I. Saliva

In contrast to ruminants, for example, equine saliva in general contains relatively more calcium and chloride, and less bicarbonate and sodium. Thus, under conditions where large amounts of saliva may not be swallowed for an extended period of time, such as a dysphagia or esophageal obstruction (“choke”), horses tend to become hypochloremic and alkalotic. This has important clinical significance when it comes to choosing which formulation of electrolyte solutions one would choose to replace electrolytes and water lost to the animal.

II. Esophagus

It is important to remember that the principle musculature of the proximal two thirds of the equine esophagus is striated, while the distal third is smooth. When a swallow is induced, the upper esophageal sphincter (UES) relaxes to allow the bolus of food to enter the esophageal lumen. The bolus is passed through the esophagus by classical peristaltic (propagating ring of contraction) motor activity. The speed of passage through the smooth muscle region is slightly slower than that through the striated muscle region. There is a clinical impression that spontaneous “choke” in the horse occurs more frequently in the upper one-half of the esophagus, but the implications of this, with respect to smooth vs striated muscle function remain to be investigated. Once the bolus reaches the distal end of the esophagus, the lower esophageal sphincter (LES) relaxes, allowing the contents to enter the stomach. Control of LES tone is complex, with relaxation involving vasoactive intestinal peptide (VIP) and nitric oxide (NO).

III. Stomach

A. Secretion

The proximal half of the mucosal surface is lined with nonglandular, squamous instead of glandular epithelium. Squamous and glandular portions are well demarcated by the margo plicatus, and the glandular mucosa is divided into cardiac, fundic and pyloric portions. The cardiac region comprises only a thin strip right adjacent to the margo plicatus and, while little is known about its secretory products, it is the site of ulceration in neonatal foals under stress. As with other monogastrics, the fundic mucosa contains parietal cells which secrete acid, zymogen cells which secrete pepsin and lipase, and enterochromaffin-like (ECL) cells that secrete histamine in response to gastrin. The pyloric mucosal region is the site of G-cells which synthesize gastrin. D-cells, which produce somatostatin that suppresses gastrin release from G-cells, are found in both fundic and pyloric mucosas.
Horses, like pigs, monkeys, rats and humans, continue to secrete acid at a variable rate when the stomach is empty. This secretion involves the interaction of histamine, since it can be markedly inhibited by histamine-2 blocking agents. In contrast to other species studied, however, the horse does not have as high an acid concentration in the gastric juice when the stomach is stimulated by gastrin, probably because of concomitant reflux of gastrin-stimulated pancreatic juice back through the pylorus. At least this is the case under experimental conditions when the stomach is empty and the contents are being continually collected via a cannula. However, continuous pH monitoring of pH in the bottom region of the stomach indicates that such reflux can occur under free-feeding conditions as well.

B. Fermentation

In all monogastric species, the stomach serves as a sort of initial holding tank for ingested food. In some, such as the pig and the horse, moreover, considerable bacterial fermentation takes place within the stomach which is probably not the case in other, non-herbivorous monogastrics such as humans, dogs, and cats. So, what's the trick with respect to support of intra-gastric fermentation in the equine stomach? It probably works like this. First of all, there are some fermenting microbes, such as Lactobacillus acidophilus and Streptococcus bovis, that do quite nicely in a relatively acidic environment; the by-product of such fermentation is lactic acid, and plenty of that can be generated within a horse’s stomach if there is a large proportion of soluble carbohydrates within the meal. On the other hand, those microbes that convert ingested carbohydrates to volatile fatty acids (VFA) are more acid-sensitive, but they are also present and active within the equine stomach. I suggest that they colonize within the coarser fibrous ingesta, which collects towards the top of the stratified mat of gastric contents where the pH of the contents is more to their liking because the mat has not been fully penetrated by gastric acid. This higher pH is augmented by swallowed parotid saliva, which amounts to 10 - 12 litres/day in an adult horse, and has a bicarbonate concentration about twice that of plasma. The regional pH variation was elegantly demonstrated by Baker and Gerring who recorded a progressive drop in value as a nasogastrically introduced weighted pH probe dropped through the gastric contents to the bottom of the stomach where the most acidic components collect. It is highly unlikely however, that much of the ingested cellulose component of the herbage is digested within the stomach of the horse; most will not be digested until it reaches the cecum and colons.

C. Non-fermentative Digestion

The two digestive enzymes of note secreted by the equine stomach are pepsin and lipase, which is in concordance with most other mammalian species. Pepsin is proteolytic in an acid medium. The primary secretion by the zymogen (chief) cells found within fundic and pyloric mucosas is pepsinogen, which is converted to pepsin when the pH of the medium is < 4.0. More than one biochemical form of equine pepsinogen exists, but the functional significance of this, with respect to intra-gastric proteolysis, is not known. We really know nothing about the extent to which pepsin contributes to the digestion of ingested dietary protein in the horse. Equine gastric lipase, which has a peak activity at pH 4.0, is also produced by zymogen cells, primarily in the fundic mucosa. Horses produce a large amount of gastric lipase but, as with pepsin, nothing is known about its role in the processing of ingesta.

D. Motility

If the stomach cannot relax to receive an ingested meal, the volume that can be ingested is very limited and there is risk of rupture if an attempt is made to exceed the limit. In our lab, Lorenzo-
Figueras et al. looked at response to two different sizes of either a hay or grain meal given to horses, which provided some interesting results. Basically, in contrast to the monophasic relaxation episode seen in humans and dogs after a liquid meal, we saw a distinct biphasic response to the ingestion of the solid meals. The first occurred during active ingestion, which we regarded as true “receptive relaxation”, and the second, more prolonged phase, which we referred to as “accommodation”, followed the active ingestion.

The duration of the receptive relaxation phase was directly related to the time it took to eat the meal, and was most distinct during ingestion of the “large” (1 g/kg body weight) hay meal. The only significant degree of accommodation (relaxation) above baseline during the second phase was after the large hay meal. We wondered if the first phase was mainly induced by mechanosensors within the pharynx and/or esophagus, whereas the second phase was more under the feedback control of sensors within the duodenum that were triggered by food entering from the stomach. These studies represent only the tip of the iceberg of what needs to be done in horses, particularly in view of the dietary manipulations fostered on them these days! Nevertheless, the results certainly suggest that, within reason, the equine stomach is fully capable of adjusting to size of meal ingested.

From the limited number of studies that have been done to date, it appears that equine gastric motility that results in emptying, *per se*, is not fundamentally different from other species. The coarser contents are moved out primarily by peristaltic contractions that start at mid-fundic level and course through the pyloric region with increasing rate and strength (“antral systole”) to force the contents into the upper duodenum. The finer, more liquid contents collect in the pyloric region and are forced into the upper duodenum by a combination of increased proximal gastric tone and “antral systole”. This contractility is governed by the enteric nervous system (ENS) and local myoeletrical events. Myoelectrically, the slow wave frequency rate, which determines the maximum frequency that the stomach can contract, is around 3/min in the horse, whereas in dogs, pigs and ruminants it is closer to 6/min. An interesting feature of equine and ruminant gastroduodenal motility is that just as a phase III myoelectrical pattern develops in the proximal duodenum, the gastric antrum stops moving for a few minutes. During this time, gastric emptying ceases which, in the horse allows backflow of duodenal contents into the stomach.

### IV. Small Intestine

As in other species, the small intestine of the horse is divided into three major portions, the duodenum, jejunum, and the ileum. Relative to its size, the horse has a relatively short small intestine, around twenty-five meters in length, with the majority being jejunum, in the average 450 kg horse. Indications are that the transit time through the small intestine of the horse is relatively rapid. Fifty percent of a liquid marker instilled into the stomach of a pony can be found in the distal ileum by the end of one hour after instillation. By the end of an hour and a half, twenty-five percent of that marker is in the cecum. However, particulate markers move much more slowly; in general, the larger the particle, the slower the transit rate.

Small intestinal contents are moved aborally by motility events that involve both rapidly and slowly propagated contractions. The myoelectrical activity of the equine small intestine, which is indicative of when contractile events occur, is similar to other species showing the 3 phases (I, II, III) of the migrating myoelectrical complex (MMC), which is only partially disrupted by feeding. These motile events are undoubtedly under both neural and hormonal control. Another factor governing transit rate in the horse is the time between meals. Under natural and most
management conditions, horses have some food in front of them at all times. However, those that have been fasted for 12 hours have a much more rapid transit of food from stomach to cecum when refed than those fed the same amount on an ad libitum basis.

Thus, numerous factors including composition, liquid or solid phase, partial size, autonomic nervous activity, gastrointestinal hormone release, and time between feedings, along with the character of the motile events, govern the rate at which the ingested bolus moves from the stomach to the large intestine of the horse. Again, the ENS is intimately involved in this process.

A considerable amount of fluid and other substances are secreted into the small intestine to aid in the digestive and absorptive functions. As with most monogastrics, the non-cellulose portions of the diet are digested and absorbed by the small intestine of the horse. Fat digestion is no doubt promoted by the presence of bile salts secreted through the biliary system and enzymes in the pancreatic juice. In contrast to most other species, the resting pancreatic secretion in horses is profuse and apparently continuous. When further secretion is stimulated by either nervous or hormonal means, the concentration of bicarbonate in equine pancreatic juice does not increase as markedly as it does in other species; that is, it remains closer to the resting concentration. Nonetheless, the pH of equine pancreatic juice is ~8.0. There is now strong evidence that gastrin is a major stimulant of pancreatic water and electrolyte secretion in horses, similar to the effect that secretion has in other species. Furthermore, the concentration of the digestive enzymes in the pancreatic juice of horses seems to be very low. Of interest is that, on a relative basis, the equine pancreas produces much more lipase than any of the other digestive enzymes. And, recent studies have shown that horses can effectively assimilate quite large amounts of dietary fat. Much research still needs to be done concerning control of pancreaticobiliary secretion in the horse.

That pancreatic juice is profuse in the horse may play an important role in preparing the small intestinal contents for microbial digestion within the large intestine. For instance, the concentration of bicarbonate in the small intestinal ingesta increases markedly as it approaches the distal ileum due, in part, to pancreatic secretion and, in part, to bicarbonate secretion by the ileum in exchange for chloride.

V. Cecum and Colons

The horse has to be regarded as having just about the ultimate large intestinal development within the mammalian species. The equine surgeon who does any degree of colic surgery is constantly reminded of this. Besides the large sacculated cecum, which can be seen in other herbivores such as rabbits and guinea pigs, there is a vast compartmentalized large intestine consisting of five distinct portions: namely the right ventral, left ventral, left dorsal and right dorsal sections of the large colon, and a smaller well-delineated small colon where final desiccation of the ingesta takes place to form fecal balls. Studies have suggested that not only is this compartmentalization an anatomical feature, but probably a functional feature as well.

The most important general function of the large intestine of the horse is that it acts as a fermentation vat for digesting ingested plant fibre to volatile fatty acids which are absorbed and used as nutrients (see below). B-vitamin synthesis also probably occurs here as it does in the rumen. In contrast to the ruminant, the fermentation vat at the end instead of at the beginning of the intestinal tract may be a more efficient herbivorous arrangement in that there is less energy expenditure in digesting and reassembling of the soluble components of the ingested nutrients.
A. Motility

Quite a bit is now known about the electromyographic aspects of equine large intestinal motility, and some, but not enough, about motor events themselves and how they are involved in the orderly movement of ingesta along the large intestinal lumen. The base of the cecum, essentially a large blind sac, has a highly sophisticated movement. For emptying, contracts strongly some twelve to fifteen centimeters from the cecocolic junction, trapping ingesta and some gas within the end of the cecal base, or "cupola". Coinciding with, or shortly after, the formation of this ring of contraction, the ileocecal area itself seems to relax so that the ingesta from the cupola spills through the junction and into the ventral colon. Then this contraction ring begins to relax and there is some reflux of ingesta from the ventral colon back into the cupola of the cecum, but the net effect is a movement of ingesta from the base of the cecum into the ventral colon. Myoelectrical activity indicative of propagated rings of contraction ("peristalsis") moving both apically and basally along the whole cecal body have also been described.

If one looks at the 5 major colonic divisions - RVC, LVC, LDC, RDC and small colon - there are definite regional differences in motility patterns. In some early studies, the pelvic flexure region, which differentiates LVC from LDC, was suggested to be a major "pacemaker" region for motility of the great colon. From a clinical point of view this is of considerable interest because a common site for impaction is the LVC just orad to the pelvic flexure. Colonic myoelectrical activity within the pelvic flexure region of a horse from which feed has been withheld for 12 hours, shows distinct, aborally migrating clusters of action potentials (spikes) which are indicative of contraction, with cluster duration of 5-8 minutes. Dispersed between these distinct events is random spike activity of up to 6 seconds each in duration that does not seem to be propagated or may have an orad orientation over a few electrodes, and intense spike bursts of 4-6 seconds duration that appear to be signaling a rapidly migrating, aborally oriented contractile event that traverses across all the electrodes. This latter event is analogous in appearance to the "migrating action potential complex" (MAPC) seen in the small intestine. Another major feature of the fasting pattern of colonic myoelectrical activity in this region is its occasional interruption by a period of very intense spike activity that lasts about 5 minutes and propagates rapidly (~3 cm/sec) in an aboral direction and is followed by a period of relative electrical quiescence. This activity is analogous to the phase III of the small intestinal migrating myoelectrical complex (MMC). In the fed animal, these "colonic migrating myoelectrical complexes" (CMMC) are much less frequent and the 5-8 minute bursts of activity are not as distinct. These, and subsequent studies do not, however, support the concept of distinct "pacemaker" properties within the pelvic flexure region.

The orally directed events, combined with those that seem to be isolated and not propagated in either direction, are thought to hold up digesta transit and promote fermentation absorption by "mixing" movements. You can appreciate how important this must be for a hind gut fermenter. The colonic transit rate of particulate markers of very small size is similar to liquid, but larger markers have a direct size-related decrease in transit rate. For instance, only about 40% of an experimentally fed nondigestible 2 cm size marker had appeared in the feces after ten days! The implication from these studies is that the larger particles of ingesta entering the great colons are held up so that maximum microbial digestion can occur, in the process of which the particle is reduced in size. Sometimes larger indigestible items, such as small stones or hardware, are spontaneously ingested by a horse and hang up within the great colon where they can form the nidus for an "enterolith" that, in itself, can become large enough to block colonic outflow and cause colic.
B. Microbial Digestion

It is the microbial digestive activity within the cecum and colons that enables the horse to obtain nutrients from ingested forage. Complex microbial interaction, no doubt analogous to what occurs in the rumen, is involved; certainly, similar kinds of microorganisms are present in both organs. Bacteria and protozoa are both involved and some seventy-two species of protozoa, primarily ciliates, have been described as normal inhabitants of the equine large intestinal tract. Some studies have shown that certain species of these protozoa predominate in certain parts of the large colon, again suggesting a compartmentalized function of this organ.

Optimal conditions are necessary for appropriate microbial digestive activity to occur within the large intestine of the horse. First of all, the pH must be maintained somewhere around 6.5; as suggested earlier, the bicarbonate secretion by the distal ileum of the horse most likely helps to adjust the pH of the small intestinal contents entering the cecum. Proper pH is not only necessary to support the microbes but it also enhances microbe produced enzyme activity occurring within the lumen and promotes volatile fatty acid (VFA) absorption. Both pH and sodium concentration within the lumen also have a strong influence on the rate of VFA absorption. Second, an anaerobic environment must be maintained within the large intestines of the horse in order for optimal microbial digestion to take place, since many of the more important flora are strict anaerobes. Third, the ingesta must be held within the lumen of the various portions of the large intestine, including the cecum, so that maximum digestion and absorption can occur. This implies, of course, normal motility patterns must be present.

Finally, for microbial digestion to take place there must be available substrate. This means that the animal must have been eating normally. Most of the soluble substrates within the diet will have been digested within the stomach and small intestine and absorbed by the latter. Any soluble carbohydrate that may have escaped this process and moved into the large intestine will be converted there by microbes to propionate, which in ruminants, and probably in horses as well, is a "gluconeogenic" volatile fatty acid. The insoluble carbohydrate material (plant fibre) which is undigestible in the upper bowel will also be converted by large intestinal flora to VFA, with molar ratios of acetate, butyrate and propionate dependant upon diet composition. In the pony (presumably the same occurs in the conventional horse), the net VFA production within any portion of the large intestine or cecum varies with the time following the ingestion of the meal and portion of the colon involved.

While colonic digestion of ingested cellulose in the horse and resultant absorption of the VFA across its mucosa seems quite straight-forward, the processing of protein nitrogen which has been trapped within the plant material, and nitrogen cycling into and out of the colon in general, appears to be a more complicated story. Recall that, within the rumen, the majority of ingested protein of all types is digested to constituent elemental components which are then reassembled by ruminal bacterial into amino acids, and that some of the nitrogen within these assembled amino acids may also come from non-protein sources such as urea which is either in the diet or has recycled back into the rumen. These protein-laden bacteria then move through into the abomasum where some are killed by the abomasal acidity and others are broken down by digestive enzymes in the small intestine to release the protein within them. The released proteins are then further digested and absorbed by the small intestine as amino acids or dipeptides. There is some fairly good evidence that colonic flora of the horse can also build their own proteins from dietary nitrogen sources and that there is recycling of blood urea nitrogen transmurally into the colonic lumen. Furthermore, protein nitrogen can move from colonic lumen into the blood stream. This cyclic appearance and disappearance of protein nitrogen
appears to be independent of digestive flow, which supports the contention that it is microbial in origin. What is not year clear, however, is if and how the colonically produced microbial protein is assimilated. That is, is it absorbed as protein, or as amino acid, or as merely nitrogen? Are there any amino acid transporters involved as there are in the small intestine? A carrier for movement from lumen into colonic mucosal cell has been described, but this amino acid is not transferred intact from cell into blood.

C. Transport of Solute and Water

The transport of VFA and protein nitrogen across the colonic mucosa has already been discussed in some detail above. It has been suggested that optimal VFA transport is dependent upon pH and sodium concentration within the ingesta. That is, as the pH at the luminal surface of the mucosa moves closer to the pK of the VFA in question through luminal membrane Na⁺/H⁺ exchange, more of that VFA is protonated and therefore is more readily absorbed. Thus, in the process of VFA absorption there is also a net absorption of sodium, chloride and water from the lumen into the blood. In the proximal part of the great colon, the Na⁺ absorption is primarily via an electroneutral pump in exchange for H⁺; in the small colon it is primarily via electrogenic pump. There is evidence that both of these pumps can be influenced by aldosterone. This has important implications regarding the effect of meal vs ad libitum food intake on the hydration of equine colonic contents. For instance, it has been shown that fermentation within the large intestine of a large single pelleted meal by a pony can cause up to 15% reduction in its plasma volume, which does not occur in ponies allowed to eat on a free choice basis. This is due to the fact that the production of VFA’s initially outstrips their absorption, thus pulling plasma water into the colonic lumen. A drop in plasma volume ultimately results in renin-angiotensin, and then aldosterone, release, promoting an enhanced Na⁺, and thus water, absorption over and above that that moves with VFA absorption. The interesting question therefore arises, “Does meal feeding of horses increase their chances of developing a colonic impaction?”

In the final analysis, it appears that the greatest amount of the water which is emptied from the small intestine through the ileoceccal junction of a horse is absorbed by the cecum. The ventral colon absorbs the next greatest amount on a daily basis. The dorsal colon actually seems to accumulate a small amount of water on a net daily basis, while the small colon mops up whatever is necessary to form normal fecal balls. Thus, in their classic studies, Argenzio et al. showed that of the 19.5 litres of water which enters the large intestinal tract of a 100 kg pony each day, only about 1.5 litres leaves a fecal water. Because the equine cecum and colons play such an active role in digestion and resultant water balance, a horse may have, for example, a disease process that results in severe small intestinal malabsorption which will not manifest as diarrhea so long as the large intestinal tract is functioning normally, which would be in contrast to what would happen in a human or a dog.