flunixin meglumine, 2 mg/g intravenously) as needed. The abdominal cavity is opened routinely<sup>2</sup> but with special caution because the abdominal wall of neonatal calves is thin and, in the case of intestinal atresia, additionally distended by the dilated intestine lying immediately underneath the peritoneum in the right flank. Puncture and gas aspiration for decompression of the intestine may be necessary before exploration of the abdomen can be performed.<sup>62</sup> Colonic atresia is easily diagnosed when a second dilated blind end (of the atretic colon) is found in addition to the dilated cecum (see **Fig. 9**). It is obviously of uppermost relevance to differentiate between the different types of colonic atresia (eg, between type II and type IV) before attempting surgical correction, because the type of malformation will impact not only on surgical options for a given case but also on prognosis for the animal. Furthermore, a thorough exploration of the abdominal cavity is indicated because malformations in other parts of the intestine or in other organs (eg, in the urogenital tract) have been associated with atresia coli.<sup>62</sup>

Four options can be considered after a diagnosis of colonic atresia is confirmed on exploratory surgery: end-to-side or side-to-side anastomosis of the proximal blind end of the colon and the colon descendens, end-to-end anastomosis of the intestine proximal and distal to the atretic segment, colostomy, and euthanasia. End-to-end anastomosis is only possible in rare cases because the distal part of the colon is, in most cases, atrophied and cannot be mobilized for end-to-end apposition and suture. Prior to anastomosis, an enterotomy must be performed at the apex of the cecum or the blind end of the proximal colon segment to drain intestinal contents, to reduce tension before attempted anastomosis, and to minimize the possibility of postoperative obstruction through impacted ingesta. The cecal apex or blind end of the colon is exteriorized, and meconium is milked out of the proximal intestine through a stab incision. The enterotomy site is flushed with warm saline solution and closed using a double inverting suture pattern (Cushing or Lembert) and absorbable suture material (eg, 3-0 polyglyconate or polydioxanone). If apposition of the proximal and the distal part of the colon is possible, intestinal clamps are placed across the proximal and distal intestinal segments, the blind ends are removed, and end-to-end anastomosis is performed with absorbable suture material (eg, 3-0 polyglyconate) by first apposing the mesenteric and antimesenteric edges of the intestine with a single perforating simple suture each, and then closing the intestinal lumen either with a double layer of interrupted continuous inverting sutures (Cushing or Lembert) or with a single row of simple perforating interrupted sutures. Surgery to create a bypass of the affected intestinal segment through side-to-side or end-to-side anastomosis between the blind end of the proximal intestinal segment and the descending colon has been described in numerous reports.<sup>2,53,60–62</sup> It is a delicate operation that needs to be performed under general anesthesia.<sup>2</sup> For this procedure, a soft flexible tube can be passed with caution 10 to 20 cm into the rectum by an assistant to allow intra-abdominal identification of the descending colon. A low-volume enema may be administered carefully to remove mucus and cellular debris.<sup>61–63</sup> An intestinal clamp is placed on the proximal colon segment and its blind end is resected. For end-to-side anastomosis, an adequate site is chosen on the descending colon and two simple interrupted Lembert traction sutures are placed approximately 5 cm apart through the antimesenteric seromuscular layer of the descending colon and at 180° intervals on the open proximal colonic segment. The lumen of the descending colon is opened between the stay

sutures and end-to-side anastomosis is performed with a single layer of sutures placed in a simple interrupted pattern (with absorbable suture material such as 3-0 polydioxanone, polyglyconate, or polyglactin 910). If necessary, a second layer of inverting sutures is added.<sup>63</sup> If side-to-side anastomosis is to be performed, the transected end of the spiral colon is sutured with a double inverting suture pattern and side-to-side anastomosis is performed in a manner similar to that described earlier for end-to-side anastomosis. The use of stapling devices for colocolic anastomosis has also been described.<sup>53,60,61,63</sup> Some investigators have recommended removing 6 to 10 cm of the dilated proximal blind end of the intestine to prevent anastomotic aperistalsis subsequently to chronic distention,<sup>60,61,63,65</sup> but this procedure did not appear to improve the short-term results of surgery.<sup>63</sup> After completion of the anastomosis, the intestine is flushed carefully with warm saline solution and repositioned in the abdomen. The body wall is closed in a routine manner.

If colostomy is chosen by the surgeon or the owner, or if this salvage procedure is the only possible option because the proximal and distal parts of the intestine cannot be apposed for anastomosis, the blind end of the proximal segment of the intestine is sutured to the skin in 3 layers in the right flank distally to the surgical incision. Localization of the stoma dorsally in the flank results in severe fecal contamination of the abdominal wall, whereas a more ventral stoma bears an increased risk for intestinal prolapse. A circular piece of skin with a diameter of 3 to 4 cm is removed in the chosen location and the muscles of the abdominal wall are prepared by blunt dissection down to the peritoneum. After opening the abdominal cavity, the peritoneum and transverse abdominal muscle are sutured to the skin on both sides of the wound with a size 0 absorbable material to protect the abdominal wall. Then, the blind end of the intestine is pulled through the incision, and the wall of the intestine is sutured to the skin with eight simple interrupted sutures (eg, 0 polyglyconate). The lumen is opened and the intestinal wall is sewn to the skin in a simple continuous pattern.<sup>2,57</sup> As an alternative to the blind end of the proximal segment of the colon, the cecal apex can be sutured to the skin. In such cases, amputation of the colon is recommended to limit impaction of its blind end.<sup>2,68</sup>

In all cases, postoperative care after correction of large intestinal atresia includes antibiotics for at least 5 days (eg, procaine penicillin, 30,000 IU/kg intramuscularly or subcutaneously once daily, or a broad-spectrum antibiotic) and anti-inflammatory drugs as needed. Correction of dehydration and acid-base and electrolyte imbalances with intravenous infusions of saline or polyionic solutions containing glucose has been described by several investigators as an essential part of the clinical management of calves that have intestinal atresia. Plasma administration is indicated in the case of failure of passive transfer of immunity.<sup>2,60-64</sup>

Feces are usually passed within 24 hours of surgery, which are first of loose consistency and become more consistent within 4 to 5 days. Oral feeding can be restored within the first 24 hours after surgery if the calf passed feces, with small amounts of milk or milk replacers fed four to six times daily.<sup>60,63</sup>

### **Prognosis**

The prognosis for atresia ani after surgical correction is principally good if the animals are presented for treatment before they become strongly debilitated and if no anal stricture develops. Although this procedure can be used for salvage, it remains questionable as to whether such animals should be further used for breeding because a genetic component of the development of intestinal atresia is not excluded.<sup>57,61</sup> In addition to combined atresia of multiple intestinal segments, congenital malformations

affecting the umbilicus, the urogenital system, the musculoskeletal system, or the eyes have been reported in 18% to 38% of calves that had intestinal atresia.<sup>60,61,63,76</sup>

Short-term survival rates as high as 71% but also as low as 27% have been reported for colonic atresia, and long-term success rates (ie, the percentage of calves born with colonic atresia corrected surgically and subsequently reaching reproductive age) are distinctly lower, ranging from 14.3% to 16.7%.<sup>61–63</sup> In two case series, between 42% and 48% of calves that had atresia coli could be released from the clinic after treatment. The most common postoperative complications were peritonitis secondary to anastomosis failure and megacolon or impaction of the anastomosis despite patent lumen.<sup>60,62</sup> Loose manure and poor growth have been reported in calves after surgery for correction of colon atresia.<sup>60,63</sup> Higher success rates (43%–72%) have been reported for the bypass procedure than for end-to-end anastomosis of the proximal and distal parts of the intestine (17%–40%).<sup>60,63</sup> The prognosis for survival is better for calves that are alert and able to stand at presentation than in severely debilitated calves.<sup>62,64,68</sup>

In one study,<sup>64</sup> creation of a stoma in the flank was unsuccessful in seven out of seven cases; thus, this method should not be recommended. If it should be performed despite these recommendations, calves that have fistulas must be slaughtered when they reach a weight of 130 to 150 kg, to avoid later complications.<sup>2,68</sup>

Because of issues related to animal welfare, because of the high frequency of multiple malformations in calves that have atresia coli,<sup>57,60,63,64</sup> and because the role of genetic inheritability in bovine intestinal atresia is unclear and therefore the impact of continued breeding of such animals on persistence of the malformation in the population is unknown,<sup>73</sup> the author does not recommend surgical correction of colonic atresia. When this diagnosis is confirmed on exploratory surgery at the author's clinic, the calf is usually euthanized immediately after informed consent has been obtained from the owner.

## SUMMARY

Surgery for correction of CDD can be performed easily under field conditions, even if cecal amputation needs to be performed. However, complicated cases requiring bypass procedures in the large intestine require general anesthesia and are best performed in hospital conditions.

In calves that have atresia coli, the author recommends celiotomy for confirmation of the diagnosis but not for surgical correction of the condition because of reasons of animal welfare and breeding hygiene. If, however, corrective surgery should be attempted, colostomy can be performed in the field, but the more complicated anastomosis procedures are best done in hospital settings. Anal reconstruction can be performed easily under epidural anesthesia and may allow fattening and salvage of calves born with anal atresia.

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