A Pathologist's View of the Scouring Calf

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The pathologist studies lesions (changes in cells, tissues and organs) in an effort to explain clinical signs, reconstruct the sequence of events leading to death, and determine the significance of infectious agents which the microbiologists tell us are present.

Lesions are studied grossly as well as with the aid of a light microscope and, frequently, with the aid of an electron microscope.

Grossly, the scouring calf in an uncomplicated case of neonatal diarrhea presents few lesions. The calf is obviously dehydrated, and the perineal region is contaminated with fecal material. The intestinal tract shows little evidence of inflammation but is often flaccid and fluid filled. Fecal material in the large intestine is more fluid than normal and may be white, yellow, pale green or tan. Milk in the abomasum is often poorly curdled.

The uncomplicated case of scours is often the exception rather than the rule, hence a variety of other organs may be involved in addition to the gastrointestinal tract. Various types of pneumonia commonly complicate calf scours. Navel infections are not uncommon nor are abomasal ulcers. As a consequence of navel infections we may see arthritis, nephritis and, occasionally, meningitis. Quite often a bacterial infection which started in the intestine as scours becomes systemic in which case there is multiple organ involvement.

As stated earlier, the uncomplicated scours case presents few gross lesions. Microscopically, however, lesions characteristic of various infectious agents or combinations of agents can often be seen provided the specimen is adequate. Fresh specimens are absolutely essential. Post mortem changes begin to obscure characteristic microscopic lesions as quickly as 10 minutes post mortem (8).

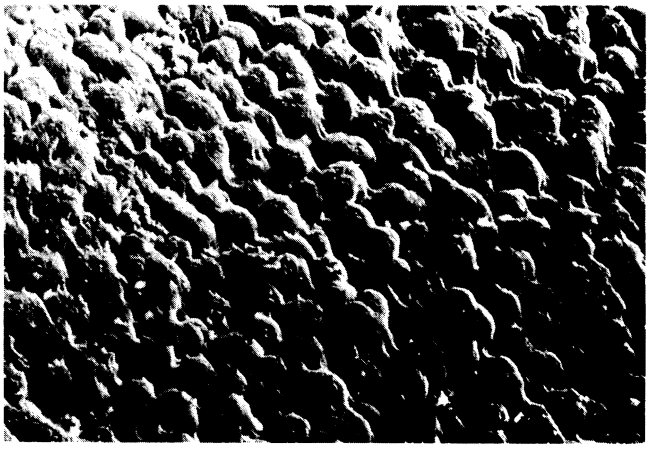
The normal small intestine is lined by long, tapering villi covered by vacuolated, columnar epithelial cells. The epithelial cells in turn are covered, on their luminal surface, by hair-like microvilli. These epithelial cells are termed absorptive enterocytes, for it is at the microvillous border of the intestinal epithelial cells that absorption of nutrients and fluid takes place.

There is a continual turn-over of epithelium on the villi. New cells are produced in the intestinal crypts and move up the villous. The most mature cells are at the villous tips. As the cells reach the end of their life span, which is approximately two days as calculated by studying replacement time using labeled cells (6), they die and are sloughed into the intestinal lumen.

The structure of the large intestine is similar to that of the small. There are more mucous-producing cells in the crypts and on the villi, and the large intestine is primarily responsible for absorbing and returning water to the system.

Infectious causes of calf scours usually attack the intestinal mucosa at the villous or the crypt epithelium.

The normal villi (finger-like projections) lining the small intestine of the calf not exhibiting diarrhea. The villi provide a greater absorbing surface and are the source of secretions necessary to the digestion and absorption of colostrum.



The villi of the small intestine have been destroyed by infectious agents causing calf diarrhea. Though the villi will regrow to some extent, digestion and absorption will also be decreased as compared to the noninfected healthy gut lining of a calf.

Rotavirus, by itself, causes slight to moderate shortening (atrophy) of villi in the jejunum and ileum. The villi are blunt and covered by immature epithelial cells (5,7,9).

When the rotavirus is accompanied by a pathogenic strain of *Escherichia coli,* the lesions are similar but more severe. There is degeneration and sloughing of cells from the villous tips, and bacteria are adherent to cell surface (7,8).

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Corona virus usually affects lower portions of the intestinal tract than does rota. The lower jejunum, ileum, colon and rectum are involved. Crypts are seen to be dilated, lined by flattened cells and the lumen filled with mucous, inflammatory cells and cell debris. These lesions are most prominent in the colon; of the infectious agents involved in calf scours, colitis is seen only with corona virus infections (2,4,7).

Escherichia coli (enterotoxigenic strains) when acting alone causes minimal changes in the gut. Small intestinal villi may be slightly shorter than normal and occasional degenerate absorptive cells are seen. Large numbers of gram negative bacteria are seen attached to villous epithelial cells; in the gut lumen, there is a mild infiltration of neutrophils (1,7).

Figures 1 and 2 illustrate normal small intestine compared to a section showing villous atrophy as seen in rota virus or rota virus plus *E. coli* infections.

Figures 3 and 4 show normal colon and a corona virus induced colitis.

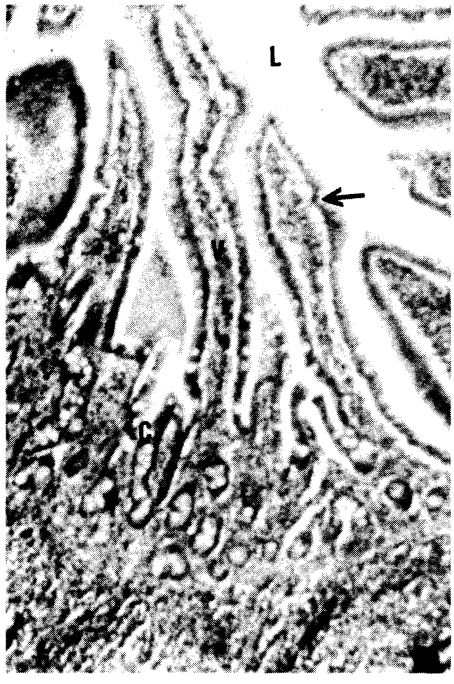

Fig. 1. Photomicrograph of a normal small intestine. Note the long, tapering villi (V), crypts (C) and vacuolated enterocytes (arrow). Intestinal lumen (L).

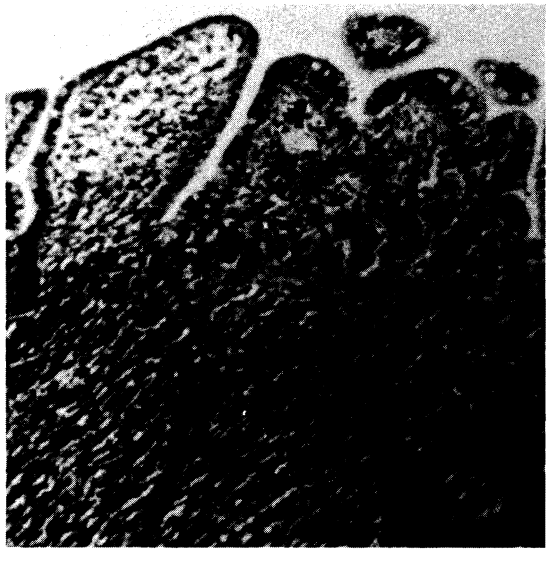

Fig. 2. Section of small intestine from a scouring calf with rota virus and *E. coli*. Note the short, blunt villi (V).

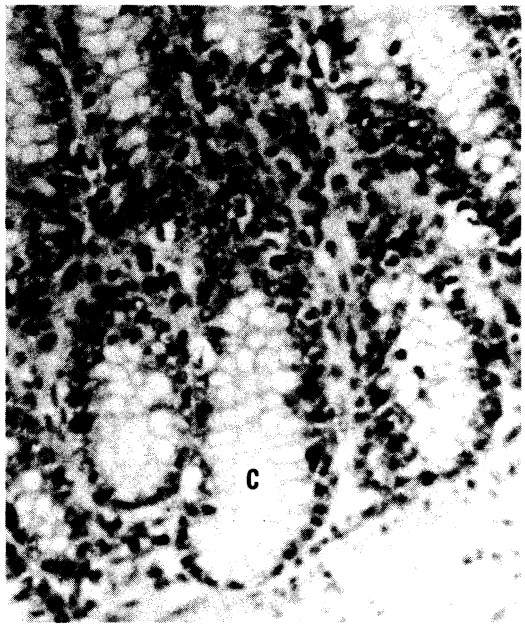

Fig. 3. Normal colon.

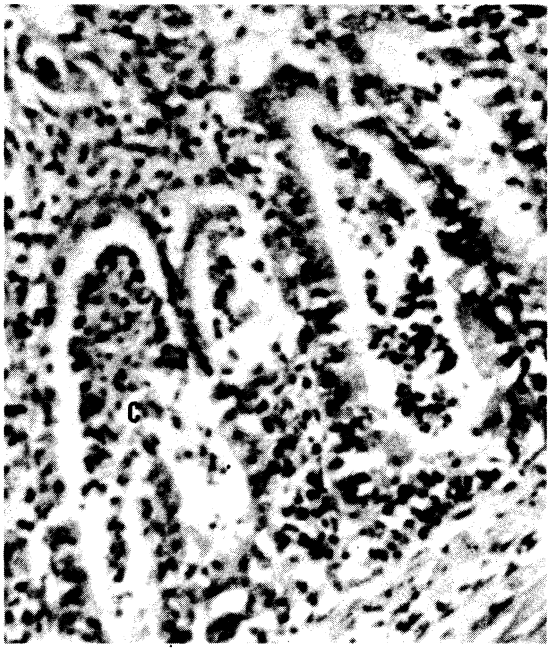

Fig. 4. Colon infected with corona virus. Note the crypts (C) filled with cell debris and lined by flattened epithelium.

Another infectious agent associated with neonatal diarrhea is cryptosporidia. Cryptosporidia is a protozoan similar to coccidia and is probably more common than is realized. It is seen attached to the microvillous border of villi in the lower jejunum and ileum. These organisms disappear rapidly after death of the calf and may not be seen in gut sections as soon as 6 hours post mortem (10). If cryptosporidiosis is complicated by one of the viruses, the lesions are more severe than with either agent alone and villous atrophy may be severe (7). Figure 5 shows cryptosporidia attached to the microvillous border of absorptive epithelial cells in the ileum.

Regardless of the point of attack of the infectious agent, i.e., villous or crypt epithelium, the ability to absorb materials is reduced. If the crypt epithelium is damaged, there is failure of replacement of absorptive cells as they live out their life span and are sloughed. If the villous epithelium is attacked, there is accelerated loss of absorptive cells. In either case there is a shrinkage (atrophy) of villi and, in the former, there is also a so-called "cryptitis."

The end result of intestinal damage is reduced absorptive capacity leading to an accumulation of fluid. Adding to the accumulation of fluid is secretion into the gut and the influx of fluid due to changes in osmotic pressure (3). Large numbers of *E. coli* in the intestinal lumen tend to increase this pressure, further increasing the inflow of fluid. Figure 6 illustrates fluid changes in normal and diarrheic intestine.

The accumulation of fluid in the intestine due to reduced absorption, active secretion and changes in pressure gradients is what leads to scours. Loss of fluid to the exterior in diarrhea leads to dehydration and dehydration is the ultimate "killer" of the scouring calf.

In addition to the factors involved in fluid loss, some infectious agents produce toxic products which, when absorbed from the damaged intestine, result in rapid death often before frank diarrhea is observed. Few, if any, gross or microscopic lesions are seen in tissues from these peracute cases.

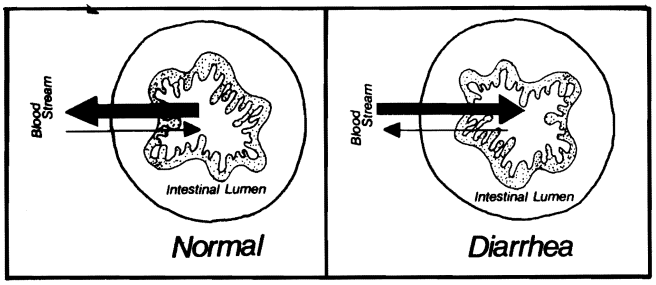

Fig. 6. In the normal intestine there is a net flow of fluid from the lumen to the blood stream as indicated by the heavy arrow. The reverse is true in intestinal diseases resulting in diarrhea.

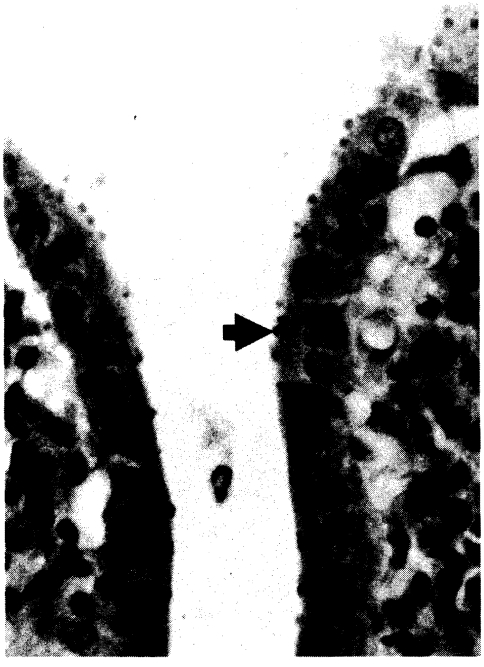

Fig. 5. A case of cryptosporidiosis. Note the cryptosporidia attached to the microvillous border of enterocytes. The magnification in this photomicrograph is 400X.

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