SOME EFFECTS OF THE REMOVAL OF VARYING LENGTHS OF THE DISTAL SMALL INTESTINE IN HORSES

by

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INTRODUCTION

The equine digestive tract is unique among herbivorous animals. The primary area for the digestion and absorption of proteins, carbohydrates, and fats is in the small intestine anterior to the large bowel, the fermentation vat, where breakdown and utilization of fiber occurs.[1-8] The small intestine of the horse is involved in many medical and surgical diseases.[9-43] The surgical problems frequently require removal of varying lengths of this organ. Time is critical during equine abdominal surgery.[44,45] Ability to remove one longer section of intestine versus two or more shorter segments would decrease surgery time and consequently would be less stressful to the horse, especially if no ill effects resulted from the removal of extra intestine.

Knowledge of specific effects of the removal of varying lengths of small intestine is lacking. This study was undertaken to quantify these effects in hopes of clarifying potential postsurgical problems. This study was also designed to provide some insight regarding how much small intestine could be removed without producing devastating and irreversible metabolic disorders.

LITERATURE REVIEW

Awareness of the location of various digestive and absorptive activities of the small intestine and its relationship to the large bowel is paramount to understanding its function.[2,46,50] The small intestine is largely responsible for the digestion and absorption of carbohydrates, proteins, and fats while the large intestine is the primary site of fiber digestion and net water absorption.[2-4,6,47-51] Carbohydrates are a major source of energy for the horse.[5,7,51-53] Soluble carbohydrates are presented in the form of grain and to differing degrees in the dry matter of roughages.[5,8,48,50-54] They are processed in the small intestine into glucose and other simple sugars.[2,3,5] Sixty-five to seventy-five percent of the soluble carbohydrates are digested in the small intestine and absorbed as glucose.[5,8,47] The remainder, in addition to the insoluble carbohydrates (cellulose, lignin, and other fibrous plant material) go to the cecum and large intestine for bacterial fermentation where the short chain volatile fatty acids (VFA) acetic, butyric, and propionic acid are produced and absorbed.[2,3,5,47,50,51,58,59] Theses VFA can account for 30% of digestible energy. Increased grain intake or changing the grain-to-hay ratio can increase the amount of soluble carbohydrate that reaches the large intestine. Furthermore, soluble carbohydrates can influence cecal fermentation patterns promoting the formation of propionate.[2,3,6,47,55,56,57]

In the horse, little digestive enzyme activity is provided by salivary secretions. Pancreas secretions, although large in volume, possess low alpha amylase activity with which to start carbohydrate digestion. However, this may be sufficient to initiate digestion in most instances.[1,5,60] The disaccharidase enzymes of the small intestine columnar epithelial cells

(enterocytes) then hydrolyse the resulting disaccharides and oligosaccharides into constituent monosaccharides on the enterocyte brush borders.[1,5,61,62] Two major classes of small intestine mucosal digestive enzymes have been demonstrated. Beta glycosidases: lactase (beta galactosidase) and cellobiase (beta glucosidase) develop during fetal life and reach maximum levels in the neonate and then decline. Alpha glucosidases: sucrase, maltase, trehalase, and isomaltase, the second major enzyme class, develop primarily after birth. Adult levels are reached between seven and nine months of age.[1,2,5,62]

The beta glycosidase enzyme lactase (neutral beta galactosidase) has its highest activity in the duodenum and proximal jejunal brush borders with decreasing activity distally. Peak activity occurs from birth through four months of age. Activity decreases by 50-70% by the end of the second year. An accelerated decrease occurs the third year and by the fourth year activity is barely detectable. However, a second enzyme capable of hydrolysing lactose also exists in the jejunum and ileum. This is the nondigestive lysosomal enzyme acid beta galactosidase which represents the lactase activity in the adult small intestine.

Cellobiase exhibits a similar distribution pattern to neutral beta galactosidase and is not found in adult small intestine.

Sucrase activity is highest in the middle and distal jejunum with decreasing activity towards both the ileum and duodenum. Trehalase has its maximum activity in the midjejunum. Maltase is a complex of a minimum of four enzymes (sucrase, glucoamylases maltase II and III, and isomaltase). Its greatest activity occurs in the middle and distal jejunum with decreasing activity toward the duodenum and ileum. Glucoamylase has a similar distribution pattern as maltase until about one year of age when its highest activity occurs in the distal small intestine.

The large bowel exhibits virtually no disaccharidase activity. Occasionally low maltase activity has been demonstrated in cecal and colonic homogenates. No other alpha glucosidases, neutral beta galactosidase, or cellobiase has been detected. However, lysosomal acid beta galactosidase associated with intracellular metabolism is found in the large intestine as well as in many other body tissues and organs.

The intestinal alkaline phosphatase mucosal enzymes are likely involved in long chain fatty acid transport and not carbohydrate digestion or transport. These are found in both the small and large bowel.[50,63]

The small intestine is also the primary site of protein digestion and amino acid absorption. Seventy percent of the dietary protein is digested and absorbed before leaving the small intestine.[2,3,7,64] Thus the absorbed amino acids depend to a large degree on dietary amino acids. The proteolytic activity which exists in the large intestine is less than 3% of that in the small intestine. The end products of protein digestion and absorption in the large intestine include ammonia and some amino acids. Significance of amino acid synthesis by bacteria and subsequent absorption is still questionable.[2,3,7,50,64-86]

The primary site of dietary fatty acid absorption is the small intestine. Continuous bile secretion occurs because the horse does not have a gall bladder. Fat absorption tests have not proven useful to this point.[2,3,50,87-90]

Fat soluble vitamins (A,D,E,K) and water soluble vitamins (various B and C) are primarily absorbed in the small intestine. B vitamins to an extent are also synthesized and absorbed in the cecum and large intestine.[2,3,6,50,91-95]

The small intestine is also the primary site of absorption of minerals.[2,3,6,50,96-100] The greatest percentage of Calcium (Ca) is absorbed in the proximal small intestine with lesser amounts absorbed distally. Magnesium, copper, and

zinc are also absorbed in the small intestine. Phosphorus is absorbed in the small intestine as well as in the dorsal large colon and small colon. As digesta travels from the small intestine to the cecum increasing amounts of bicarbonate accumulate within the intestinal lumen.[2,3,6,50,58,96-106]

The large intestine is the primary site of fiber digestion and net water absorption.[2,3,4,6,49,50,54,107-109] The large intestine daily receives fluid from the small intestine and the large intestine mucosa approximately equal to the horse's blood volume. Upwards of 95% of this fluid is resorbed.[107] Secretion and absorption of water is closely related to microbial activity, sodium (Na), and VFA regulation.[4,107,109,110] Feces are usually 66-70% water.[2,3] Hay diets yield feces with higher water content than high grain diets. Pelleted diets also tend to increase fecal water.[2,3]

Besides phosphorus the large intestine also absorbs and secretes the electrolytes Na, Chloride (Cl), and potassium (K).[2-4,50,58,107,111]

Digestion of fiber in the horse is approximately 60-80% as efficient as in ruminants. The difference tends to decrease as roughage quality increases. Reasons for the difference include faster digesta passage in the horse and ruminal bacteria being more efficient.[2,3,50,56,112-118]

Physiologically the equine large intestine is divided into four functional units: cecum, ventral colons, dorsal colons, and small colon. Mixing occurs in each segment but no retrograde movement occurs between functional units.[107,108]

The physical nature of the ingesta greatly affects its digestive tract transit time. Liquid passes most rapidly from the stomach followed by small particles and then larger particles. Transit time from the stomach to the cecum is rapid and may occur in 90 minutes for fluid material.[2,3,24,107,120-122]

The large bowel produces the majority of the VFA. The highest concentration occurs in the ventral colons while lactic acid concentration remains low throughout the entire large bowel. The stomach also produces some acetic,

butyric, propionate, and lactic acid.[2,3,6,50,58,59,107,123-125] Acetic acid accounts for approximately 75% of the VFA in the stomach and small intestine while propionic acid represents roughly 25% with butyrate accounting for the remainder. Concentration fluctuations do occur with propionate showing reciprocal changes to acetate. Butyrate remains fairly constant.[124] The gastric mucosa absorbs small amounts of the organic acids, but transports little to the blood.

The gastric pH ranges from 4.5 to 6 depending on time since feeding. The acid environment results from the release of gastric hydrochloric acid. Small intestine pH increases from 6 in the duodenum to above 7 in the mid jejunum to 7.3 in the ileum. Cecal and large intestine pH is fairly constant between 6 to 6.5. The concentration of the VFA, lactic acid, and pH ranges change with time and are related to diet intake.[124,125]

The horse uses both circulating glucose and free fatty acids (FFA) for metabolic energy.[2,4,50,126,127-130] The more conditioned a horse becomes the more FFA are utilized for energy.[126,131,132] Since the primary source of glucose is from small intestinal absorption, grain fed horses tend to secrete more insulin, be more insulin sensitive, and are more glucose tolerant than roughage fed horses.[128,130,133] VFA do not affect blood glucose levels with the exception of elevation of blood glucose to propionate in fasted ponies.[127] Hence VFA do not appear to stimulate gluconeogenesis. Blood glucose levels are maintained by counteractions of insulin and glucagon, catecholamines, and endogenous corticosteroids.[134-136] Free fatty acids are mobilized for energy from fat depots by epinephrine and glucocorticoids to help preserve blood glucose levels. Since nervous tissue cannot store glucose, other tissues are used to maintain blood glucose levels. Fasted horses maintain blood glucose levels at the expense of fat depots and muscle tissue.[137-144]

The ratio of the length of the horses' digestive tract to total body length is approximately 10:1.[145] The digestive tract is beset with many medical and surgical problems. The nervous and vascular supply of the intestines are involved in a large number of these problems.[24] Intrinsic intestinal smooth muscle contractions and basal secretory action of the gut occur spontaneously and rhythmically. Both occur independently of systemic innervation and are regulated by the intestinal intramural nervous system.[46,146,147] The autonomic nervous system (ANS) provides regulated motor activity and can override the intramural nervous system. The ANS innervates the muscular coat, glands, and spincters of the intestine. The sympathetic nervous system tends to inhibit the gastrointestinal tract due to alpha adrenergic fibers located in the smooth muscle of the gut and blood vessels.[46,146,147,148] Some $beta_2$ receptors are located in the gastrointestinal smooth muscle. Parasympathetic activity stimulates the intestinal tract motility and increases secretions. Postganglionic fibers which supply endocrine cells, glands, and the smooth muscle of the intestinal wall are cholinergic. Visceral organ pain sensation is derived from ischemia, distension, spasms, and chemical influences.[149-152] Direct intestinal inhibition results from alpha adrenergic stimulation.[46,146,153] Hence fright, stress, or pain will cause decreased motility and decreased intestinal tract blood flow. Consequently medical therapy of the equine digestive tract depends on the direct action of drugs on the intestinal smooth muscle or on the ANS.[146,149,151]

The arterial blood supply of the small and large intestine comes primarily from the cranial and caudal mesenteric arteries. The celiac artery and internal iliac arteries provide some supply to the proximal duodenum and rectum respectively.[24,152] The digestive tract except for the ileum has good collateral circulation.[30] The veins from the entire intestinal tract go to the portal vein. The rectum is drained into the caudal vena cava.[152]

The cranial mesenteric artery is predisposed to traumatic assault from the migration of Strongylus vulgaris larvae.[40,153-161] The larvae penetrate intestinal mucosa and migrate along the arterial intima causing edema, arterial dilation, fibrosis, thrombi, and arteritis.[156,160,161] The three major branches of the cranial mesenteric artery are closely associated with the celiacomesenteric ganglion.[24,46] Hence damage to these vessels can affect the gut innervation.[24,46,162-164] Decreases in motility have been associated with parasitic infection, direct neurogenic inhibition, and other causes.[148,153-155, 157-159,165-168]

The small intestine is involved in many abdominal problems.[13,32,33] These problems include displacement, torsion, obstructions (simple and strangulating), intussusception, muscular hypertrophy, neoplasia, focal and multifocal ischemia, etc.[169] Surgical correction of ileocecal area problems is difficult because of the poor accessability of the area.[32] Many of the surgical problems of the abdomen of the horse necessitate removal of sections of the small intestine.[32]

In humans and other species functional and anatomical intestinal adaptation occurs following partial intestinal resection.[170-184] Small intestine epithelium has the most rapid turnover rate, from replication through migration to extrusion, of healthy body tissue.[170] Furthermore, the epithelial proliferation can be stimulated to occur more rapidly by several factors, one of which is intestinal resection.[170]

Intestinal adaptation occurs by compensatory growth of all layers of the small intestinal wall, especially notable is villous hyperplasia. An increase in crypt depth and villous height are associated with an increase in the length and diameter of the remaining intestinal segment. Adaptation appears more efficient and greater following proximal small intestine resection with maximal adaptation reported to occur around anastomatic sites.[170,184,185]

Mucosal hyperplasia is directly related to the length of intestine resected.[170, 175,178] Theoretically, improved absorption could be due either to an increased functional ability of the enterocytes or to an increased number of cells. Enhanced segmental glucose absorption in laboratory animals is directly proportional to adaptive increments in villous height and an increase in the number of epithelial cells.[170,184,185] Enterocytes in the hyperplastic mucosal areas exhibit normal or decreased disaccharidase and dipeptidase activities.[170,181,186] Hence functional adaptation is due to an increased number of enterocytes and not to an increase in the absorptive capacity of the individual cells.[170,186]

Digestion and absorption tests are employed to assess the functional ability of the small intestine.[1,2,187-195] The oral glucose tolerance test (OGTT) is used for this purpose. It exhibits two phases. The first phase assesses the absorptive capacity of the small intestine by the exhibition of a blood glucose peak equal to or greater than 80% of the resting blood glucose level by 120 minutes following the oral administration of glucose. The second phase assesses the endocrine function of the pancreas in response to elevation in blood glucose. Return to resting levels is usual 360 minutes following oral glucose administration.[187,193] Other oral absorption tests employed to assess the small intestine function include starch, lactose (in foals), galactose, sucrose, maltase, and D-xylose tolerance tests.[1,2,188-195] The oral D-xylose tolerance test is sometimes preferred over the oral glucose absorption test.[188-191,193] D-xylose, a five carbon sugar, is absorbed from the proximal small intestine, is not metabolized by the body to any significant degree, and is not affected by body regulatory mechanisms following as absorption glucose. However, following the oral administration of xylose other factors can affect the absorption curve as occurs with glucose.[1,2,188-195] These factors include gastric emptying time, intestinal transit time, renal clearance, and mucosal blood supply. Oral glucose tolerance tests are accepted, however, as an

accurate measure of small intestine function.[191,193] Length of fasting, excitement, quantity, and concentration of glucose are among factors that may affect the glucose curves.

MATERIALS AND METHODS

Nine mixed breed horses, geldings and mares, between three and seventeen years of age, were randomly divided into three groups (Table 1) following the conditioning program described below.

During the three weeks prior to the study, the horses were vaccinated for equine influenza,[1] rhinopneumonitis,[2] tetanus,[3] and Eastern and Western Equine Encephalomyelitis.[3] An anthelmintic combination of trichlorfon[4] and pyrantel pamoate[5] was administered. Teeth and foot care were performed as needed. A standardized pelleted ration[6] was fed throughout the conditioning period and for six weeks following surgery at the rate of 1.5# feed/cwt/day. No other concentrate or bedding was provided. Horses G and I required one week of postoperative hay supplementation. None of the other horses received any roughage. The post surgical diet consisted of .5# feed/cwt the first day and was increased to the preoperative level by the fourth postoperative day.

Abdominocentesis and a blood chemistry profile were performed on all horses in conjunction with assessment of complete blood count (CBC). The profile consisted of blood urea nitrogen (BUN), creatinine, glucose, aspartate

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[1]Equine Influenza Vaccine-Bayvet Division, Cutter Laboratories, Inc., Shawnee, Kansas 66201.

[2]Pneumabort K-Fort Dodge Laboratories, Inc., Fort Dodge, Iowa 50501.

[3]Encevac-T with Havlogen-Bayvet Division, Cutter Laboratories, Inc., Shawnee, Kansas 66201.

[4]Combot-Bayvet Division, Cutter Laboratories, Inc., Shawnee, Kansas 66201.

[5]Strongid T-Pfizer Inc., New York, New York 10017.

[6]Horse Chow #200-Ralston Purina Company, St. Louis, Missouri 63188.

aminotransferase (AAT), serum alkaline phosphatase (SAP), total serum protein (TSP), albumin, calcium (Ca), phosphorus (P), sodium (Na), potassium (K), chloride (Cl), and total carbon dioxide (CO_2). A physical examination was performed. All horses were clinically normal prior to the commencement of the investigation.

Body weights were recorded twice weekly for four weeks to insure maintenance or increase of body weight with time. Gross examination of feces was recorded daily (consistency and color). A fecal consistency scale of one to eight was employed (Table 2).

Following completion of the conditioning program, the physical examination was repeated, CBC and chemistry values were reassessed, and a preoperative oral glucose tolerance test (OGTT) was performed on each horse. An indwelling intravenous jugular catheter was placed the day prior to the test to eliminate the potential excitement related to venipuncture. Each horse was fasted but permitted water for twelve (12) to fourteen (14) hours prior to the OGTT.

Fifteen milliliters of blood was withdrawn through each catheter and the pretest blood sample was collected in a clot tube. The catheter was then flushed with heparinized saline. Following clot formation, the sample was centrifuged and the serum recovered and frozen. Glucose concentration determination was performed at a later date.

Twenty percent glucose in water was administered via nasogastric tube at the rate of 1.0 gram anhydrous glucose/kg of body weight.[187,193] Care was taken not to excite the horse during intubation. Blood samples were subsequently collected at 30, 60, 90, 120, 150, 180, 240, 300, and 360 minutes. The indwelling intravenous catheter was removed following the glucose tolerance test.

One week following the oral glucose tolerance test abdominal surgery was performed. Feed was withheld the night prior to surgery.

Procaine penicillin G and dihydrostreptomycin[7] were given intramuscularly three hours preceding surgery. Each horse was sedated with xylazine[8] (1.1 mg/kg of body weight) intravenously. Anesthesia was induced via guaifenesin[9] (5% solution) followed by sodium thiamylal[10] (6.6 mg/kg of body weight). Anesthesia was then maintained with halothane inhalation anesthesia employing a semiclosed system with the horse positioned in dorsal recumbency.

A 35 cm ventral midline incision was made starting at a point 5 cm posterior to the umbilicus and progressing rostrally. The cecum was exteriorized and the ileocecal ligament, ileum, and ileocecal junction was identified. The small intestine was exteriorized and measured, progressing retrograde from the ileocecal junction to the pylorus. The measurement was taken using premeasured umbilical tape placed next to the intestine. The intestines were not stretched. The segment of the small intestine was measured intraabdominally.

Once the length of the small proximal intestine had been ascertained the ileum was transected 30 cm proximal to the ileocecal junction, blind-stumped, oversewn, and returned to the abdomen. A side to side ileal-cecal anastomosis was then done in the control group (Group I).

In Group II 4.6 meters (15 feet) of the ileum and distal jejunum were removed. The distal end of the jejunum was blind-stumped and oversewn. A side to side jejunal-cecal anastomosis was performed on the dorsal border of the cecum and the mesentery was sutured.

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[7]Combiotic-Pfizer Inc., New York, New York 10017.

[8]Rompun-Bayvet Division, Cutter Laboratories, Inc., Shawnee, Kansas 66201.

[9]Glycodex-Burns-Biotec Laboratories Division, Chromalloy Pharmaceutical, Inc., Omaha, Nebraska 68103.

[10]Bio-tal-Bio-Ceutic Laboratories, Inc., A division of Philips Raxane, Inc., St. Joseph, Missouri 64502.

In Group III 9.2 meters (30 feet) of the ileum and jejunum were removed. The distal end of the proximal small intestine and ileal stump were blind-stumped and oversewn. A side to side jejunal-cecal anastomosis was performed on the dorsal cecal border.

In all cases the body wall was closed in routine fashion using 22 gauge[11] wire in the linea alba.

Following recovery from anesthesia a sterile, absorbent, nonadherent pad[12] was placed over each incision and held in place with an abdominal support bandage of elastic tape.[13] Abdominal bandages were left in place for seven to ten days.

Procaine penicillin G and dihydrostreptomycin were continued for three postoperative days at the recommended dosage. Phenylbutazone[14] was administered orally daily for five days at a dosage of 1 gram/200 kg of body weight. Body weight was determined three times weekly for two weeks and then every fourth day. Blood chemistry values and a CBC were evaluated twice weekly for three weeks, then once weekly for three weeks. Feces were observed daily for three weeks then three times per week for the remaining three weeks. OGTT were performed during the first and fourth postoperative weeks.

At the completion of the sixth postoperative week the horses were gradually switched from the pelleted ration to brome hay and winter pasture ad libitum.

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[11]Surgical Wire-Steri-spool, P.O. Box 7114, Los Angeles, California 90022.

[12]Telfa surgical dressing-3M Company, 3M Center, St. Paul, Minnesota 55101.

[13]Elastikon-Johnson & Johnson, 501 George Street, New Brunswick, New Jersey 08903

[14]Phen-Buta-Vet Tablets, Anthony Products Company, El Monte, California 91732.

TABLE I

GROUP	HORSE	SEX	AGE yr.	HEIGHT		WEIGHT (kg) AT SURGERY	WEIGHT (kg) AT STUDY COMPLETION	LENGTH OF SMALL INTESTINE		% SMALL INTESTINE REMOVED
				Hand	Cm			Meter	Feet	
I	A. Cracker Jack	M-C	4	15.1	155	432	444	21.1	69	0
	B. Charlie	M-C	17	15.0	152	414	434	15.9	52	0
	C. Trigger	M-C	7	14.3	150	364	391	21.4	70	0
II	D. Chubbs	Fe	8	13.2	137	343	361	15.3	50	30
	E. Amy	Fe	3	14.1	145	386	416	19.6	64	23
	F. Star	M-C	3	14.1	145	336	337	22.0	72	21
III	G. Molly	Fe	3	13.2	137	304	264	15.6	51	59
	H. Goose	Fe	3	15.0	152	414	421	19.9	65	46
	I. Snowflake	Fe	8	13.3	140	309	273	16.8	55	54

Table 2

FECAL CONSISTENCY

Score	Consistency
1	Formed, hard
2	Formed, firm
3	Formed, moist
4	Formed, crumble
5	Cow pie
6	Loose
7	Watery
8	Other

RESULTS

A. Clinical Observations

With one exception prolonged gross changes did not occur in fecal consistency. Horse F developed transient diarrhea from day one through day seven post surgery. A group C_2 salmonella was isolated. The diarrhea was attributed to the stress of the preoperative oral glucose tolerance test (OGTT) and the pretest fasting being done one day prior to the abdominal surgery. Ventral midline incisional dehiscence of the skin and subcutaneous tissue which healed via second intention occurred concommitant with the diarrhea.

Horses in Groups I and II and horse H in Group III maintained or gained weight on the diet begun during the conditioning period. Two horses in Group III, G and I, lost weight on the conditioning diet.

Prior to surgery all horses had readily consumed the daily pelleted ration. All of the horses except G and I continued to do so following surgery. Horses G and I exhibited a marked lack of interest in their pelleted ration. Their feed consumption averaged ¼ to ½ of their allotted daily allowance.

As a result of partial anorexia horse G was fed ½ of her pelleted ration as a gruel via a nasogastric tube postoperative days three through ten. During this time weight loss was not as rapid.

Horse I readily returned to feed following intestinal resection. However, seven days later a clockwise displacement of the large bowel occurred. Surgical correction was done. This was followed by a ventral midline incisional dehiscence of the skin and subcutaneous tissue. Although the skin healed via second intention, a linea alba hernia developed. Anorexia occurred following the second surgery and five days later tube feeding was begun and continued for seven days. Body weight loss was not as rapid during this period.

Tube feeding of horses G and I was supplemented with a total of 2 kg/day of prairie hay. The hay was readily consumed and was stopped one day after the cessation of the tube feeding.

Routine postoperative convalescence occurred in the remaining horses. In some instances mild transient neutrophilia, increased fibrinogen, and slight temperature rises were evident following surgery (appendix). Leukocytosis and increased fibrinogen values persisted slightly longer in horses G and I.

Throughout the study the following blood chemistry values: BUN, creatinine, glucose, P, Ca, Cl, K, and Na remained within normal limits for all of the horses. Transient postoperative decreases in total CO_2 in all horses were attributed to increased respirations resulting from pain.

Subjective evaluation at the termination of the study in conjunction with physical examination revealed horses A through F and horse H had maintained or improved their physical status. All were healthy, active, in good flesh, and possessed glistening hair coats. Horses G and I had lost weight, were thin, had rough hair coats, and lacked good appetites. However, they had stabilized and were active. When placed on pasture after the termination of the study their appetites improved.

B. Group I

Transient increases in aspartate aminotransferase (AAT) and serum alkaline phosphatase (SAP) occurred immediately following surgery. AAT and SAP values decreased and remained within normal limits following the third postoperative week for all three horses. Horse B exhibited more marked AAT and SAP increases than either horse A or C following surgery. (Table 3 and 4)

Total serum protein (TSP) remained within normal limits for the duration of the study. Horses B and C exhibited decreases following surgery. Horse B returned to presurgical levels during the second week following surgery.

TSP for horse C remained decreased (Table 5).

Albumin concentrations decreased following surgery and gradually returned to normal presurgical levels (Table 6).

Blood samples were evaluated two to four weeks following the trial period. At that time the serum albumin levels for all three horses had rebounded to a point within the upper limits of normal.

Results of the oral glucose tolerance tests (OGTT) varied. Horse A had an essentially flat preoperative absorption curve (Figure 1). The first postoperative test indicated some glucose absorption with a 29% increase at 90 minutes (Figure 2). A 92% increase occurred by 90 minutes during the fourth postoperative week (Figure 3).

Horse B possessed a normal preoperative tolerance test (Figure 1). The first postoperative test revealed some glucose absorption (36%) but no significant peak occurred and this was considered to be less than normal absorption (Figure 2). The second postoperative test had 71% increase of the pretest level at 90 minutes (Figure 3).

The preoperative test for horse C showed a 71% increase by 150 minutes (Figure 1). A peak of 105% occurred at 240 minutes during the first postoperative test (Figure 2). The final tolerance test only peaked at 24% (Figure 3).

Consequently the preoperative and first postoperative tests for horse A, both postoperative OGTT for horse B, and the preoperative and second postoperative tests for horse C indicated abnormal glucose absorption.

C. Group II

AAT and SAP transiently increased after surgery for all three horses. AAT returned to normal limits in all three horses by the third postoperative week. A slight, transient, elevation occurred in horse F the third day

following the onset of diarrhea (Table 3). SAP declined to preoperative activity (Table 4).

A postoperative drop in both the TSP and albumin occurred in all of these horses. The TSP tended to rebound the second postoperative week, while the albumin levels remained low longer (Table 5 and 6).

Oral glucose tolerance tests revealed pre and post surgical glucose peaks indicative of some glucose absorption. Horses D and E showed progressive decreases in the peaks from the first to the third tolerance tests. Horse F had peak increases of at least 100% in each OGTT (Figure 4, 5, and 6).

D. Group III

AAT levels increased after surgery and returned to normal for H and I. Levels for G fluctuated in and out of the normal range throughout the study (Table 3).

SAP was elevated prior to surgery in both horses H and I and within normal range for horse G. Postoperative levels increased and remained elevated above pretrial values throughout the study for all three horses (Table 4).

TSP and albumin levels gradually decreased following surgery (Table 5 and 6). The albumin levels tended to be subnormal as were the TSP levels for G and I.

Presurgical OGTTs on horses G and H indicated normal absorption with blood glucose peaks at 150 and 120 minutes respectively. Horse I had maximum absorption with a resultant blood glucose peak of 56% of the pretest level at 60 minutes (Figure 7).

Postoperative OGTTs were performed on horse G at the first, fourth, and tenth postoperative weeks. The blood glucose peaks were 51%, 52%, and 63% above the pretest levels for the respective first, second, and third postoperative tests. These were indicative of some intestinal glucose absorption (Figure 8 and 9).

Horse H had glucose absorption but only achieved 44% and 46% peaks in the first and fourth weeks following surgery. In a clinical situation these results coupled with weight loss would indicate malabsorption.

The first postoperative OGTT for horse I revealed a flat absorption curve. The four week test showed the absorption of some glucose with a blood glucose peak of 40% of the resting blood glucose level (Figure 8 and 9).

Complications which arose from the abdominal surgery and intestinal resection were the development of postoperative diarrhea, the occurrence of a large bowel displacement, incisional dehiscence, and minor skin suture abscesses.

Table 3

ASPARTATE AMINOTRANSFERASE
(AAT)

Mutiple samples for a week are listed with the earliest value upper most in the square.

Transient post surgical increases occurred in all horses. Horse F exhibited a second transient elevation 3 days following the beginning of transient diarrhea. In horse G, postsurgical levels fluctuated between normal and slightly above normal.

Table 3

ASPARTATE AMINOTRANSFERASE (AAT)
(I.U./L)

GROUP	HORSE	WEEK 0		WEEK I	WEEK II	WEEK III	WEEK IV	WEEK V	WEEK VI
I	A. Cracker Jack	224		335 319	257	242 225	191	191	232
	B. Charlie	196	SURGERY	925 745	502 432	273 259	199	198	225
	C. Trigger	145		187	174	199	179	194	221
II	D. Chubbs	245		1360 900 785	725 676 628	392 322	244	231	305
	E. Amy	364	SURGERY	2000 1205	1370	554 472	383	323	253
	F. Star	177		557 360	385 244	182	167	164	226
III	G. Molly	190		484 534 470	434 446	525 433	313	270	487 477 482
	H. Goose	244	SURGERY	439 359 298	194	191	202	193	252
	I. Snowflake	274		800	1245	944	318	243	245

Table 4

SERUM ALKALINE PHOSPHATASE
(SAP)

Multiple samples for a week are listed with the earliest value upper most in the square.

Post surgical elevations occurred for all horses. Horses B and C returned to normal presurgical values during convalescence. Horses A, D, E, F, and H had a elevated presurgical values which increased further following surgery and subsequently declined to presurgical activity. Horses G and I remained increased at the study's end.

Table 4

SERUM ALKALINE PHOSPHATASE (SAP)
(I.U./L)

GROUP	HORSE	WEEK 0		WEEK I	WEEK II	WEEK III	WEEK IV	WEEK V	WEEK VI
I	A. Cracker Jack	307		366 357	307	315 325	312	295	288
	B. Charlie	213	SURGERY	276 303	327 371	298 221	225	204	172
	C. Trigger	149		203	233	213	192	155	123
II	D. Chubbs	297 257		290 352 333	351 355 372	341 288	280	297	278
	E. Amy	286	SURGERY	288 292	427	278 277	263	253	232
	F. Star	388		351 447	449 406	462	387	407	417
III	G. Molly	177		260 444 399	474 423	460 563 533	743	805	892 754 684
	H. Goose	298 351	SURGERY	367 378 353	317	381	447	369	369
	I. Snowflake	266		430	501	522	345	405	406

Table 5

TOTAL SERUM PROTEIN TABLE
(TSP)

Multiple samples for a week are listed with the earliest value upper most in the square.

All horses exhibited decreases in TSP following surgery. Horses A and B had higher values at the conclusion of the study than the presurgical levels while C, D, E, F, and H had final levels below presurgical values. G and I had fluctuating levels that were below normal at the study conclusion. Samples taken 6 weeks following the end of the study with the horses on grass indicated TSP levels of 5.7 g/dl and 7.0 g/dl for horses G and I respectively.

Table 5

TOTAL SERUM PROTEIN (TSP)
(g %)

GROUP	HORSE	WEEK 0		WEEK I	WEEK II	WEEK III	WEEK IV	WEEK V	WEEK VI
I	A. Cracker Jack	6.9		6.8 7.1	6.8	7.7 7.5	7.7	7.9	7.8
	B. Charlie	7.8	SURGERY	7.2 7.0	7.1 7.7	8.7 8.5	8.3	8.0	8.0
	C. Trigger	8.0		7.1	6.8	7.0	6.5	6.8	6.5
II	D. Chubbs	7.8		7.2 6.0 6.0	6.3 6.1 6.2	7.0 6.6	6.7	6.9	6.7
	E. Amy	9.1 9.3	SURGERY	7.9 7.6	8.2	7.6 8.0	7.6	7.4	7.3
	F. Star	7.5		8.0 6.6	6.9 6.4	6.3	6.6	6.5	6.6
III	G. Molly	8.0		7.8 6.6 6.8	6.9 6.6	6.5 6.6 6.2	5.9	6.1	5.7 5.6 5.3
	H. Goose	7.6	SURGERY	6.8 6.5 5.5	7.5	7.0	7.3	7.6	7.0
	I. Snowflake	8.0		8.1 6.1	6.0	6.8	6.6	6.8	6.0

Table 6

ALBUMIN

Multiple samples for a week are listed with the earliest value upper most in the square.

Albumin levels decreased in all horses following surgery. Group I levels returned to or above presurgical levels. Horse D remained within normal limits while values returned to normal limits in horse E after being subnormal. Values in horse F decreased to subnormal even with a rebound in the final three weeks. Horses G and I finished with subnormal levels while H ended with low normal values.

Table 6

ALBUMIN (g/%)

GROUP	HORSE	WEEK 0		WEEK I	WEEK II	WEEK III	WEEK IV	WEEK V	WEEK VI
I	A. Cracker Jack	3.2		3.2 3.2	3.1	3.2 3.2	3.1	3.1	3.4
	B. Charlie	3.5	SURGERY	3.4 3.2	3.0 3.2	3.6 3.2	3.1	3.0	3.5
	C. Trigger	3.0		2.6	2.9	3.1	3.0	3.3	3.4
II	D. Chubbs	3.6 3.4		3.3 2.8 2.9	2.9 2.8 3.0	3.2 3.1	3.0	3.0	3.2
	E. Amy	3.1	SURGERY	2.7 2.4	2.4	2.7 2.9	2.8	3.0	3.0
	F. Star	2.7		2.7 2.2	2.3 1.9	2.1	2.5	2.3	2.4
III	G. Molly	3.5 3.0		3.5 3.0 3.0	3.0 2.6	2.7 2.8 2.5	2.1	2.0	2.0 1.9 1.9
	H. Goose	3.6	SURGERY	3.3 2.9 2.7	2.8	2.7	2.6	2.6	2.6
	I. Snowflake	3.3		3.3	2.2	2.4	2.4	2.3	2.3

Figure 1

PREOPERATIVE ORAL GLUCOSE TOLERANCE TEST
Horses A, B, C

Horses A and C exhibited abnormal glucose absorption curves. Horse B possessed a normal absorption curve because the peak blood glucose level was 96% of the resting level. Normal glucose absorption is considered to have occurred if the peak blood glucose is > 80% of the resting level. (See appendix table 7)

Horse	Peak % of resting level
A	7%
B	96%
C	71%

Figure 1

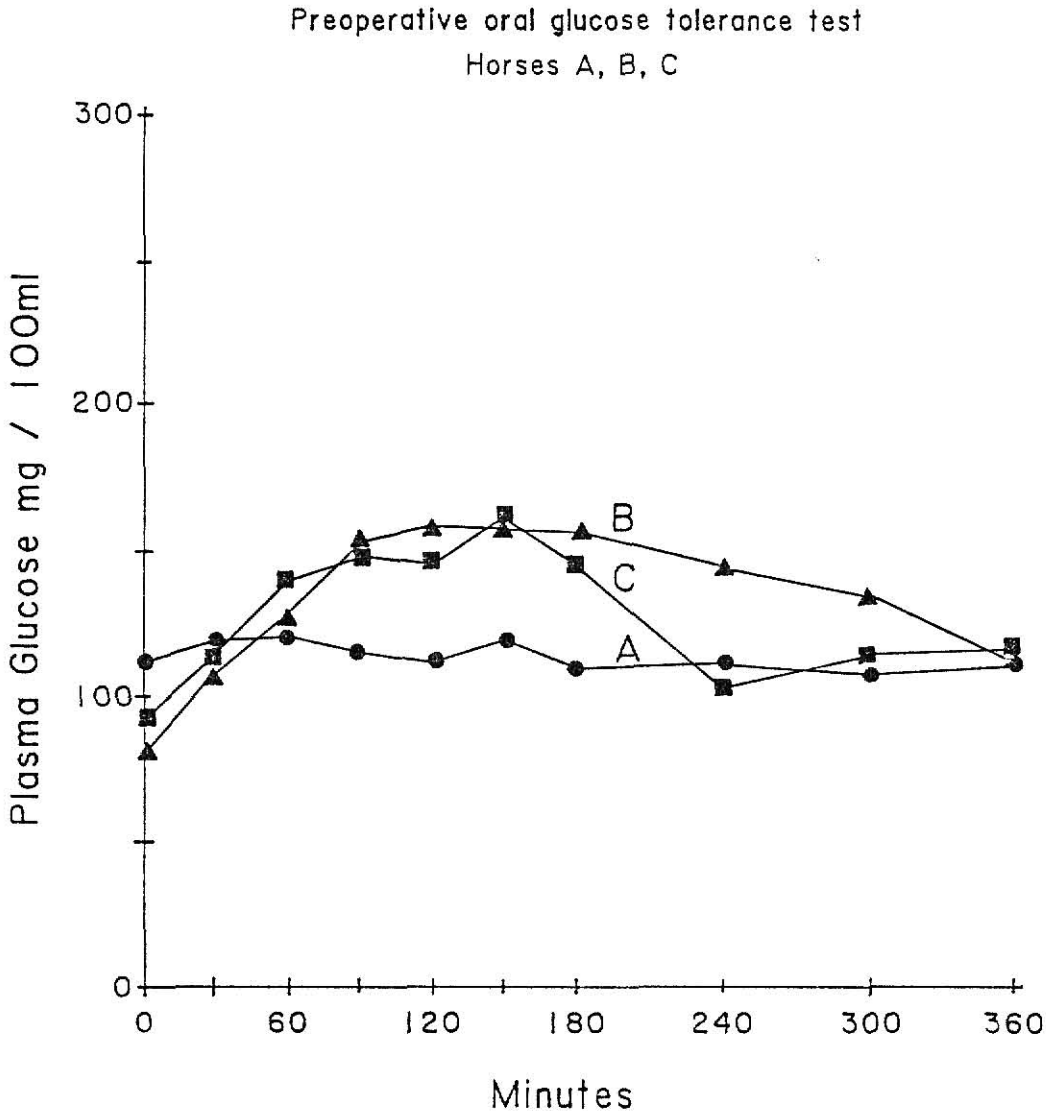

Preoperative oral glucose tolerance test
Horses A, B, C
Plasma Glucose mg / 100ml
300
200
100
0
0
60
120
180
240
300
360
Minutes
B
C
A

Figure 2

1 WEEK POSTOPERATIVE ORAL GLUCOSE TOLERANCE TEST
Horses A, B, C

Horses A and B exhibited some glucose absorption. However their % peaks were less than normal. Horse C exhibited a normal absorption curve because the peak blood glucose level was 105% of the resting level. Normal glucose absorption is considered to have occurred if the peak blood glucose is > 80% of the resting level. (See appendix table 8).

Horse	Peak % of resting level
A	29%
B	36%
C	105%

Figure 1

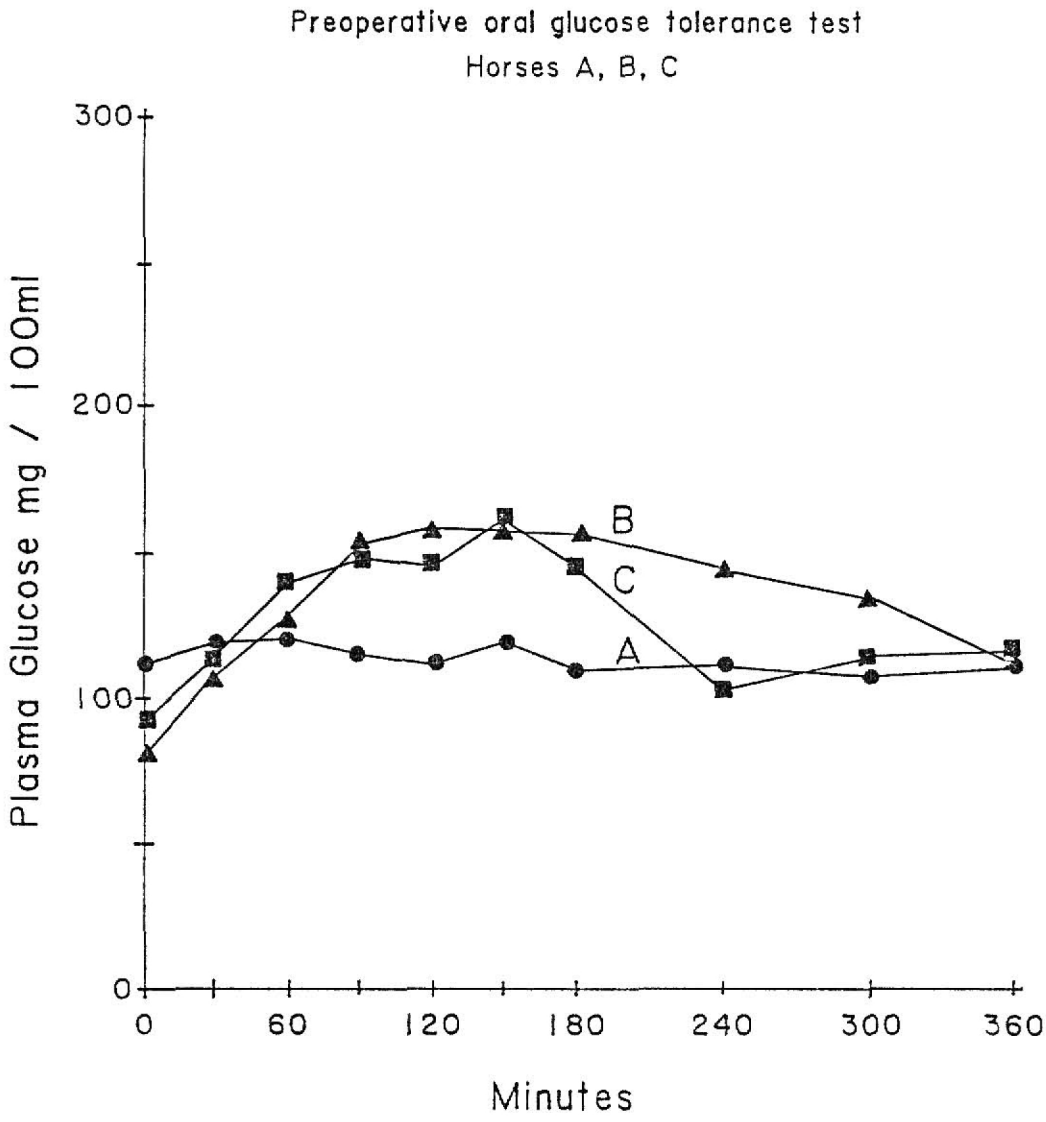

Preoperative oral glucose tolerance test
Horses A, B, C
Plasma Glucose mg / 100ml
300
200
100
0
0
60
120
180
240
300
360
Minutes
B
C
A

Figure 3

4 WEEK POSTOPERATIVE ORAL GLUCOSE TOLERANCE TEST
Horses A, B, C

Horse A exhibited a normal glucose absorption curve with a peak blood glucose of 92% of the resting level. Horses B and C absorbed some glucose. The exhibited abnormal glucose absorption curves because their peak blood glucose levels were <80% of their resting blood glucose levels. (See appendix table 9).

Horse	Peak % of resting level
A	92%
B	71%
C	24%

Figure 3

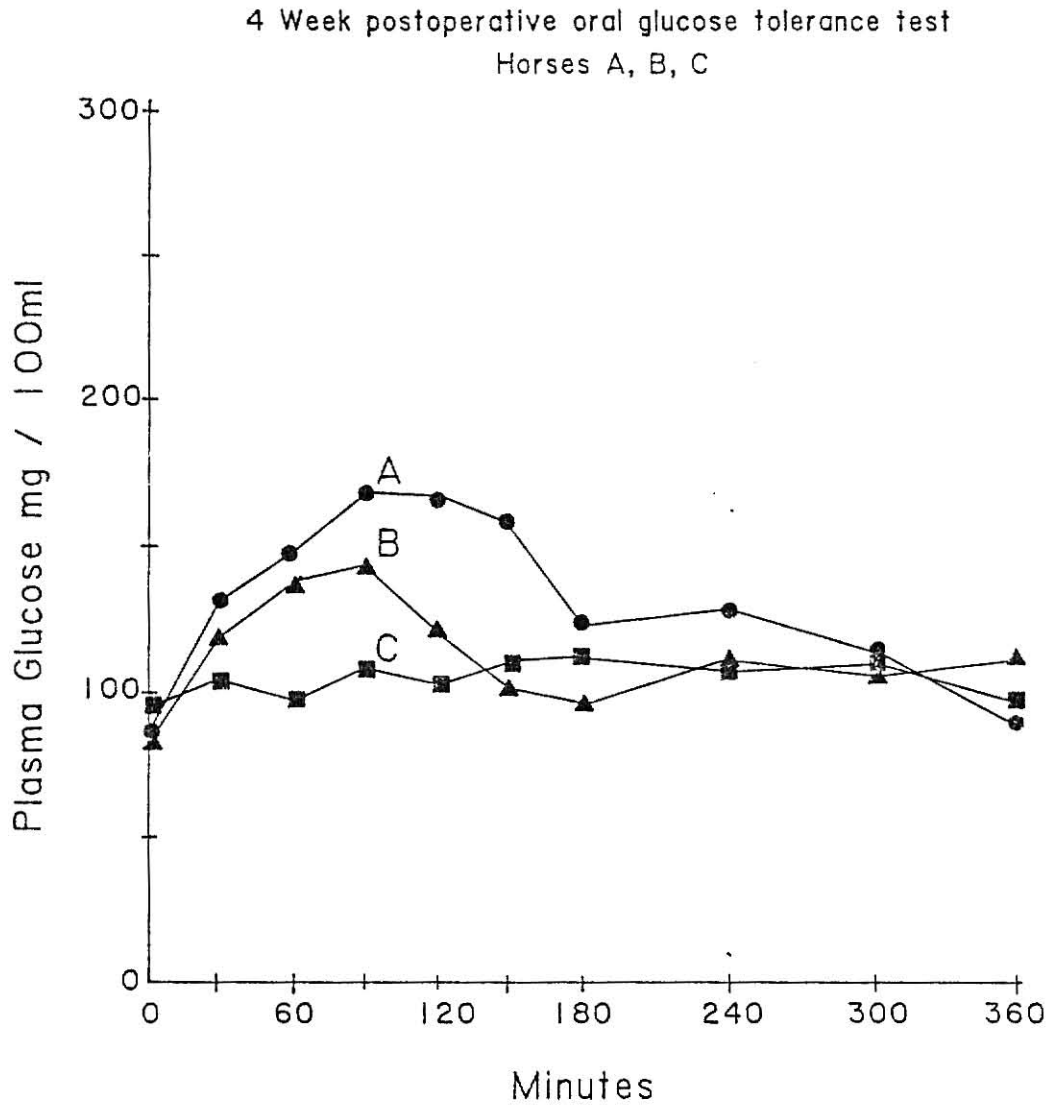

4 Week postoperative oral glucose tolerance test
Horses A, B, C
Plasma Glucose mg / 100ml
300
200
100
0
A
B
C
0
60
120
180
240
300
360
Minutes

Figure 4

PREOPERATIVE ORAL GLUCOSE TOLERANCE TEST
Horses D, E, F

Horses D and F exhibited normal glucose absorption curves. Horse E exhibited glucose absorption but because the peak blood glucose was < 80% of the resting blood glucose level the absorption was considered abnormal. (See appendix table 7).

Horse	Peak % of resting level
D	104%
E	67%
F	103%

Figure 4

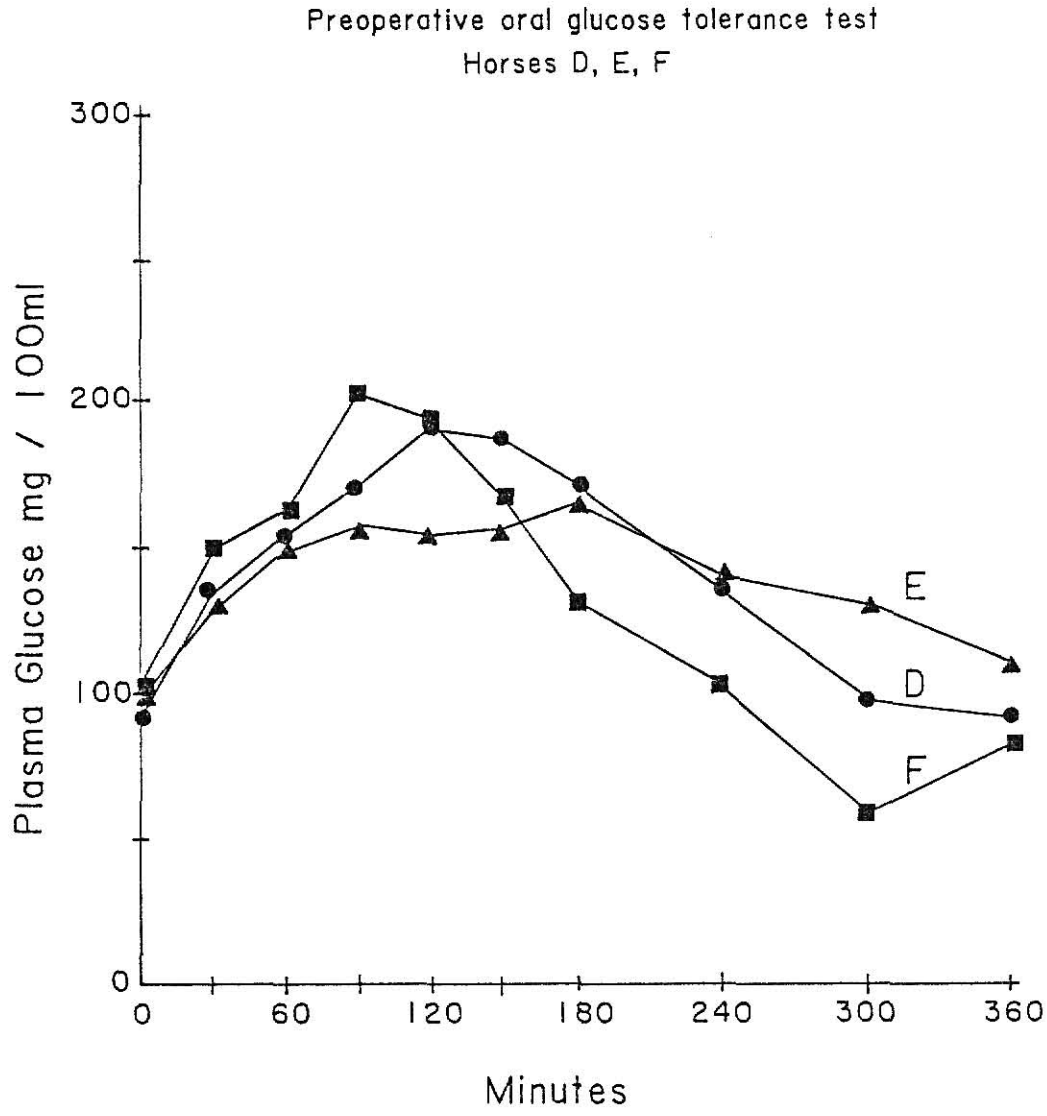

Preoperative oral glucose tolerance test
Horses D, E, F
Plasma Glucose mg / 100ml
300
200
100
0
0
60
120
180
240
300
360
Minutes
E
D
F

Figure 5

1 WEEK POSTOPERATIVE ORAL GLUCOSE TOLERANCE TEST
Horses D, E, F

Horses D and F exhibited normal glucose absorption curves. Horse E exhibited glucose absorption. However, the peak blood glucose was < 80% of the resting blood glucose level and therefore considered abnormal. (See appendix table 8).

Horse	Peak % of resting level
D	80%
E	51%
F	142%

Figure 5

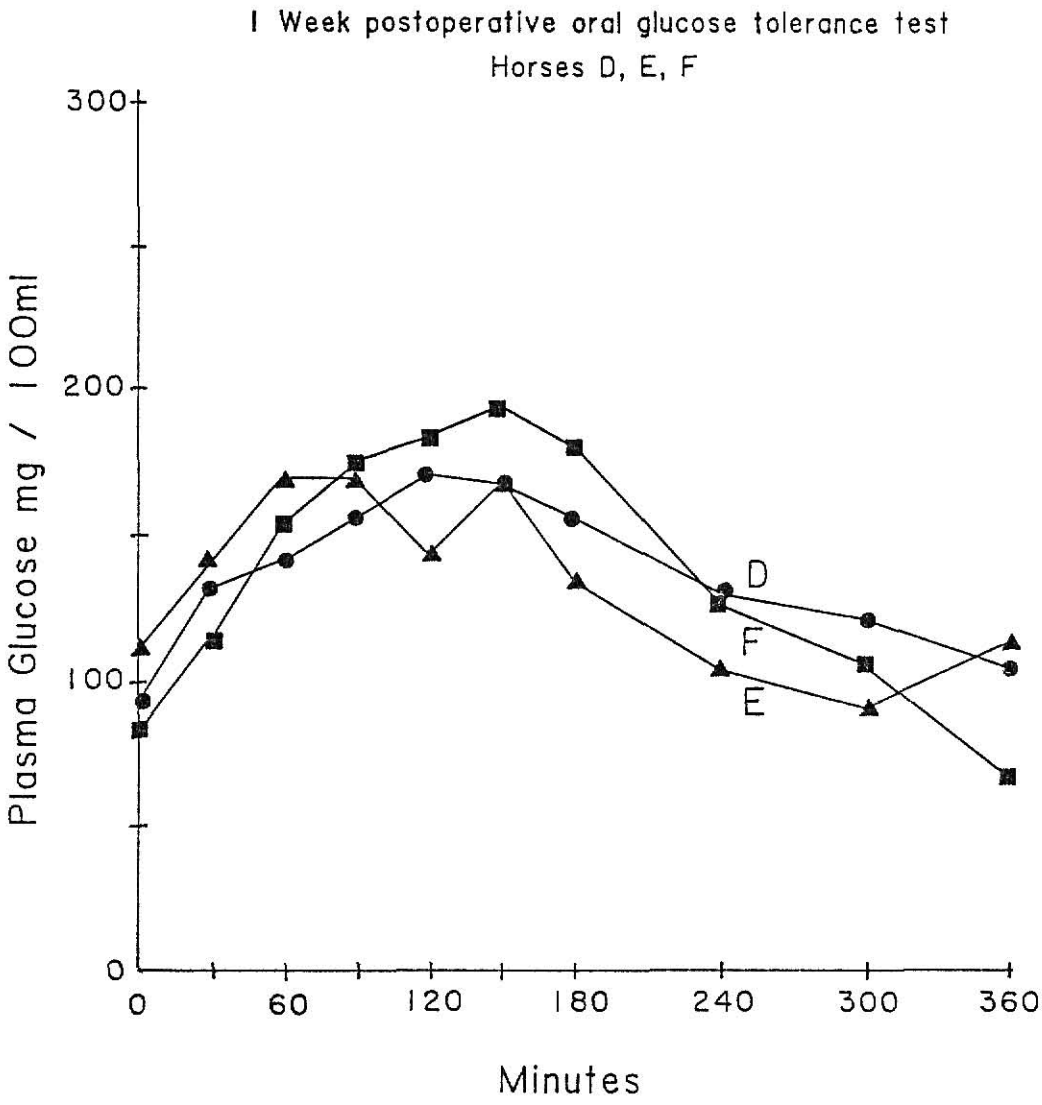

1 Week postoperative oral glucose tolerance test
Horses D, E, F
300
200
100
0
Plasma Glucose mg / 100ml
0
60
120
180
240
300
360
Minutes
D
F
E

Figure 6

4 WEEK POSTOPERATIVE ORAL GLUCOSE TOLERANCE TEST
Horses D, E, F

Horses D and E exhibited glucose absorption. However the peaks were < 80% of the resting blood glucose levels and were considered abnormal. Horse F exhibited a normal glucose absorption curve. (See appendix table 9).

Horse	Peak % of resting level
D	55%
E	46%
F	216%

Figure 6

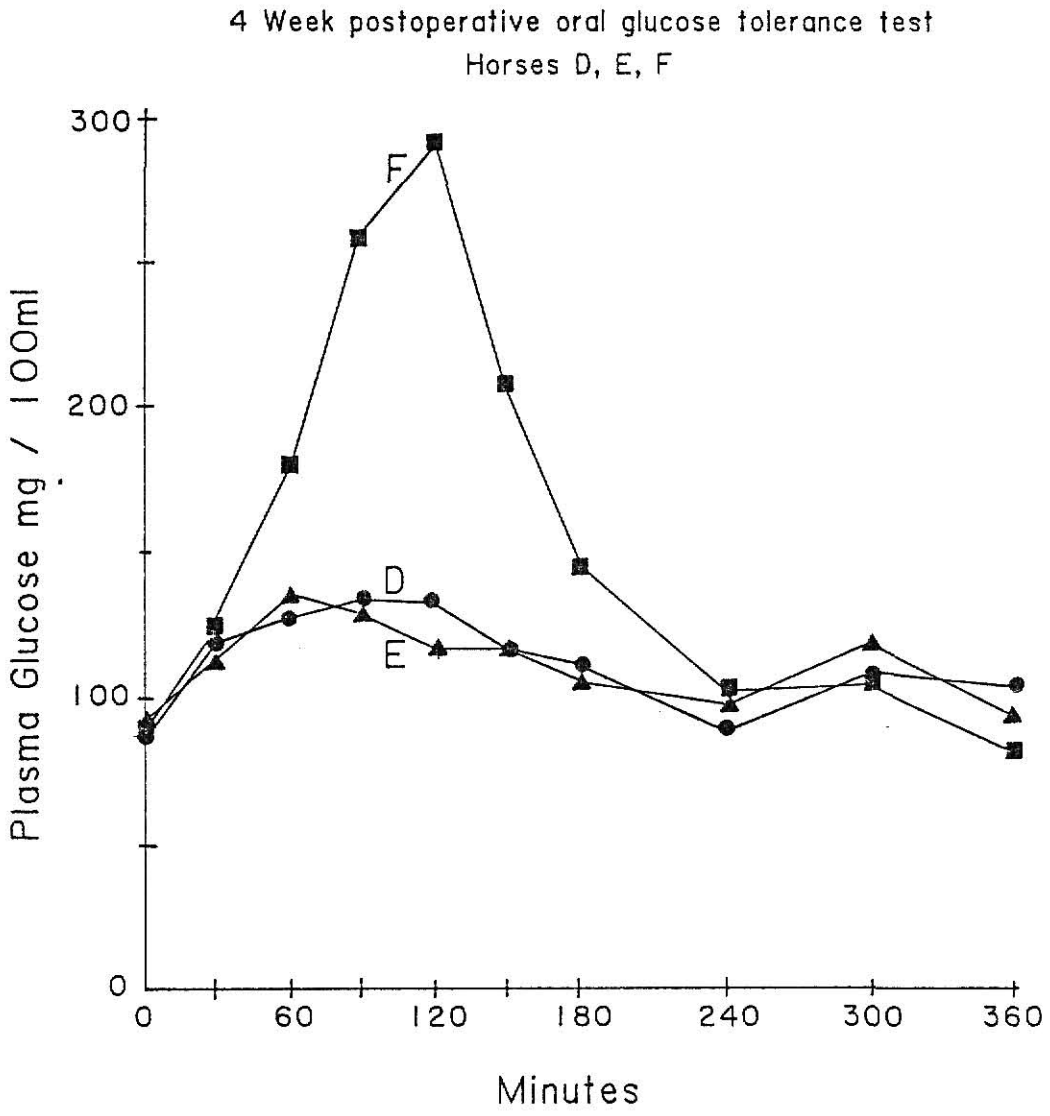

4 Week postoperative oral glucose tolerance test
Horses D, E, F
Plasma Glucose mg / 100ml
300
200
100
0
F
D
E
0
60
120
180
240
300
360
Minutes

Figure 7

PREOPERATIVE ORAL GLUCOSE TOLERANCE TEST
Horses G, H, I

Horses G and H possessed normal glucose absorption. Horse I absorbed glucose but because the peak blood glucose was < 80% of the resting level the glucose absorption was considered abnormal. (See appendix table 7).

Horse	Peak % of resting level
G	129%
H	123%
I	56%

Figure 7

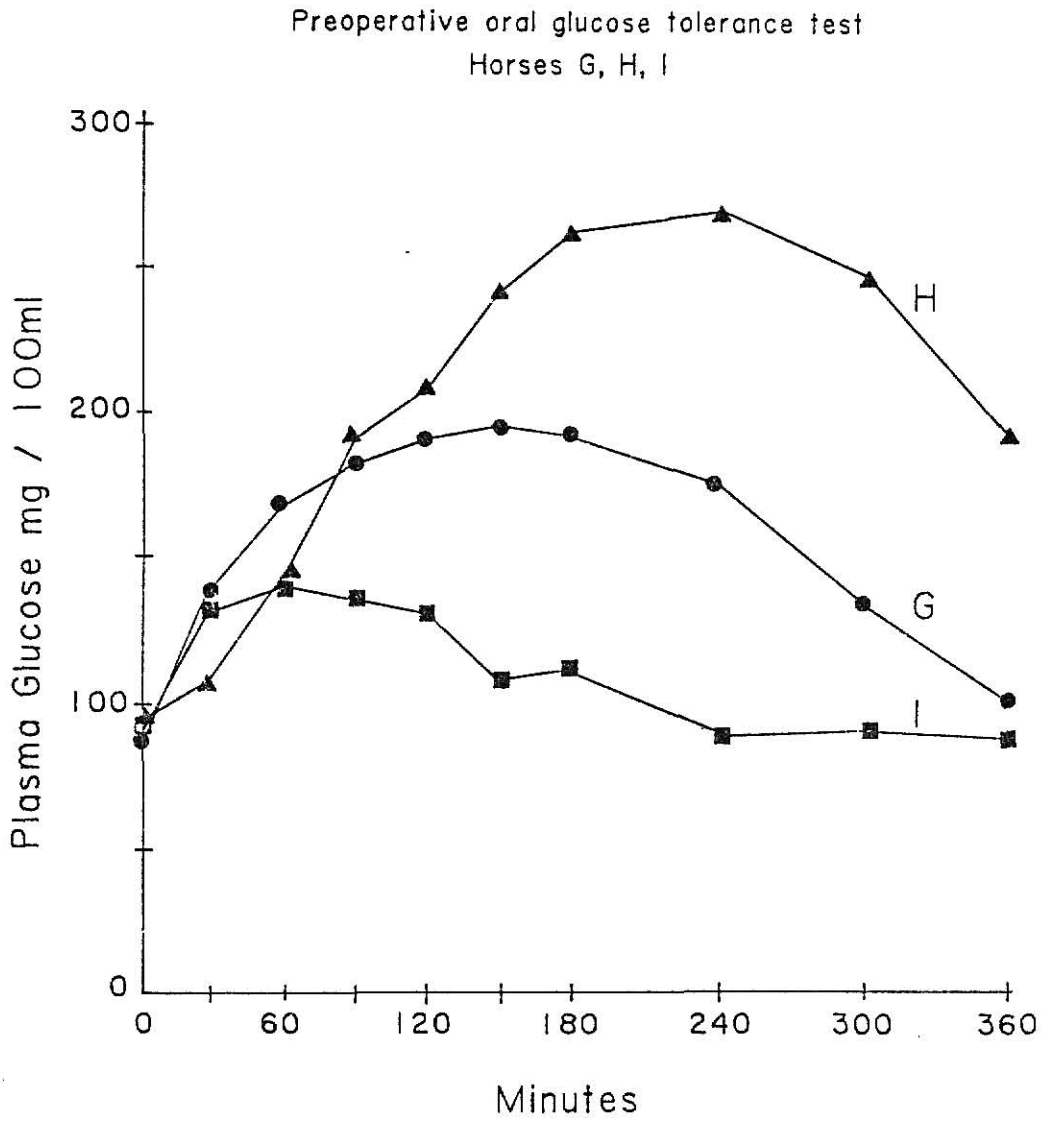

Preoperative oral glucose tolerance test
Horses G, H, I
Plasma Glucose mg / 100ml
300
200
100
0
0
60
120
180
240
300
360
Minutes
H
G
I

Figure 8

1 WEEK POSTOPERATIVE ORAL GLUCOSE TOLERANCE TEST
Horses G, H, I

All three horses exhibited some glucose absorption. However all three curves were considered abnormal because the peak blood glucose levels were < 80% of the resting blood glucose levels. (See appendix table 8).

Horse	Peak % of resting level
G	51%
H	44%
I	18%

Figure 8

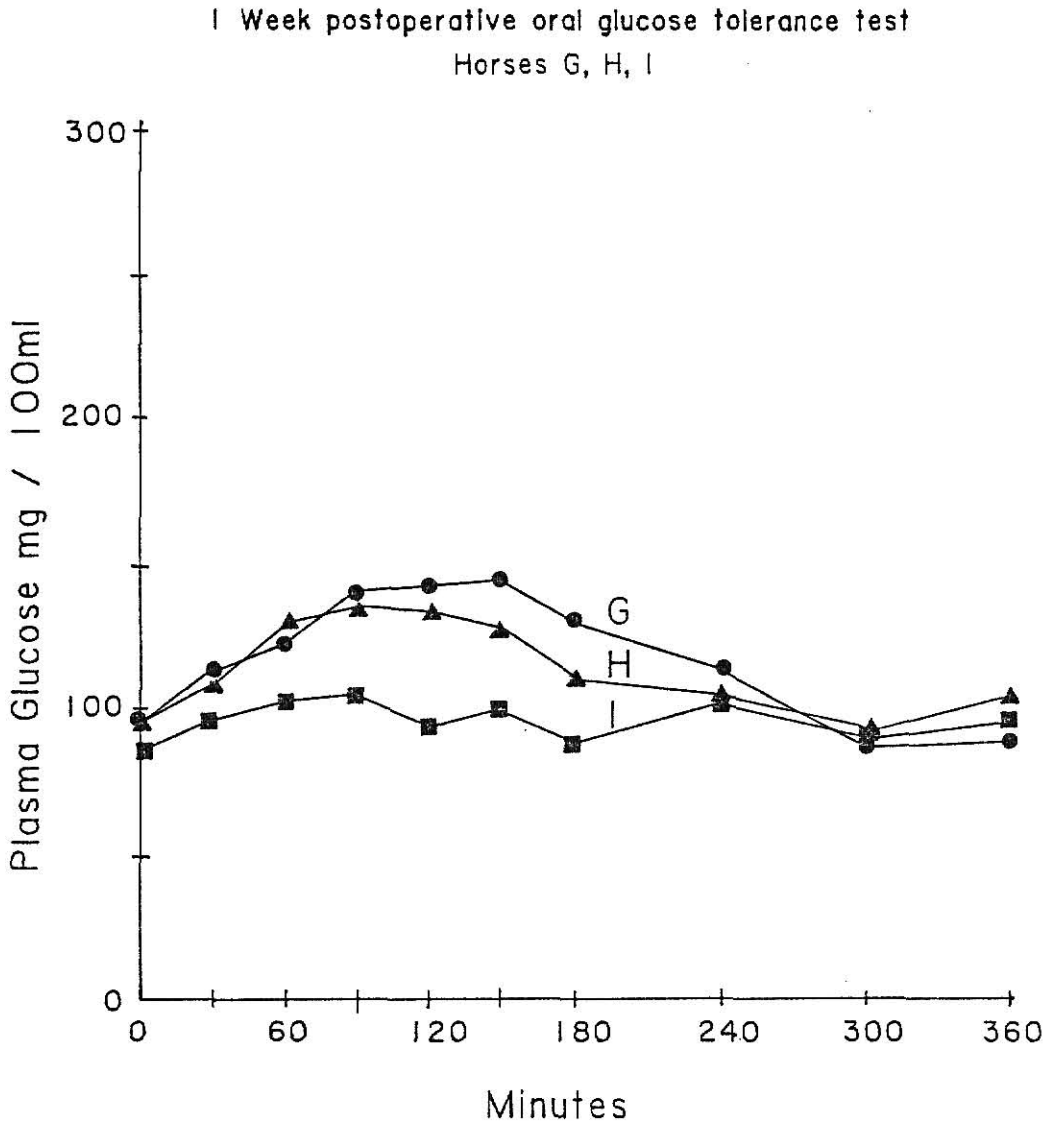

1 Week postoperative oral glucose tolerance test
Horses G, H, I
300
200
100
0
Plasma Glucose mg / 100ml
G
H
I
0
60
120
180
240
300
360
Minutes

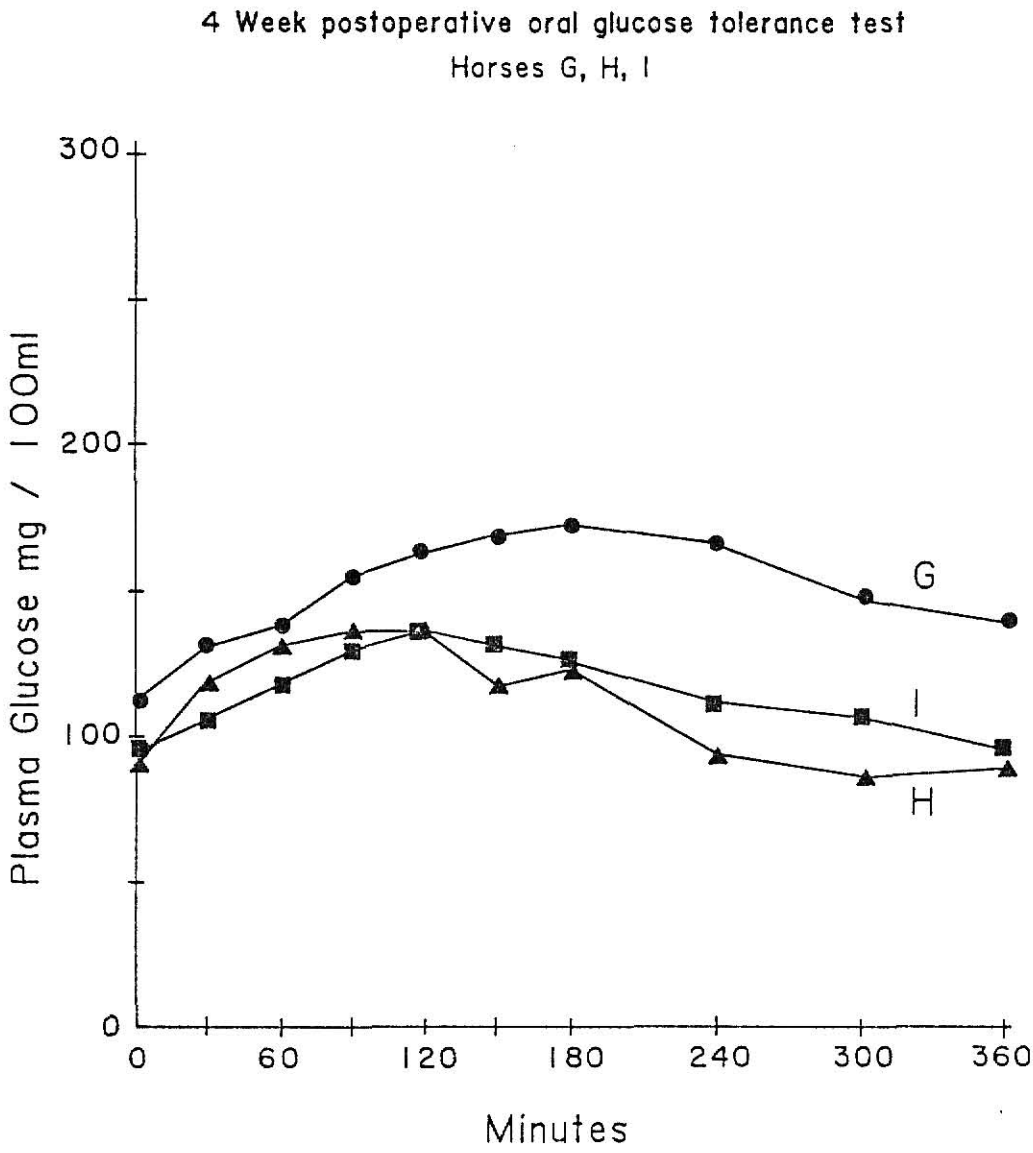
Figure 9

4 WEEK POSTOPERATIVE ORAL GLUCOSE TOLERANCE TEST
Horses G, H, I

All three horses exhibited some glucose absorption. However all three curves were considered abnormal because the peak blood glucose levels were < 80% of the resting blood glucose levels. (See appendix table 9).

Horse	Peak % of resting level
G	52%
H	44%
I	40%

Figure 9


4 Week postoperative oral glucose tolerance test
Horses G, H, I
300
200
100
0
Plasma Glucose mg / 100ml
0
60
120
180
240
300
360
Minutes
G
I
H

DISCUSSION

None of the horses died from the removal of varying amounts of small intestine. Weight loss occurred in horses G and I which had 9.2 m (30') or > 50% of the small intestine removed. Both of these horses were partially anorectic and required tube feeding for one week. Horse G was tube fed starting the third postoperative day and horse I was tube fed starting 5 days following a second surgery to correct a large bowel displacement that occurred one week following small intestine resection. The rate of weight loss decreased while these horses were bring tube fed.

In horse H where 9.2 m represented < 50% of the small intestine a weight gain of 4.5 kg was observed by the sixth postoperative week and a total of 7.7 kg by the ninth postoperative week. The seventh through ninth post-operative weeks free choice grass pasture was added to the test diet.

Horses in group I and group II gained weight with the exception of horse F. Horse F contracted transient diarrhea following surgery. Salmonella type C was cultured and the underlying cause was attributed to stress. This horse regained weight to the preoperative level and continued weight gain following the completion of the study.

All of the horses except G and I ate the full ration throughout the project. Horses G and I consumed ¼ to ½ of their daily ration following surgery. Group III horses seemed to prefer roughage following the surgery both during and after the study while the others did not. The lack of a large segment of small intestine and consequently soluble carbohydrate digestive and absorption area in conjunction with the ability of the equine large intestine to convert nonsoluble carbohydrates into volatile fatty acids

may indicate the horses in Group III were changing their diet to adapt to the altered digestive tract. The two horses (G and I) that lost weight while partially anorectic on the pelleted ration, exhibited a slower weight loss when tube fed and when placed on pasture. Their appetite for and consumption of the pelleted ration gradually improved several weeks following surgery although their weights continued to decrease. Other possible reasons for a roughage preference may be related to a change in the volatile fatty acid ratios or pH levels in the large bowel as a result of the removed small intestine.[2,5,8,25]

Anorexia may have been caused by the large amount of small intestine removed and possible resultant pain from stretching of mesentery and intestinal ligaments due to altered small and large bowel position. Part of the weight loss may well have been due to anorexia as evidenced by the slower decrease in weight loss while horses G and I were being tube fed. However, the removal of > 50% of the total small intestine in horses G and I have resulted in the lack of sufficient small intestine digestive and absorptive area. Hence the possible lack of carbohydrate energy and proteins may have been primarily responsible for the weight loss. The mechanisms of weight loss in these horses warrants further study. Certainly malabsorption of protein and ample simple sugars could be a contributing factor.

Weight gain in horse H and groups I and II may be explained by the existence of sufficient digestive and absorptive area in the remaining small intestine or to the production and absorption of additional volatile fatty acids in the large bowel. Sufficient small intestine surface area seems the likely explanation for the weight increases. In horse H and horses in groups I and II where < 50% of the small intestine was removed weight gain occurred. Hence the weight gains may be directly related to sufficient small intestine surface area and function.

Absence of changes in fecal consistency support the hypothesis that diarrhea, at least in adult horses, is related primarily to malfunction or inadequate function of the large bowel.[4,107,197] The large capacity and bacterial flora of the equine large intestine is apparently able to handle the nonmetabolized dietary components leaving the small intestine.[119] Characterization of alterations in compartmentilization, ingesta flow pattern, or fluid fluxes in the large bowel would be needed to define the mechanisms of these actions.

As expected evaluation of BUN, creatinine, Cl, K, and Na revealed no evidence of abnormalities (appendix). Normal liver function with synthesis of urea, and normal renal function would preclude any elevation caused by the possible increase in catabolism of protein. Normal renal function and normal large bowel physiology would permit the maintenance of normal Cl, K, and Na equilibrium.

Because of the inability of the central nervous system to store energy, normal resting blood glucose values would be expected even with a lack of carbohydrate absorption.[133] The balancing influences of insulin versus glucagon, epinephrine, glucocorticoids, and a direct effect of decreased glucose on the liver would keep blood glucose levels within normal limits. Maintenance of blood glucose levels as well as electrolytes and minerals has been demonstrated in fasted horses.[139-141,143]

Because calcium is absorbed in the proximal small intestine and phosphorus is absorbed in the small intestine as well as the large bowel, they would also be expected to be within normal limits for this short term study. Absorption studies with labelled Ca and P over a longer period in conjunction with urinary clearance values would be necessary to determine any effects of massive small intestine resection.

Transient postoperative neutrophilia and increased fibrinogen may be related to the surgery and the inflammation associated with healing of the abdominal incision and enterotomies, as well as to varying degrees of peritonitis. A more prolonged mature neutrophilia in horse F was attributed to the development of diarrhea and incision dehiscence. Horse I also displayed a prolonged but transient mature neutrophilia related to peritonitis and dehiscence of the abdominal incision following the second abdominal surgery.

Post surgical increases in AAT can be attributed to muscle damage and intestinal manipulation as well as to hepatocellular changes associated with general anesthesia.[196] Horse F's second transient elevation following the development of diarrhea may be attributed to intestinal damage and to destruction of hepatocytes. The fluctuation of AAT observed in horse G is presumed to be the result of conversion of muscle protein to glucose. Intestinal damage resulting from stretch and direct injury, ongoing intestinal adaptation, and partial small bowel obstruction are also considered possible sources. Ongoing hepatocyte damage in horse G due to hypoxia, endotoxin, cholestasis, and fibrosis may be a plausible explanation for the increase in AAT. In spite of elevated AAT levels the final three weeks of the study, however, sorbitol dehydrogenase (SDH) values, revealed no liver involvement (appendix).

Serum alkaline phosphatase (SAP) levels varied widely in each group of horses both preoperatively and postoperatively. Because SAP comes from several different sources a number of plausible explanations exist.[63,198,200] All horses exhibited transient peaks possibly from liver and intestinal sources following surgery. Baseline increases for horses A, E, F, and H could be related to osteoblastic sources since these horses were 3 to 4 years old. Horse D displayed elevated SAP levels which would seem to be nonhepatic in origin.

Group III displayed the most marked, long lasting elevation in SAP. Since assays of AAT and SDH activities did not indicate hepatotoxicity, intestinal mucosa was a likely source. Roberts has shown horse intestinal mucosa to be rich in AP except for the terminal small colon and rectum.[63] Prolonged small intestinal adaptation, continuous intestinal damage resulting from functional changes, damage due to abnormal stretch as a result of organ placement, partial obstruction, or venous and lymphatic obstruction due to mesentery closure, could account singly or collectively for this type of an increase. Induction of SAP from endogenous glucocorticoid release in response to stress and malabsorption was also possible. However, concomitant eosinopenia, lymphopenia, or hyperglycemia would be expected and this did not occur.

Irrespective of the cause of SAP elevation it is reflective of sustained metabolic or structural changes attendant to extensive resection and the poor prognosis traditionally associated with such interventions.

Total serum protein (TSP) and albumin concentrations are best interpreted together because they are closely related. Consideration of the albumin:globulin (A:G) ratio also lends insight into clinical events.

Abrupt decreases of both TSP and albumin in all horses were related to blood loss at surgery. Changes in the A:G ratio were associated with disproportionate increases or decreases in the globulin fraction related to inflammatory and immunologic reactions (appendix). Horse F displayed low normal albumin (2.7 g %) at the beginning of the study with a high normal TSP (7.5 g %) and high globulin fraction (4.8 g %). The consistently low albumin and decrease in TSP were associated with protein losing gastroenteropathy as a sequel to diarrhea.[201-204]

Group III exhibited the most marked decrease in albumin and TSP levels with albumin levels continuing to drop throughout the study. Loss was attributed to trauma of handling intestines, loss around the anastomotic site, and

possible loss due to lymphatic blockage as a result of mesenteric closure or to stretch resulting from abnormal intestinal location following surgery.

Decreases in albumin and globulin synthesis could account for hypoalbuminemia over a more extended period of time. However, in the horse, the half life of albumin is 18-21 days and approximately 11 days for gamma globulins and this would not account for the dramatic and rapid decreases observed. In most cases decrease of albumin levels precede the development of hypoproteinemia [111] in dietary protein deficiencies.[204-209]

These alterations in protein values also explain in part why extensive intestinal surgery in horses results in so many complications. Loss of immunologlobulins, osmotic pressure, and metabolite transport and binding potential are serious events which are usually difficult to quantify and correct.

Oral glucose tolerance tests (OGTT) are employed to assess the functional absorptive capacity of the small intestine. Normal small intestine function is considered to be characterized by a post-glucose-administration blood glucose peak of 80% or more above pretest levels.[1,133,135,187] Four of the horses (A, C, E, and I) displayed abnormal presurgical tolerance tests. If simultaneous weight loss occurred this would have been indicative of malabsorption. The other horses had normal presurgical OGTT by achieving glucose peaks above 80% of pretest levels. Group I and II experienced mixed results for the 1 week and 4 week postoperative OGTT. All of these horses gained weight throughout the study and consequently no correlation with malabsorption could be made. Unlike the other horses in Group I and II, horse F did exhibit a marked increase in peak blood glucose levels. The first postoperative OGTT was done following the cessation of diarrhea. The peak was achieved 60 minutes later than in the presurgical OGTT. The four week OGTT reached peak levels only 30 minutes later than in the presurgical OGTT, suggesting that diarrhea increased time to peak blood levels.

The postoperative OGTT for Group III horses exhibited decreases in glucose peaks. However, with increasing time from surgery the peak blood glucose levels increased. A 10 week post surgical OGTT on horse G continued this trend. Although the peak blood glucose percentages were less than 80% following surgery, some glucose was being absorbed even in the horses with > 50% of their small intestine removed.

In several instances the blood glucose peaks occurred following the normally accepted 120 minute period and the 6 hour levels were often above the pretest ranges. In addition wide fluctuations sometimes occurred in the final glucose assays of several tolerance tests. These latter changes may be related to insulin regulation of elevated blood glucose levels and exemplify the reason some investigators prefer xylose absorption to glucose absorption tests.

The increasing post surgical peaks in blood glucose levels in Group III support the possibility of the occurrence of adaptive small intestine changes. These changes could either be the result of larger numbers of epithelial cells or from an increased functional capacity of the cells already present. The SAP levels in this group could be further evidence of the intestinal adaptation process especially with horses G and I. Further work needs to be done on horses with a majority of their small intestine removed to clarify and quantify these effects. The poor correlation between OGTT, length of bowel removed, and weight gain (or loss) strongly suggests that the role of OGTT in diagnosis of malabsorption should be re-assessed.

The changes observed and discussed appear to be related to intestinal and not hepatic or renal changes.

Complications arising from the abdominal surgery and intestinal resection included the development of postoperative diarrhea, the occurrence of a large bowel displacement, incisional dehiscence, and minor skin suture abscesses.

SUMMARY AND CONCLUSIONS

Nine mixed breed horses of both sexes between 3 and 17 years of age were used to study certain effects of the removal of varying lengths of the distal small intestine. Following the completion of a conditioning program which included a standardized pelleted ration, the horses were randomly divided into three equal groups with 0, 4.6 m (15'), and 9.2 m (30') of distal small intestine removed from Group I, II, and III respectively. Monitoring of patient progress was accomplished by clinical observations, serial body weights, serial blood counts and blood chemistry, fecal evaluation, and preoperative and postoperative oral glucose tolerance tests. The laboratory data from the nine horses is discussed. At the termination of the study, the horses were returned to a roughage diet in preparation for further digestion trials.

Although the number of animals used in this project did not permit statistical analysis, the results do suggest general trends. Gross fecal changes did not occur in any group, supporting the hypothesis that diarrhea in adult horses is a result of malfunctioning of the large bowel.

All horses lived and weight gain occurred in Group I, II and 1 horse in Group III in which the 9.2 m of small intestine removed was < 50% of the total length of the small intestine. Weight loss occurred in the two horses in Group III in which the 9.2 m of small intestine removed constituted > 50% of the total length of the small intestine.

Transient albumin and globulin deficits, attributed to loss at surgery, occurred in all three groups. However, Groups II and III displayed more prolonged deficits possibly related to lymphatic obstruction, protein losing gastroenteropathy, and peritonitis.

Little correlation was observed between the glucose absorption tests, weight loss or gain, and possible malabsorption. The horses in Group III displayed some change in postoperative glucose absorption. In each case improvement in absorption occurred with increasing time from surgery. Intestinal adaptation which occurs in humans and other animals may occur in horses. Histopathologic examination is needed for verification.

Group III horses preferred roughage when compared to Groups I and II, possibly as a means of adapting dietary intake to the residual functional capacity of the digestive tract. This would be consistent with decreased soluble carbohydrate and protein digestion and absorption in the reduced small intestine, thereby increasing the demands on the large intestine and its conversion of carbohydrates into volatile fatty acids. Since the horse appears to be more efficient in deriving energy from carbohydrate as compared to protein utilization in the large intestine, long term effects of the removal of a major portion of the small intestine absorptive area may be more devastating to protein metabolism than to carbohydrate metabolism.

Further investigation to determine the specific effects of small intestine function, surface area, and intestinal length with respect to total body surface area in regards to carbohydrate and protein digestion and absorption needs to be done.

BIBLIOGRAPHY

1. Roberts, M. C.: Carbohydrate digestion and absorption studies in the horse. Res. Vet. Sci. 18:64-69, 1975.

2. Hintz, H. F.; Schryver, H. F.: Digestive physiology of the horse. J. Eq. Med. Surg. 2:147-150, 1978.

3. Hintz, H. F.: Digestive physiology of the horse. J. S. Afr. Vet. Ass. 46:13-16, 1975.

4. Argenzio, R. A.: Functions of the equine large intestine and their interrelationship in disease. Cornell Vet. 65:303-330, 1975.

5. Roberts, M. C.: Carbohydrate digestion and absorption in the equine small intestine. J. S. Afr. Vet. Assa. 46:19-27, 1975.

6. Hintz, H. F.; Schryver, H. F.; Stevens, C. E.: Digestion and absorption in the hindgut of nonruminant herbivores. J. An. Sci. 46:1803-1807, 1978.

7. Reitnour, C. M.; Baker, J. P.; Mitchell, G. E.; Little, C. O.: Nitrogen digestion in different segments of the equine digestive tract. J. An. Sci. 29:332-335, 1969.

8. Householder, D. D. 1978. Prececal, postileal and total tract digestion and growth performance in horses fed concentrate rations containing oats or sorghum grain processed by crimping or micronizing. Ph.D. Dissertation. Texas A & M University.

9. Peterson, F. B.; Stewart, J. V.: Experimental ileocecal anastomosis in the horse. J. Eq. Med. Surg. 2:461-464, 1978.

10. Lowe, J. E.: Intussusception of the ileum in a horse. A case report. Cornell Vet. 56:51-53, 1966.

11. Pearson, H; Gibbs, C.: Review of Sixty Equine Laparotomies. Eq. Vet. J. 2:60-63, 1970.

12. Svendsen, C. K.; Hjortkjaer, R. K.; Hesselholt, M.: Colic in the horse. A clinical and clinical chemistry study of 42 cases. Nord. Vet. Med. 31 (suppl. 1):1-32, 1979.

13. Robertson, J. T.: Surgical diseases of the small intestine, in proceedings 26th Annual Meeting, Am. Assoc. Eq. Pract., 201-209, 1980.

14. Freeman, D. E.: Koch, D. B.; Boles, C. L.: Mesodiverticular bands as a cause of small intestinal strangulation and volvulus in the horse. J. Am. Vet. Med. Assoc. 175:1089-1094, 1979.

15. Schneider, J. E.; Kennedy, G. A.; Leipold, H. W.: Muscular hypertrophy of the small intestine in a horse. J. Eq. Med. Surg. 3:226-228, 1979.

16. Austin, R. J.: Disseminated phycomycosis in a horse. Can. Vet. J. 17:86-89, 1976.

17. Adams, I. B.; McIlwraith, C. W.: Abdominal crisis in the horse: a comparison of presurgical evaluation with surgical findings and results. Vet. Surgery 7:63-69, 1978.

18. Robertson, J. T.; Johnson, F. M.: Surgical correction of cecocolic intussesusception in a horse. J. Am. Vet. Med. Assoc. 176:223-224, 1980.

19. van der Gaag, I; can Bruinessen-Kapsenberg, E. G.; Dik, K. J.; Kroneman, J.: Congenital duodenal stenotic diaphragm in a foal. Vet. Quarterly 3:131-135, 1981.

20. Barth, A. D.; Barber, S. M.; McKenzie, N. T.: Pyloric stenosis in a foal. Can. Vet. J. 21:234-236, 1980.

21. Kopf, V. N.; Niebaur, G. W.; Rettenbacher, G.: Innere verletzungen als ursache oder folge von ileus beim pferd. Wien tierarztl Mschr 66:233-247, 1979.

22. Wagner, P. C.; Grant, B. D.; Schmidt, J. M.; Hallowell, A. L.: Duodenal stricture in a foal. Eq. Pract. 1:29-35, 1979.

23. Norrie, R. D.: Heistand, D. L.: Chronic colic due to an omental adhesion in a mare. J. Am. Vet. Med. Assoc. 167:54-55, 1975.

24. Bennet, D. G.: Predisposition to abdominal crisis in the horse. J. Am. Vet. Med. Assoc. 161:1189-1194, 1972.

25. Mason, T. A.: Johnston, D. E.; Wallace, C. E.: Christie, B. A.: Laparotomy in equine colic - a report of thirteen clinical cases. Austral Vet. J. 46:349-355, 1970.

26. Horney, F. D.; Funk, K. A.: Ileal myotomy in the horse. Mod. Vet. Pract. 52:49-50, 1971.

27. Lindsay, W. A.; Confer, A. W.; Ochoa, R.: Ileal smooth muscle hypertrophy and rupture in a horse. Eq. Vet. J. 13:66-67, 1981.

28. Rooney, J. R.; Jeffcott, L. B.: Muscular hypertrophy of the ileum in a horse. Vet. Rec. 83:217-219, 1968.

29. Valdez, H.; McLaughlin, I. A.; Taylor, T. S.: A case of colic due to an abscess of the jejunum and its mesentery. J. Eq. Med. Surgery 3:36-38, 1979.

30. Edwards, G. B.: Obstruction of the ileum in the horse: a report of 27 clinical cases. Eq. Vet. J. 13:158-166, 1981.

31. Donawick, W. J.; Christie, B. A.; Stewart, J. V.: Resection of diseased ileum in the horse. J. Am. Vet. Med. Assoc. 159:1146-1149, 1971.

32. Owen, R.; Physick-Sheard, P. W.; Hilbert, B. J.; Horney, F. D.; Butler, D. G.: Jejuno- or illocecal anastomosis performed in seven horses exhibiting colic. Can. Vet. J. 16:164-169, 1975.

33. Hjortkaer, R. K.; Svendsen, C. K.: Simulated small intestinal volvulus in the anesthetized horse. Nord. Vet. Med. 31:466-483, 1979.

34. Scott, E. A.; McDole, M. G.: Fibrinous enteritis with fibrin cast formation in a horse. Vet. Med. Sm. An. Clinic 74:382-384, 1979.

35. Coffman, J. R.: Diagnosis and management of acute abdominal diseases in the horse (part 1). Vet. Med. Sm. An. Clinic 65:669-673, 1970.

36. Pearson, H.; Messerny, A.; Pinsent, P. J. M: Surgical treatment of abdominal disorders in the horse. J. Am. Vet. Med. Assoc. 159:1344-1352, 1971.

37. Meuten, D. J.; Butler, D. G.; Thompson, G. W.; Lumsden, J. H.: Chronic enteritis associated with malabsorption and protein-losing enteropathy in the horse. J. Am. Vet. Med. Assoc. 172:326-332, 1978.

38. Merritt, A. M.; Cimprich, R. E.; Beech, J.: Granulomatous enteritis in nine horses. J. Am. Vet. Med. Assoc. 169:603-609, 1976.

39. White, N. A.: Intestinal infarction associated with mesenteric vascular thrombotic disease in the horse. J. Am. Vet. Med. Assoc. 178:259-262, 1981.

40. Dixon, R. J.; Brownlow, M. A.: Parasitic arteritis and its consequences in a foal. Austral Vet. J. 54:494-495, 1978.

41. Baker, R. H.: Acute necrotizing pancreatitis in a horse. J. Am. Vet. Med. Assoc. 172:268-270, 1978.

42. Lowe, J. E.: Intussusception of the ileum in a horse. A case report. Cornell Vet. 56:51-53, 1966.

43. Schoeb, T. R.; Panciera, R. J.: Pathology of blister beetle (Epicanta) poisoning in horses. Vet. Pathol. 16:18-31, 1979.

44. Huskamp, B.: Some problems associated with intestinal surgery in the horse. Eq. Vet. J. 9:111-115, 1977.

45. Vaughan, J. T.: Surgical management of abdominal crisis in the horse. J. Am. Vet. Med. Assoc. 161:1199-1212, 1972.

46. de Boom, H. P. A.: Functional anatomy and nervous control of the equine alimentary tract. J. S. Afr. Vet. Assoc. 46:5-11, 1975.

47. Crawford, B. H., Jr. 1971. Digestion of energy sources in different segments of the equine digestive tract. Ph.D. Dissertation. University of Kentucky, Lexington.

48. Bienfet, V.: Physiologie digestive du cheval. Ann. Med. Vet. 121: 257-261, 1977.

49. Janis, C.: The evolutionary strategy of the equidae and the origins of rumen and cecal digestion. Evolution 30:757-774, 1976.

50. Robinson, D. W.; Slade, L. M.: The current status of knowledge on the nutrition of equines. J. An Sci. 39:1045-1063, 1974.

51. Fonnesbeck, P. V.: Digestion of soluble and fibrous carbohydrate of forage by horses. J. An. Sci. 27:1336-1344, 1968.

52. Hintz, H. F.: Energy. J. Eq. Med. Surg. 2:300-304, 1978.

53. Crawford, B. H.; Baker, J. P.; Lieb, S.; Mitchell, G. E.: Starch and cellulose digestion by equine intestinal microorganisms. J. An. Sci. 27:1151, 1968.

54. Fonnesbeck, P. V.; Lydman, R. K.; Vander Noot, G. W.; Symons, L. D.: Digestibility of the proximate nutrients of forage by horses. J. An. Sci. 26:1039-1045, 1967.

55. Hintz, H. F.; Argenzio, R. A., Schryver, H. F.: Digestion coefficients, blood glucose levels and molar percentage of volatile acids in intestinal fluid of ponies fed varying forage - grain ratios. J. An. Sci. 33: 992-995, 1971.

56. Kern, D. L.; Slyter, L. L.; Weaver, J. M.; Leffel, L. C., Samuelson, G.: Pony cecum vs. steer rumen: the effects of oats and hay on the microbial ecosystem. J. An. Sci. 37:463-469, 1973.

57. Stillons, M. C.; Teeter, S. M.; Nelson, W. E.: Equine digestive volatile fatty acid concentration, in proceedings 2nd Eq. Nutr. Conf., Ithaca. (1970):21.

58. Argenzio, R. A., Southworth, M.; Lowe, J. E.; Stevens, C. E.: Interrelationship of Na, HCO_3, and volatile fatty acid transport by equine large intestine. Am. J. Phys. 233:E469-478, 1977.

59. Glinsky, M. J.; Smith R. M.; Spires, H. R.; Davis, C. L.: Measurement of volatile fatty acid production rates in the cecum of the pony. J. An. Sci. 42:1465-1470, 1976.

60. Alexander, F.; Hickson, J. C. D.: The salivary and pancreatic secretions of the horse in Phillipson AT (ed): Physiology of Digestion and Metabolism in the Ruminant. Newcastle upon Tyne, England, Oriel, 1970, pp 375-389.

61. Roberts, M. C.; Hill, F. W. G.: The mucosa of the small intestine of the horse: a microscopic study of specimens obtained through a small intestine fistula. Eq. Vet. J. 6:74-80, 1974.

62. Roberts, M. C.; Hill, F. W. G.; Kidder, D. E.: The development and distribution of small intestinal disaccharidases in the horse. Res. Vet. Sci. 17:42-48, 1974.

63. Roberts, M. C.: The development and distribution of alkaline phosphatase activity in the small intestine of the horse. Res. Vet. Sci. 16: 110-111, 1974.

64. Hintz, H. F.; Hogue, D. E., Walker, E. F., Lowe, J. E.; Schryver, H. F.: Apparent digestion in various segments of the digestive tract of ponies fed diets with varying roughage grain ratios. J. An. Sci. 32:245-248, 1970.

65. Slade, L. M.; Robinson, D. W.; Casey, K. E.: Nitrogen metabolism in nonruminant herbivores. I. The influence of nonprotein nitrogen and protein quality on the nitrogen retention of adult mares. J. An. Sci. 30:753-760, 1970.

66. Slade, L. M.; Robinson, D. W.: Nitrogen metabolism in nonruminant herbivores. II. Comparative aspects of protein digestion. J. An. Sci. 30:761-763, 1970.

67. Wootton, J. F.; Argenzio, R. A.: Nitrogen utilization within equine large intestine. Am. J. Phys. 229:1062-1067, 1975.

68. Reitnour, C. M.: Effect of cecal administration of corn starch on nitrogen metabolism in ponies. J. An. Sci. 49:988-991, 1979.

69. Glinsky, M. J.; Tyler, T. R.: Ammonia kinetics in the equine cecum. Fed Proceeding 36:1180, 1977.

70. Slade, L. M.; Bishop, R.; Morris, J. G.; Robinson, D. W.: Digestion and absorption of N-labelled microbial protein in the large intestine of the horse. B. Vet. J. 127:11-13, 1971.

71. Pulse, R. E.; Baker, J. P.; Potter, G. D.: Effects of cecal fistulation upon nutrient digestion and indicator retention in horses. J. An. Sci. 37:488-492, 1973.

72. Reitnour, C. M.; Baker, J. P.; Mitchell, G. E.; Little, C. O.; Kratzer, D. D.: Amino acids in equine cecal contents, cecal bacteria and serum. J. Nutr. 100:349-354, 1970.

73. Wysocki, A. A.; Baker, J. P.: Bacterial protein digestion in the equine lower gut. J. An. Sci. 35:225, 1972.

74. Reitnour, C. M.; Salsbury, R. L.: Digestion and utilization of cecally infused protein by the equine. J. An. Sci. 35:1190-1193, 1972.

75. Johnson, R. J.; Hart, J. W.: Utilization of nitrogen from soybean meal, buiret and urea by equine. Nutrition Reports Internal 9:209-216, 1974.

76. Ott, E. A.; Asquith, R. L.; Feaster, J. P.: Lysine supplementation of diets for yearling horses. J. An. Sci. 53:1496-1503, 1981.

77. Godbee, R. G.; Slade, L. M.: The effect of urea or soybean meal on the growth and protein status of young horses. J. An. Sci. 53:670-676, 1981.

78. Reitnour, C. M.; Salsbury, R. L.: Utilization of proteins by the equine species. Am. J. Vet. Res. 37:1065-1067, 1976.

79. Nelson, D. D.; Tyznik, W. J.: Protein and nonprotein nitrogen utilization in the horse. J. An. Sci. 32:68-73, 1971.

80. Reitnour, C. M.: Response to dietary nitrogen in ponies. Eq. Vet. J. 10:65-68, 1978.

81. Meyer, H.; Pferdekamp, M.: Avswirkungen uberhohter proteingaben beim pferd. Zbl Vet. Med. A. 27:746-757, 1980.

82. Yoakam, S. C.; Kirkham, W. W., Beeson, W. M.: Effect of protein level on growth in young ponies. J. An. Sci. 46:983-991, 1978.

83. Prior, R. L.; Hintz, H. F., Lowe, J. E. Visek, W. J.: Urea recycling and metabolism of ponies. J. Ani. Sci. 38:565-571, 1974.

84. Fonnesbeck, P. V.; Symons, L. D.: Effect of diet on concentration of protein, urea nitrogen, sugar and cholesterol of blood plasma of horses. J. An. Sci. 28:216-219, 1969.

85. Reitnour, C. M.; Treece, J. M.: Relationship of nitrogen source to certain blood components and nitrogen balance in the equine. J. An. Sci. 32:487-490, 1971.

86. Fonnesbeck, P. V.; Symons, L. D.: Effect of diet on concentration of protein, urea nitrogen, sugar and cholestrol of blood plasma of horses. J. An. Sci. 28:216-219, 1969.

87. Coffman, J. R.: Equine Clinical Chemistry and Pathophysiology, ed 1. Bonner Springs, Kansas, Vet. Med. Pub. Co., 1981, pp 4.

88. Bowman, V. A.; Fontenot, J. P.; Webb, K. E.; Meacham, T. N.: Digestion of fat by equine, in proceedings. 5th Eq. Nutr. Symposium, St. Louis, (1977):40.

89. Hintz, H. F.; Ross, M. W.; Lesses, F. R.; Leids, P. F.; White, K. K.; Lowe, J. E.; Short, C. E.; Schryver, H. F.: Dietary fat for horses. Proc. Cornell Nutr. Conf. 87-90, 1977.

90. Kane, E; Baker, J. P.: Utilization of a corn oil supplemented diet by the horse, in proceedings, 5th Eq. Nutr. Symp., St. Louis, (1977):114.

91. Stillions, M. C.; Teeter, S. M.; Nelson, W. E.: Ascorbic acid requirements of mature horses. J. An. Sci. 32:249-251, 1971.

92. Stillions, M. C.; Teeter, S. M.; Nelson, W. E.: Utilization of dietary vitamin B_{12} and cobalt by mature horses. J. An. Sci. 32:252-255, 1971.

93. Linerode, P. A.: Studies on the synthesis and absorption of B-complex vitamins in the horse, in proceedings. 13th Annual Meeting, Am. Assoc. Eq. Pract., 283-314, 1967.

94. Carroll, F. D.; Goss, H.; Howell, C. E.: The synthesis of B vitamins in the horse. J. An. Sci. 8:290-299, 1949.

95. Davies, M. E.: Studies on the microbial flora of the large intestine of the horse by continuous culture in an artificial colon. Vet. Sci. Commun. 3:39-49, 1979.

96. Schryver, H. F.; Hintz, H. F.; Craig, P. H.; Lowe, J. E.: The calcium and phosphorus requirements of the horse, in proceedings, 16th Annual Meeting, Am. Assoc. Eq. Pract., 117-125, 1970.

97. Argenzio, R. A.; Lowe, J. E.; Hintz, H. F.; Schryver, H. F.: Calcium and phosphorus homeostasis in horses. J. Nutr. 104:18-27, 1974.

98. Whitlock, R. H.; Schryver, H. F.; Krook, L.; Hintz, H. F.; Craig, P. H.: The effects of high dietary calcium in horses, in proceedings. 16th Annual Meeting, Am. Assoc. Eq. Pract., 127-133, 1970.

99. Schryver, H. F.; Van Wil, S.; Danilisk, P.; Hintz, H. F.: The voluntary intake of calcium by horses and ponies fed a calcium deficient diet. J. Eq. Med. Surg. 2:337-340, 1978.

100. Schryver, H. F.; Hintz, H. F.; Lowe, J. E.: Calcium metabolism, body composition, and sweat losses of exercised horses. Am. J. Vet. Res. 39:245-248, 1978.

101. Krosk, L.: Dietary calcium-phosphorus and lameness in the horse. Cornell Vet. 58:59-73, 1968.

102. Wasserman, R. H.; Taylor, A. N.: Some aspects of the intestinal absorption of calcium, with special reference to vitamin D, in Mineral Metabolism III, comor. ed., New York, Academic Press, 1969, pp 321-393.

103. Schryver, H. F.: Intestinal absorption of calcium and phosphorus by horses. J. S. Afr. Vet. Assoc. 46:39-42, 1975.

104. Schryver, H. F.; Hintz, H. F.; Lowe, J. E.: Calcium and phosphorus inter-relationships in horse nutrition. Eq. Vet. J. 3:102-109, 1971.

105. Harrington, D. D.; Walsh, J. J.: Equine magnesium supplements: evaluation of magnesium oxide, magnesium sulphate and magnesium carbonate in foals fed purified diets. Eq. Vet. J. 12:32-33, 1980.

106. Schryver, H. F.; Hintz, H. F.; Lowe, J. E.: Absorption, excretion and tissue distribution of stable zinc and zinc in ponies. J. An. Sci. 51:896-902, 1980.

107. Argenzio, R. A.; Lowe, J. E.; Pickard, D. W.; Stevens, C. E.: Digesta passage and water exchange in the equine large intestine. Am. J. Phys. 226:1035-1042, 1974.

108. Dyce, K. M.; Hartman, W.: A cinefluoroscopic study of the caecal base of the horse. Res. Vet. Sci. 20:40-60, 1976.

109. Applegate, C. S.; Hershberger, T. V.: Evaluation of in vitro and in vivo cecal fermentation techniques for estimating the nutritive value of forages for equine. J. An. Sci. 28:18-22, 1969.

110. Meyer, H.; Pferdekamp, M.; Huskamp, B.: Untersuckungen uber die verdaulichkeit und vertraglichkeit verschiedener futtermittel bei typhletomierten ponys. Dtsch tierarztl Wschr 86:377-420, 1979.

111. Argenzio, R. A.; Stevens, C. E.: Cyclic changes in ionic composition of digesta in the equine intestinal tract. Am. J. Phys. 228:1224-1230, 1975.

112. Tasker, J. B.: Fluid and electrolyte studies in the horse. III. Intake and output of water, sodium, and potassium in normal horses. Cornell Vet. 57:649-657, 1967.

113. Hintz, H. F.; Argenzio, R. A.; Schryver, H. F.: Digestion coefficients, blood glucose levels and molar percentage of bolatile acids in intestinal fluid of ponies fed varying forage - grain ratios. J. An. Sci 33: 992-995, 1972.

114. Adam, K. M. G.: The quantity and distribution of the ciliate protozoa in the large intestine of the horse. Parasit. 41:301-311, 1951.

115. Alexander, F.; Macpherson, M.J.D.; Oxford, A. E.: Fermentative activities of some members of the normal coccal flora of the horse's large intestine. J. Comp. Path. 62:252-259, 1952.

116. Koller, B. L.; Hintz, H. F.; Robertson, J. B.; Van Soest, P. J.: Comparative cell wall and dry matter digestion in the cecum. J. An. Sci. 47:209-215, 1978.

117. Hintz, H. F.: Review article: equine nutrition. Comparisons of digestive coefficients obtained with cattle, sheep, rabbits, and horses. Veterinarian 6:45-51, 1969.

118. Slade, L. M.; Hintz, H. F.: Comparison of digestion in horses, ponies, rabbits and guinea pigs. J. An. Sci. 28:842-843, 1969.

119. Kern, D. L.; Slyter, L. L.; Leffel, E. C.; Weaver, J. M.; Oltjen, R. R.: Ponies vs. steers: microbial and chemical characteristics of intestinal ingesta. J. An. Sci. 38:559-564, 1974.

120. Alexander, F.: The rate of passage of food residues through the digestive tract of the horse. J. Comp. Path. 56:266-267, 1946.

121. Moore, J. M.; Traver, D. S.; Johnson, J. H.; Coffman, J. R.; Tritchler, L. G.; Garner, H. E.: Particulate fecal markers in the diagnosis of large intestine obstruction. J. Eq. Med Surg. 2:541-544, 1978.

122. Vander Noot, G. W.; Symons, L. D.; Lydman, R. K.; Fonnesbeck, P. V.: Rate of passage of various feedstuffs through the digestive tract of horses. J. Am. Sci. 26:1309-1311, 1967.

123. Alexander, F.; Davies, M. E.: Production and fermentation of lactate by bacteria in the alimentary canal of the horse and pig. J. Comp. Path. 73:1-8, 1963.

124. Argenzio, R. A.; Southworth, M.;Stevens, C. E.: Sites of organic acid production and absorption in the equine gastrointestinal tract. Am. J. Phys. 226:1043-1050, 1974.

125. Willard, J. G.; Willard, J. C.; Wolfram, S. A.; Baker, J. P.: Effect of diet on cecal pH and feeding behavior of horses. J. Am. Sci. 45:87-93, 1977.

126. Coffman, J. R.: Equine Clinical Chemistry and Pathophysiology, ed. 1. Bonner Springs, Kansas, Vet. Med. Pub. Co., 1981, pp 4-5, 14, 20-25.

127. Argenzio, R. A., Hintz, H. F.: Glucose tolerance and effect of volatile fatty acid on plasma glucose concentration in ponies. J. An. Sci. 30:514-518, 1970.

128. Argenzio, R. A., Hintz, H. F.: Volatile fatty acid tolerance and effect of glucose and VFA on plasma insulin levels in ponies. J. Nutr. 101:723-730, 1971.

129. Argenzio, R. A.; Hintz, H. F.: Effect of diet on glucose entry and oxidation rates in ponies. J. Nutr. 102:879-892, 1972.

130. Evans, J. W.; Thompson, P. G.; Winget, C. M.: Glucose and insulin biorhythms in the horse. J. S. Afr. Vet. Assoc. 45:317-329, 1974.

131. Anderson, M. G.: The effect of exercise on blood metabolite levels in the horse. Eq. Vet. J. 7:27-33, 1975.

132. Lindholm, A.: Glycogen depletion pattern and the biochemical response to varying exercise intensities in standardlred trotters. J. S. Afr. Vet. Assoc. 45:341-343, 1974.

133. Coffman, J. R.: Equine Clinical Chemistry and Pathophysiology, ed. 1. Bonner Springs, Kansas, Vet. Med. Pub. Co., 1981, pp 22-24.

134. McDonald, L. E.: Veterinary Endocrinology and Reproduction, ed. 2. Philadelphia, Lea and Febiger, 1975, 115-130.

135. Kaneko, J. J.: Clinical Biochemistry of Domestic Animals, ed. 3. New York, Academic Press, 1980, pp 9, 22-23, 25, 28, 40-43, 133, 320.

136. Gay, C. C.; Sullivan, N. D.; Wilkinson, J. S.; McLean, J. D.; Blood, D. C.: Hyperlipaemia in ponies. Austral Vet. J. 54:459-462, 1978.

137. Robie, S. M.; Janson, C. H.; Smith, S. C.; O'Connor, J. T.: Equine serum lipids: serum lipids and glucose in Morgan and thoroughbred horses and Shetland ponies. Am. J. Vet. Res. 36:1705-1708, 1975.

138. Carlson, G. P.; Mansmann, R. A.: Serum electrolyte and plasma protein alterations in horses used in endurance rides. J. Am. Vet. Med. Assoc. 165:262-264, 1974.

139. Carlson, G. P.; Ocen, P. O.; Harrold, D.: Clinicopathologic alterations in normal and exhausted endurance horses. Therio. 6:93-104, 1976.

140. Naylor, J. M.; Kronfeld, D. S.; Acland, H.: Hyperlipemia in horses: effects of undernutrition and disease. Am. J. Vet. Res. 41:899-905, 1980.

141. Tasker, J. B.: Fluid and electrolyte studies in the horse. IV. The effects of fasting and thirsting. Cornell Vet. 57:658-667, 1967.

142. Johnson, R. J.; Hart, J. W.: Influence of feeding and fasting on plasma free amino acids in the equine. J. An. Sci. 38:790-794, 1974.

143. Finocchio, E. J.: Rehabilitation of the recumbent malnourished horse (equine starvation syndrome), in proceedings. 19th Annual Meeting, Am. Assoc. Equine Pract. 167-170, 1973.

144. Coffman, J. R.: Equine Clinical Chemistry and Pathophysiology, ed. 1 Bonner Springs, Kansas, Vet. Med. Pub. Co., 1981, pp 13.

145. Schummer, A.; Nickel, R.; Sack, W. O.: The Viscera of the Domestic Mammals. ed. 2. New York, Springer-Verlag, 1979, pp 108.

146. Davis, L. E.; Knight, A. P.: Review of the clinical pharmacology of the equine digestive system. J. Eq. Med. Surg. 1:27-35, 1977.

147. Coffman, J. R.: Equine Clinical Chemistry and Pathophysiology, ed. 1 Bonner Springs, Kansas, Vet. Med. Pub. Co., 1981, pp 7.

148. Schummer, A.; Nickel, R.; Sack, W. O.: The Viscera of the Domestic Mammals, ed. 2. New York, Springer-Verlag, 1979, pp 113.

149. Stashak, T. S.: Colic. Eq. Vet. Data, pp 117-126, 1980.

150. Donawick, W. J.: Metabolic management of the horse with an acute abdominal crisis. J. S. Afr. Vet. Assoc. 46:107-110, 1975.

151. Stashak, T. S.: Medical management of the acute abdomen in the horse. Florida Vet. J. 7:32-33, 1978.

152. Stashak, T. S.: Medical management of the acute abdomen in the horse. Part III. Florida Vet. J. 7:10-15, 1979.

153. Gay, C. C.; Speirs, V. C.: Parasitic arteritis and its consequences in horses. Austral Vet. J. 54:600-601, 1978.

154. Rous, R. C.: Mesenteric thrombosis. J. S. Afr. Vet. Assoc. 46:79-80, 1975.

155. Drudge, J. H.; Lyons, E. T.: Pathology of infections with internal parasites in horses. Blue Book. 27:267-275, 1977.

156. Slocombe, J. O. D., Owen, R. R.; Pennock, P. W.; McCraw, B. M.; Rendano, V. T.: Arteriographic presentation of the early development of vascular lesions in ponies infected with Strongylus vulgaris, in proceedings. 23rd Annual Meeting, Am. Assoc. Eq. Pract., 305-311, 1977.

157. Howell, L. M.: Verminous colic in horses: Vet. Med. Small An. Clin. 72:857-860, 1977.

158. Wright, A. I.: Verminous arteritis as a cause of colic in the horse. Eq. Vet. J. 4:169-174, 1972.

159. Vibe-Petersen, G.; Nielsen, K: Verminous enteritis and thrombo-embolic colic in the horse. Nord. Vet. Med. 31:385-391, 1979.

160. Duncacn, J. L.; Dargie, J. D.: The pathogenesis and control of strongyle infection in the horse. J. S. Afr. Vet. Assoc. 46:81-85, 1975.

161. Duncan, J. L.; Pirie, H. M.: Life cycle of strongylus vulgaris. Mod. Vet. Pract. 54:45, 1973.

162. Ruckebusch, Y.; Fioramonti, J: Colonic myoelectrical spiking activity: major patterns and significance in six different species. Zbl. Vet. Med. A. 27:1-8, 1980.

163. Bueno, L.; Ruckebusch, Y.; Dorchies, P.: Disturbances of digestive motility in horses associated with strongylus infection. Vet. Parasit. 5:253-260, 1979.

164. Ruckebusch, Y.: Elektromyographische analyse der sunndarmmotorik bei haustieren. Zbl. Vet. Med. A. 24:1-12, 1977.

165. Nelson, A. W.; Adams, O. R.: Intestinal infarction in the horse: acute colic arterial occlusion. Am. J. Vet. Res. 27:707-710, 1966.

166. Rollins, J. B.; Clement, T. H.: Observations on incidence of equine colic in a private practice. Eq. Pract. 1:39-42, 1979.

167. Wheat, J. Q.: Causes of Colic and types requiring surgical intervention. J. S. Afr. Vet. Assoc. 46:95-98, 1975.

168. Lunkenheimer, P. P.; Pircher, W; Klinke, F; Lunkenheimer, A.; Druen, B; Weritz, D.; Dittrich, H.: Das ischamisch-septisch krankheitsbild nach akutem verschlub der arteria mesenterica superior im tierversuch. Zbl. Vet. Med. A. 27:452-468, 1980.

169. Boles, C.: Surgical techniques in equine colic. J. S. Afr. Vet. Assoc. 46:115-119, 1975.

170. Williamson, R. C. N.; Chir, M.: Intestinal adaptation. Structural, functional and cytokinetic changes. New Eng. J. Med. 298:1393-1402, 1978.

171. Williamson, R. C. N.; Chir, M.: Intestinal adaptation. Mechanisms of Control. New Eng. J. Med. 298:1444-1450, 1978.

172. Althausen, T. L.; Doig, R. K.; Uyeyama, K.: Digestion and absorption after massive resection of the small intestine. II. Recovery of the absorptive function as shown by intestinal absorption tests in two patients and a consideration of compensatory mechanisms. Gastroenterology 16: 126-139, 1950.

173. Nygaard, K.: Resection of the small intestine in rats. III. Morphological changes in the intestinal tract. Acta Chir Scand 133:233-248, 1967.

174. Hanson, W. R.; Osborne, J. W.; Sharp, J. G.: Compensation by the residual intestine after intestinal resection in the rat. I. Influence of amount of tissue removed. Gastroenterology 72:692-700, 1977.

175. Hanson, W. R.; Osborne, J. W.; Sharp, J. G.: Compensation by the residual intestine after intestine resection in the rat. II. Influence of poaroperative time interval. Gastroenterology 72:701-705, 1977.

176. Scheflan, M.; Galli, S. J.; Perrotto, J.: Intestinal adaptation after extensive resection of the small intestine and prolonged administration of parenteral nutrition. Surg. Gynecol. Obstit. 143:757-762, 1976.

177. Obertop, H; Nundy, S.; Malamud, D.: Onset of cell proliferation in the shortened gut: rapid hyperplasia after jejunal resection. Gastroenterology. 72:267-270, 1977.

178. Williamson, R. C. N.; Bauer, F. L. R.; Ross, J. S.: Proximal enterectomy stimulates distal hyperplasia more than bypass or pancreaticobiliary diversion. Gastroenterology 74:16-23, 1978.

179. Wilmore, D. W.; Dudrick, S. J.: Daly, J. M.: The role of nutrition in the adaptation of the small intestine after massive resection. Surg. Gynecol. Obstet. 132:673-680, 1970.

180. Bochkov, N. P.: Morphological changes in the jejunum and ileum of rats after wide resection of the small intestine. Bull. Exp. Biol. Med. 47:339-343, 1959.

181. Weser, E.; Henandez, M. H.: Studies of small bowel adaptation after intestinal resection in the rat. Gastroenterology 60:69-75, 1971.

182. Tilson, M. D.; Wright, H. K.: The effect of resection of the small intestine upon the fine structure of the intestinal epithelium. Surg. Gnecol. Obstet. 134:992-994, 1971.

183. Knudtson, K. P.; Priest, R. E.; Jacklin, A. J.: Effects of partial resection on mammalian small intestine. I. Initial autoradiographic studies in the dog. Lab. Invest. 11:433-439, 1962.

184. Dowling, R. H.; Booth, C. C.: Structural and functional changes following small intestinal resection in the rat. Clin. Sci. 32: 139-149, 1967.

185. Booth, C. C.; Evans, K. T.; Manzies, T.: Intestinal hypertrophy following partial resection of the small bowel in the rat. Br. J. Surg. 46:403-410, 1959.

186. Bury, K. D.: Carbohydrate digestion and absorption after massive resection of the small intestine. Surg. Gynecol. Obstet. 35:177-187, 1972.

187. Roberts, M. C.; Hill, F. W. G.: The oral glucose tolerance test in the horse. Eq. Vet. J. 5:171-173, 1973.

188. Dietz, H. H.: D-xylose absorption test in the horse. Nord. Vet. Med. 33:114-120, 1981.

189. Balton, J. R.; Merritt, A. M.; Cimprich, R. E.; Ramberg, C. F.; Streett, W.: Normal and abnormal xylose absorption in the horse. Cornell Vet. 66:183-197, 1976.

190. Dietz, H. H.: Metoder til vurdering af tarmfunktionen has hest. Nord. Vet. Med. 33:172-177, 1981.

191. Roberts, M. C.: The D-xylose absorption test in the horse. Eq. Vet. J. 6:28-30, 1974.

192. Loeb, W. F.; McKenzie, L. D.; Hoffsis, G. F.: The carbohydrate digestion absorption test in the horse. Technique and normal values. Cornell Vet. 62:525-531, 1972.

193. Coffman, J. R.: Equine Clinical Chemistry and Pathophysiology, ed. 1 Bonner Springs, Kansas, Vet. Med. Pub. Co., 1981, pp 22, 33, 243, 244, 255.

194. Kaneko, J. J.: Clinical Biochemistry of Domestic Animals, ed. 3 New York, Academic Press, 1980, pp 25, 28-29, 242-243, 322-323.

195. Anderson, N. V.: Veterinary Gastroeneterology, ed. 1 Philadelphia, Lea and Febiger, 1980, pp 252, 260, 641.

196. Johnson, B. D.; Heath, R. B.; Bowman; Phillips; Rich; Voss: Serum chemistry changes in horses during anesthesia: a pilot study investigating the possible causes of postanesthetic myositis in horses. J. Eq. Med. Surg. 2:109-123, 1978.

197. Argenzio, R. A.: Physiology of diarrhea-large intestine. J. Am. Vet. Med. Assoc. 173:667-672, 1978.

198. Kaplan, M. M.: Alkaline phosphatase. New Eng. J. Med. 198:200-202, 1972.

199. Coffman, J. R.: Equine Clinical Chemistry and Pathophysiology, ed. 1 Bonner Springs, Kansas, Vet. Med. Pub. Co., 1981, pp 97-98.

200. Kaneko, J. J.: Clinical Biochemistry of Domestic Animals, ed. 3 New York, Academic Press, 1980, pp 183-184, 238-240, 270, 476.

201. Coffman, J. R.: Equine Clinical Chemistry and Pathphysiology, ed. 1 Bonner Springs, Kansas, Vet. Med. Pub. Co., 1981, pp 52-53, 61.

202. Scrutchfield, W. L.: Protein losing gastroenterology, in proceedings 21st Annual Meeting, Am. Assoc. Equine Pract. 203-210, 1975.

203. Davenport, H. W.: A hypothesis about protein-losing gastroenteropathy. Gastroenterology 58:600-601, 1970.

204. Dietz, H. H.; Nielsen, K.: Turnover of I-labelled albumin in horses with gastrointestinal disease. Nord. Vet. Med. 32:369-373, 1980.

205. Mattheeuws, D. R. G.; Kaneko, J. J.; Loy, R. G.; Corneluis, C. E.; Wheat, J. D.: Compartmentalization and turnover of I-labelled albumin and gamma globulin in horses. Am. J. Vet. Res. 27:699-705, 1966.

206. Kaneko, J. J.: Clinical Biochemistry of Domestic Animals, ed. 3 New York, Academic Press, 1980, pp 66.70.101, 105-106.

207. Rothschild, M. A.; Oratz, M.; Schreiber, S. S.: Albumin synthesis (first of two parts). New Eng. J. Med. 286:748-757, 1972.

208. Rothschild, M. A.; Oratz, M.; Schreiber, S. S.: Albumin synthesis (second of two parts). New Eng. J. Med. 286:816-821, 1972.

209. Coffman, J. R.: Equine Clinical Chemistry and Pathophysiology, ed. 1 Bonner Springs, Kansas, Vet. Med. Pub. Co., 1981, pp 50-56.

APPENDIX

Table 7

ORAL GLUCOSE TOLERANCE TEST (mg %)
(OGTT)
Preoperative

Horse A exhibited an essentially flat absorption curve. Horses C, E, and I exhibited glucose absorption although peak levels were below the minimum accepted normal peak level of 80% above the pretest value. Horses B, D, F, G, and H had normal peak glucose levels.

Peak levels occurred in horses A, B, D, F, and I by the second hour. However, in horses C, E, G, and H peaks occurred after 2 hours. Horses A, D, F, and I had 6 hour levels within the presample range as normally expected. Horses B, C, E, G, and H had 6 hour levels higher than the expected pretest ranges.

Table 7

ORAL GLUCOSE TOLERANCE TEST (mg %)
(OGTT)
Preoperative

GROUP	HORSE	HRS. / MIN.	0 Presample	½ 30	1 60	1½ 90	2 120	2½ 150	3 180	4 240	5 300	6 360	Peak % of resting level
I	A. Cracker Jack		114	121	122	119	114	120	113	114	112	114	7%
	B. Charlie		80	110	128	152	157	155	155	144	137	115	96%
	C. Trigger		91	118	139	149	148	156	145	105	116	117	71%
II	D. Chubbs		93	136	156	173	190	183	163	136	99	95	104%
	E. Amy		99	131	154	160	155	158	165	140	133	114	67%
	F. Star		101	150	164	205	192	162	132	107	65	87	103%
III	G. Molly		85	136	163	179	190	195	190	175	130	100	129%
	H. Goose		94	107	147	190	210	243	260	270	249	190	123%
	I. Snowflake		92	131	144	134	128	108	110	91	93	89	56%

Table 8

ORAL GLUCOSE TOLERANCE TEST (mg %)
(OGTT)
One Week Postoperative

Horses A, B, E, G, H, and I displayed glucose peaks below the normal accepted minimum of 80% above initial glucose levels. Horses C, D, and F had peak levels > 80% above the initial glucose level.

Horses F and G peaks occurred after the anticipated 2 hour time period. At six hours assay values had returned to pretest levels in Horses A, B, E, F, G, H, and I. In horses C and D, 6 hour levels remained elevated above pretest levels.

Table 8

ORAL GLUCOSE TOLERANCE TEST (mg %)
(OGTT)
One Week Postoperative

GROUP	HORSE	HRS. / MIN.	0 Presample	½ 30	1 60	1½ 90	2 120	2½ 150	3 180	4 240	5 300	6 360	Peak % of resting level
I	A. Cracker Jack		104	114	127	134	121	113	56	65	52	105	29%
	B. Charlie		89	121	116	98	107	108	117	89	98	82	36%
	C. Trigger		77	94	107	126	132	143	141	158	123	114	105%
II	D. Chubbs		93	130	144	156	167	162	156	129	122	104	80%
	E. Amy		111	142	168	165	145	162	141	104	91	114	51%
	F. Star		81	118	154	175	183	196	178	125	106	69	142%
III	G. Molly		96	115	124	142	143	145	128	118	87	92	51%
	H. Goose		96	113	131	138	135	126	112	109	95	103	44%
	I. Snowflake		89	98	104	105	96	100	89	101	91	97	18%

Table 9

ORAL GLUCOSE TOLERANCE TEST (mg %)
(OGTT)
Four Week Postoperative

Normal glucose absorption with $\geq$ 80% absorption by 2 hours occurred with horses A and F. Horses D, E, H, and I had peak levels by 2 hours with < 55% absorption and horse B a peak of 71% by 2 hours. Horses C and G had peaks $\leq$ 52% after 2 hours.

Six hour levels near pretest concentration occurred for horses A, C, E, F, H, and I.

Table 9

ORAL GLUCOSE TOLERANCE TEST (mg %)
(OGTT)
Four Week Postoperative

GROUP	HORSE	HRS. / MIN.	0 Presample	½ 30	1 60	1½ 90	2 120	2½ 150	3 180	4 240	5 300	6 360	Peak % of resting level
I	A. Cracker Jack		85	128	149	163	161	155	123	127	117	88	92%
	B. Charlie		84	121	131	144	122	101	99	117	106	117	71%
	C. Trigger		94	105	99	107	102	112	117	112	109	95	24%
II	D. Chubbs		86	121	127	133	131	119	115	88	110	102	55%
	E. Amy		92	117	134	130	119	119	109	99	118	96	46%
	F. Star		91	122	182	261	288	213	148	101	109	78	216%
III	G. Molly		113	129	135	155	162	169	172	166	149	138	52%
	H. Goose		96	113	131	138	135	126	112	109	95	103	44%
	I. Snowflake		96	106	118	130	134	130	126	116	109	94	40%
	G. Molly (10 week OGTT)		81	85	104	104	120	122	128	132	121	123	63%

Table 10
A. CRACKER JACK

	WEEK 0		WEEK I	WEEK II	WEEK III	WEEK IV	WEEK V	WEEK VI	WEEK IX
Fibrinogen mg %	300		400, 600	500	400, 300	300	300	200	300
PCV %	32, 28, 28		28, 25	25	32, 32	31	33	33	33
Hb g/dl	12, 11, 11		11, 10	9	12, 12	12	12	13	13
RBC $x10^6$	7.7, 6.9, 6.8		6.7, 5.9	5.8	7.7, 7.4	7.4	7.9	7.9	8.0
WBC $x10^6$	9.1, 8.9, 16.2		8.4, 10.5	12.5	14.6, 14.1	9.8	10.6	10.3	11.0
bands	46, -, -		-, 525	-	146, -	-, -	-	-	-
segs	4095; 10,530		4200, 5933	7313	10,585; 8742	4998	5512	5459	6050
lymphs	3913, 4212		3108, 3307	4062	3285, 4724	4263	4558	4429	4400
mono	182, 648		504, 630	750	438, 70	294	212	206	55
eos	819, 810		588, 105	375	146, 564	245	318	206	495
baso	45, -		- -	-	- -	-	-	-	61
BUN mg %	17,11,15		13, 14	12	13, 12	15	14	13	16
glucose mg %	88, 89, 94		111, 114	107	103, 93	102	102	101	98
calcium mg %	11.1,10.8,12.0		11.0, 11.2	11.1	11.9, 11.4	11.0	12.5	12.6	12.3
Phos mg %	4.3 4.5, 4.6		4.1, 3.8	4.6	3.8, 3.8	4.9	4.5	3.0	4.4
Na meg/L	142, 136, 138		139, 140	140	140, 141	143	147	142	137
K meg/L	4.3, 3.3, 3.9		3.6, 3.4	3.7	4.0, 3.9	3.2	3.3	3.2	4.2
Cl meg/L	116, 97, 105		105, 107	102	106, 102	104	107	108	103
Tot CO_2 meg/L	29, 31, 32		26, 29	31	31, 35	32	32	31	31
fecal consistency	3-4		2, 2, 3	3, 4, 4	3-4, 3-4, 3-4	3-4, 3-4, 3-4	3-4, 3-4, 3-4	3-4, 3-4, 3-4	3
weight (kg)	432		414, 404, 409	412, 414, 415	416, 420	424, 425, 427	428, 432	436, 444	
SDH I.U.						2.3	2.7	2.8	2.2

Multiple samples for a week are listed with the earliest value first in the square.

Table 11

B. CHARLIE

	WEEK 0	WEEK I	WEEK II	WEEK III	WEEK IV	WEEK V	WEEK VI	WEEK IX
Fibrinogen mg %	300, 400	300, 500	400, 400	400, 300	300	200	100	300
PCV %	35, 32	33, 29	25, 25	34, 31	30	31	33	34
Hb g/dl	14, 13	13, 11	10, 9	13, 12	11	12	13	12
RBC $x10^6$	7.8, 7.3	7.4, 6.5	5.7, 5.4	7.7, 6.8	6.7	6.8	7.5	7.0
WBC $x10^6$	9.9, 10.0	8.5, 6.7	12.4, 14.6	14.3, 12.0	9.7	9.3	9.7	9.1
bands	-, -	595, 134	434, 73	- -	-	-	-	-
segs	6732, 7000	4760, 3685	9052; 11,899	9867, 9480	6693	6417	6402	5500
lymphs	2673, 2250	2210, 2211	2108, 1825	3575, 2160	2377	2692	2474	2600
mono	99, 300	255, 134	558, 365	72, 240	242	186	194	400
eos	297, 400	680, 536	186, 438	643, 120	340	-	533	300
baso	99, 50	- -	62, -	143, -	48	-	97	90
BUN mg %	19, 23	21, 17	15, 13	23, 20	21	23	19	20
glucose mg %	93, 77	75, 110	107, 95	99, 90	98	86	103	95
calcium mg %	12.6, 11.9	11.1, 10.9	11.1, 11.1	11.7, 11.2	11.8	12.4	12.2	12.3
Phos mg %	4.9, 4.0	2.7, 4.1	2.6, 3.8	3.6, 3.6	4.7	4.3	2.3	2.7
Na meg/L	149, 140	138, 145	145, 141	149, 140	145	147	138	139
K meg/L	3.1, 3.5	3.8, 3.4	3.2, 3.5	3.5, 3.7	3.6	4.0	4.3	4.0
Cl meg/L	110, 106	106, 109	110, 107	111, 98	103	108	108	106
Tot CO_2 meg/L	31, 30	29, 27	31, 28	31, 36	34	31	29	30
fecal consistency	4, 4, 4	4, 4, 4	4, 4, 4	4, 4, 4	4, 4, 4	4, 4, 4	4, 4	3
weight (kg)	415	413, 414, 412	414, 417, 419	422, 424, 430	427, 430	432, 432, 434	435, 434, 436	
SDH I.U.					3.6	4.4	2.4	2.8

Multiple samples for a week are listed with the earliest value first in the square.

Table 12
C. TRIGGER

	WEEK 0		WEEK I	WEEK II	WEEK III	WEEK IV	WEEK V	WEEK VI	WEEK IX
Fibrinogen mg %	300		200	200	300	500	300	300, 300	200
PCV %	27		34	31	36	33	36	37, 33	38
Hb g/dl	9.8		12.7	11.7	13.0	11.5	13.8	13.6, 12.5	14.0
RBC $x10^6$	5.9		7.4	6.8	7.5	6.8	8.0	7.9, 7.2	8.2
WBC $x10^6$	8.7		7.2	8.0	7.0	6.5	7.3	6.0, 6.6	6.7
bands	87		-	-	-	-	-	-	-
segs	5481		4104	5120	3920	3770	5183	3300, 4422	3283
lymphs	2958		2952	2560	2870	2535	1752	2280, 2112	2948
mono	-		72	160	70	65	146	120, 66	268
eos	174		72	160	70	-	219	240. -	201
baso	-		-	-	70	130	-	60, -	-
BUN mg %			25	16	16	16	-	19, 13	14
glucose mg %	105		83	91	89	86	-	105, 87	95
calcium mg %	12.2		10.3	11.4	12.8	11.5	-	11.5, 11.1	12.1
Phos mg %	3.2		4.4	3.1	3.2	3.0	-	3.4, 2.9	2.9
Na meg/L	137		141	141	139	142	-	138, 141	140
K meg/L	3.7		3.4	3.9	4.3	3.2	-	4.6, 3.9	4.0
Cl meg/L	105		109	104	108	107	-	110, 108	107
Tot CO_2 meg/L	27		25	36	28	32	-	31, 30	29
fecal consistency	4, 4, 4		4, 4, 4	4, 4, 4	4, 4, 4	4, 4, 4	4, 4, 4	4, 4, 4	3
weight (kg)	365		348, 341, 341	348,350, 360	358, 371, 375	380, 382	382, 385	386, 389, 391	
SDH I.U.	3.3		2.7	2.0	1.5	2.4	2.7	4.1, 3.3	.3

Multiple samples for a week are listed with the earliest value first in the square.

Table 13
D. CHUBBS

	WEEK 0	WEEK I	WEEK II	WEEK III	WEEK IV	WEEK V	WEEK VI	WEEK IX
Fibrinogen mg %	200	400, 400, 400	300, 400, 300	200, 200	100	300	100	200
PCV %	28	34, 31, 28	29, 28, 29	28, 31	27	29	34	34
Hb g/dl	11	13, 12, 11	11, 11, 11	11, 11	10	11	13	13
RBC $x10^6$	6.4	7.6, 7.2, 6.4	6.7, 6.3, 6.5	6.4, 6.7	6.0	6.3	7.5	7.3
WBC $x10^6$	6.0	5.1, 5.4, 5.9	8.7,11.6,14.3	9.2, 8.0	6.4	6.8	7.6	8.1
bands		- , 59	174, 71	-	-	-	-	-
segs	3240	2856, 2950	5133; 11,011	6716, 4880	3232	3332	4636	4293
lymphs	2340	2091, 2537	3045, 2932	2392, 3040	2560	3060	2736	3402
mono	180	102, 118	348, 286	92, -	160	68	76	81
eos	240	51, 177	- -	- , 80	384	340	76	324
baso	-	- , 59	- -	- -	64	-	76	-
BUN mg %	16	14, 22, 16	14, 14, 13	21. 18	16	16	15	15
glucose mg %	87	113, 105, 98	82, 89, 82	105, 88	94	118	103	111
calcium mg %	11.8	12.3,11.0,10.3	11.5,10.4,10.8	11.4, 11.5	10.7	12.8	12.5	13.3
Phos mg %	3.5	3.6, 3.5, 3.7	2.6, 2.7, 2.2	2.9, 3.0	3.7	2.9	2.6	2.7
Na meg/L	141	139. 139. 139	142. 141. 145	139. 142	139	145	142	143
K meg/L	3.4	3.5, 3.4, 2.8	3.2, 3.5, 3.2	2.9, 3.9	3.9	3.8	4.2	3.9
Cl meg/L	109	105, 103, 103	110, 109, 110	110, 106	104	112	107	109
Tot CO_2 meg/L	29	28, 32, 33	29, 28, 30	28, 32	30	27	31	29
fecal consistency	4	4, 4, 4	4, 4, 4	4, 4, 4	4, 4, 4	4, 4, 4	4, 4, 4	3
weight (kg)	343	342, 341, 341	340. 343, 350	351, 352, 349	350, 354, 355	356, 359, 359	362, 362, 361	
SDH I.U.					2.6	2.8	1.7	1.4

Multiple samples for a week are listed with the earliest value first in the square.

Table 14
E. AMY

	WEEK 0		WEEK I	WEEK II	WEEK III	WEEK IV	WEEK V	WEEK VI	WEEK IX
Fibrinogen mg %	400		200, 500	800	300, 400	200	300	300	300
PCV %	30		25, 32	24	30, 34	30	32	32	35
Hb g/dl	10.9		9.5, 12.1	9.2	11.9, 12.8	11.4	11.8	11.8	12.0
RBC $x10^6$	7.4		6.6, 8.4	6.6	8.2, 8.8	7.9	8.4	8.2	9.0
WBC $x10^6$	12.2		14.5, 11.0	12.8	11.3, 11.4	12.2	12.1	10.9	11.1
bands	-		435, 605	512	56	-	-	-	-
segs	7564		12,398; 6160	7168	5707, 4788	5429	4054	4087	4385
lymphs	4209		1522, 3080	4800	5142, 6384	6222	7683	6213	6549
mono	305		145, 715	320	169, 228	244	242	-	55
eos	122		-, 440	-	226, -	244	121	-	111
baso	-		-	-	-	61	-	-	
BUN mg %	14		14, 10	13	16, 12	14	17	16	11
glucose mg %	90		110, 106	93	85, 79	95	104	94	94
calcium mg %	12.1		10.7, 10.8	10.4	11.1, 12.4	11.9	11.9	12.3	12.3
Phos mg %	4.1		2.7, 4.2	4.8	3.3, 4.5	4.1	4.7	3.8	3.7
Na meg/L	137		141, 136	139	137, 139	141	139	140	138
K meg/L	4.0		3.0, 4.5	4.3	3.7, 4.3	4.2	4.3	4.5	4.7
Cl meg/L	104		101, 102	106	101, 103	108	105	110	104
Tot CO_2 meg/L	26		29, 30	27	35, 31	31	30	29	29
fecal consistency	4		4, 4, 4	4, 4, 4	4, 4, 4	4, 4, 4	4, 4, 4	4, 4, 4	3
weight (kg)	386		384, 384, 373	373, 376, 375	380, 383, 389	392, 395	401, 407	414, 417, 416	
SDH	3.1		4.5, 2.2	2.3	1.6, 1.5	2.1	2.0	4.3	0.3

Multiple samples for a week are listed with the earliest value first in the square.

Table 15
F. STAR

	WEEK 0	WEEK I	WEEK II	WEEK III	WEEK IV	WEEK V	WEEK VI	WEEK IX
Fibrinogen mg %	100	200, 200	400, 500	400	200	200	200	300
PCV %	28	26, 32	31, 26	29	29	32	35	33
Hb g/dl	10.5	9.6, 11.8	11.7, 9.9	11.0	11.4	11.6	13.1	12.1
RBC $x10^6$	6.8	6.4, 7.6	7.4, 6.3	6.8	7.2	7.5	8.3	6.1
WBC $x10^6$	9.4	4.4, 7.4	13.0, 18.3	17.0	10.3	11.9	12.7	10.4
bands	-	88	520	85	-	-	-	-
segs	4230	2640, 4662	9035; 14,000	13,175	6695	6545	6985	5616
lymphs	4042	1682, 2590	3120, 3294	3060	3245	4582	5017	3744
mono	376	-, 148	325, 234	425	206	178	254	104
eos	752	44, -	-, 732	255	154	535	317	884
baso	-	-	-	-	-	60	127	-
BUN mg %	16	14, 34	28, 12	10	13	12	13	15
glucose mg %	100	115, 97	103, 89	83	98	101	112	98
calcium mg %	11.7	9.5, 10.3	10.5, 10.6	10.7	11.2	11.7	11.5	11.4
Phos mg %	5.4	4.7, 5.4	5.0, 3.1	3.7	4.6	4.4	4.8	4.6
Na meg/L	139	136, 136	136, 139	140	139	140	140	141
K meg/L	4.0	3.1, 3.7	4.0, 4.3	4.1	3.9	4.1	4.4	4.1
Cl meg/L	102	99, 101	100, 106	105	104	103	108	106
Tot CO_2 meg/L	34	29, 24	28, 27	30	31	27	31	32
fecal consistency	4	4, 5→7, 7, 7	6,5,3,3,4,4	4, 4, 4	4, 4, 4	4, 4, 4	4, 4, 4	3
weight (kg)	336	333, 330, 325	327, 330, 329	331, 332, 331	330, 332, 332	333, 333, 340	334, 335, 337	
SDH I.U.	2.6	9.3, 3.9	10.1, 4.8	3.8	2.0	1.6	4.7	2.0

Multiple samples for a week are listed with the earliest value first in the square.

Table 16
G. MOLLY

	WEEK 0		WEEK I	WEEK II	WEEK III	WEEK IV	WEEK V	WEEK VI	WEEK IX
Fibrinogen mg %	200		500, 500, 400	600, 300	300	300		200, 100, 100	200
PCV %	30		33, 39, 33	33, 32	40	41		35, 38, 32	38
Hb g/dl	11.2		12.6,14.7,12.6	12.4, 11.5	14.7	14.8		13, 14, 12	14.8
RBC $x10^6$	6.0		6.4, 7.7, 6.7	6.7, 6.4	7.9	8.0		7.0, 7.8, 6.6	8.0
WBC $x10^6$	10.4		8.8,11.6,12.4	13.7, 12.3	11.1	11.3		10.9, 9.9, 9.7	12.4
bands	52		- , 1240	41, -	-	-	-	54, -	-
segs	7332		6204, 7564	9316, 9471	7104	6924		8775, 6257	7564
lymphs	2652		2244, 3348	3357, 2337	3552	3955		2017, 3249	3968
mono	156		220, -	499, 369	333	282			372
eos	208		88, 248	137, 123	111	339		54, 97	496
baso	-		44, -	-	-	-	-	-, 97	-
BUN mg %	21		20, 20, 22	15, 11	16, 14, 13	21	17	24, 22, 12	15
glucose mg %	97		113, 96, 114	88, 104	105, 108, 102	114	110	111, 98, 88	104
calcium mg %	10.9		11.1,10.3,10.7	11.0,10.7	11.3,11.8,11.2	11.0	11.4	10.6,10.5,10.9	10.4
Phos mg %	3.3		4.3, 5.1, 4.8	4.0, 2.9	2.6, 2.5, 2.8	3.5	3.3	2.0, 2.4, 2.4	2.4
Na meg/L	142		142, 143, 143	140, 141	142, 148, 145	147	145	136, 143, 142	139
K meg/L	3.2		3.3 3.0 4.5	3.6, 3.3	3.8, 4.2, 3.7	3.9	3.8	3.7, 4.2, 3.6	3.1
Cl meg/L	108		108, 110, 110	109, 113	113, 116, 107	110	114	109, 114, 112	108
Tot CO_2 meg/L	30		24, 24, 26	25, 27	28, 30, 31	31	28	26, 26, 29	24
fecal consistency	3		1-2, 2, 3, 3	2, 2, 2, 2	3, 2, 5, 3,	2, 2, 2, 2	2, 2, 2, 2	2, 2, 3, 2	3
weight (kg)	304		296, 295, 297	287, 286, 290	288, 286, 285	285, 282, 281	280, 276	272, 269, 264	
SDH I.U.					6.4	5.2		3.6, 2.9, 2.0	2.5

Multiple samples for a week are listed with the earliest value first in the square.

Table 17
H. GOOSE

	WEEK 0		WEEK I	WEEK II	WEEK III	WEEK IV	WEEK V	WEEK VI	WEEK IX
Fibrinogen mg %	200		400, 400, 400	500	400	300	100	100	200
PCV %	35		35, 33, 31	32	33	35	38	34	36
Hb g/dl	13.6		13.3,12.3,12.1	11.9	11.9	13.0	14	12.9	14
RBC $x10^6$	7.5		7.6, 7.2, 7.0	7.0	7.0	7.7	8.2	7.6	8.1
WBC $x10^6$	9.9		7.3, 6.2, 9.3	15.7	13.2	12.1	13.2	12.6	13.0
bands	100		- , 248, -	-	-	-	-	-	72
segs	5550		2628, 6417	11,697	9108	7563	7920	7623	5372
lymphs	3500		4307, 2232	3375	4026	3993	4752	4221	6413
mono	250		365, 465	471	66	363	132	567	540
eos	550		- , 186	157	-	121	396	189	540
baso	50		- , -	-	-	60	-	-	-
BUN mg %	17		27, 25, 20	12		17	13	16	13
glucose mg %	104		98, 114, 106	94	108	103	95	108	112
calcium mg %	12.7		11.7,11.0,10.3	11.6	11.2	11.9	12.8	12.2	10.7
Phos mg %	5.3		4.1, 4.7, 4.6	3.5	3.2	4.1	4.2	3.2	4.0
Na meg/L	142		143, 140, 138	140	143	147	150	141	134
K meg/L	3.6		3.8, 3.7, 3.5	3.2	4.0	3.9	4.6	4.2	4.0
Cl meg/L	106		112, 110, 106	107	101	111	113	106	103
Tot CO_2 meg/L	29		24, 26, 28	32		31	31	30	29
fecal consistency	4		4, 4, 4	4, 4, 4	5, 5, 4	4, 4, 4	4, 4, 4	4, 4, 4	3
weight (kg)	414		406, 400, 395	385, 385, 384	390, 394, 389	394, 400, 404	409, 402, 407	411,413, 418	
SDH I.U.						2.8	3.1	2.6	2.5

Multiple samples for a week are listed with the earliest value first in the square.

Table 18
I. SNOWFLAKE

	WEEK 0		WEEK I	WEEK II	WEEK III	WEEK IV	WEEK V	WEEK VI	WEEK IX
Fibrinogen mg %	500		100	500, 1000	900	300	300	300	200
PCV %	35.2		38.4	26.7, 26.3	28.5	28.1	29.1	29.5	40.2
Hb g/dl	13.4		14.4	9.7, 9.7	10.4	10.6	10.3	10.9	14.7
RBC $x10^6$	7.23		7.68	5.34, 5.24	5.79	5.93	5.94	6.30	8.67
WBC $x10^6$	6.8		6.2	9.6, 5.6	17.4	4.6	8.3	8.3	12.1
bands	-		-	864, -	87	-	-	-	-
segs	3876		3720	4896, 4424	15,051	1978	5229	6557	8652
lymphs	2448		1860	3168, 1120	1479	2070	2241	1743	2964
mono	68		310	672	522	322	581		121
eos	408		186	- , -	87	138	166	-	363
baso	-		124	- , 56	174	92	83	-	-
BUN mg %				21	28	21	18	16	17
glucose mg %	91		103	79	64	88	99	84	106
calcium mg %	13.2		11.5	9.9	11.4	11.2	10.7	10.7	12.8
Phos mg %	3.4		2.9	3.2	2.3	2.7	2.6	2.3	2.8
Na meg/L	139		148	139	142	141	139	137	145
K meg/L	3.8		1.9	3.2	4.1	3.9	3.2	4.5	3.8
Cl meg/L	110		114	104	110	109	107	109	108
Tot CO_2 meg/L	27		23	29	21	29	24	24	31
fecal consistency	4		4, 4, 4	4, 4, 4	4, 4, 4	3-4, 3-4, 4	3-4, 3-4, 4	3, 4, 4	3
weight (kg)	309		295, 290, 287	285, 279, 284	281, 280, 279	278, 276, 277	275, 279, 276	277, 279, 277	
SDH I.U.	2.3		2.3	2.8	3.5	2.3	2.0	3.4	.7

Multiple samples for a week are listed with the earliest value first in the square.

SOME EFFECTS OF THE REMOVAL OF VARYING LENGTHS OF THE DISTAL SMALL INTESTINE IN HORSES

by

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Although the small intestine of the horse is involved primarily and secondarily in many medical and surgical diseases, knowledge of specific effects of the removal of varying lengths of small intestine is lacking. This study was undertaken to quantify these effects in hopes of clarifying potential postsurgical problems. This study was also designed to provide some insight regarding how much small intestine could be removed without producing irreversible weight loss, diarrhea, or metabolic disorders.

Nine mixed breed horses of both sexes between 3 and 17 years of age were randomly divided into three equal groups with 0, 4.6 m (15'), and 9.2 m (30') of distal small intestine removed from Group I, II, and III respectively, following the completion of a conditioning program which included a standardized pelleted ration. Monitoring of patient progress was accomplished by clinical observation, serial body weights, various serial blood counts and blood chemistry, fecal evaluation, and preoperative and postoperative oral glucose tolerance tests. At the termination of the study the horses were returned to a roughage diet in preparation for further digestion trials.

Although the number of animals used in this project did not permit statistical analysis, the results do suggest trends. No gross fecal changes occurred in any group, supporting the hypothesis that diarrhea in adult horses is a result of malfunctioning of the large bowel.

All horses lived and weight gain occurred in Group I, II and I horse in Group III in which the 9.2 m of small intestine removed was < 50% of the total small intestine length. Weight loss occurred in the two horses in Group III where the 9.2 m of small intestine removed was > 50% of the total small intestine length.

Transient albumin and globulin deficits, attributed to loss at surgery, occurred in all three groups. However, Groups II and III displayed more prolonged deficits possibly related to lymphatic obstruction, protein losing

gastroenteropathy, and peritonitis.

Little correlation was observed between the glucose absorption tests, weight loss or gain, and possible malabsorption. The horses in Group III displayed some change in postoperative glucose absorption. In each instance improvement in absorption occurred with increasing time from surgery. Intestinal adaptation which occurs in humans and other animals may occur in horses.

Group III horses preferred roughage compared to those in Groups I and II, possibly as a means of adapting dietary intake to the residual functional capacity of the digestive tract. This would be consistent with decreased soluble carbohydrate and protein digestion and absorption in the reduced small intestine, thereby increasing the demands on the large intestine and its conversion of carbohydrates into volatile fatty acids. Since the horse appears to be more efficient in deriving energy from carbohydrate as compared to protein utilization in the large intestine, long term effects of the removal of a major portion of the small intestine absorptive area may be more devastating to protein metabolism than to carbohydrate metabolism.

Further investigation to determine the specific effects of small intestine function, surface area, and intestinal length with respect to total body surface area in regards to carbohydrate and protein digestion and absorption needs to be done.