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PROCEEDINGS

FIRST INTERNATIONAL EQUINE VETERINARY CONFERENCE – PRETORIUSKOP, KRUGER NATIONAL PARK, AUGUST 5-10, 1974

VERHANDELINGE

EERSTE INTERNASIONALE HIPPIATRIESE KONGRES, PRETORIUSKOP, KRUGER NASIONALE WILDTUIN, AUGUSTUS 5-10, 1974

SECTION B: THE ALIMENTARY TRACT OF THE HORSE
The Aetiology, Diagnosis and Therapy of Diseases and Metabolic Complications

Papers and Discussions — **Referate en Besprekings**

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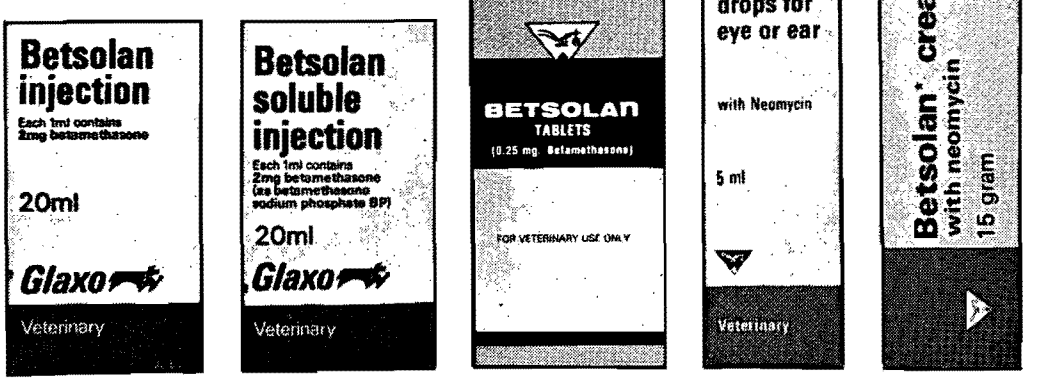

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CORRIGENDA: VOL. 45 Corrigenda: Vol. 45 No. 4 1974

Article by H.H. Krzywanek: "Lactic acid concentrations and pH values in trotters after racing." Page 358, first column, line 4: For: **60m/min** read **600 m/min.**
Page 359, Reference 7: For **Acta vet. scand. 15:1** read: **Acta vet. scand. 15:310.**
Reference 10: For: **Bagdanow** read: **Bogdanow.**

Open Discussion on Respiration, Biochemistry, Electrolytes and Performance.
Page 371, first column, discussion by H.H. Krzywanek, 2nd paragraph, first line; for: **water and soap** read: **water and alcohol.**

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VERHANDELINGE VAN DIE EERSTE INTERNASIONALE HIPPIATRIESE KONGRES PRETORIUSKOP, KRUGER NASIONALE WILDTUIN, AUGUSTUS 5 – 10, 1974

SECTION B: THE ALIMENTARY TRACT OF THE HORSE
The Aetiology, Diagnosis and Therapy of Diseases and Metabolic Complications.

FIRST SESSION: DIGESTION

Chairman: P. BOYAZOGLU

FUNCTIONAL ANATOMY AND NERVOUS CONTROL OF THE EQUINE ALIMENTARY TRACT*

H.P.A. DE BOOM**

SUMMARY

By outlining its ontogenetic development, the topographical anatomy of the equine digestive tract is presented in concise form. Those aspects of particular significance from the functional and clinical point of view are stressed. The anatomy of that part of the autonomic system supplying the digestive tract is sketched in brief with some remarks pertaining to function.

INTRODUCTION

This paper merely serves as a general theoretical background to those that are to follow in this particular section of the Conference. Perforce, it will be restricted to generalities: it is a quick overall memory-refresher and not a presentation of research results.

ONTOGENESIS

The digestive system develops from a simple, entodermal tube, abutting rostrally on the pharyngeal membrane, which temporarily closes it off from the ectodermal bay or stomodeum, the anlage of the oral cavity, and caudally on the temporary cloacal membrane, separating it from the proctodeum which represents the future anal canal of ectodermal origin. The persistence of the cloacal membrane forms the basis of various forms of atresia ani occasionally encountered in the new-born. Atresia coli is regarded as a lethal factor in horses [18 23].

From the midgut region of the entodermal tube the yolk sac, initially a very prominent structure in the horse, is suspended by the yolk stalk with its vitelline duct. A remnant of this connection, namely Meckel's diverticulum, is best known in sheep.

From the hindgut the allantois develops as an evagination. Separation caudalwards of this evagination, between the allantoic stalk, or urachus, ventrally, and entodermal tube dorsally, produces the eventual separation between alimentary and genito-urinary tracts. Incompleteness of separation and permanence of the cloacal membrane produces the congenital recto-vaginal fistula so often associated with atresia ani. The alimentary tract undergoes relatively simple changes in principle, despite the apparent complexity of the final product.

The entoderm differentiates either into squamous stratified epithelium – oesophagus and initial part of the stomach – or simple columnar epithelium, as in the rest of entodermal component of the gut, *i.e.*, up to and including the rectum, while the surrounding mesenchyme produces the muscular and connective tissue layers.

The liver and pancreas arise initially as diverticula from the entodermal tube, the liver and major duct component of the pancreas from two closely associated ventral diverticula, the dorsal pancreatic component from a dorsal diverticulum, thus dictating the eventual relationship of their points of entry into the duodenum.

The stomach region of the tube develops a dorsal convexity, the major curvature, and a concave ventral one, the minor curvature. Rotation along a longitudinal (craniocaudal) axis to the left, approximation of cardia and pylorus, swinging the duodenum to the right, and final rotation of the stomach along a transverse axis, the fundus region tilting caudalward in keeping with diaphragmatic movement and configuration, produces the adult morphology and topography.

Elongation of the entodermal tube in great excess

* For illnstrations the reader is referred to standard texts on Anatomy and Embryology.

**Department of Anatomy, Faculty of Veterinary Science, P.O. Box 12580, Onderstepoort 0110.

relative to the lengthening of the body as a whole produces loops, coils and festooning of the intestine. In the horse this relationship will eventually attain a ratio of 10:1, compared to 5:1 for the carnivorous dog, 15:1 for the omnivorous pig and 20:1 to 25:1 for ruminants.[16]

The future intestinal tract undergoes a primary or umbilical loop ventralward, with the cranial mesenteric artery as axis, which loop then rotates through almost 360° around this axis, in clockwise fashion as seen from the dorsal aspect. It is this rotation which results in the basic pattern of ascending (right), transverse and descending (left) colon, as well as in cranial flexure, descending limb, caudal flexure and ascending limb of the duodenum. The two systems fit into one another like the intercalation of an inverted and an upright U. The proximity of the duodenum to the initial part of the colon is thus effected. It is maintained by the duodenocolic ligament – a useful practical landmark in differentiating duodenum from jejunum at the duodenojejunal flexure.

In herbivores the ascending limb of the colon becomes greatly elongated and is consequently thrown into loops and coils. In the Perissodactyla this morphogenetic manoeuvre produces a long loop cranialward, which, within the confines of the abdominal cavity, is forced to the left and caudally, thus falling into an inverted U-pattern with the well-known components: Right ventral colon, sternal flexure, left ventral colon, pelvic flexure, left dorsal colon, diaphragmatic flexure and left dorsal colon. In the process, the mesentery of the ascending limb becomes drawn out and narrowed, finally presenting itself as a narrow ligament connecting the two limbs rather than as a mesentery, thus leaving the greater part of the colic loop potentially mobile.The caecum appears as an evagination at the ileocolic junction. Enlargement of its lumen concomitantly with that of the colon crowds the ileum to the left and gives the latter the morphological appearance of being a side-passage entering the main stream by means of a T-junction. Adopting a teleological mode of argumentation merely for ease of comprehension, one may state that the Perissodactyla, faced with the choice of site for a continuous culture system for cellulose-splitting bacteria, chose the post-intestinal region in contrast to the more logical pre-intestinal one selected by the ruminants, much to the former's detriment, and to the veterinarian's chagrin. The lumen of caecum and ascending and initial part of the transverse colon increased tremendously to supply the necessary capacity. The relatively faster passage of a barium meal through the large intestine of a suckling foal compared to the rate in a weaned foal [1] is also indicative. Besides developing a marked narrowing of lumen at the pelvic flexure and in the initial part of the transverse colon, the Equidae – as if to test the veterinarian's mettle further – developed a marked, sudden constriction in the initial part of the ascending colon (right ventral colon), whereby the caecum became enlarged at expense of the colon: the caecocolic orifice of adult anatomy is thus in reality a narrowing in the lumen of the initial part of the colon, and the cranial part of the base of the caecum is ontogenetically speaking the initial part of the ascending colon.

The Rhinoceros, representing a remaining living example of equine ancestry, retains the simpler ontogenetic pattern.

ANATOMY

For the purposes of this paper attention will be limited to certain aspects of the tubular components of the alimentary tract, *i.e.*, from oesophagus to anus.

Oesophagus: Initially it lies in the median plane, between cervical vertebral column and associated musculature dorsally, and trachea ventrally. At the caudal third of the neck (about the level of the fifth cervical vertebra) it inclines to the left, very occasionally to the right, [2 11] where it is most easily accessible. Here, in the lowest position of the oesophagus, the common carotid artery lies dorsolaterally, with the associated vago-sympathetic trunk dorsally and the recurrent nerve medioventrally to the vessel. The deep ventrovertebral fascia forms a sheath around it.[6] Disruption of the tracheal side of the sheath would give entry to the peritracheal adventitia. From this region, as well as from the perioesophageal adventitia, it is but a brief, descending pathway to the mediastinal endothoracic fascia. Further warning is unnecessary.

At the thoracic entrance the oesophagus reverts to its dorsotracheal position. I have often wondered whether its proximity here to the left cervicothoracic ganglion, hard against the upper end of the first rib, could not play a rôle in the deleterious effects of choke.

At the level of about the 14th vertebra and about 12cm below it[16] (closer to it in soft or frozen specimens)[22] and slightly to the left of the median plane, the oesophagus traverses the obliquely disposed oesophageal hiatus of the diaphragm and upon emergence therefrom enters the cardia.

The horse's powerful oesophageal musculature is of the striated type only up to the root of the lung, where it is 4-5 mm thick, [16 20] to be replaced caudalwards by smooth muscle of increasing thickness (up to 1,2-1,5 cm) [16 20] at the cardia.

Initially the outer coat forms elliptical turns in double stranded loops, which are more closely spiralled in the middle region, to go over into the outer longitudinal layer. The deeper-lying circular coat is mainly responsible for the increase in thickness of the muscular tunic towards the cardia, where many of the muscle bundles cross in irregular plexiform manner.

The oesophagus can be distended to an average internal diameter of 6 mm.[20] There are minor variations in calibre noticeable in the gas– or fluid–distended oesophagus. Succeeding a narrow entrance, there is a moderate elliptical dilatation with a slight constriction, followed by a relatively large dilatation in the cervical region and a distinct narrowing at the thoracic entrance; a widened thoracic region gradually narrows down to a minimum towards the cardia.[2]

The mucous membrane of squamous stratified epithelium is freely movable relative to the musculature owing to the loose submucosa. Only at the oesophageal entrance from the pharynx is lubrication provided in the form of mucous glands.

Stomach: The unilocular, compound stomach, with a meagre physiological capacity of 8–15 l,[16] is nestled against the diaphragm cranially. In the ventral two-thirds of this contact area the left lobe of the liver is wedged. Laterally, the diaphragm, curving to its attachment to the closely approximating rib cage, also covers the stomach. Along its greater curvature the spleen becomes interposed between stomach and left body wall. To the right, liver and large colon adjoin

the stomach. Caudally the visceral face of the stomach is related dorsally to the relatively firmly fixed transverse colon (transition from large to small colon) and, more ventrally, to the coils of small colon forms a veil between the visceral face of the stomach and the above mentioned intestinal coils. The greater curvature of the stomach normally rests upon the dorsal colon; when the stomach is empty, coils of small intestine are interposed; when it is distended, it may push the left dorsal colon aside.

Dorsally, from the expanded portion of fundus peculiar to horses *(the saccus caecus)* the gastrophrenic ligament arises to form a firm link with crura of the diaphragm. When the stomach is empty, this attachment causes the *saccus caecus* to be drawn out into a cone. The stomach is thus firmly held in position, yet direct pressure on the stomach by contraction of abdominal musculature is not possible. Abnormal distension is bound to produce considerable discomfort. Clinically, the last couple of ribs on the left side will be raised, producing an asymmetrical appearance when viewed from the front.[6] Trocarization, as advocated through the dorsal end of the 17th intercostal space, does make one feel uneasy when considering the spleen's position, itself possibly enlarged because of expected compression of the gastrolienal vein. Even at the 15th intercostal space danger is not entirely eliminated. In any case the pleural cavity must be traversed and lung damage at deep inspiration is not necessarily obviated.

The cardia is characterized by the oblique entry of the oesophagus, the very thick muscular coat of the latter at this site and the formation of the powerful cardiac sphincter, which appears not as a typical sphincter but as two semilunar folds, one dorsal and one ventral, which are slightly staggered in position. The more proximal, ventral one is formed by a loop of the circular fibre coat, the more distal, dorsal one by a loop of the internal oblique fibre coat. This arrangement would provide even better occlusion than a typical sphincter. Numerous folds of mucous membrane are stated to occlude the opening, so that distension of the stomach to the point of rupture may be produced by forcing air or fluid into the stomach from the pyloric side without ligating the oesophagus [22].

The edges of the layer of internal oblique fibres, lying close to the lesser curvature, form a groove, the *sulcus ventriculi*, spanned crosswise in its floor by the circular fibres. In the pyloric part the longitudinal fibres rather suddenly become thick and powerful and the circular fibres at this point form a slight retraction, which demarcates the more proximal antrum from the more distal, thick walled pyloric canal.*I doubt whether the physiological significance of these structures has been considered adequately.

Small Intestine: The *duodenum* of the horse is relatively well fixed in position, the cranial part being held to the liver by the terminal part of the small omentum (hepatoduodenal ligament). The mesoduodenum, supporting the descending transverse and ascending parts, is shortened by the fact that the base of the caecum, on account of its size and that of the caecum as a whole, has been forced against the abdominal roof; its serosal covering has fused secondarily with whatever peritoneal membranes were in the way, namely mesoduodenum containing the pancreas and ventral surface of the right kidney. One thus finds the initial part of the descending duodenum running dorsocaudally, skirting the right lobe of the liver and the right dorsal colon, the rest of it then forming a wreath around the base of the caecum. At the duodenojejunal flexure the short duodenocolic fold fixes it to the transverse colon. In view of its course, it must be subject to considerable pressure in cases of tympany of the caecum. In the first part of the duodenum the *ansa sigmoidea*, an ∿-shaped curve, presents itself as two dilatations with a constriction between them. The first one is clearly demarcated between the pyloric sphincter and the above-mentioned constriction, thus producing the *ampulla duodeni*, typical of horses. This anatomical arrangement also opens one's mind to speculation as to its physiological significance.

Beyond the constriction in the second part of the *ansa sigmoidea*, about 12-15 cm from the pylorus, the *ampulla hepatopancreatica* [10] (formerly known as the *diverticulum duodeni*) is situated, in which the common hepatic duct (ductus choledochus of other species) and the major pancreatic duct open.

The musculature of the ampulla, although oblique and overwhelmed by longitudinal fibres and not independent of the duodenal musculature as in man, is now regarded as forming the *M.sphincter ampullae hepatopancreaticae*. [10] Nearly opposite the ampulla is the opening of the minor pancreatic duct.

The *jejunum* occupies mainly the left sublumber region, dorsal to the large colon and caudal to the stomach. Here it lies intermingled with coils of the small colon, which are generally more dorsally disposed.[6]

Because of the very wide mesojejunum, (about 50 cm), coils of small intestine may interpose themselves between left dorsal colon and stomach (when the latter is fairly empty), between left dorsal colon and left flank, extending to the right flank behind the caecum, (when it is relatively empty), and between the ventral colon and caecum to the abdominal floor. The width of the mesentery is sufficient also to allow coils of the jejunum to enter the pelvic cavity and even descend into the scrotum, provided of course the vaginal canal, *i.e.*, the peritoneal lining of the inguinal canal, is abnormally wide – normally it will admit just about one finger in a large stallion – or provided it has been torn. While on this aspect, the possibility of a loop of gut being caught through a tear in the *mesoductus deferens (plica ductus deferentis)* produced *inter alia* by rough handling at castration, should be borne in mind. Part of the jejunum may even enter the vestibule of omental bursa through the epiploic foramen. Apart from allowing abnormal positions to be attained, the width of the mesentery is a prerequisite for the development of volvulus and intussusception.

The last part of the small intestine is regarded as *ileum*. In formalin-preserved cadavers it is usually tightly contracted and firm to the touch, and about 20 cm long; if relaxed and atonic, it measures 50-70 cm [21] Arbitrarily, and for purposes of definite measurement (or identification), the ileum may be regarded as that part of the small intestine which moves away from the edge of the mesentery, leaving an antimesenterial fold, which subsequently attaches to the

* There is a discrepancy between the terminology in Anglo-American and Continental literature. The former is followed here, in apparent accordance with the NAV.

dorsal band of the caecum (the ileocaecal fold). The ileum in the final part of its course proceeds almost straight dorsalwards just right of the median plane at about the middle of the lumbar region or slightly cranial thereto, and ends at the lesser curvature of the base of the caecum. Here, at the ileal ostium, the ileum is telescoped into the caecum and causes a papillalike projection into the lumen of the caecum, the *papilla ilealis,* with a central system of radiating folds. Remarkably enough, the ileal musculature diminishes in thickness at the point of entry; there is no sphincter but a rich venous plexus is present in the submucosa [21]. The latter, together with the powerful ileal musculature – it can cause a threefold shortening of the ileum – represents a regulating mechanism for proper transport of jejunal contents and prevention of entry of gas from the basis caeci [21]. The relatively fixed ileum can act as a pivot for the development of a volvulus by the more mobile jejunum.

Large Intestine: The *caecum* has the classically described 'comma' shape. From the ileal ostium cranialwards, the caecal base represents the dilated and recurved initial portion of the colon, as explained under the heading 'Ontogeny'. From its highest point in the sublumbar region it curves cranial- and ventralwards to about the 15th or even 14th rib, below the middle of the latter, here touching the liver, depending on the degree of fullness of the right dorsal colon. From here it curves back, forming the blind sac ventrally. At its caudal extremity the caecocolic ostium points dorsocaudally. Consequently the two ostia, ileal and caecocolic, are in close proximity to one another (±5 cm) with an intervening fold of mucous membrane between them. The caecocolic ostium is slit-like or elliptical in outline, about 5 cm long, and is guarded by a ventrally situated valve (*valva caecocolica*) and a ring of muscle (*M.sphincter caeci*). Anatomically speaking there is no direct passage of ileal contents to the colon: The caecum forms a huge diverticulum of about 25-30 *l* capacity

The body of the caecum curves ventrocranialward to the abdominal floor, its lesser curvature roughly parallel to the costal arch and some 10–15 cm caudal to it. The apex normally lies on the abdominal floor, about a hand's breadth behind the xiphoid, fitting snugly between right and left ventral colon. It has no attachments and depends for its position on the loop of the ventral colon. A case of rotation of body and apex through 540° and one of spiralling thereof, both with prolonged signs of colic, have been described.[13] Gas accumulating in the carcum will tend to rise to the highest point, namely the most dorsal part of the caecal base in the sublumber region. If this occurs to abnormal degree, trocarization must be performed at the highest point of the bulge halfway between *tuber coxae* and last rib on the right side.

Of its four taeniae, the dorsal one is attached for a short distance to the ileocaecal fold. It extends to the apex. The medial one also extends to the apex and bears the medial caecal vessels. The ventral one is free and joins the medial one near the apex. The lateral one, bearing the lateral caecal vessels, is attached to the right ventral colon for a considerable distance, thus the details of identity and topography in this region are obscured, although in its caudal part the band can be felt as a concave projecting edge [22]. It may peter out before reaching the apex.

The *large colon*, of more than double the capacity of the caecum, anatomically speaking begins at the narrow caecocolic ostium; it immediately widens and curves sharply dorsally, caudally and then ventrally and cranially.

This initial portion lies against the right flank immediately below the caecal base; by virtue of its shape and course it can form a gas trap. In tympany of this segment, it can push the caecal base medialward and usurp its position, thus leaving the site for trocarization unchanged. As right ventral colon, the gut continues cranialward in the position indicated by its name. For about half its initial distance its ventrolateral band is attached to the lateral band of the caecum by the caecocolic fold. At the sternal flexure it turns to the left and continues caudalward to the pelvic flexure. This flexure, being unattached, usually assumes a recurved course and a very variable position: As a rule it is directed towards the right flank and may lie against the caudal part thereof, or it might be found in the right inguinal region. From the pelvic flexure the left dorsal colon runs cranialward. At the diaphragm and left lobe of the liver it forms the diaphragmatic flexure and continues as the left dorsal colon against the liver and, below the latter's edge, against the left body wall and the diaphragm curving to its attachment to the ribs and lower rib cage. The duodenum rides atop of it. It then skirts the caecal base, partially below it but mainly to the left thereof, to turn to the left and dorsally, caudal to the stomach and cranial to the cranial mesenteric artery, to narrow considerably into the small colon under the left kidney. This transverse segment at about the level of the 17th to 18th thoracic vertebra, cranial to the root of the mesentery, represents the transverse colon. The terminal part of the large colon is attached broadly by peritoneum and areolar connective tissue to the left face of the caecal base and dorsally to the ventral face of the pancreas. This represents an area of secondary fusion owing to the large size and position of the viscus. The adherence is continued over the whole transverse colon. In the process, the origin of the omentum (dorsal mesogastrium) has become trapped between colon, pancreas and body wall and thus appears to take origin from the transverse colon. The right dorsal colon is also attached to a fold from the right lateral ligament of the liver. Apart from the attachments mentioned, the whole large colon is free to move, its size and mass (with contents) within the confines of the abdominal cavity being the only restricting factors. The mesocolon plays no part in this restriction: it has become greatly extended in width with the development of this remarkable loop of ascending colon, but so shortened fore to aft, that it merely finds the two limbs of the loop together, becoming obliterated in the process and merely recognizable as a mesenterial structure at the pelvic flexure: Elsewhere it is replaced by a broad connective tissue attachment between the two limbs of the loop. Furthermore the mesocolic axis can act as an axis of rotation, around which dorsal and ventral colons can rotate. This is most likely to occur in the left part of the colic loop, and could then be diagnosed by rectal palpation. Huskamp[9] has described amputation, closing of the stumps and anastomoses between the latter as surgical treatment where the twisted loops of colon were not viable.

As on the caecum the taeniae are useful for identi-

fying a particular section of gut at autopsy or clinically (Fig.). The entire ventral colon has four taeniae: The ventromedial and ventrolateral bands are free and exposed, except for the first portion of the ventrolateral band which is attached to the lateral taenia of the caecum by means of the caecocolic fold as already described. The dorsal bands are concealed in the area of broadened mesocolic adherence between ventral and dorsal colons. They can be palpated. The dorsomedial one is followed by the *ramus colicus* (arterial and venous) which is the specific supply and afflux for the initial part of the ascending colon in all species. The colic lymph nodes accompany the vessels. The left dorsal colon has only one band, clearly palpable per rectum at the pelvic flexure, and swinging up from the dorsomedial one on the left ventral colon to continue ventrally along the mesocolic attachment, indicating also the course of the *A. and V. colica dextra*, the specific blood supply and afflux for the final half of colon ascendens. Sacculations are absent here. As the diaphragmatic flexure is approached, two dorsal bands are acquired, which are free and exposed.

This pattern is continued on the right dorsal colon. The lateral taenia is wide and rather indistinct, the medial one narrow and distinct. Only towards the terminal part of the dorsal colon do sacculations – such a feature of caecum and ventral colon – reappear, and then only indistinctly so.

The variations in lumen diameter are of particular importance. From a mere 5 to 7,5 cm at the origin of the ventral colon, it enlarges rapidly to 20–30 cm, reducing to 6 to 10 cm at the pelvic flexure, increasing rapidly towards the diaphragmatic flexure and beyond, to attain the enormous diameter of 30–50 cm at its transition to small colon, where it forms the *ampulla coli*, narrowing down at this point to 7,5–10 cm to continue as small colon. The implications are obvious.

The presence of more goblet cells in the right dorsal than in the right ventral colon[15] could have some physiological significance.

The *small colon* begins after the terminal funnel–like narrowing of the large colon, behind the fundus of the stomach and ventral to the left kidney. It turns caudalward; as *colon descendens* it pursues in principle a straight course to the rectum at the pelvic inlet. In horses its great lenght (about 3,5 m) and the considerable width of the mesentery cause it to be thrown into coils, which occupy the left upper quadrant of the abdominal cavity, where they lie partly intermingled with coils of the jejunum.

The small colon is thrown into definite sacculations by the presence of two taeniae, the dorsal one in the mesenteric attachment and the ventral one exposed and free. Normally the faeces here form balls which occupy the sacculations. The occasional occurrence of a singly disposed faecal ball might mislead the hasty rectal palpator or surgeon.

The *rectum* is arbritrarily considered to begin at the pelvic inlet. It takes a relatively straight or moderately oblique course to end at the anus at the level of second to third caudal vertebra. The first part is covered by peritoneum, the extent depending on the degree of full-ness of the bowel. At usually the fourth or fifth sacral segment the peritoneum is reflected cranialwards, leaving the considerable retroperitoneal portion devoid of a serosa and surrounded only by connective tissue. Here the rectum forms the flask–like *ampulla recti*. From the powerful longitudinal muscle coat the *Mm. rectococcygei* arise; they are visible as two bands ventral to the raised tail.

The anal canal is a short (5 cm) tube, lined by glandless, squamous stratified epithelium, and forming a distinct caudalward projection covered by thin hairless skin, well firmed up by the underlying well–developed *M.sphincter ani internus* (smooth muscle), *M.sphincter ani* externus (striated) and supported by the fibres of the rectal part of the smooth *M.retractor penis/clitoridis* as well as by the striated *M.levator ani*, which in the horse form strong subanal loops. The *M.levator ani* is attached to the perineal body – the perineal septum in the mare – in which its action, together with that of the anal sphincters, is responsible for the gaping of rectovaginal tears [6].

Omentum: In passing it may be pointed out that the equine omentum, although extensive enough to be able to enter the *cavum vaginale* ('scrotal cavity'), does not form an extensive covering between intestines and body wall. Consequently this 'policeman' of the peritoneal cavity cannot fulfil its function – also likened to that of a self–sealing inner tube – as effectively as in other species.

NERVE CONTROL

The nerve control of the equine alimentary tract may be accepted to follow the general pattern of autonomic control of the gut, *i.e.*, by parasympathetic excitory and sympathetic inhibitory effects on secretory cells and particularly on smooth muscle, which has its own inherent rhythmicity. The efferent parasympathetic innervation is by way of the vagus and the pelvic nerves (cranio–sacral outflow) with preganglionic fibres entering the gut wall, whereas the sympathetic fibres stem from the thoracolumbar outflow, mainly via the greater and lesser splanchnic nerves and coeliac and mesenteric ganglia, from whence post–ganglionic fibres extend as plexuses around the arteries, probably passing mainly to the *muscularis mucosae*,[7] and to the ganglion cells of Auerbach's plexus, so that inhibition occurs predominantly by blocking parasympathetic ganglionic transmission[12 17 19] or by vasoconstriction, and from epinephrine and norepinephrine released from the adrenal gland.[7]The myenteric (Auerbach's) plexus and the submucous (Meissner's) plexus, together with associated ganglion cells form a diffuse generalized system with the formation of local reflex arcs. Afferent fibres pass to the central nervous system through both sympathetic and vagal routes.

In the stomach the mobility and tone are increased greatly by cholinergic impulses, and are usually decreased moderately by adrenergic impulses acting on beta adrenergic receptors [14]. Sphincters are usually relaxed moderately by cholinergic action, and usually moderately contracted by mediation of alpha receptors of adrenergic impulses [14].

Secretion is strongly stimulated by cholinergic impulses; its inhibition by adrenergic impulses is questionable [14].

The same mechanisms hold good in the case of the intestine, although here mobility and tone are decreased by both alpha and beta adrenergic receptors [14].

Obviously, the mechanics of peripheral control are meaningless without control from hierarchy upon hierarchy of higher control centres: spinal cord and medulla oblongata, hypothalamus, limbic system, striatum and cerebral cortex. Considerations of this aspect fall outside the scope of this paper. Too little is known about details pertaining specifically to the horse.

The motor nerve supply routes are summarized briefly. The initial part of the oesophagus appears to be innervated from the pharyngeal plexus, mainly by the vagus nerve and probably the glossopharyngeus as well. From the plexus a large branch extends along the oesophagus.

The cervical and cranial thoracic regions are considered to be supplied by the recurrent laryngeal nerve from the vagus, whereas more caudally the dorsal and ventral vagal rami take over. A myenteric plexus is regarded as being present only where smooth muscle has supplanted the striated musculature. Sympathetic supply to the oesophagus is mainly via the cervicothoracic ganglion; whether it ultimately innervates the muscle, is unknown. The peristaltic contraction caused by a bolus placed into the oesophagus does not occur if the vagus nerves are sectioned [8].

The dorsal vagal trunk, upon reaching the stomach, divides into gastric branches to its visceral face and coeliac branches to the left and right coeliacomesenteric ganglion. The ventral vagal trunk supplies the parietal face of the stomach, the pylorus, the duodenum and the liver. From the coeliacomesenteric ganglion, where no synapses occur, vagal fibres are distributed with those of the sympathetic system to the small intestine and via the intermesenteric plexus and caudal mesenteric ganglion to the large intestine. Their target territory is usually regarded as ending at the transverse colon.

The sympathetic supply to the abdominal part of the gut[4 5 22] separates from the sympathetic trunk to form the greater splanchnic nerve, its roots originating from the sixth or seventh to fourteenth or fifteenth thoracic ganglia. Classically it passes medioventrally to the trunk to enter the abdominal cavity through the lumbocostal arch to join the coeliacomesenteric ganglion. The lesser splanchnic nerves arise from the last two or three thoracic ganglia and go to the coeliacomesenteric ganglion which also receives filaments from the most cranial lumbar paravertebral ganglia (first lumbar splanchnic nerve). The coeliac plexus is a relatively extensive dense network at the aorta and the coeliac and mesenteric arteries. The close relationship here has given rise to the well-known speculation concerning verminous aneurisms of these vessels as a possible cause of neurogenic colic. From the plexus secondary plexuses proceed, laced around the major vessels (abdominal aorta, left gastric, hepatic, splenic and cranial mesenteric arteries) to reach the various abdominal organs and viscera. The right and left coeliacomesenteric ganglia (fusion of coeliac and cranial mesenteric ganglia) are situated in the plexus, on each side of the aorta, at the origin of the homonymous arteries, and are interconnected. From them the *plexus intermesentericus* arises, seemingly part of the aortic plexus [5], and generally appearing as two trunks, with transverse connecting strands and joining the mesenteric ganglia along the face of the aorta. The caudal mesenteric ganglion is a single mass in the horse, immediately cranial to the caudal mesenteric ganglion, splitting to pass on either side of the artery, and ventrally on the vessel, giving rise to the caudal mesenteric plexus. It is supplied also by lumbar spanchnic nerves ($L_2$and interganglionic part between L_2 and L_3[5]) via the intermesenteric plexus. From it in turn the hypogastric nerves, left and right, arise and proceed in close proximity to the ureters to the pelvis, running at first in the mesocolon, then inclining laterally and finally in the lateral vesical ligaments.

In the horse the 'colic tract',[4]a single nerve of considerable size, and a contributary nerve dividing usually into three trunks, together form a bypass from the cranal mesenteric plexus and left coeliac ganglion (and in small part from the coeliac plexus), directly to the caudal mesenteric plexus.

The lumbar sympathetic trunk is not regularly segmented and for a while the left and right components are fused in the sagittal plane. The sympathetic supply to the pelvic viscera is by way of the hypogastric nerves, aortic plexus and the sacral part of the sympathetic trunk. The parasympathetic supply is derived from sacral nerves 2, 3 and 4, which arise with the roots of the pudendal nerve and form the pelvic splanchnic nerves; it is also contained in the caudal rectal nerve to supply the end of the rectum and anal sphincter.

Accessory nerve fibres which bypass the pelvic plexus and establish connections between sacral nerves and pelvic organs, so that the atony of the bladder after total bilaterial resection of the pelvic plexus later clears up completely, have not yet been demonstrated in animals.

The anatomical arrangements of the abdominal and pelvic autonomic systems give a very complex and variable appearance, yet are simple in terms of general principles. Nevertheless, one would like to have more detailed and clear-cut definite data. The structural complexities and the impossibility of tracing microscopic fibre distribution to its ultimate termination, as well as the problem of differentiating between afferent, sympathetic and parasympathetic fibres anatomically, call for an experimental, anatophysiological research attack on a species, the size and cost of which tend to place it beyond the realm of being a practical research subject. It is a fantastic piece of static and dynamic machinery with a most troublous sewage system.

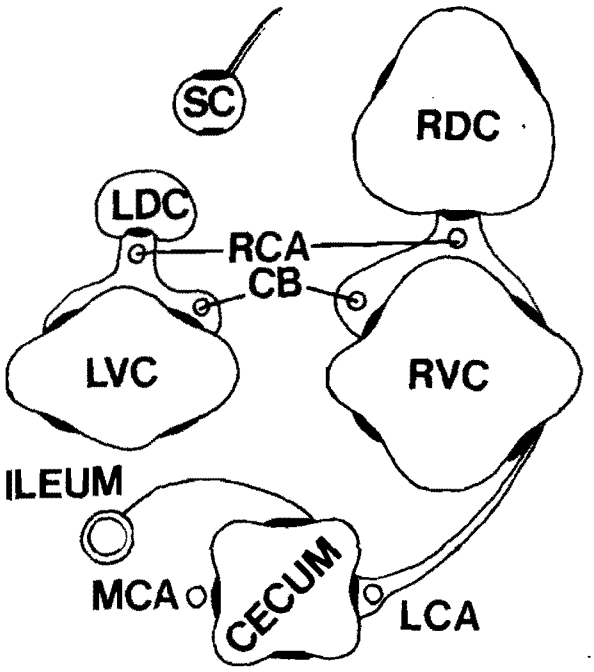

Figure: Schematic cross section of the large intestine of the horse. The longitudinal bands of smooth muscle (teniae) are shown as dense black sections. CB, colic branch of the ileocolic artery; LCA, lateral cecal artery; LDC, left dorsal colon; LVC, left ventral colon; MCA, medial cecal artery; RCA, right colic artery; RDC, right dorsal colon; RVC, right ventral colon; SC, small colon. The mesocolon connects the mesocolic bands of the large colon and contains the blood vessels; it is also attached to the small colon. The ileocecal fold connects the ileum to the dorsal band of the cecum. The cecocolic fold connects the lateral band of the cecum to the lateral free band of the right ventral colon.
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DISCUSSION

F.J. Milne: You described briefly the fact that sometimes the omentum is found as far back as the inguinal canal. In our experimental work on investigating the healing of peritoneum, we found in normal horses and also in foetal surgery, when doing an approach through the midline, that the omentum was very rarely present as in the dog, where it is the first structure one meets. Yet, *when we were following* through some of those cases that had adhesions to plastic implants, we found that the omentum had tended to migrate towards the adhesion. This reminds me of the words of one human worker, who described the omentum as 'that interfering busy-body, who always wants to interfere in someone else's affairs.' Can you explain why the omentum has this capacity to migrate to tissues, such as the midline peritoneum, and also sometimes to the inguinal region, following castration, when one would be called back a week later to deal with the omentum? Is there any reason for it?

H.P.A. De Boom: I am afraid, I cannot give a scientifically documented reason. If one looks at the anatomy of an animal, one must always remember that one is looking at the anatomy of a dead animal. In other words, one looks at one picture frame, a still, taken at a given instant from a moving film. I think what actually happens in practice is that the omentum is continuously wandering. With the movement of the intestines the omentum will be shifted along. I have never done any accurate detailed observations to support the idea, but when looking at successive dissecting room cadavers one observes considerable variation in the precise location of the mobile part of the omentum. I think it is continuously moving and it must be exceedingly responsive to local inflammatory reactions. So the moment one has such a reaction, the omentum, as it passes by, probably respond immediately by vascular permeability to fibrinogen, so sealing off the danger area, and forming and adhesion. Hence, despite the fact that, in the horse, it does not form a permanent and fairly complete cover to the visceral mass, it still can be reasonably effective by virtue of its wandering habit.

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DIGESTIVE PHYSIOLOGY OF THE HORSE

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SUMMARY

Recent studies on the digestive physiology of the horse are reviewed. It was suggested that the small intestine is the primary site of digestion and absorption of protein, soluble carbohydrates, most minerals, fats, fat soluble and water soluble vitamins. The large intestine is the primary site of fibre digestion and net water absorption. Significant amounts of phosphorus are also absorbed from the large intestine. Many factors such as rate of passage, processing of feeds, level of intake, work and maturity of plant may influence digestive ability.

INTRODUCTION

In 1963, Alexander[2] wrote an excellent review on digestive physiology of the horse. His review contains much of the basic information still applicable today. In the last ten years, however, there has been an increased interest in equine research in the United States. The following is a brief review of some of the more recent experiments dealing with digestive physiology of the horse.

MICROBIOLOGY

Smith[37] suggested that the flora of the caecum of the horse and of the rumen of the sheep and cow were qualitatively similar, that is, they consisted of the same types of organisms. Kern *et al.*[23] fed steers and ponies hay with or without oats and found the numbers of bacteria per volume of caecal ingesta increased when oats were fed to the ponies but not when oats were fed to the steers. Rods, Gram-negative and -positive, predominated in the Gram smear counts of both ponies and steers. Cellulolytic bacteria in numbers per gram of ingesta were similar in the ponies' caecum and steers' rumen, whether or not oats were included in the diet.

Kern and co-workers[22] also compared the microbial population of steers and ponies fed timothy hay. The results are shown in table 1. The total bacterial, viable bacterial and ammonia concentrations and pH were higher in the rumen of steers than in the caecum of ponies.

SITE OF DIGESTION

Protein

Recent studies indicate that the small intestine is the primary site of protein digestion and amino acid absorption[14 17 33]. At least 60–70 per cent of the dietary protein may be digested and absorbed before reaching the large intestine. Thus, the absorbed amino acids are dependent on the dietary amino acids. This illustrates why the amino acid content of the diet is important for young, growing horses[5 20]. The end pro-

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Table 1: CHEMICAL AND MICROBIAL CHARACTERISTICS FOR INGESTA FROM INTESTINES OF PONIES AND STEERS FED TIMOTHY HAY[4]

Characteristic	Pony gut regions			Steer gut regions			
	Ileum	Caecum	Colon, terminal	Rumen	Ileum	Caecum	Colon, terminal
Ingesta pH	$7,4^{w}$	$6,6^{w}$	$6,6^{w}$	$6,9^{ax}$	$7,3^{aw}$	$7,0^{aw}$	$7,2^{ax}$
Viable bacteria/gmx10^{-7}	36^{b}	492^{cw}	363^{d}	1658^{ax}	$5,4^{b}$	230^{c}	$12,7^{d}$
Rods %							
Gram-negative	$9,2^{a}$	$63,8^{bw}$	$54,3^{b}$	$33,1^{x}$	20,3	$29,9^{x}$	25,7
Gram-positive	38,9	6,4	11,2	2,4	0,9	2,7	5,5
Cocci %							
Gram-negative	6,4	33,1	22,6	44,1	33,9	46,9	45,9
Gram-positive	$36,2^{a}$	$5,6^{a}$	$11,2^{b}$	$19,4^{a}$	$44,7^{c}$	$22,7^{b}$	$25,3^{b}$
DNA µg/g	$9,9^{b}$	$8,4^{cw}$	$6,2^{c}$	$51,6^{x}$	$25,5^{b}$	$24,8^{bx}$	$16,0^{c}$
NH_3–N mg/100 ml	$5,2^{w}$	$2,9^{w}$	5,4	$10,6^{x}$	$15,5^{x}$	$18,2^{x}$	13,1

a,b,c,d Means on the same line (within species) bearing different superscript letters, differ significantly ($P<0,01$).

w,x Comparison: fundic vs. abomasum: pyloric vs. abomasum; small intestine vs. small intestine; caecum vs. caecum; colon vs. colon; caecum vs. rumen. Means (between species) on the same line bearing different superscript letters for each comparison differ significantly ($P<0,01$).

ducts of protein digestion and absorption in the large intestine include ammonia[13] and some amino acids[34]. The importance of the amino acids synthesized by bacteria and absorbed from the large intestine is still open to question. Bacterial amino acids certainly do not supply all the needs of young horses. It is known that mature horses can use some non-protein nitrogen to increase nigrogen retention[19 36]. Nevertheless, the increased retention could result from utilization of bacterial protein or perhaps because of the synthesis of non-essential amino acids in the liver. Although there is proteolyic activity in the large intestine, it is much less than that in the other segments of the tract[22] (Table 2).

Table 2: PROTEOLYTIC ACTIVITY OF INTESTINAL TRACT INGESTA OF PONIES

Region	Protein hydrolyzed[a]
Stomach (fundic)	3
Stomach (pyloric)	54
Small intestine (ileum)	530
Caecum	12
Colon (terminal)	15

[a] Each unit equivalent to 0,045 µg protein hydrolysed per milligram ingesta per minute.

Carbohydrates

Most of the soluble carbohydrates are digested in the small intestine and the absorbed end-products of the carbohydrate digestion are glucose and other simple sugars[14 17]. In cattle, the end-products of grain digestion are volatile fatty acids (VFA) which are used less efficiently than glucose. Thus, the horse may be more efficient in the utilization of grain than cattle. Some soluble carbohydrates reach the large intestine where they are converted to VFA. The fermentation pattern in the caecum can be influenced to change the acetate: propionate ratio by feeding high levels of soluble carbohydrate to horses[16 38]. The changes in the VFA pattern are quite similar to those produced in the rumen of cattle fed high levels of soluble carbohydrate[16] (Table 3). Similar changes in the VFA pattern of caecal fluid are also observed when the diet is changed from long hay to pellets[42].

The large intestine is the primary site of fibre digestion and the end-products are VFA, primarily acetate, propionate and butyrate[14 17].

Table 3: COMPARISON OF RATIOS OF VOLATILE FATTY ACIDS IN RUMEN FLUID AND CAECAL FLUID OF CATTLE AND PONIES FED RATIONS WITH VARYING HAY-GRAIN RATIOS [14 16]

Hay-Grain Ratio	Species	Molar Percentage		
		Acetate	Propionate	Butyrate
1:0	Pony	73	17	8
	Cattle	74	18	8
1:4	Pony	59	25	11
	Cattle	62	22	16

Fat

The small intestine is probably the primary site of dietary fatty acid absorption. The composition of the body fat is influenced by the composition of the dietary fat[10]. This suggests that the fats are absorbed from the small intestine before they can be altered by the bacteria in the large intestine. The horse does not have a gall bladder but this does not appear to hinder the digestion of fat. Mature horses can tolerate diets containing at least up to 18 per cent fat. For example, we found that the fat in diets containing 15 per cent beef tallow and 85 per cent alfalfa pellets was highly digestible[27]. Bryant[6] reported that the fat in diets containing 9 per cent corn oil was 90 per cent digestible.

Vitamins

Information on the site of absorption of vitamins is lacking. Presumably, the fat soluble vitamins are absorbed primarily in the small intestine. The small intestine is probably the primary site of absorption of dietary B vitamins. More work is needed to determine the availability of B vitamins produced by bacteria in the large intestine. About 25 per cent of S^{35}-thiamine introduced into the caecum was absorbed[25].

Water

The water content of the digesta in the colon is much less than that in the caecum and it was suggested that the colon is the primary site of net water absorption[17]. Recent studies, however, using a polyethylene glycol marker, demonstrate that although significant amounts are absorbed from the colon, the caecum is the primary site of net water absorption[4].

Estimates of site of digestion of the various nutrients are summarized in table 4.

Table 4: ESTIMATES OF SITE OF DIGESTION AND NET ABSORPTION

Dietary fraction	Small intestine %	Caecum and colon %
Protein	60–70	30–40
Soluble carbohydrates	65–75	25–35
Fibre	15–25	75–85
Fats	Primary [1]	–
Calcium	95–99	1–5
Magnesium	90–95	5–10
Phosphorus	20–50	50–80
Vitamins	Primary [1]	–

[1] Estimates of percentage absorbed in various segments not available, but small intestine is probably primary site of absorption of dietary sources of these nutrients.

RATE OF PASSAGE OF DIGESTA

When indicators such as chromic oxide or coloured particles are added to hay-grain diets, about 10 per cent of the indicator is excreted within 24 hours, 50 per cent within 36 hours and 95 per cent within 65 hours[1 12 18 41]. The rate of passage can be influenced by the physical form of the diet. Pelleted diets have a faster rate of passage than chopped or long hay[12 18]. Fresh grass moves more rapidly through the tract than does hay[29].

Liquid leaves the stomach and passes rapidly through the small intestine. Argenzio, *et al.*[4] reported that the majority of a liquid marker left the stomach within 30 minutes and reached the caecum less than two hours after the oral administration.

Particle size will also influence rate of passage. Ponies were given polyethylene tubing (OD 2mm) of either 2mm, 1 cm or 2 cm in length. After six days, about 75 per cent of 2mm material appeared in the faeces compared to 55 per cent and 25 per cent for the 1 cm and 2 cm materials, respectively. There was no evidence of retrograde movement of liquid or solid markers from the caecum to ileum or from dorsal to ventral colon[4].

FACTORS AFFECTING DIGESTION

Processing of Feeds

The method of processing may influence digestion. For example, the pelleting of roughage decreases fibre digestion about 9–15 per cent[11 18]. Rolling or breaking of the kernel is important in the digestion of small grains, such as wheat and milo. Processing of larger grains, such as corn and oats, does not seem to improve digestibility greatly. Morrison [28] concluded that there is no advantage in cooking, fermenting or predigesting feed for horses. Steeping of wheat bran in warm water (50° C) for one hour did not affect dry matter, protein or phosphorus digestibility[21]. Incidentally, a wet bran mash is usually considered to have a more laxative effect than dry bran[28] but our trials indicated that there was little difference in the laxative effect between wet bran and dry bran. The dry matter content of faeces of ponies fed either wet or dry bran was similar (Table 5). Furthermore, it appears that the laxative qualities of wheat bran often may be exaggerated, at least for ponies. The water content of the faeces of ponies fed a pelleted diet containing 16 per cent wheat bran was significantly higher than the water content of faeces of ponies fed similar diets but without wheat bran; the actual difference, however, was only 3 per cent. If we could digress further, faeces of ponies fed alfalfa pellets did not contain much more water than did faeces of ponies fed other pelleted feeds. One of the easiest ways to produce dry faeces is to feed oats only (Table 5).

Table 5: EFFECT OF DIET ON WATER CONTENT OF FAECES OF PONIES

Diet	No. Observations	Water content %
Wet bran mash [1]	4	68,2 ± 2,3
Dry bran	4	68,5 ± 4,2
Basal [2]	16	66,4 ± 3,6
Basal + wheat bran	16	69,8 ± 2,5
Alfalfa pellets	5	68,8 ± 4,4
Oats	8	50,7 ± 5,6

[1] Diet consisted of 50% wheat bran, 50% beet pulp.

[2] Both diets were pelleted; contained 40% ground timothy hay.

Level of Intake

The level of intake does not appear to effect the digestibility of all-roughage diets but the digestibility of diets containing forage and grain may be decreased with increasing increments of dietary intake[32].

Frequency of Feeding

Frequent feedings (two or three times per day) are usually recommended for horses. The horse has a relatively small stomach and severe over-eating at one time can produce colic or even a ruptured stomach[30]. Overeating may also produce founder, therefore, the policy of feeding the grain at least twice daily seems reasonable. Frequency of feeding does not appear to affect digestibility, at least of complete pelleted diets. Three ponies were used in a 3 x 3 latin square digestion trial. The daily feed was divided into 1, 2 or 6 feedings. The results shown in table 6 demonstrate that digestibility was not influenced by frequency of feeding[7].

Table 6: EFFECT OF FEEDING FREQUENCY OF DIGESTIBILITY OF COMPLETE PELLETED HORSE FEED[1]

Feeding frequency	Digestibility (%)			
	Dry matter	Crude protein	Neutral detergent fibre	Acid detergent fibre
1 x day	71,0	82,3	45,9	28,2
2 x day	71,0	80,6	44,6	27,6
6 x day	72,0	79,5	44,2	28,1

[1] Means are average of 3 values.

Work

Early studies indicate that activity may have some influence on digestion in the horse. Olsson & Ruudvere[29] suggest that light exercise might slightly improve digestibility but heavy work may slightly inhibit it.

Individuality

The individuality of the horse is sometimes listed as a factor affecting feed digestibility[41]. Fonnesbeck *et al.*[9] reported that horses differed significantly in their ability to digest crude protein and nitrogen free extract.

Associate Effects

No associative effects or interactions were observed when ponies were fed diets containing all hay, 50 per cent hay and 50 per cent grain, or 20 per cent hay and 80 per cent grain, that is, the addition of the grain did not appear to influence the digestion of the hay [16]. There may be special associative effects, for example, the addition of wheat bran may decrease the absorption of calcium because of the phytin phosphorus but does not appear to influence the digestion of fibre or protein. Leonard *et al.*[24] reported that the addition of dried distiller's grains to the caecum improved fibre digestion.

Maturity of Plant

Stage of maturity of the roughage at harvesting, affects digestibility. Delay of cutting alfalfa hay for 10

or 20 days significantly decreased digestibility of dry matter, crude protein and crude fibre[8] (Table 7).

Table 7: EFFECT OF MATURITY ON DIGESTIBILITY OF ALFALFA[36]

Fraction	Date of cutting		
	June 3	June 13	June 23
	Digestion coefficient (%)		
Dry matter	69	62	57
Crude protein	75	72	55
Crude fibre	58	55	53

Fistulation of the Intestine

Pulse *et al.*[31] reported that caecal fistulation of three Thoroughbred geldings increased the digestion of the crude fibre but not of dry matter, energy or protein. They suggested the improved fibre digestion resulted from the decreased rate of passage of digesta in the fistulated horses. No differences in digestibility or rate of passage were observed in ponies with a small caecal fistula[26].

SPECIES COMPARISON

Most studies indicate that the horse is as efficient as the ruminant in the digestion of crude protein[15 40 41]. The horse digests fibre about 60–70 per cent as efficiently as the ruminant[15 29 40], although the difference between species decreases as the quality of the roughage increases. Some of the decreased efficiency can be attributed to the faster rate of passage of digesta in the horse. Bacteria from the large intestine of the horse were as efficient in the digestion of cellulose as bacteria from the rumen of cattle under *in vitro* conditions[2]. Differences may also exist because much of the dietary protein is absorbed prior to the caecum. Nitrogen is essential for the bacteria to digest fibre. We found that soybean meal, given *via* caecal fistula, improved fibre digestion of a low protein diet.

The horse is much more efficient in the digestion of fibre than is the rabbit, even though the rabbit practises coprophagy[41]. Digestion coefficients are similar for horses and ponies[35]. Further studies are needed, comparing other equidae. One report suggests that the mule is more efficient in the digestion of fibre than the horse[3]. Preliminary studies indicate that the onager or wild ass may also be more efficient in digestion than the horse.

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T. Gumas: I would like to raise the question concerning the rationale of feeding high fat diets. Most of us are dealing with horses that are working in temperate, tropical or subtropical climates. In human nutrition there are fats that play an important rôle in nutrition. Where humans are working in Arctic or very cold climates, there are individuals who are very deficient in vitamins. Fats form the basis by which vitamins are synthesized. What is the rationale of feeding more than 3 or 4% fat in the diet of a horse which is well supplemented with vitamins and working in a temperate or subtropical climate?

H.F. Hintz: As for fat being used in cold climates, it is primarily as an energy source. In humans fats have 2,25 times as much energy per gram as carbohydrates. I do not think that there is any rationale in the idea that fat is needed for vitamin synthesis. That is what I understood you to say. The exact energy requirements of race-horses for the kind of condition they should be in, and what the best sources of energy are, are still open questions. I think fat is one thing we should look at as a potential source. I am not recommending it at all yet. There is no truth to the old myth that horses cannot utilize fat, in fact, they can utilize it just as efficiently as any other species.

C.J. Roberts: In New Zealand, we use a lot of milk powder; we give it to our young horses for development. I would like to ask you whether, with the increase in weight that one gets, is it possible to determine if that increase in weight is due to fat deposition or to skeletal development, or both?

H.F. Hintz: That is a good question. We did not slaughter our horses, so we did not get any body composition. We did ask commissions to evaluate the animals. In all cases it was claimed that the animals looked more muscular and had a glossier coat, the latter we think is due to the good protein. We also used ultrasonics on these animals and so we were able to measure the fat thickness over the rump, which most studies indicate is a pretty good indication of total body fat in the horse. We found no evidence to indicate that our particular horses were fat but in fact they were well muscled. I want to pin-point a problem with regard to growth gains in horses. There is still a big controversy concerning how fast one wants these horses to grow. We have seen many cases ending up with epiphysitis and so forth, and always it is the Quarter Horse that grows the fastest and is the fattest. I think it is important to keep the whole diet in balance, because if one is going to foster this faster growth, the horse is going to have higher calcium and phosphorus requirements, besides others.

S.G.B. Persson: Human athletes, we know, can only take up 70g protein per day which they can utilize. I wonder, taking digestibility into account, what is about the amount that a race-horse can take up per day and which he can utilize.

H.F. Hintz: We have no idea. As a matter of fact, we are running a trial right now, in which we are measuring protein requirements for endurance in horses. Maybe there are other factors in addition to the protein, which are to be reckoned with, such as ammonia production. How much ammonia can be produced? If we get too much ammonia produced, is this inclined to slow down bone metabolism? We are working now on protein levels in relation to ammonia production and metabolism. How much a horse can utilize and how much he can take up, we do not have any idea.

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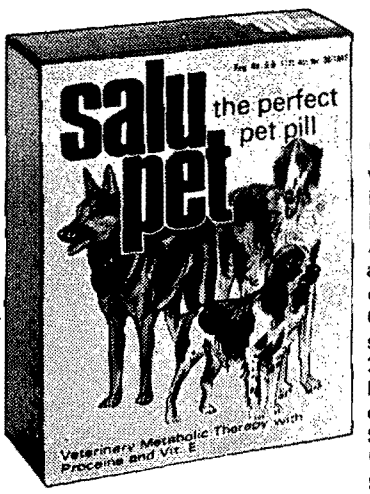

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CARBOHYDRATE DIGESTION AND ABSORPTION IN THE EQUINE SMALL INTESTINE

MALCOLM C. ROBERTS*

SUMMARY

Dietary carbohydrates, which constitute a most important source of equine nutrition, are digested and absorbed by a series of complex processes principally in the small intestine, beginning with intraluminal starch hydrolysis by the action of pancreatic amylase. The continuous secretion of a copious volume of pancreatic juice, low in enzyme activity, presumably releases sufficient oligosaccharides for further hydrolysis at the intestinal cell surface by brush border enzymes. Active carrier mediated mechanisms then transport the final hexose products across the intestinal cell for uptake in the hepatic portal system.

Brush border disaccharidase activities in the equine small intestine are of the same order of magnitude, and have a similar distribution pattern, to those reported in omnivorous and carnivorous species. The disaccharidase development patterns are characteristic and reflect the ability of the horse to digest the major nutrient sources adequately at various stages of life.

The efficiency of the mucosal disaccharidases and the monosaccharide transport systems in the equine small intestine have been established by a series of oral disaccharide and monosaccharide tolerance tests. Horses older than three years of age are unable to hydrolyse lactose, but young and adult horses are fully capable of rapidly hydrolysing sucrose and maltose loads.

Several tests have clinical application for assessing small intestinal dysfunction in the investigation of diarrhoea and malabsorption. The deficient digestion or absorption of carbohydrate, whether primary or secondary, can almost always be localized to a defect in the enzymic or transport capacity of the small intestinal surface cell. The continued ingestion of lactose could be detrimental in severely diarrhoeic foals.

INTRODUCTION

Carbohydrates contribute an important part of the equine diet, providing the principal source of energy, whether the horse is at pasture or being fed relative to a specific performance. The soluble carbohydrate content of hay and pasture grass is approximately 5 per cent of the dry matter[1]; horses in work or training will be receiving a much higher proportion of soluble carbohydrates, as oligosaccharides and disaccharides predominate in concentrate rations. Alexander[1] reported that glucose was wholly absorbed in the equine small intestine, and Hintz, Hogue, Walker, Lowe & Schryver[10] concluded from feeding trials, using fistulated ponies, that 65 – 75 per cent of the soluble carbohydrates were digested pre-caecally and absorbed as glucose. The remainder, as well as the insoluble carbohydrates, cellulose, lignin and fibrous plant material, undergoes bacterial fermentation in the large intestine with the production and absorption of the short chain volatile fatty acids: acetic, propionic and butyric[6].

The equine small intestine, although representing 30 per cent of the capacity of the alimentary tract and thus providing a vast surface area for digestive and absorptive processes, has received scant attention in comparison to the large intestine; the rôle of enzymic digestion has largely been inferred by analogy with other species.

In omnivores and carnivores, carbohydrate digestion is initiated by salivary and pancreatic α-amylases and continued in the small intestine by the disaccharidase enzymes located in the microvillous membrane (brush border) of the lining columnar epithelial cells (enterocytes). In this way, starch, sucrose and lactose, not normally absorbed as such by the small intestine[11], are hydrolysed to their constituent monosaccharides, the form in which absorption takes place. The transport mechanisms involved in this intestinal absorption are located in the brush border of the enterocytes at a site internal to the disaccharidase activity[11]. Glucose and galactose, being of similar structure, are both absorbed by the same sodium- and energy-dependent active transport process involving a specific carrier, thus accumulating the sugars against a concentration gradient[5]. The absorption of fructose is slower, requiring a separate carrier-mediated active transport system[8], contrary to the former theory of facilitated diffusion[7].

In the horse, salivary secretion provides very little digestive enzyme activity, and pancreatic secretion, whilst copious in volume and continuously secreted, is low in α-amylase activity, the output of α-amylase per unit mass of pancreatic tissue being only 5-6 per cent of that attained in the pig after vagal nerve stimulation[2]. Nevertheless, under normal conditions, this may be adequate for initial polysaccharide digestion, furnishing sufficient substrate for sequential hydrolysis by the disaccharidases present in the equine small intestine[15]. These enzymes located in the brush border are strategically placed to bind and hydrolyse oligosaccharides coming into contact with the cell surface[9].

The following mucosal enzymes have been demonstrated in the equine small intestine; the α-glucosidases-sucrase, maltase, trehalase[22] and glucoamylase[19], and the β-glycosidases, lactase (β-galactosidase)[23], and cellobiase (β-glucosidase)[22]. In addition, the low level of pancreatic α-amylase compared to other species has been confirmed by assaying small intestine mucosal homogenates[19], this enzyme being adsorbed to the brush border. Alkaline phosphatases, another group of mucosal enzymes in the equine small intestine, are probably involved in

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long chain fatty acid transport and not carbohydrate digestion or transport[17].

This paper outlines the development and distribution of the disaccharidases in the small intestine, their efficiency in dealing with carbohydrate loads much greater than normal, emphasizing the importance of the small intestine in carbohydrate digestion and absorption, and the use of clinical tests dependent on these processes in assessing small intestinal involvement in alimentary dysfunction in the horse.

THE DISACCHARIDASES

Lactase

There are two enzymes with lactose hydrolysing activity in equine small intestine mucosal homogenates: the brush border digestive enzyme neutral β -galactosidase and the non-digestive lysosomal enzyme acid β -galactosidase[23] (Table). The true lactase is probably responsible for cellobiase activity, a consistent relationship of 5 to 1 being maintained throughout the small intestine[22].

Lactase is detected in the foetal intestine after three months' gestation but there is little change in the level of activity by the ninth month (Fig. 1d), with peak activity in the duodenum and upper jejunum, decreasing distally (Fig. 1a–d). A rapid increase in lactase activity must occur throughout the tract in the two months before birth, as maximum levels are reached at birth, persisting during the suckling period for four months (Fig. 1e,f), the distribution pattern being similar to that in the foetus. There is a progressive decline in activity in the first two years,

Table : SUMMARY OF THE KNOWN PROPERTIES OF THE EQUINE BETA-GALACTOSIDASES

Properties	Equine Beta-galactosidase	
	Acid	Neutral
Localization	Cytoplasmic or lysosomal Soluble Present in supernatant after centrifugation at 100 000g for 60 min	Presumably in brush border. Particulate Major fraction sediments after centrifugation at 100 000g for 60 min
Substrates	Lactose, BNG	Lactose, Cellobiose'
Reaction with 0,2mmol p-CMB	Inhibited	Not inhibited
pH Optimum	4,2	6,0
Activity at acid pH Optimum	Maximal	26% of activity at pH 6,0 (in young horse)
Activity at neutral pH Optimum	23% of activity at pH 4,2	Maximal

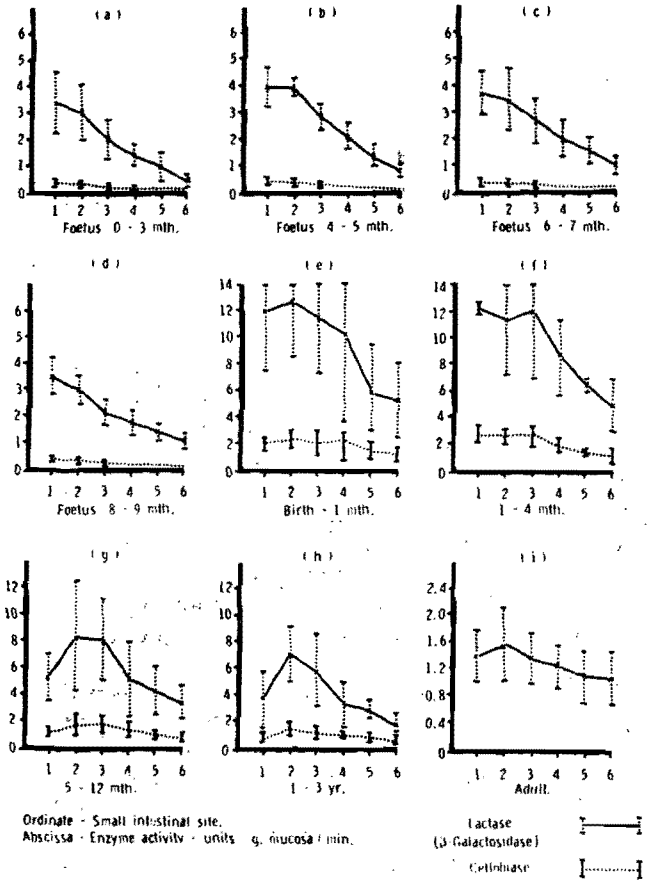

Fig. 1: Distribution and development of β -glycosidase activities in the equine small intestine.
(Mean and Standard Deviation).

rapidly accelerating in the third year; lactase is not detected in the small intestine of horses older than four years of age (Figs 1g–i,2).

Acid β -galactosidase, present in the jejunal and ileal mucosa of the 3-month foetus, is almost trebled in activity by the ninth month, increasing further to reach maximal levels at birth (Fig. 2). The proportion of acid to neutral enzyme is consistently greater in the ileal than in the jejunal mucosa of the foetus. By 15 weeks of age, the high birth levels have decreased to the range of activity recorded subsequently along the small intestine throughout the horse's life, and this non-digestive enzyme represents the lactase activity in the adult small intestine (Figs 1i, 2). The distribution patterns of the two lactase enzymes are very similar in the foetus and the young foal, highest activities being present in the upper small intestine and decreasing distally.

The pattern of lactase development reflects the capability of the foal to utilize lactose, the principal source of energy at birth and in the immediate perinatal period, without provoking any detrimental digestive effects.

Alpha-Glucosidases

Maltase, a complex of at least four enzymes (isomaltase, sucrase and the glucoamylases, maltase II and III) in man and other animals[5], is just demonstrable in the 3-month equine foetus with little increase in activity by the ninth month (Fig. 3). Birth levels of maltase, representing only 12 – 15 per cent of the adult levels, are almost three times higher than in the oldest foetus, and subsequently, during the suck-

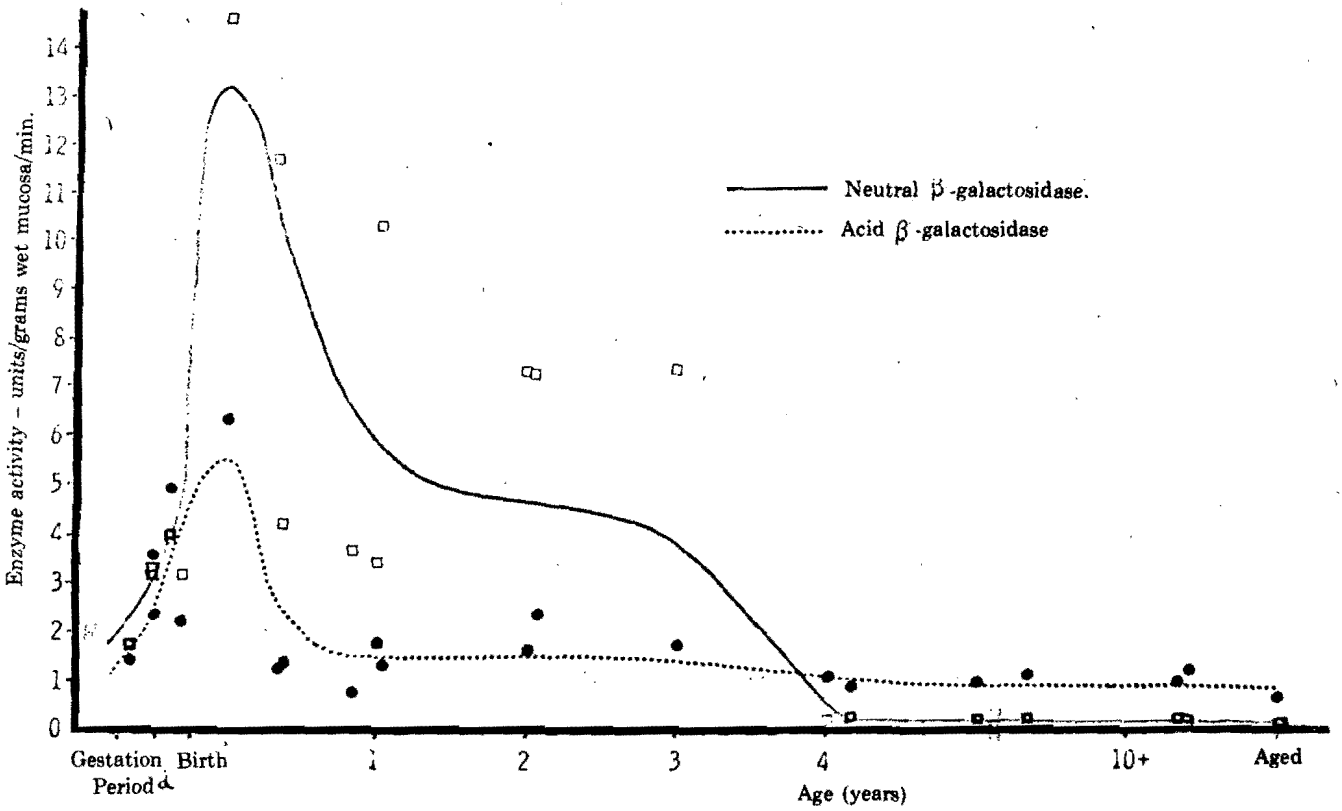

Fig. 2: Development of β-galactosidase activities in the horse.

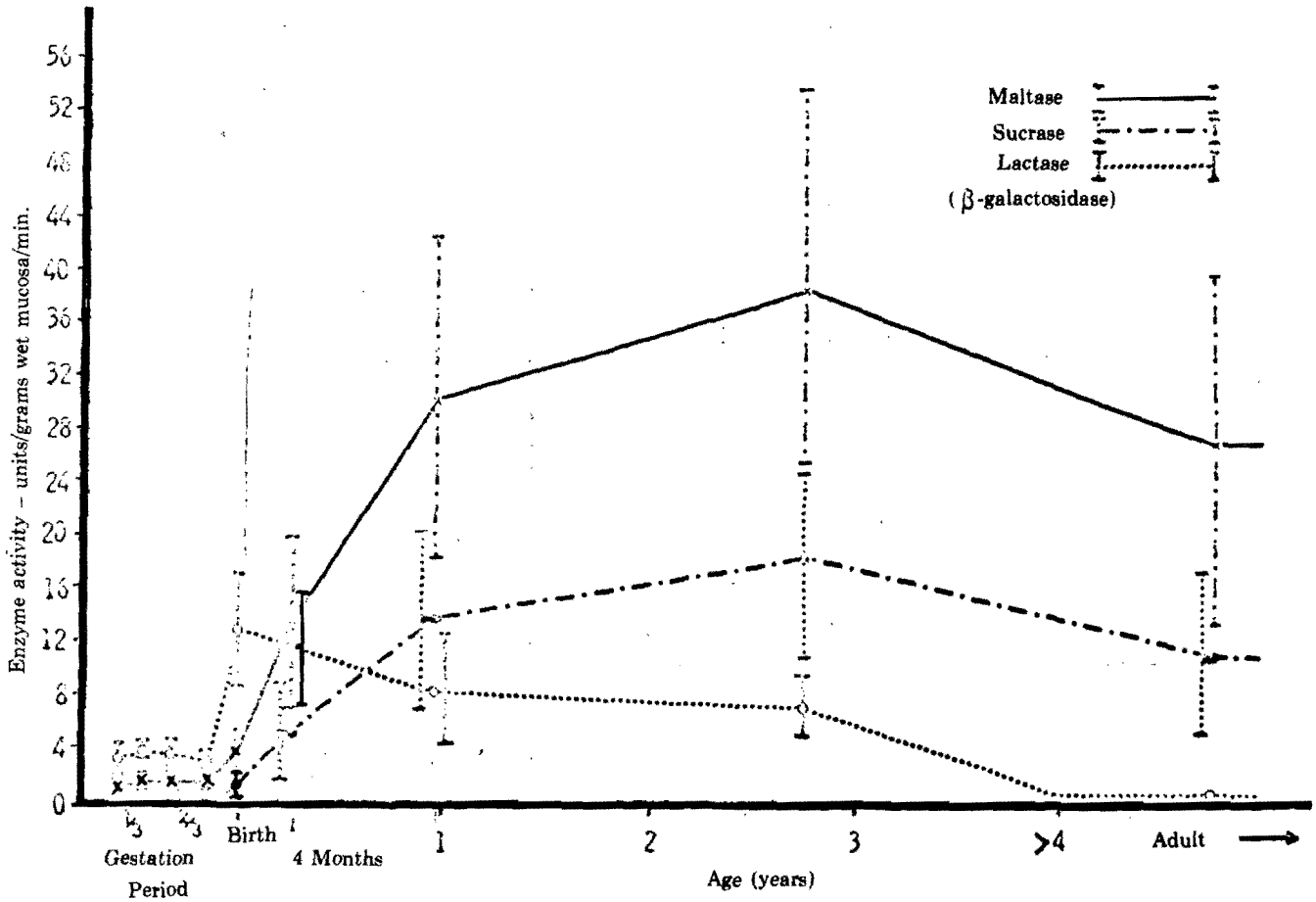

Fig. 3: Development of disaccharidase activities in the equine small intestine (Site 2. Upper Jejunum. Mean and Standard Deviation)

ling period, there is a steady increase in activity to reach adult levels by seven months of age (Fig. 3). The distribution pattern during development is remarkably constant, highest activity being present in the middle and lower jejunum, decreasing toward both the ileum and the duodenum (Fig. 4a–i).

Sucrase, barely discernible in the foetal intestine, is just demonstrable at very low levels at birth (Fig. 4e) and thereafter sucrase parallels maltase development, attaining adult levels by seven months of age (Fig. 3), the distribution patterns being identical (Fig. 4). In the neonate the level of sucrase activity is 29,5 per cent of the total maltase activity, although rising to 39,1 per cent by four months of age, indicating a slower development rate for sucrase compared to other components of the maltase complex [22].

Glucoamylase is detected in the first week of life and reaches adult levels of activity in the tenth month, and although the distribution pattern is similar to maltase in the neonate intestine, by one year of age highest activity is recorded in the distal small intestine[19].

Trehalase, absent from foetal and neonatal intestine, is present at four months of age, reaching adult values by the seventh month (Fig. 4) but showing no definite relationship to the other φ-glucosidases. The midjejunum is the site of highest activity. The substrate, trehalose, is found in insects, mushrooms, plants, yeast and other microorganisms and is probably a constituent of the equine diet.

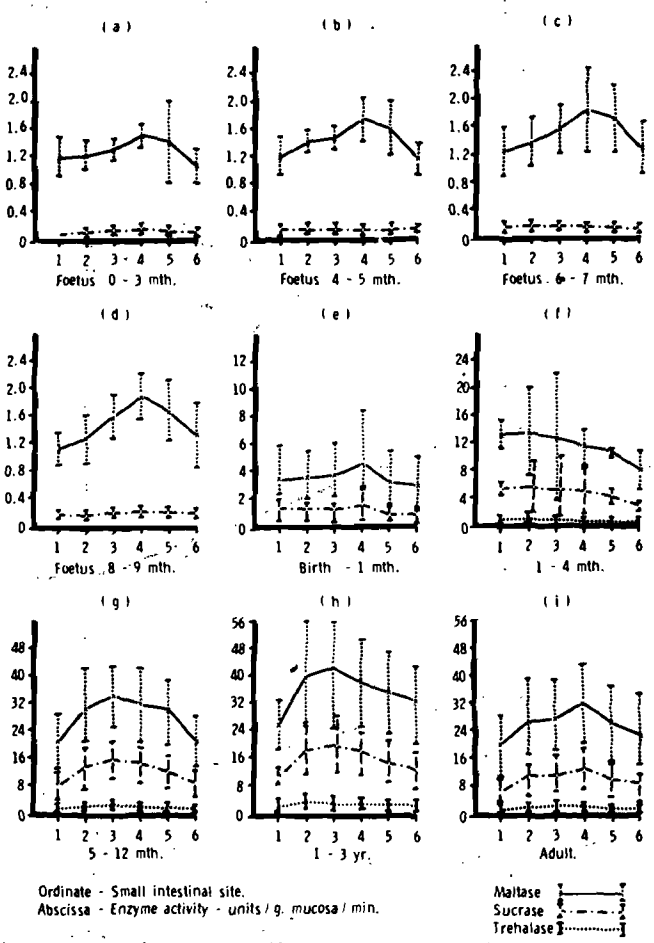

Fig. 4: Distribution and development of α-glucosidase activities in the equine small intestine.
(Mean and Standard Deviation)

The enzymic digestion of carbohydrates is unlikely to be of any importance in the large intestine, there being a virtual absence of disaccharidases in this part of the tract[22]. The lysosomal acid β-galactosidase concerned with intracellular metabolism is found throughout the large intestine and in many other organs and tissues of the body.

The measured concentrations of the various enzyme activities throughout the small intestine of the horse are comparable to those recorded in omnivorous and carnivorous species[15], indicating the potential capacity of the small intestine for sequential hydrolysis of starch breakdown products released by pancreatic α-amylase activity.

The pattern of disaccharidase development in the horse is similar to that in other mammals[24], indicating the importance of lactose as the major dietary constituent at birth and the increasing capability of the young animal to digest other carbohydrates through the suckling period to become fully competent at, or around, the time of weaning. The human neonate is an exception, for, in addition to high birth levels of lactase, the baby is born with demonstrable α-glucosidase activities which closely approximate those found in the adult[3]. It is thus able to digest a wider selection of carbohydrates without suffering detrimental digestive effects than the new-born foal.

The pattern of β-galactosidase activity in the horse closely parallels that present in man with racial lactose intolerance in traditional non-milk-drinking areas, representing the majority of the world's population[12]. In this condition, which becomes evident as early as four years of age, the brush border enzyme is missing or barely detectable at this age, despite being present at comparable levels to lactose-tolerant persons at birth, the acid enzyme being unaffected.[12].

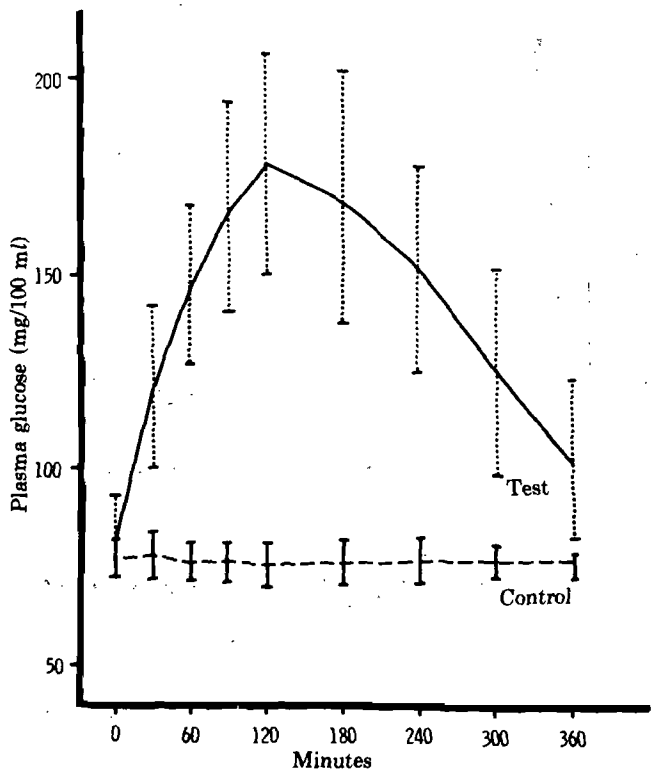

Fig. 5: Oral glucose tolerance test in the horse.
(Mean and Standard Deviation)

CARBOHYDRATE ABSORPTION

The efficiency of the disaccharidase enzymes and the sugar transport mechanisms in the small intestine of horses has been assessed by administering doses of disaccharide and monosaccharide greatly exceeding those normally presented to the horse, and evaluating the plasma glucose response and any untoward side-effects. Thus a series of oral disaccharide and monosaccharide tests was devised and standardized[18]; they have subsequently proved of value in the investigation of small intestinal disease in the horse. The basis of these tests is the active transport of glucose across the intact, functioning mucosa of the small intestine.

In the horse, a dose of glucose at 1g/kg body mass and prepared as a 20 per cent solution, produces a peak plasma glucose level after 120 minutes, greater than twice the resting level which is regained four hours later. (Fig. 5). This normal alimentary response to glucose has been utilized as the oral glucose tolerance test (OGTT)[20]; similar test procedures were adopted for the disaccharide and monosaccharide tolerance tests, the same group of horses and ponies receiving the disaccharide, followed the next day by an equivalent concentration of the constituent monosaccharides[18].

The actual kinetics of carbohydrate absorption were not investigated, although an attempt was made to determine the efficiency and rate of absorption of various sugars using established intestinal perfusion techniques in fistulated ponies[15].

LACTOSE TOLERANCE TESTS

In horses under three years of age, a characteristic sugar tolerance curve was recorded after dosing with lactose (Fig. 6a). When horses between three and four years of age and older were subjected to the test, there was a flat tolerance curve, indicating little or no absorption of glucose during the 6-hour test period (Fig. 6 b,c). Several adult ponies showed mild abdominal discomfort and became diarrhoeic in the 24 hours following the test, passing mushy faeces with soiling of the tail and perineum. Subjecting the same groups of horses to the glucose/galactose mixture resulted in a rise in plasma glucose levels (Fig. 6a–c), similar to that produced by an equimolecular concentration of glucose (Fig. 8a). These results indicated an impairment of digestion rather than absorption, the flat tolerance curves resulting from lactase enzyme deficiency, there being no interference with the monosaccharide transport system. Thus the lactose tolerance test results closely correspond to the established development pattern of lactase activity in the small intestine, the adult horse being lactose intolerant.

The detrimental effect of lactose in the adult horse was demonstrated in those horses which developed

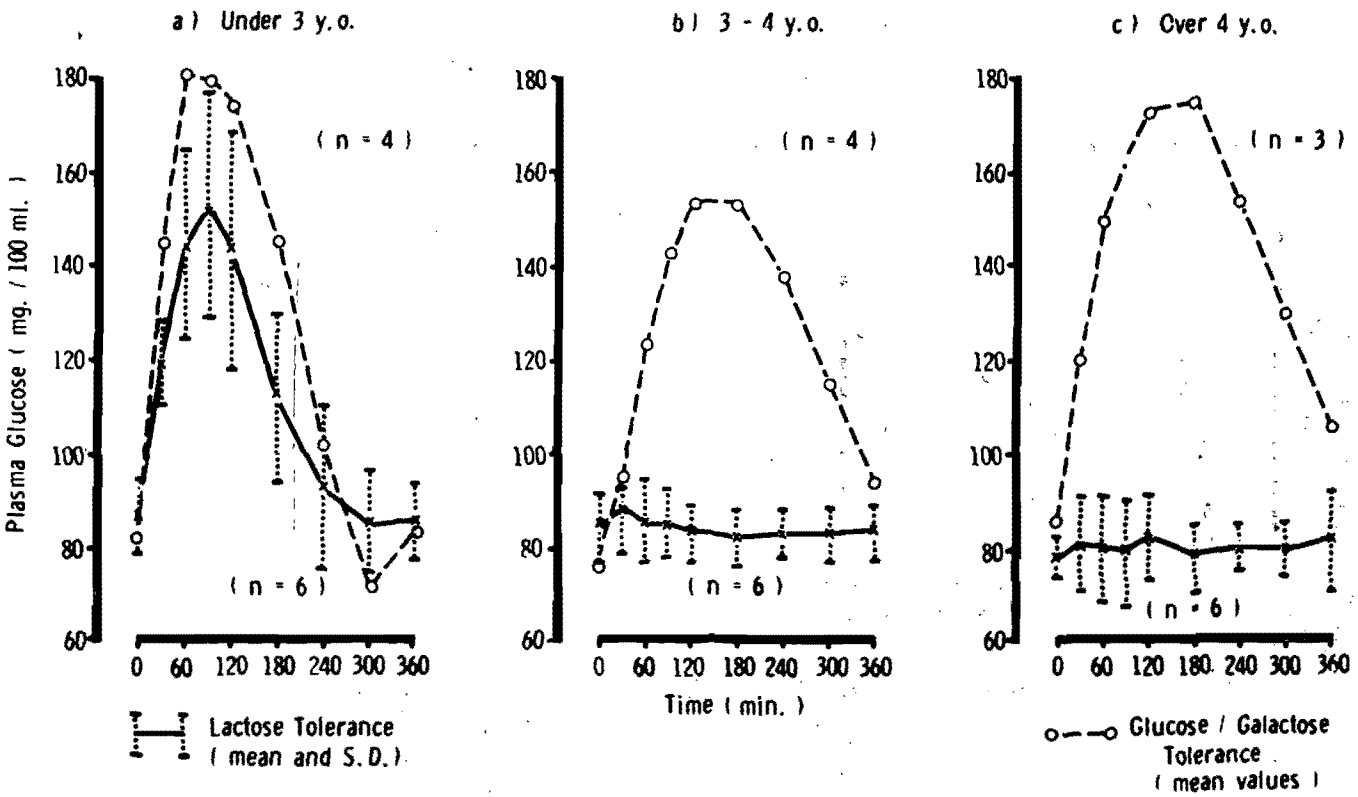

Fig. 6: Comparison of lactose and glucose/galactose tolerance tests in the horse.

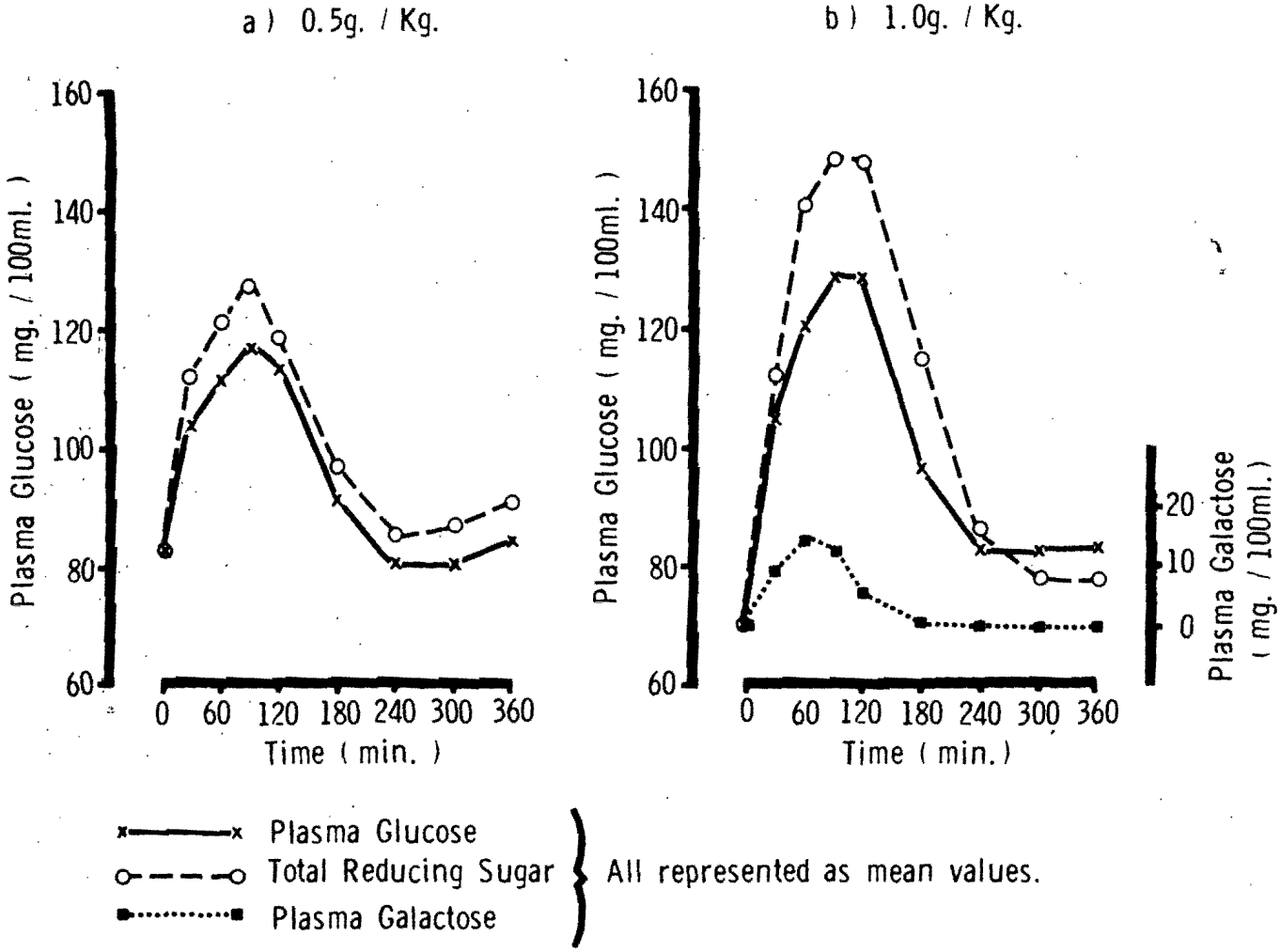

Fig. 7: D (+) galactose absorption in the horse.

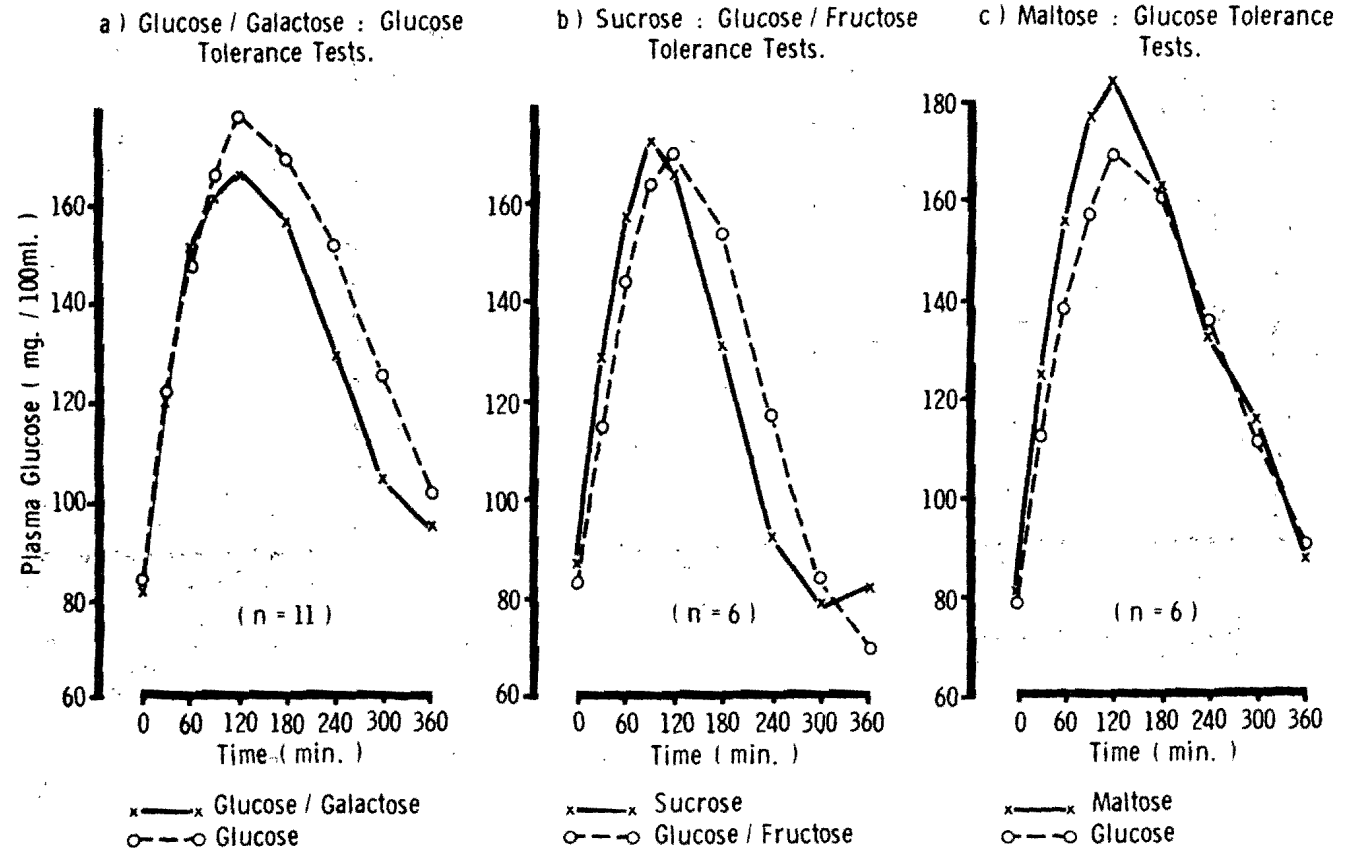

Fig. 8: Carbohydrate absorption in the horse.

diarrhoea. In disaccharidase deficiency in many species, dosing with the corresponding and therefore indigestible sugar causes fermentative acid diarrhoea and can lead to body wasting and ultimately to the subject's death[14]. Lactose-induced diarrhoea in human lactase deficiency is considered to result from a combination of the osmotic effect of unabsorbed disaccharide in the small intestine and interference with fluid absorption in the large intestine by the products of lactose fermentation[4]. This procedure may operate in the horse.

Plasma galactose was absent from horses subjected to the lactose and glucose/galactose tolerance tests, but was detected in the peripheral circulation of horses receiving the higher galactose dosage rate 30 to 120 minutes after dosing, the peak level being reached by 60 minutes[18] (Fig. 7b). Although galactose and glucose are absorbed by the same active transport mechanism, the equine data support the finding that galactose is absorbed at a slightly faster rate[5]. Furthermore, the plasma glucose increase following galactose administration implied a rapid conversion of galactose to glucose in the equine liver.

SUCROSE AND MALTOSE TOLERANCE TESTS

The shape of the plasma glucose curve following sucrose administration was very similar to that after giving glucose and fructose, implying rapid hydrolysis of the disaccharide (Fig. 8b) but, the peak plasma glucose level for the sucrose dose was reached 30 minutes earlier than that for the monosaccharide mixture, the resting level being regained one hour earlier (Fig. 8b). The tolerance curves produced after maltose and glucose dosing in the adult horse were almost identical, the peak plasma glucose level being reached at 120 minutes, with the resting level being regained after six hours (Fig. 8c).

Thus the sucrose and maltose loads were effectively hydrolysed by the disaccharidase enzymes and the products of digestion were rapidly absorbed. Adult levels of sucrase and maltase are reached by seven months of age and, in these groups of adult horses, the mucosal enzymes were capable of dealing with disaccharide doses greatly exceeding those normally presented to the horse. Indeed, comparison of the results for the disaccharide and monosaccharide tolerance tests indicated that the disaccharidase enzyme activities were not limiting the subsequent absorption rate. Monosaccharide transport is apparently the rate limiting factor, for sufficient monosaccharide is released on hydrolysis to saturate the active transport mechanisms. Furthermore, the close association of the enzymatic and carrier functions of the brush border could confer a kinetic advantage for the absorption of disaccharides as compared to monosaccharides[11]. Examination of the sucrose and glucose/fructose tolerance curves in the horse supported the observation that glucose was better absorbed when given as sucrose than as free glucose[7].

In man, lactose hydrolysis appears to be rate-limiting, the maximum hydrolytic rate of lactose being too slow to saturate the glucose-galactose transport mechanism[9]. This probably holds true for the foal and young horse, lactose hydrolysis being slower than absorption of the equivalent glucose-galactose mixture (Fig. 6a).

The sites of highest disaccharidase activity in the small intestine could correspond to the sites of maximum absorption of the constituent monosaccharides rather than reflecting the differential development of brush border structures[15]. On the other hand, Neale[14] suggested that the disaccharidase activities bore little relationship to the rate of sugar hydrolysis in normal healthy subjects able to utilize the dietary sugars.

CLINICAL ASSESSMENT OF SMALL INTESTINAL DISEASE

Clearly, the small intestine of the horse, as in other species, is an important site for the digestion and absorption of carbohydrates and most probably of proteins and fats. Consequently, any impairment or deficit of normal physiological processes could predispose to intestinal dysfunction clinically exhibited as diarrhoea and malabsorption. Thus, selected sugar tolerance tests could be used on clinical cases of intestinal dysfunction to assess changes in mucosal integrity, in particular the hydrolytic and cellular transport mechanisms, leading to a specific diagnosis, rational lines of treatment including adjustment of the diet, and a reasoned prognosis.

Oral Glucose Tolerance Test (OGTT)

This test, devised for the horse[20] (Fig. 5), can be used to assess pancreatic endocrine function in suspected cases of diabetes mellitus, but more particularly to determine small intestinal function, as pathological changes in the mucosa interfere with cellular transport mechanisms and hinder glucose absorption as reflected in the shape of the tolerance curve. Two horses with symptoms of malabsorption and subjected to the test gave flat tolerance curves indicative of small intestinal dysfunction, later confirmed at *post-mortem* examination, normal intravenous glucose tolerance curves having eliminated any pancreatic involvement. In both horses, the mucosa and submucosa of the small intestine and the associated lymph nodes were extensively infiltrated with cells of the lymphocytic series, producing gross changes in villous structure from partial to total villous atrophy, accompanied by lowered or barely detectable levels of α-glucosidase activities[15].

In coeliac disease in man, the flattened mucosa of total villous atrophy reduces the mucosal surface area to 3 per cent of normal and requires only 3 per cent of the normal adult cell population for complete epithelial cover[25]. The microvilli, which increase the surface area of an individual enterocyte by a factor of 30 and number between 3 000 and 6 500 per cell, are also affected, becoming shortened, stunted and irregular[25], thus further reducing disaccharidase activities. Undoubtedly, similar marked changes in the mucosal surface area and enzyme content occurred in these two horses, affecting the capability for digestion and absorption of many dietary constituents and emphasising the importance of the small intestine as the major site of these functions under normal conditions.

A starch tolerance test developed by Loeb, McKenzie & Hoffsis[13] is probably of less use in determining small intestinal dysfunction than the OGTT, the test being dependent initially on the action of pancreatic α-amylase, and to a much lesser extent glucoamylase, and then requiring maltase enzymes to complete the digestion of starch breakdown products. The final

product is glucose, but the brush border enzymes may be deficient or missing in the presence of normal glucose transport mechanisms[20].

D (+) Xylose Absorption Test

A specific and efficient test of upper small intestinal function in man, the D (+) xylose absorption test, was developed for use in the horse[16], the pentose sugar D (+) xylose being absorbed partly by passive diffusion and partly *via* the same active transport system responsible for glucose and galactose, although at a much lower rate. A xylose dose rate of 2 g/kg body mass is required to produce a peak plasma xylose level of 30 mg/100 m*l* after two hours without causing alimentary discomfort or diarrhoea in healthy horses (Fig. 9). The test would probably prove too expensive for routine clinical screening but should be considered as an adjunct to the OGTT in determining the site and degree of small intestinal dysfunction.

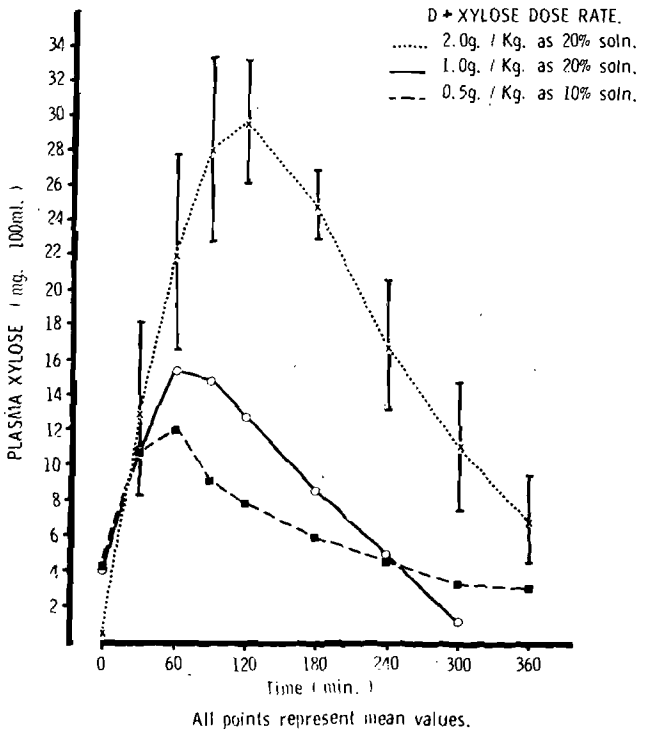

Fig. 9: D (+) xylose absorption test in the horse.

Oral Lactose Tolerance Test

Diarrhoea is not uncommon in the suckling foal. A consequence of enteritis is damage to the epithelium of the small intestine accompanied by depression of brush border disaccharidase activities and a resulting malabsorption of sugars. The ability to hydrolyse lactose is severely affected in humans with diffuse intestinal mucosal damage: lactase activity is depressed more than the other disaccharidases and is the last to return to normal after clinical recovery of the primary illness[14]. An apparent improvement in symptoms in such patients may result from a reduction in lactose intake[14].

The oral lactose tolerance test (OLTT) has proved a valuable screening test in human malabsorption; a blood glucose rise of less than 20 mg/100 m*l*, 15 to 60 minutes after ingestion of the test dose, coupled with the production of diarrhoea, is indicative of an enzyme deficiency[9].

An OLTT, devised for use in the horse[17], has highlighted the possible detrimental effects of continued lactose ingestion in foals suffering from severe diarrhoea. A foal reared artificially on a lactose-containing diet and suffering from a profuse watery diarrhoea of five days duration, was subjected to the test.

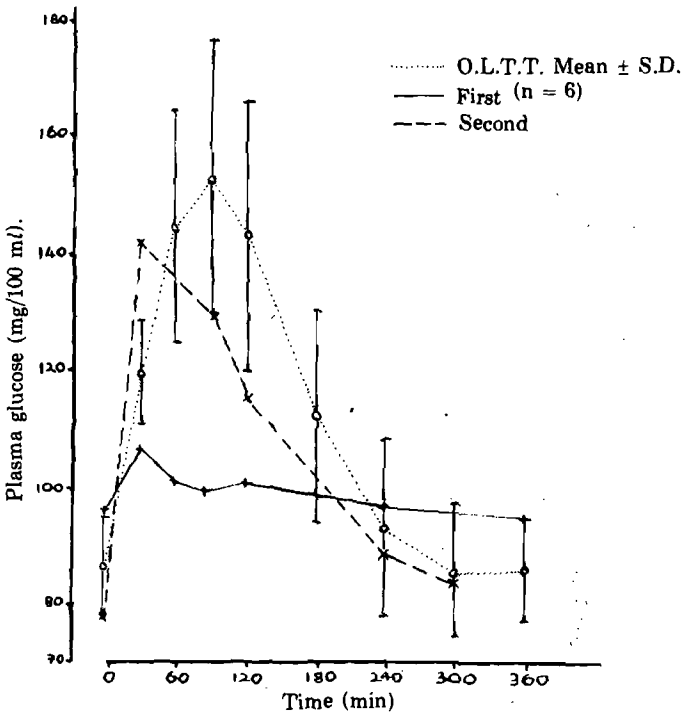

Fig. 10: Oral lactose tolerance test in the horse.

Thirty minutes after dosing, the plasma glucose rose by 10,6 mg/100 m*l* (Fig. 10). A change to a non-lactose diet, together with supportive therapy, produced a marked improvement in the foal's condition and faecal consistency. A second OLTT, performed 15 days after the first, gave a plasma glucose rise of 63,8 mg/100 m*l* at 30 minutes and a tolerance curve similar to the mean curve (Fig. 10). In the absence of mucosal biopsy, impractical in the horse except under experimental conditions[21], this test provides a valuable means of detecting transient, acquired lactase deficiency in foals and young horses: dietary adjustments necessary to improve the clinical picture can thus be made.

ACKNOWLEDGEMENTS

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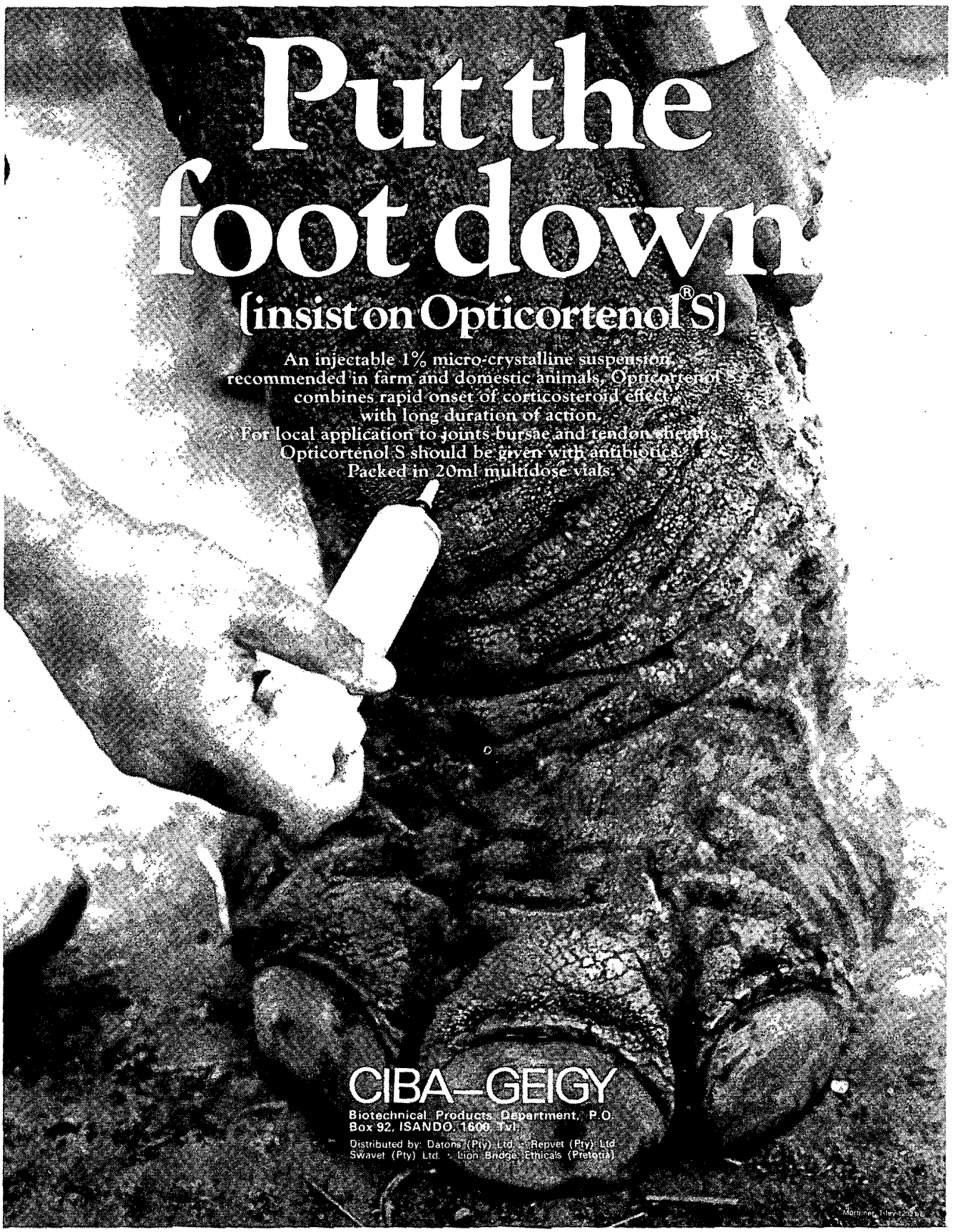
DISCUSSION

H.F. Hintz: How much lactose can one put in the diet of this sort of horse without problems? Also, what would happen, do you suppose, if the horse were to be continued on lactose in his diet? Would he still develop his lactase enzymes?

M.C. Roberts: The dosage of lactose that I was using was the standard 1gm/kg as a 20% solution, I found in a group of six horses that I could induce diarrhoea by fasting them over-night, then dosing them with lactose first thing in the morning and then doing the test. Within twenty-four hours of starting the test, two of these horses developed diarrhoea, passing a lot of flatus and dung of a 'cow-like' consistency, with soiling of the tail and perineum. When I gave these animals, which were approximately 5 or 6 years of age, 2gms/kg lactose, then I could induce diarrhoea in five of these animals. With regard to adaptation of lactase enzyme systems, if one feeds lactose throughout the animal's life, it has no effect on this decrease in lactase enzymes. One still gets this decrease between three and four years of age; this has been shown in laboratory animals and also in the human. There has been some work done on trying to adapt the human by feeding large doses of lactose to humans naturally intolerant to lactate to see whether one could actully raise the level of lactase enzymes throughout the small intestinal tract. At one stage there were two schools of thought: one postulating it could be done and one that it could not. Those who thought it could be done, were basing their work on Chinese who had emigrated to Australia. Undoubtedly, there is genetic selection for the ability to utilize milk. The work that the Australians did, has not been substantiated in other groups of originally non-milk-drinking races who have emigrated to predominantly milk-drinking areas. Again, a group working in Australia tried to induce lactase levels *i.e.*, to retain lactase levels at a high level in rats, by feeding about 30% lactose in the diet. They continued this for about eight weeks and they showed there was a slight increase in the rat, in which species this marked decrease of lactase activity occurs at about nineteen to twenty-one days of life. They could prevent this decrease occurring for about another two to three weeks. On the other hand, they could also do this by feeding high levels of glucose. This work has not been substantiated by other workers. The conclusion now is that lactase activity cannot be adapted by feeding of lactose.



Jl S. Afr. vet. Ass. 46(1) 29-37 1975
(Proc. 1st int. equine vet. Conf., Pretoriuskop)

DIETARY VALUE OF CUBES IN EQUINE NUTRITION

J. A. VAN DER MERWE*

If we put bits into horses' mouths to make them obey our will, we can direct their whole bodies

James 3 : 3.

SUMMARY

The revised NRC's Nutrient Requirements of Horses is changing the outlook on equine nutrition, dominated for so long by the traditional belief in oats. This has lead to the need for properly balanced diets, in the compounding of which dietary cubes offer the distinct advantage of providing a standardized diet of constant quality in keeping with modern knowledge. Additional factors are: longer storage, freedom from dust, palatability, refractoriness to mould infection, and facilitation of routine feeding. Cube size and hardness are important considerations for the manufacturer. Horses appear to favour smaller-sized cubes, but on the whole preferences for size and hardness appear to be equivocal.

The nutritional status of four commercial brands of horse feed marketed in South Africa has been analyzed and compared to commonly used raw feedstuffs. The results of feeding cubes have been assessed by means of a questionnaire. There appears to be a need for more knowledge concerning equine nutrition on the part of trainers, as well as for more specialized advisory services. The establishment of training centres has been suggested.

INTRODUCTION

This quotation from the holy scripture has been selected as it strikingly illustrates the parallel resemblances in restraining the tonque in man and animal. The metallic mouthpieces, the bits, physically direct, control and guide the movements of horses. Hence it is logical to reason that the context of this analogy revolves on the power and function of bits placed in mouths.

There is however, another meaning to the word 'bits', namely a small quantity, a portion, a morsel. For the purpose of our discussions, I wish to draw a similarity by way of a question. Is it not the bits which we place in mouths, constituting the diets of horses, which promote or hold in subjection, development, performance and action of the body?

This statement stands unchallenged. But before we delve into our subject matter, namely the Dietary Value of Cubes in Equine Nutrition, let us examine a number of facts which singly and in combination, precipitate a state of affairs necessitating changes in our outlook on equine nutrition.

The last three decades revealed to the world radical upheavals in the scientific and social spheres of mankind's activities and behaviour. It would not be exaggeration to state that almost all strata of society in the Western civilizations witnessed a metamorphosis, affecting their ways of living.

Everyone, in all walks of life, was involved and subjected to the gruelling process of constantly adapting to reorientation, as some existing and recognized standards of values, whether principles or materials, assumed modified dimensions.

In this respect, no attempt will be made to offer an explanation or analyse the causative agents other than saying that the advancement of the mechanical revolution and resultant automation had tremendous bearing on developments. So rapid was the expansion, that within a space of 30 years, the buggy carts had been converted to moon-ships and elevated missions to outer space explorations.

These achievements displaced the first horse power, known and employed by man since time immemorial. No wonder that equine scientists and lovers watched with great concern the declining populations of the aristocrat of the animal kingdom. The horse lost out on economic importance, with the result that research in this field of scientific endeavour waned to near non-existence.

Despite the horse being deprived of its utilitarian value, there appeared to be a re-awakening of its usefulness as an element providing fun, enjoyment and recreation. Today, reports indicate that legendary Pegasus in reality is returning in increasing popularity to once again fulfil a rôle in entertaining society. Racing, show jumping, polo, gymkhanas and hunting are becoming regular enthralling events while the number of riding schools and urban horse owners is increasing in size and number. The very fact that we are assembled here today, is convincing proof of the validity of this assertion.

A stable-mate to the sketched background is the manifestation of another phenomenon, namely that of short supplies and escalating costs of feedstuffs traditionally used in equine nutrition. This poses the question, especially to race horse breeders, owners and trainers: How should their steeds be nourished adequately and economically under present-day conditions?

Perusing literature on equine nutrition, which of late has become more prolific, the impression is indelibly gained that a dire need exists for information which would guide adoption and application of newer and different feeding practices, in an endeavour to overcome problems confronting equine establishments. The inadequacy of technical 'know-how' is being replenished in the updating of the NRC Nutrient Requirement of Horses, 1973. The opinion is held that this publication will stimulate equine feeding regimen to move from the pathway of art to that of science.

Looking out of the paddock, we see in contradistinction to the slow-paced equine nutrition developments, how tremendous strides have been made in the last decade on the broad nutritional front, as highlighted through the advancements of scientific discoveries involving the areas of other domestic

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animals, particularly poultry, pigs and ruminants. Nevertheless, there is no need to cast an envious eye, as Baker & Potter[3] appropriately have postulated: 'Perhaps the most significant difference in feeding horses and other domestic animals, is the unique purpose for which they are used. The horse is an athlete, and the entire equine nutritional programme is geared to his production and development in the efficient use of athletic ability'.

Breuer[5] supplemented by stating 'In practice, the criteria for judging the adequacy of horse feeds and feeding programmes differ from those traditionally employed by nutritionists. Growth rates, feed efficiency and cost of grain or product utilised in important farm species, are replaced by such criteria as size, development, appearance and performance at maturity or near maturity. With such subjective measures and in the absence of definitive research on horse nutrition, horse feeding practices evolved over the years, based on recipes for combining selective raw and nutritional supplements to accomplish the desired results. This amounted to an art which was highly developed by a few successful horsemen.'

These references clearly spotlight that the current views and outlook on feeding practices of the trainer, raw materials and equine nutrition are inseparable entities. If we are to attain our objectives, we must consider these three aspects as integral parts of a success structure.

THE TRAINER

The entire equine feeding programme employed by owners, breeders and trainers is founded mainly on tradition. Admittedly, experience is knowledge, which could be scientifically unsound, because in most cases there is limited scientific back-up to guard against the influx of fads, fancies and fallacies. These facts are allied to the hyperaesthetic nature of the race horse, making it difficult for the trainer, however wise or dedicated he may be, to depart from the 'art' and 'magic' of treating each horse as an individual, meeting its specific needs. No one realizes more than the trainer that nutrition of the horse is fundamental to its performance. The constant aim is to get maximum expressive performance out of the genetic ability. This places a demand on the trainer with regard to feeding routine adjustments.

To mention but a few: some horses are fast eaters, others are slow, some prefer different feeds and will consume more hay. There are fixed likes and dislikes, even different psychological attitudes. No two horses are the same. It is no overstatement of fact that successful feeding of horses is completely dependent on the skill, ability and finesse of the horseman, who should constantly be alert, studying the peculiarities of the horse. Table 1 indicates a complicated feeding schedule used for a race-horse in training and racing.

Table 1: DAILY DIETARY ALLOWANCE FOR A RACE HORSE (500kg) IN TRAINING

	Morning	Night
Oats (rolled)	3 kg	4 kg
Dry Lucerne Hay	1,5 kg	1,5 kg
Wheat Bran	250 g	250 g
Oat Hay	500 g	500 g
Fresh Shredded Carrots	600 g	600 g
Salt	40 g	40 g
Teff Hay	2–3 kg (as needed)	2,5 kg
Green Fresh Lucerne	–	500 g
Nutrient Ingestion:		
Protein	± 824 g	± 946 g
T.D.N.	± 4,68 kg	± 5,48 kg

Molasses	As desired
Wheat germ meal	50 – 100 g
or	or
Wheat germ oil	10 ml
Pro-Nutro	200 – 500 g alternate days
Brewers Yeast	As needed

Vitamins:

Vitamin B_{12} Injections
Vitamin B_1 Injections
Vitamin B-Complex – Oral administration

Seaweed, folic acid, calcium carbonate, vitamins A,C,E, and other undeclared (miracle) constituents may be added.

RAW MATERIALS – OATS DOMINANCE

As pointed out, the entire nutritional programme is designed to promote and ensure the development and expression of physical fitness. In providing the nutritive needs of horses, the first consideration is the actual nutrients required, rather than the particular feeds to be used. In the animal industry, many operators classify certain feeds as: bull feeds, cow meal and horse feeds, etc. More often than not, the choice of

Table 2 : LIVESTOCK NUMBERS [1)]

Species	1946	1950	1955	1960	1964	1971[2]
	Thousands					
Cattle	12 593	11 513	11 689	12 295	12 243	7 773
Sheep	30 832	31 361	37 042	38 789	39 717	29 424
Goats	5 189	5 185	5 137	5 057	5 667	2 134
Pigs	1 118	1 350	1 127	1 381	1 165	776
Horses	687	679	558	472		197

Source – 1) Agricultural Census
– 2) Preliminary – White areas only

such a feed commodity is identified with a belief as to its nutritive suitability.

In the equine world, oats certainly is a true example, being accepted and recognized as the traditional feed. This concept has become so ingrained that many horse owners refuse to use other cereals. They simply state emphatically that a substitute for oats does not exist. What aggravates matters, is the misconception that oats are oats. Nothing could be further from the truth. Analytical quality variances do occur from time to time, as well as mass, (plumpness), freedom from mould, mustiness, foreign matters, etc.

In addition to tables 2 and 3, revealing the Republic's livestock numbers and quantity production of oats, the South African Wheat Industry Control Board revealed the following tonnages of oats available to the trade:

1972 – 1973 : 32 477 metric tons
1973 – 1974 : 20 864 metric tons

It is evident that about 80% of total oats production is not reaching the markets. Apparently oats production has not kept pace during the last two years, as a result of increasing demand. The static rate of production had the effect of necessitating interest in other feed commodities.

Table 3 : PRODUCTION OF OATS, BARLEY, RYE AND LUPINS (WHITES ONLY)[1]

YEAR	Oats	Barley	Rye	Lupins
	Tons in Thousands			
1965/66[2]	93	36	9	28
1966/67[2]	88	38	7	33
1967/68[2]	169	51	13	35
1968/69	116	37	8[2]	35[2]
1969/70[2]	90	19	7	16
1970/71[2]	121	30	7	28
1971/72[2]	98	32	6	11
1972/73[2]	105	33	7	13
1973/74[3]	103	32	6	8

Source – 1) Agricultural Census
– 2) Estimates by the Division of Agricultural Marketing Research
– 3) First Estimate

'Built in' Horse Feeds

There is evidence suggesting that sophisticated equine entrepreneurs are beginning to examine the introspect and prospect of feeding standards and materials, and are less concerned about retrospect systems and methods.

Other than oats, commonly used feed ingredients are periodically coming into short supply, such as wheat bran, lucerne and oil cakes. Adding gravity to the feed quality situation, is the fact that the denaturing of cereal by-products is causing alarm in nutritional quarters. Advancements in milling techniques resulted in by-products, especially wheat bran (digestive), becoming denuded of germ and endosperm, yielding a product to which feeders were not accustomed. Hence there is mounting interest in the 'built in' horse nutrition and feeding methods.

Ott [15] welcoming the revised NRC bulletin, Nutrient Requirement of Horses, suggested: 'The horseman and the feed manufacturer should take the release of this publication as an opportunity to review current products and programmes.' It must be realized that horse nutrition is not limited to oats and a few other favoured 'horsey' feeds.

To the contrary, feed manufacturers are examining the characterizing merits and properties of oats in an effort to evolve substitutes, should oats supplies become inconsistent and unavailable. Simultaneously, on research farms, time and effort are devoted to study and project experimentation of new products and techniques and that despite the fact that in accordance with statistics released by the South African Association of Balanced Feed Manufacturers, formula feeds totalled 1,8 million metric tons in 1973, and for 1974 prognostication based on current growth rate will exceed two million tons. Horse feeds marketed comprise a mere 0,85% of total aggregate. This is convincing proof of the high esteem in which the equine industry is held. In 1971 (Table 2), the equine population in the Republic numbered 197 000. There are indications that this will continue to increase, especially around the metropolis.

NEW ERA IN EQUINE NUTRITION

Equine nutrition is not covered in a veil of mysticism, as we sometimes make out or like to believe. True, it must be acknowledged that scientific evidence is lacking in precision and perfection, because metabolic investigations on horses have been lagging in the past. In consequence, dietary needs of horses are based on the NRC recommended nutrient requirements. This was confirmed by Ott[16], and Breuer[5], who respectively used NRC recommendations as the foundation for computing horse feed formulae, and regarded the revised edition as improved and comprehensive information. Taking these specifications as our guidelines and combining it with our empirical but practical observations, we adopt feeding techniques which are nutrition-wise less based on trial and error, although we may err on the side of generosity. Perhaps unwittingly we follow wasteful feeding schemes, especially where Thoroughbreds are involved.

As a result of this, many a diet becomes imbalanced, and we fail to achieve our objectives of promoting optimum and sustained performance. Furthermore, feeding practices are influenced by other extraneous factors, such as quality and quantity of forages fed in conjunction with concentrates, and interactions of management, environment and health, which must be maintained in a balance of harmony. Quality is closely linked to quantity.[15 24]

Zintzen[24] concluded that the horse is altogether more dependent than the ruminant on the quality of the diet. The nutrient requirements are intermediate between those of the pig and the cow. The nutritional requirements of horses could vary enormously in relation to breed and type, age, temperament, severity of work, etc. These are the facts which complicate the precision and perfection of feeding.

Ott[15] emphasized that the nutrient requirements of a horse are influenced by the animal's size and activities. Each animal has a requirement for each nutrient to maintain its current state. He lists activity factors to be considered in horse feeding, which include growth, breeding, gestation, lactation and work.

In other domestic animals, we can measure pro-

ductivity, or set standards, and determine and compare milk yields, livemass gains in beef, broilers and pigs, employing specific production efficiencies in compliance with a time factor. We are able to make biomass calculations. In a horse we are merely observing physical performance through muscular energy expenditure. Our criterion is speed in time and distance, a revelation of relationship between nutrients ingested, vigour and endurance. Performance becomes our unit of measurement in unlocking and releasing horse power.

Processed Feeds

Cubes, pellets and crumbles are processed feeds, obtained by employing presses. This is not an innovation. An illustrated London weekly newspaper, *The Graphic*, in its July to December 1877 issue, published the following report: 'A new cooking apparatus has been invented for the Russian troops. It is fitted on a two-wheeled cart, and consists of a cylindrical pot with furnace and chimney so arranged, as to be always upright, in spite of the severest joltings. The food is cooked by steam, and a single apparatus, in three hours, will supply a company of 250 men with ample *stchi* and oatmeal soup. As the kitchen only occupies half of the cart, uncooked food, plates, cups, etc, can be carried at the other end. Condensed forage is also supplied by three Russian manufacturers, one at St Petersburg, turning out 30 000 lbs *per diem*. The forage is composed of small biscuits of oat-meal, pea flour, rye-meal and ground linseed, and 28 biscuits form a single ration for a horse, containing as much nutriment as 12 lbs of oats, and being about one-fifth of the bulk.'

In this country processed equine feeds, that is, cubes, pellets and crumbles, have not created a fanfare in the market-place. This is generally attributable to the fact that oats, singly or in combination, is universally an accepted complete and safe feed for horses. Presently there is increasing interest in cubes and pellets as equine feeds, and it is envisaged that this kind of feed will steadily become more popular. It deserves mentioning that feed compounders have experienced resounding success in supplying beef production schemes with complete feeds. In other parts of the world complete horse pellets have established recognition and are making headway.

Earle [7] arrived at the conclusion that a complete pelleted horse ration offered several advantages over feeding practices comprising separate allowances of grain mixtures and hay. Hintz & Loy[11] conducted experiments to study the effect of pelleting horse rations on the efficiency of feed utilization, digestibility, rate of passage of digestion and amount of time required to consume the ration. It was established that pelleting the ration did not influence rate of gain or efficiency of feed utilization.

Haelein *et al.*[10] reported on the digestibility and voluntary intake of a complete, pelleted ration by horses. They concluded that an alfalfa-corn complete pelleted ration appeared to be of high nutritive value and of high palatability to the horse, but a problem of wood chewing associated with pelleted rations needed further study.

Yuhso Nagata[23] as result of considerable research designed three formulae of complete, pelleted rations for race-horses, using oats, wheat bran, soybean, oatmeal, potato protein and molasses. Pellet size was 10 mm diameter and 12 mm in length, with a hardness of 23 kg pressure.

Stillions[20] emphasized that pelleting the feed will not overcome a nutritional imbalance, but generally pelleted feeds are complete feeds. Pelleted feeds provide adequate nutrition. Pelleted, complete feeds are available for different life phases and activities of the horse. A table was presented showing the digestibility coefficients for pelleted diets of different nutrient quality. He proposed a pellet size of 6 x 6 cm.

Ott[16] commented that complete rations, those containing the roughage, grain and appropriate supplementation, offer considerable opportunity for the feed manufacturer. Complete feed pellets are attractive. He warned that management must compensate for boredom.

The manufacture of cubes, pellets and crumbles is a two-stage process. Several makes and types of cubing machines are used. All ingredients, in accordance with the prescribed computerized formula, are blended in a mixer and conveyed into the cascader. Here the meal is steam – and heat – conditioned; molasses, if provided in the formula, is incorporated. When the desired temperature of 65°C is reached, the heated, moistened material is pressed through the die, which resembles a sausage machine, transforming the meal into cylindrical miniature ingots.

In the die, the material is exposed to a high pressure of between 1 000 – 5 000 kg/cm^2 for a duration of 3 – 4 seconds. Friction may cause inside temperature to rise to 150°C, and drop down to an exit temperature of 75 – 80°C. From the die, the cubes pass over a cooler, where moisture is reduced from 14 – 15% to a 10 – 11% level. Pellets are made the same way, only a die with different apertures is used. After cooling pellets are passed through a roller crushing system to obtain crumbles.

Physical Size of Cubes

Physical size and form vary and change in relation to the manufacturers' recommendations and market requirements.

The following measurement variations exist:

Category	**Diameter**	**Length**
Crumbles	0,8 – 1,0 mm	1,0 – 2,0 mm
Pellets	1,5 – 3,0 mm	4,5 – 6,0 mm
Cubes	9,5 – 18,0 mm	12,0 – 20,0 mm

From practical feeding trials horses appear to favour smaller sized cubes.

MANUFACTURING PROBLEMS

Hardness of Cubes

Cube durability or hardness presents a problem to the animal feed compounder, who has to consider acceptability to the animal, and the inevitable abrasion during handling and in transit. A high percentage of 'fines' in cubes or pellets is undesirable. It is a distracting feature to the buyer who wants to feed cubes and not meal.

MacBain[13], in dealing with the problem of maintaining constant pellet quality, listed mechanical, environmental and operational factors which affect pelletability. Similarly, Sebestyen[19] postulated 'Hardness is a factor in the palatability of a pellet. As

such it is obviously limited to the degree of hardness which is not only suitable, but also acceptable to the animal, which may refuse it if it cannot chew it comfortably. Excessive hardness may also affect digestibility, even if it is accepted by the animal. It is not the objective of this abrasion resistance test to determine the limitation of hardness, but it must be taken into account in the evaluation of results.'

Pfost[17] reported 'Durability tests have been proposed and tested from the standpoint of controlling pellet quality to prevent fines during the handling of pellets. The requirements from an animal acceptance importance should be conducted to determine that feeding trial results will correlate with the tumbling can device used'. Wornick [22] expressed the viewpoint: 'The very nature of feed pelleting seems to render it almost immune to precision and standardisation. The operation itself might be considered a variable, since it is generally very difficult to duplicate conditions from day to day, or even from run to run.' He submitted some major variables in feed pelleting, such as feed composition, texture, mash uniformity, steam pressure and temperature, conditioning, pellet pressure in die, and pellet size. He expressed concern about the destruction of micro-ingredients owing to heat and moisture.

Hardness and durability tests were conducted on different commercial brands of equine cubes with the object of ascertaining acceptability. The results are presented in tables 4, 5 and 6. The Kahl pellet hardness tester[12] and the Gutekumst tumbling can device[9] were used. Where bursting strength was tested on cubes made by one manufacturer, using the same machine, variances were not significant, while in commercial brands the results fluctuated over a wide scale. Durability indices (Table 4) also showed a similar pattern.

Table 4: HORSE CUBE HARDNESS TESTS

Cubes	Number of Samples	Variances	Average Reading
Race Horse Cubes	10	4 – 10	6
Yearling Cubes	10	6 – 10	8
Riding Horse Cubes	10	5 – 8	6
Special 'BR' Race Horse Cubes	10	4 – 8	5
Supplement Cubes	10	3 – 7	6

All readings are in kg Bursting Strength.
Samples tested five days after date of manufacture.
Manufactured on same cubing machine (press).

Table 5 : HARDNESS TESTS CONDUCTED ON DIFFERENT COMMERCIAL BRANDS OF EQUINE CUBES

Codes	Number of Samples	Variances	Average Reading
EP	10	4 – 7	5
EM	10	4 – 12	9
LL	10	8 – 19	13
MW	10	9 – 18	14

All readings are in kg Bursting Strength.
Samples tested 5 – 10 days after date of manufacture.
A Kahl Pellet Hardness Tester was used.
Cubes manufactured on different types of cubing presses.

Table 6 : DURABILITY TEST ON DIFFERENT COMMERCIAL BRANDS OF EQUINE CUBES

Number of Samples	Brand Codes	Durability Index (%)
2	MW	95,0
2	EM	88,0
3	LL	79,5
4	EPV	92,0
3	EPSF	92,5
1	EPR	90,5
2	EPCT	78,5
1	EPCC	93,5
3	EPD	62,0

Samples tested 6 – 9 days after date of manufacture.
A Gutekumst Tumbling Can Device was used.

Equine Reaction to Hardness

Horse cubes compounded by employing one formula can vary in size, colour and hardness. The appetite inclinations, preferences and behaviour differed greatly amongst individual horses. Tests on ingestion rates of cubes of the same composition were conducted to clarify the effects of various degrees of size and hardness of cubes, and the responses of horses unaccustomed to cube feeding. The results revealed that the duration of feed intake at a measured period of time became longer and the number of chewing movements appeared to decrease when the size of the cubes was smaller. Some horses prefer the smaller-sized (9 x 12 mm) cubes, which were relished with ease. Others were partial to the larger size of cube (12 x 12 mm). Preference differences are not easy to explain, but may involve a physical property or need for a bulky material factor.

Yuhso Nagata [23] adjusted the hardness of pellets by the degree of fineness, which was controlled by changing the mesh of a sieve used for grinding. It was measured roughly by means of Asker's hardness scale. He determined that there was no difference in palatability between any two kinds of pellets. Pellets were not big or hard enough to change the rate of eating, even though it was more or less difficult to eat pellets. The total feeding time of an allowance of 2 kg of pellets was about 20 minutes. With palatable compositions a horse was inclined to eat the kind of pellets in the manger to which he had turned his head first, without being influenced by the size of pellets. Two kinds of pellets in each manger were provided for the horse simultaneously. Concerning hardness, preferences varied, but ultimately differences between pellet hardness and sizes were not significant. Horses will tolerate hardness if pellets are palatable. Smaller-sized pellets seemed to increase duration of ingestion, and chewing rate was decelerated.

Effects of Cubing on Stability of Micro-ingredients

Frequently one hears the prediction that heat and moisture in the cubing operation are destructive to vitamins. Equine rations are highly fortified with all known synthetic vitamins. Mullin[18] intimated that high temperature, moisture, pH and exposure duration are factors involved in vitamin decomposition. Some components, like pantothenic acid, are heat labile, while others are less heat sensitive. Surveys showed that specialized poultry feeds suffered insign-

ificant potency losses as a result of the pelleting operation.

Surmizing that stability problems do exist, feed manufacturers routinely add safety levels of vitamins to obviate deficiencies arising. Prompt feeding of pelleted rations is prudent to reduce losses during storage.

Components and Formulation

A wide selection of ingredients is used in compounding the different horse feeds offered on the market. Raw materials are purchased on a quality specification basis, and include maize (corn), wheat bran, oats, maize and wheat germ meal, lucerne (alfalfa), groundnut and sunflower oilcakes, fish meal, calcium and phosphatic supplements, trace mineral mixtures, and vitamin pre-mixes.

All equine feed formulations are computerized, basing quality standards on the NRC nutrient specifications and other authoritative sources.

Purpose of Feeding Cubes

The purpose of feeding cubes, pellets and crumbles to horses, is to evolve a feeding system which will solve the feeding problems of ingredient quality and constancy, and to provide as accurately as possible all the nutrients required for a specific purpose at an economical price. The inclusion of cubes to supply part, if not all of a horse's ration, is gaining momentum. Some handicaps must be overcome. One is prejudice on the part of the buyer, owing to lack of understanding as to the nutritional virtues of cubes. Discussions frequently develop into controversies as to the advantages and disadvantages. In a way this is understandable, as horse owners and trainers are accustomed to their secret ways and theories of what constitutes the proper manner of feeding racehorses in particular.

It needs to be explained that including cubes in racehorse diets, means only a difference in material and not in practice. The governing factor still remains the human element. There must be healthy relationship and understanding between trainer, groom and horse. Irrespective of how perfect the cubes may be, unless the horse is treated and managed to consume the required allocation daily, the exercise is doomed to failure.

A large variety and range of equine feeds is compounded and marketed, namely for yearlings, brood mares, race-, riding- and draught horses. Vitamin, protein and mineral grainfree concentrates are designed to be blended with oats and other farm-produced materials.

Manufacturers of compounded equine feeds follow different ingredient compositions and chemical analyses, depending whether the cubes are to be used alone or in combination with other feeding materials.

Cubes, pellets and crumbles are processed feeds. It is a packaged feed, fully balanced and constant in composition for each cube. It is known that frictional heat treatment exerts a sterilizing effect and improves digestibility through gelatinization of starches. The plus properties are: longer storage tolerance, freedom from dust and mould, palatability and facilitation of feeding routine.

Quality Evaluation of Cubes

Evaluation of feedstuffs must be viewed from two angles. The oats and other essential ingredients which the owner or trainer wishes to buy, must be compared with a package feed such as cubes, pellets or crumbles, and the processed feed must be evaluated on an analytical specification basis.

Unfortunately the feed tag provides only part of the information needed for evaluating a particular horse feed designed and compounded for a specific purpose. The analysis on the feed tag must, by law, be a reflection of the contents. Five classes of nutrients, namely, protein, fibre, fat, calcium and phosphorus are enumerated in percentages complying with the minimum and maximum specifications as determined by Government regulations.

The raw materials incorporated in compounding a feed, are not listed. While this may be deemed empirical quality control, cognizance must be taken that responsibility for quality uniformity and constancy rests on the reputation of a balanced feed manufacturer. The competitive spirit of commercialism is a reliable quality security measure, apart from the fact that deviations from registered analysis are infringements of the law, subjecting the manufacturer to prosecution.

The fact that chemical composition is merely a guideline, frequently creates a resistant attitude amongst horse breeders. Some of them are not prepared to rely on the judgement of the horse, and view the nutritional virtues of cubes with prejudice.

Realizing that feed analysis is no warranty of nutritive merits, feed manufacturers support their products on a basis of providing technical advisory services. After all, a good axiom, is not what is printed on the feed label, or placed in the feed trough, but that which is utilized by the horse that counts. The integrity of the feed manufacturer is the buyer's guarantee.

Factors Complicating Feeding Practices

From the foregoing it is evident that equine feeding practices are as yet not clearly defined nutritionally. At this juncture it is necessary to draw a line of demarcation, dividing work horses from Thoroughbreds. The feeding of ordinary riding horses is far more simplified and less onerous than that of racehorses. Bourke [4] stated 'Few, if any, specific nutrient requirements of Thoroughbreds in training have been established'. This assertion is true, and therefore only known facts could be applied, at the same time employing as wide a variety of ingredients as possible to minimize risk of deficiencies. Against this statement is the fact that feeding standards for all horses were established many years ago, based mainly on experience. In modern times, these standards have been modified and updated in relation to applicable research discoveries, and borrowing information from experimental evidence gained on other species. While these simplified feeding systems and practices yield satisfactory results with pleasure and working horses, mixtures composed of hay, chaff, maize, wheat bran, oats, germ meal, etc, are inadequate for race-horses.

Training race-horses requires considerable skill and patience, as the minutest detail must be attended to, considering the idiosyncracies of the individual. This makes standardization difficult.

Horse owners sometimes do not know the live mass of their animals and disallow a calculation on maintenance allocation. Also, it is difficult to assess the severity of work done, hence production requirements are guesswork, and the whole feeding schedule becomes rule of thumb.

Taking all into account, it is not an easy task to compile rations for race-horses, arithmetically particularly those prepared and trained for an approaching race. What proved adequate, or even excessive to one horse, can be insufficient for another. Nervous, excitable horses may require more food of a kind than one with a placid nature. Distance races place a greater nutrient demand on the horse, and therefore the skilful trainer may be compelled constantly to revise and rearange feeding schedules in compliance with the dietary needs of horses training and racing. It is under these conditions that cube or pellet feeding can make a positive contribution as a balanced composite nutritional unit.

In an endeavour to test the validity of the above assumption, the following two-fold investigation was instituted.

A. DETERMINING THE NUTRITIONAL STATUS OF COMMERCIAL HORSE FEEDS AVAILABLE ON THE MARKET

The analytical data presented in tables 7 and 8, reflect the composition of four brands of horse feeds, as well as ingredients routinely included in computing horse diets. The composition of Canadian Oats and a product labelled 'Oats Constituted' (synthetic), were included, the objective being comparison of their intrinsic values. The latter, computerized blend of feedstuffs, was rated high on palatability and acceptance. Preliminary results indicated that it would be practicable to substitute it for oats.

Table 7: AMINO ACID ANALYSIS OF DIFFERENT BRANDS OF COMMERCIAL RACE HORSE FEEDS.

PERCENTAGE CONSTITUENT	BRAND CODE			
	EP	LL	EM	MW
PHENYLALANINE	0,55	0,75	0,51	0,56
TYROSINE	0,42	0,52	0,37	0,42
LEUCINE	1,21	1,20	1,01	1,20
ISOLEUCINE	0,43	0,57	0,39	0,49
METHIONINE	0,26	0,26	0,21	0,21
VALINE	0,60	0,73	0,58	0,67
T.S.A.A.	0,40	0,46	0,34	0,34
ALANINE	0,81	0,86	0,72	0,83
GLYCINE	0,65	0,86	0,68	0,75
PROLINE	0,97	0,98	0,82	0,95
GLUTAMIC ACID	2,31	2,76	2,06	2,21
SERINE	0,66	0,79	0,61	0,70
THREONINE	0,56	0,63	0,45	0,60
ASPARTIC ACID	1,14	1,53	1,15	1,19
ARGININE	0,73	1,26	0,75	0,85
HISTIDINE	0,37	0,45	0,33	0,38
LYSINE	0,60	0,71	0,53	0,59
FAT	3,80	3,85	3,10	3,69
PROTEIN	12,90	15,60	11,90	13,80

Samples received on 11/6/74

B. ASSESSING THE RESULTS AND EXPERIENCE FEEDING CUBES TO THOROUGHBREDS AND PLEASURE HORSES

Technical exploratory Investigations.

A survey was undertaken through the channels of technical advisory services aimed at determing the

Table 8: NUTRIENT DIFFERENCES IN INGREDIENTS EMPLOYED IN EQUINE NUTRITION

PERCENTAGE CONSTITUENT		YM	WB	WP	DC	O	CO	LM	GN	FM
T.D.N.		83,0	60,0	65,0	70,0	68,0	72,0	55,0	72,0	74,0
METHIONINE		0,20	0,23	0,25	0,25	0,18	0,18	0,20	0,48	1,55
FIBRE		2,6	10,0	7,0	12,0	11,8	11,0	30,0	6,2	1,0
T.S.A.A.		0,39	0,56	0,51	0,48	0,36	0,36	0,32	1,10	2,54
N.E. CAL/Kg		1480	1000	1125	—	1100	1160	740	1160	1320
M.E. CAL/Kg		3430	1430	1800	2475	2560	2600	880	2200	3000
AVAILABLE P		0,07	0,30	0,17	—	0,12	—	0,20	0,21	2,22
PHOSPHORUS		0,20	0,90	0,50	0,33	0,35	0,35	0,20	0,65	2,22
CALCIUM		0,02	0,13	0,07	0,46	0,15	0,10	1,20	0,25	3,50
THREONINE		0,34	0,49	0,51	—	0,37	—	0,64	1,15	2,80
TRYPTOPHANE		0,09	0,21	0,19	0,18	0,15	0,18	0,28	0,48	0,80
ARGININE		0,37	1,00	1,05	0,70	0,70	0,71	0,58	4,44	3,70
WATER		11,5	10,0	10,0	9,0	12,0	6,0	6,0	6,0	8,0
LYSINE		0,27	0,62	0,65	0,53	0,45	0,40	0,77	1,55	5,00
FAT		3,80	3,50	4,20	3,20	4,00	4,00	2,00	1,00	9,00
PROTEIN	Min	7,20								
	Ave	9,30								
	Max	10.90								
			15,50	16,50	14,02	9,40	12,00	15,75	42,50	64,00

YM = Yellow Maize Meal
WB = Wheat Bran
WP = Wheat Pollard
OC = Oats Constituted
O = Oats
CO = Canadian Oats
LM = Lucerne Meal
GN = Groundnut Solvent Extracted
FM = Fish Meal

penetration of cube feeding systems in the equine establishments. The response to the questionnaire involving some 400 Thoroughbreds and 150 pleasure horses revealed that stud breeders, owners and trainers are beginning to recognize that cubed feeds, as a sole diet or in combination with oats, yield the desired results. It indicated that human insistence on the quality goodness of oats for man and horse was disappearing. Scientific knowledge is beginning to occupy a position of prominence in the minds of people concerned with feeding horses.

Many owners declared their interest in experimentation in the development of new products.

Allow me, in conclusion, to quote Tyznik[21] who stated: 'Of all animals that have been entrusted to the care of man, the horse is probably the critically maltreated. This is especially true of the young animal from birth to about two years of age. It becomes imperative that the horse be given every opportunity to develop at the most rapid rate possible, so that when the horse is expected to perform, he is ready, not only to react, but to do it well.'

QUESTIONS	ANSWERS	QUESTIONS	ANSWERS
a) What is the reason for feeding cubes? Is it the unavailability of oats, or is the objective to ensure dietary quality maximization?	Both, but mainly seeking to provide optimum nutrition.	b) Do individual horses show brand preference?	Yes.
c) What is the quantity allowed per race horse per day?	Quantity depends on severity of work, condition of the animal, age, live mass, and whether in training or racing; it varies from 4 – 7 kg.	d) Do horses differ in behaviour concerning rate and time of ingestion (chewing)?	Observations indicated that horses differ distinctly in rate and time of ingestion.
e) Is hardness of cubes important in avoidance of a percentage of fines?	Yes, but if cubes are too hard inappetence occurs.	f) Do horses unaccustomed to cubes need adaptation time?	Horses adapt to the new feeds in a period of 2 – 6 days. Refusal of cubes does occur, maximum 4%. When cubes are gradually introduced mixed with daily feed, no problems were experienced.
g) Are cubes fed only to horses prepared for the race-track or are mares and yearlings included in the feeding regimen?	Cubes are fed to all denominations, as part of the diet. There were exceptions in the case of pregnant mares.	h) What are the other ingredients fed in conjunction with cubes?	Oats, pearl barley, bran, wheat- and maize germ, green feed and undisclosed compounds. Hays commonly used were: oat, chaff, lucerne (alfalfa) and teff-grass.
i) Do digestive disturbances occur?	A few incidences of colic occurred when green feed was fed after cubes.	j) Were abnormalities in behaviour observed?	Some horses showed tendency towards wood chewing and cribbing ascribed to high level cube allocations.
k) Is oats regarded an essentiality in cubes?	Not necessarily, but a definite attraction.		

REQUESTS AND SUGGESTIONS

It was interesting to learn that from several quarters requests and suggestions came for:

(1) A training centre (educational) to be established where trainers and feed attendants could participate in an equine nutrition course.

(2) Advisory services to be of a more specialized nature.

This message rings clear and loud, but if our ideals are to be achieved, it will be necessary to follow the pathway of feeding young horses on a basis of nutrient parameters, and not remain in the realm of 'art'.

This presentation may be regarded as a 'gift horse' but it is anticipated that, if 'looked in the mouth', it will be with understanding. Naturally 'horse sence' will be enrichment of knowledge, but compilation of facts is a formidable task if not coming 'straight from the horses's mouth.'

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DISCUSSION

K. Dalzell: Am I correct in saying that Dr. van der Merwe had said that colic did not supervene after feeding of cubes unless green lucerne had also been fed?

J.A. van der Merwe: This fact came out of a survey. Approximately 4 per cent of horses on different brands of cubes had developed colic. I do not know which brands were incriminated but colic arose shortly after green lucerne had been fed. In other words after the horse had had its allocation of cubes for the day, a quantity of green lucerne had been fed and only then the horses developed colic. The accuracy of this survey is very difficult to assess: it was done by several technical people and these particular observations were made in Natal. Perhaps Dr. Baker could shed some light on the question.

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INTESTINAL ABSORPTION OF CALCIUM AND PHOSPHORUS BY HORSES*

H.F. SCHRYVER**

SUMMARY

Recent knowledge regarding the absorption of calcium and phosphorus from the intestine of the horse has been reviewed. Consideration has been given to sites, mechanisms and factors affecting absorption as well as to the availability of calcium and phosphorus from feedstuffs commonly used in Eastern North America. Although the anatomy of the digestive tract might appear to impose restrictions on the ability of the horse to assimilate minerals, studies of absorption show that the horse utilizes the calcium and phosphorus of many feeds very efficiently.

INTRODUCTION

The small intestine is the major site of calcium and phosphorus absorption in many species [4 18 19 21 32]. Some of the herbivores have special digestive mechanisms which may facilitate mineral absorption. In the ruminant, microbial action degrades fibrous materials and chelating agents such as the complex organic phosphate, phytic acid, before the digesta reach the absorptive sites of the small intestine. In contrast, microbial digestion occurs in the lower digestive tract of the horse, so that feedstuffs must enter the small intestine without benefit of extensive prior digestion. Coprophagy is an important mechanism for mineral utilization in the rat [30] and in small, non ruminant herbivores, such as the guinea-pig [8] which has a gastrointestinal tract similar to that of the horse. Although foals have been observed eating faeces, coprophagy is not considered to be a common practice by horses.

Since the most important natural sources of calcium are roughages and since some phosphorus sources are complex organic molecules, the horse might appear to be at a disadvantage in obtaining minerals from feedstuffs. Thus, studies of sites, mechanisms and efficiency of absorption as well as availability of minerals from feeds are of importance to the understanding of horse nutrition and provide information of practical importance in feeding programs.

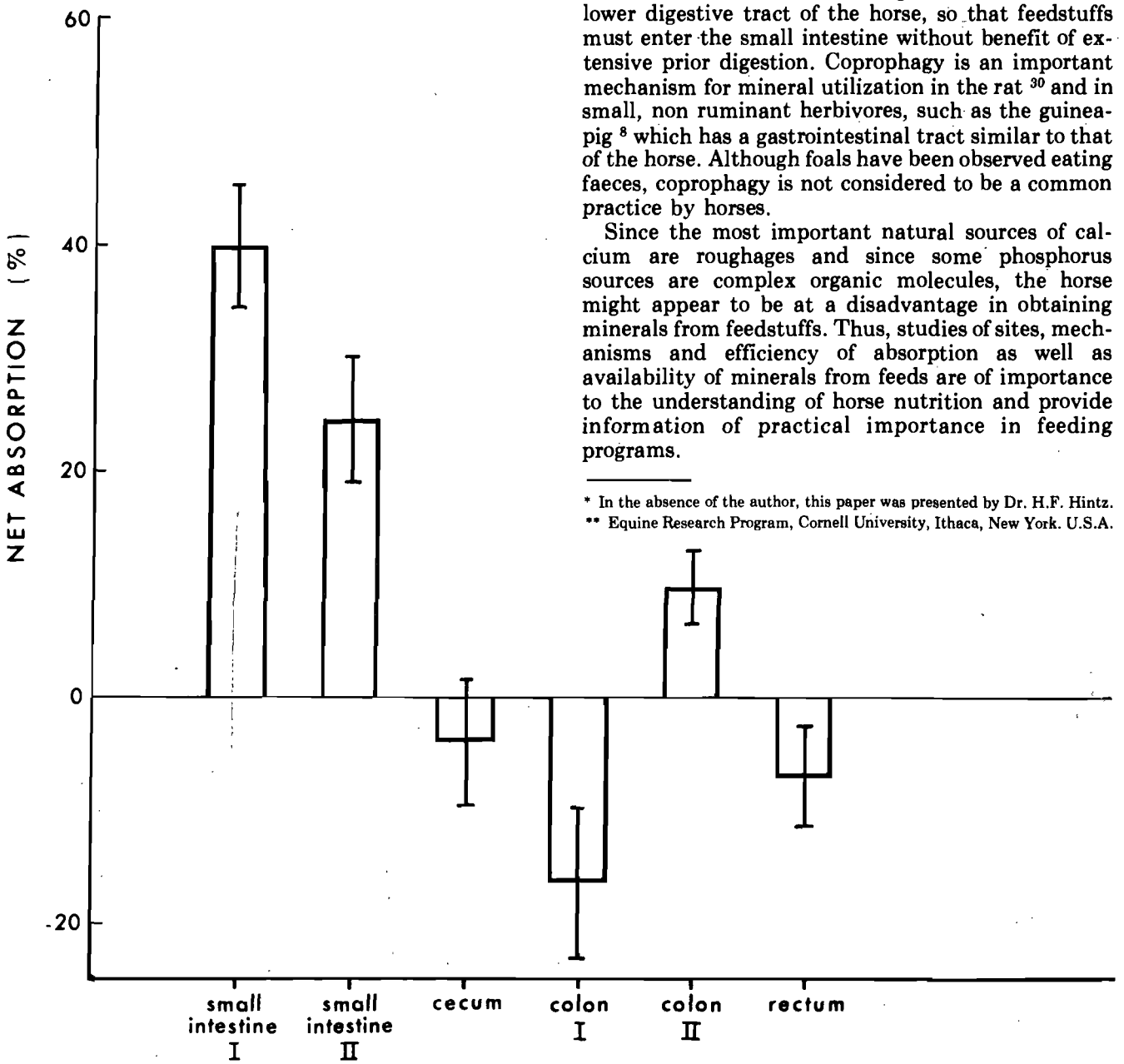

Figure 1: Net fractional absorption of Ca from the first (I) and second (II) half of the small intestine, caecum, ventral (I), dorsal (II) and small colon of horses. Standard errors shown. Forty percent of the Ca entering the first half of the small intestine was absorbed while 25 per cent of the remaining Ca was absorbed by the second half of the small bowel. An amount nearly equal to 20 per cent of the Ca entering the ventral colon was excreted in this site which may be an important source of endogenous faecal Ca.

* In the absence of the author, this paper was presented by Dr. H.F. Hintz.

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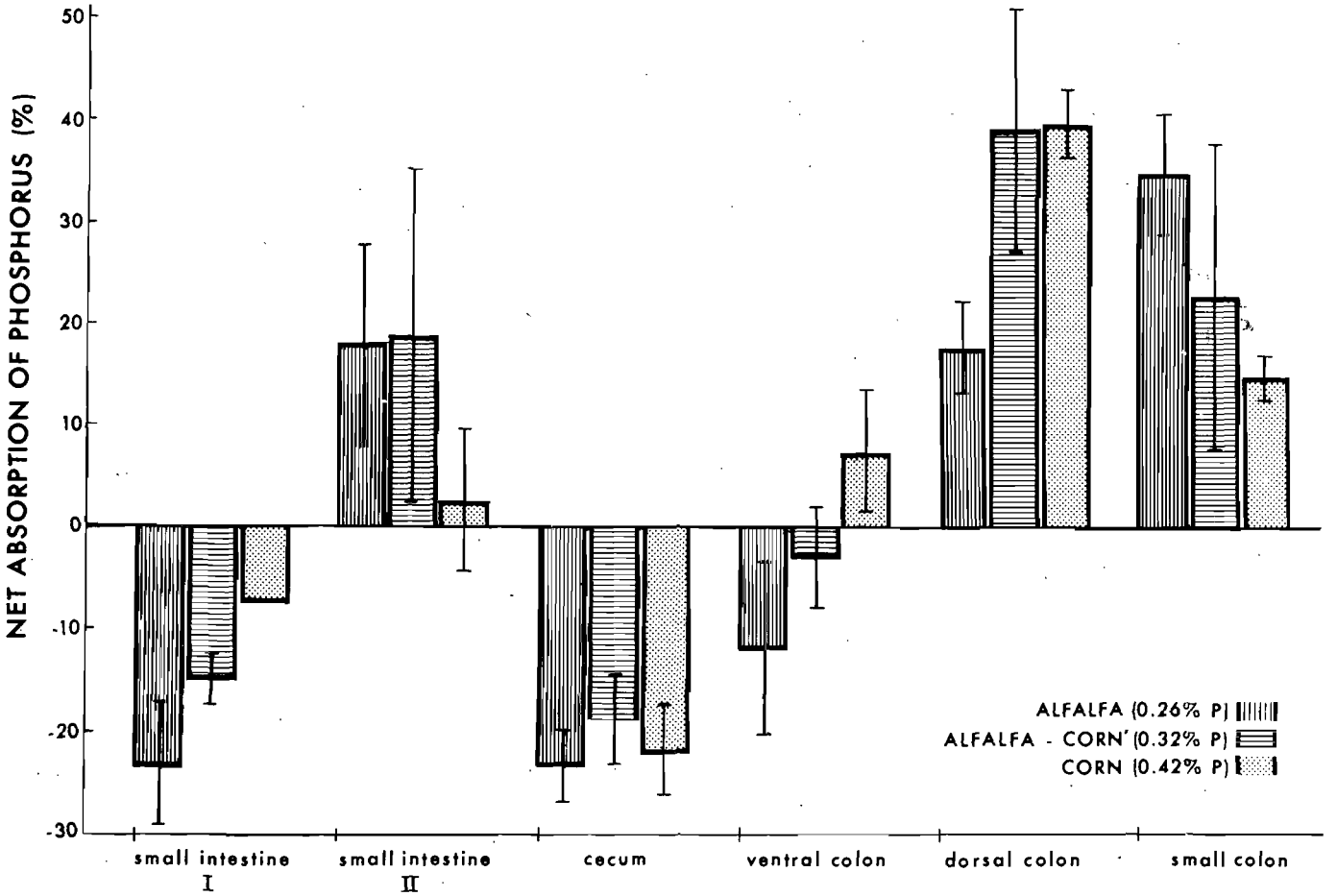

Figure 2: Net fractional absorption of P from various regions of the intestine of ponies fed an alfalfa, alfalfa-corn or high corn diet (II). Standard errors shown. Net P secretion occurred in the upper small intestine and caecum, net absorption in the lower small intestine, dorsal and small colon. The type of diet had little effect on site of absorption.

SITES OF ABSORPTION

Studies using several techniques have shown that the upper half of the small intestine of the horse has the greatest Ca absorptive potential and is the main effective site of Ca absorption [24]. Very little Ca is absorbed from the large intestine under normal circumstances (Fig 1).

Phosphorus is absorbed from both the small and large intestine. On the other hand, a large amount of phosphate is secreted into the caecum and ventral colon [1 2 28], and buffers the organic acids that are synthesized in the large intestine [3 9]. Much of the secreted phosphate is reabsorbed from the dorsal colon and small colon (Fig 2) which appear to be the major effective sites of P absorption [28].

MECHANISM OF ABSORPTION

Ca is absorbed from the small intestine by passive or facilitated diffusion and by active transport [31]. The relative importance of these processes has not been defined in the horse.

A calcium-binding protein (CaBP) similar to the vitamin D-dependent CaBP which actively transports Ca across the intestinal mucosa in other species [32] has been identified in the duodenum of the horse [6]. The amino acid composition of the protein is very similar to that isolated from cattle, but the protein appears to be somewhat more abundant in the equine small intestine. In addition, the Ca binding activity (presumably CaBP) of equine duodenal mucosa has been shown to be inversely related to dietary Ca intake [33]. Although Vitamin D is essential for the active transport of Ca across the intestinal wall, the requirement of the vitamin has not been adequately determined for the horse. The NAS-NRC Subcommittee on Horse Nutrition [20] suggests that horses require 6,6 IU of Vitamin D daily per kg of body mass. Vitamin D supplementation, at 10 X , 50 X and 500 X the recommended amount, increased Ca and P absorption for a short period but little effect was noted after several months [13].

At this time, there were no differences in the CaBP content of duodenum between control and supplemented ponies[6].

The mechanism for the transport of P across the intestinal mucosa in the horse has not been studied.

FACTORS AFFECTING THE EFFICIENCY OF ABSORPTION

The absorption of Ca and P is influenced by many factors, such as the absolute and relative amount of each of the minerals in the diet; vitamin D status; the presence of such dietary factors as oxalate, phytate, certain amino acids and carbohydrates; age; intensity of mineral metabolism. Few of these factors have been studied in detail in the horse.

The horse absorbs Ca and P with considerable efficiency which appears to be little affected by age. Metabolic balance studies which measured absorption by difference or by using ^{47}Ca or ^{32}P in young [10 23 26 33], mature [11 12], and in two old animals [14] indicate that horses fed diets containing adequate amounts of Ca and P absorb about half to two-

thirds of the Ca and slightly less than half of the P present from a variety of feedstuffs. Efficiency of absorption increases at lower levels of intake of Ca and decreases at higher levels [23 33]. Young horses, however, fed two or three times their Ca requirement, were found to absorb nearly 50 percent of the Ca present in the diet (Table 1). In addition, horses appear to absorb Ca more efficiently than cattle or sheep of equivalent age and fed equivalent amounts of Ca.

Table 1: COMPARISON OF CALCIUM ABSORPTION IN YOUNG HERBIVORES

Species	Age (months)	Intake mg Ca/kg	Absorbed %
Equine [23 25 33]	6–9	29	70
	6–9	240	46
	10	85	73
	10	410	55
	14	130	51
	18	63	70
	18	285	45
	24	103	68
Bovine [7 22]	6	112	41
	12	73	34
	12	270	28
Ovine [5]	6	130	37
	6	200	39

Table 2 : AVAILABILITY OF CALCIUM AND PHOSPHORUS IN SOME COMMON HORSE FEEDS AND SUPPLEMENTS*

Primary Dietary Source	Ca %	P %
Corn	–	38
Timothy hay	70	42
Alfalfa hay	77	38
Linseed meal	68	45
Milk products	77	57
Wheat bran	–	34
Limestone	67	–
Dicalcium phosphate	73	44
Bone meal	71	46
Monosodium phosphate	–	47

* In determining availability, the ingredients listed were the primary dietary source of either calcium or phosphorus in the experimental diets. The calcium and phosphorus contents of the diets were close to the requirements of the experimental animals.

Calcium and phosphorus in milk products are very available to horses and a greater proportion of the minerals present in these sources is retained and presumably used in bone formation [10]. The Ca and P availability in milk products may be due to the presence of such factors as lysine and lactose which facilitate absorption [32], but the addition of lysine to diets which are relatively poor in this amino acid did not increase calcium or phosphorus absorption.

Common inorganic Ca and P supplements such as bone meal, dicalcium phosphate, limestone and monosodium phosphate are readily utilized by horses (Table 2), contrary to suggestions that horses are unable to assimilate Ca and P from inorganic sources. These supplements are inexpensive but very effective mineral sources for the horse.

Balance experiments indicate that high levels of phosphate, either as mineral phosphate or phytate, inhibit calcium absorption [25]. These inhibitory effects may have little importance over a long period when adequate Ca is present in the diet, because the mineral composition of the whole body or bone of young horses fed such diets for one year or longer was not appreciably altered [29]. On the other hand, the effect of high P diets may be of considerable importance when the content of Ca in the diet is low or marginal [15 16 17]. High levels of dietary calcium have little effect on phosphorus absorption [23 27 28].

Little is known about the effect of gastrointestinal diseases, such as chronic diarrhoea, on mineral absorption in the horse.

CONCLUSIONS

Although the anatomy of its digestive tract might appear to impose restrictions on the ability of the horse to assimilate minerals, studies of absorption show that the horse utilizes the calcium and phosphorus of many feeds very efficiently. Studies of the mechanisms and control of Ca and P absorption in the horse should provide data of considerable biological interest and importance.

The availability of calcium and phosphorus has been estimated in experiments in which the minerals were fed at levels close to the requirement [11 12]. The calcium present in the common feeds and supplements used in North America is readily available to horses, while slightly less than half of the phosphorus present in these sources is available (Table 2). The relatively low availability of phosphorus in wheat bran and corn may be due to the large amount of phytate, an organic phosphate compound present in these feeds which may not be readily digested by horses. Ponies, supplemented with Vitamin D, utilize phytate phosphorus with slightly greater efficiency than unsupplemented ponies [12] but the reason for increased efficiency has not been determinded.

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DISCUSSION

M.A.J. Azzie: Would you elaborate on the significance of a high blood phosphate level. You mentioned the significance of a low blood phosphate level, but not a high one.

H.F. Hintz: That is a very good question. In the field we encounter some cases of high blood phosphorus and we know if we feed extremely high levels of phosphorus, we can pick it in the blood. Exactly what the homeostatic mechanisms are, I do not know. Under certain circumstances, feeding high levels of phosphorus will cause a high blood level of phosphorus. As Dr. Jeffcott mentioned, age will have an effect. There is also a circadian rhythm in phosphorus levels.

M. Roberts: What is still worrying me is the commercialization of the horse particularly the young horse. Our problem in New Zealand arises when we want them to grow fast and to look well as yearlings. Can you tell us whether calcium and phosphorus are needed in greater levels? In other words, what would be the required curve of utilisation when one increases the concentrate impact?

H.F. Hintz: Yes, this is what we were interested in this morning. If one gets a faster growth rate in these animals, they will have to have a super-diet with a lot of protein. They will need a lot of calcium and phosphorus. In weanlings and yearlings, which do not have interrupted growth and where one starts off early and gets a good growth rate right away, 0,7 to 0,8 per cent calcium of the total diet will suffice. If one is feeding grass hay, maybe 1,5 per cent calcium will be required in the grain mix, because grass hay has much less calcium.

Now, if young horses are on a limited diet, and suffer interrupted growth, one encounters problems. We started a project where we had to buy some Standardbreds and, being a University, where funds are limited, we tried to buy the cheapest Standardbreds we could find. One farmer had yearlings, which he had back in the field, which he had not looked at for six months and which we bought very cheaply. They were in a poor state of nutrition and were heavily parasitized. We brought them into the university, put them on our super-diet and we had 100 per cent contracted tendons within a short time, I think, four months. We also bought some weanlings from the same farmer at the same time. They were seven months of age and were put on the super-diet, which contained a high level of both protein and calcium, they have not had any problem yet. In interrupted growth, where they are held down a bit and then boosted, one gets compensatory growth. Requirements have to be greater and are more critical. If one is able to put the horse on a high plane of nutrition at weaning one has no problems, which is another reason why we believe in quick feeding and properly supplemented diet.

G.L. Faull: Could you tell me if any trace elements have been incriminated with the absorption of phosphorus and calcium?

H.F. Hintz: Not in the horse. We have fed high levels of iron and thereby have decreased calcium and phosphorus absorption, but the level of iron we had in the feed was so high it was unlikely to occur under normal circumstances. We have tried magnesium and extremely high levels of magnesium at that. Magnesium has an unusual effect: certain levels of magnesium will improve calcium and phosphorus absorption and certain levels will decrease it. Extremely high levels of magnesium will, in fact, increase calcium absorption, other levels will decrease it. But if one follows the N.R.C. recommendations one should not encounter interference in this respect. As far as trace minerals are concerned, it is just a matter of relative mass. Their presence in such small amounts in relation to the mass of calcium and phosphorus makes it very difficult to pick up the effect.

H.M. Williamson: I wonder if Dr. Hintz could comment on the value of alkaline phosphatase levels in the blood in the diagnosis of bone conditions in the horse, in relation to calcium and phosphorus metabolism.

H.F. Hintz: The use of alkaline phosphatase levels in our hands has been very equivocal; it comes from so many sources that we have not been able to align it to any diagnostic criteria, at least in our clinic. Age affects it. There may be certain circumstances where it could be of value. It has been used in and is still sometimes being used in studies on nutrition of dogs. As a routine, we do not use it.

GENERAL DISCUSSION ON DIGESTION

Chairman: P.A. Boyazoglu

P.A. Boyazoglu: To set the discussion going I wish to make some comments on the mineral problems encountered in some of the well-known horse-breeding areas of South Africa. Whereas this country is well endowed with many minerals, unfortunately we also have many areas where deficiencies occur and troubles arise as a result of them. Possibly the biggest single problem we have in a large area which is wellmapped today, is magnesium deficiency. When we do have these imbalances, unfortunately all our tables of requirements go by the board. We have a similar pattern regarding zinc problems and we certainly have problems in other parts of the country regarding manganese. We have picked up bone abnormalities of a very wide range and which are all of considerable financial concern to our breeders.

A.M. Merritt: I have a couple of questions I would like to direct to Dr. Roberts. We had a few suckling foals that had contracted diarrhoea and that we thought had been removed from the mare directly from the start. I wondered if you had seen any of these and if you had done any lactose tolerance tests on them. Secondly, have you any opinions about the rôle of the stomach in polysaccharide digestion in mature horses, which might compensate for the low level of amylase or some other enzymes, but in particular amylase from the pancreas?

M.C. Roberts: Firstly with regard to the possible detrimental effect of lactose on young foals, I was going to discuss this tomorrow, when it will be brought out in full detail. May we leave it till then.

With regard to the second point, the work of Professor Alexander, Dr. Hickson and D. Comlyn in collaboration has shown that salivary secretion is very, very low in alkaline amylase activity, far lower than in other species. Professor Alexander was unable to find any digestive enzyme activity in the stomach of horses when the horse is slaughtered, and the stomach is examined immediately after slaughter. Obviously, I have had a look at mucosa enzymes in the stomach of horses, again immediately after slaughter. I could determine low levels of alkaline phosphatase activity, none of the other enzymes that I was concerned with could I find in the stomach. Professor Alexander and Dr. Hickson in their paper on the "Physiology of Rumination", in the proceedings of a conference in Cambridge in 1970, virtually disregard the stomach as a centre of digestive activity in the horse. They do make this point, that pancreatic secretion determined by cannulating the pancreatic duct of the horse, is copious in volume, and that it is continously secreted over twentyfour-hour periods, but the level of enzyme activity is low, as I mentioned. I have spoken to Dr. Hickson about this and he agrees with me that this low level of activity of a continuously secreted, large volume of secretion in an animal that is continuously eating, would provide sufficient enzyme for the initial start of breakdown.

W.H.S. Bellinge: I should like to ask Dr. Roberts a question. In our part of the world (Western Australia) molasses is used as a supplementary feed, for dilution and also as a laxative. I would like his comment on this effect of molasses. Is it the association with lactose or is it a mechanical effect?

M.C. Roberts: I think basically with molasses one is going to overload the maltase enzyme system. Basically one is going to overload it mechanically, so that, as Dr. Hintz mentioned, there is an increased passage time of a liquid diet through the instestinal tract. The majority goes through to the caecum within half to three quarters of an hour. By overloading the enzyme activity throughout the intestinal tract, one is going to get some hydrolysis in the small intestine but I think a large amount of the molasses will go to the large intestine, where it is probably going to exert an osmotic effect, drawing water from the large intestine's mucosa and thus having a false laxative effect.

W.H.S. Bellinge: I have another question here, this time for Dr. Hintz. I did not quite understand the point with regard to the excess use of calcium producing a lower intake of some of trace elements like magnesium. If both calcium and phoshorus are overfed but in correct ratio, do you still get this depressive effect on the trace elements?

H.F. Hintz; We have not done that particular trial in which we have really measured trace element absorption where Ca and P are both fed at high levels. I would suspect, though, that the problem would be less. If one has a calcium to phosphorus ratio at a normal level, but both at a high plane, I think the effect on trace element absorption would probably be less than if one had a high calcium only. The high calcium load is more detrimental than high phosphorus.

H.M. Williamson: I would like to ask Prof. Hintz for his comments on the use of colloidal calcium with Vitamin D, as an injection, as the main treatment of calcium deficiency in animals.

H.F. Hintz: I am not familiar with colloidal calcium or what the particular product may be. There is certainly nothing wrong with using calcium gluconate. I do not see how the animal can benefit by injecting Vitamin D. If one gets it into the blood stream, it is probably alright. I would recommend Vitamin D to bring about increase in absorption rate from the intestine. I know Dr. Adams in Colorado feels that epiphysitis, for example, is a problem of calcium deficiency. Whenever he uses calcium carbonate, he always gives Vitamin D with it. He claims that he can solve the problem that way.

J.D. Steel: I do not have a specific question to ask, but Dr. Hintz alluded to a conversation which we had earlier today and I would like to make a few comments. About twenty-two years ago there began a revolution in the feeding and in training and racing of horses in Australia. The person I think primarily responsible was T.J. Smith. He has been ably assisted in developing this revolution by Mr. Sykes. One could say that since Mr. Smith and Mr. Cummings became the winners of more than a million dollars in stake money last year, that their success was largely related to the measures they had taken. These consisted in feeding fairly small quantities, which would vary, I would say, for individual constituents from about 150g to 500g a day, of feeds added to the diet. All the feeds I shall be mentioning are not in every diet: there will be two or three of these in the diets that are commonly being fed by a large number of trainers in Australia. They are: milo or grain sorghum, cow peas, New Zealand Tickbeans, soy beans and malt. We went through the milk powder phase in diets. I think it is still being used quite extensively in weanlings and up till about the same time the animals go to the yearling sales. As far as horses in training are concerned, it seems to have been substituted by cottonseed meal. There is a lot of cotton-seed meal being fed and then, of course, there are things like sunflower seeds and safflower seed, also being fed quite extensively. These practices used quite frequently by a number of our trainers are at least partially explained, on a scientific basis by some of the work which Dr. Roberts and Dr. Hintz spoke about. In Australia for a long time it has been quite difficult to get trainers to push the calcium intake up and up. They will only go up to a certain level; they do it quite empirically.

M. Griesel: I would like Dr. Hintz's comments on the following statement. Horse rations in South Africa are condemned if they do not contain oats. We have always held the view that it is a matter of supplying nutrient elements and not specific raw materials. Oats is becoming very difficult to obtain and I would like some comments from Dr. Hintz.

Secondly, he has indicated to us that phytic acid phosphorus utilization by horses is somewhere in between that of a chicken and that of the ruminant. In the chicken, we normally work on a ratio of roughly 50/50 inorganic to an organic phosphorus. Would he care to give an opinion as to what percentage of that 0,4 per cent to 0,5 per cent requirement should be inorganic and what percentage organic.

H.F. Hintz: The first comment on supplying nutrients and not specific raw materials, I can asscribe to whole-heartedly. There are many rations in the U.S.A., which are not providing any oats at all. Most of our horses at Cornell University are not fed oats because of economics. We raise them on corn from weaning at least 4 years of age. On the West coast a lot of barley is used, and a lot of milo is used very successfully, so we don't think there is anything magic about oats, but oats is a very safe feed. It is high in fibre and it is easier to avoid over-feeding oats than it is on any other grains. So, particularly if one has a poor manager it is a good idea for him to be feeding oats. The problem is that most people use 'coffee cans' as a measure: there is twice as much energy in a 'coffee can' of corn as there is in a 'coffee can' of oats. This again is because of the difference in digestibility and difference in bulk weight. It is much easier to over-feed with corn than it is with oats. Corn takes a little more management. We have never been able to see this heating effect, as long as we feed it on the same energy intake basis. Because oats has more fibre in it and the fibre make for more heating effect, theoretically, if one feeds the same amount of energy in the form of oats as one would of corn, the oats should provide more heat than the corn. Many of our trainers take oats, they clip off the ends and try to get rid of a s much fibre as they can, so what they really are doing is that they are trying to get closer to corn. So we are recommending these people to buy the cheaper oats and mix it 50/50 with corn; one will have about the same energy concentration as you do with the 'jockey oats'.

The second question concerns the desirable ratio of inorganic to organic phosphorus and the supply of phytic acid phosphorus to young, growing horses. We recommend any supplement, the phosphorus being in the form of the inorganic compound. We are talking about 0,5 per cent phosphorus. Most feedstuffs would contain roughage with about 22 per cent phosphorus content; grains might contain around 0,3 per cent or 0,4 per cent, so we think enough phytic acid is supplied. We would recommend the use of inorganic phosphorus as a supplement and not any organic phophorus sources.

G. Buys: I would like to ask Dr. Hintz's opinion on the important roughage fraction in the equine diet. First of all, the quality of the roughage is important, as far as I am concerned, namely the ratio of cellulose content to that of the more indigestable lignin. Considering the limited microbial activity in the large intestine, don't you think it is better to use a soft type of hay, like timothy, or teff, as we call it, and rather avoid the harder type of lignified fibre, as found in lucerne or even wheat straw? Is it not better to use the last-mentioned for roughage when it is in a younger stage and where one does not have this degree of lignification?

The second question: I would like your opinion on whether the ingredients of a cube must be coarse or fine, considering the rôle of fatty acid production. I think you have indicated earlier that you prefer the glucuronic acid type of fermentation, because it is more efficient, it is glycolytic.

H.F. Hintz: Concerning hay quality, there is no doubt that the stage of maturity will greatly influence the utilization of hay by the horse. One can get as much as a 1 per cent decrease in utilization per day by delaying cutting in certain fast hays. By harvesting the plant a later stage the age of the plant is going to cause a greatly decreased Fibre digestion. One can get the same effect with timothy, but because in general it has a low digestibility to start with, the difference is not pronounced. There is a very big difference between early cut alfalfa and late-cut alfalfa. The early-cut alfalfa contains a much lower level of cellulose and a much lower level of lignin, than the early cut timothy. We see nothing at all wrong with feeding straight alfalfa. We have not had any problems with that. If one takes alfalfa at a comparable stage of maturing as that of timothy, the horse does a better job of digesting the cell walls in the alfalfa. The second question concerned the size of pellets. We have not done any work on particle size in pellets. The pellets that we feed are all finely ground. We found it much easier that way; we do not have any pellets that are coarsely ground, so I cannot say if there is a difference because of particle size. But I have an idea there would not be too much difference, because the time one gets to processing the pellets, I think one is going to have a pretty uniform product.

P. Boyazoglu: I think it is relevant to mention fermentation residues here, with the back to front type of animal Prof. de Boom described to us earlier in the day in which fermentation comes right at the end and with the broadening spectrum of the types of ingredients that we are using in horse diets. We started a project whereby we tried to assist the digestive processes by the introduction of several fermentation residues in small volume. These fermentation residues were obtained from various different sources, including residues in the formation of antibiotics. The initial results at this stage are most encouraging, certainly with regard to the utilization of protein and energy. Whether this is having any effect on the mineral side of nutrition, we have no idea at this stage.

M.A.J. Azzie: There is one important question that always comes up when considering cubes in feeding. I think each and everyone of us is quite satisfied that the nutritional level attained by the animal on cubes is fine; there are no problems involved in that. But what seems to be our two major problems are: firstly, that the type of colic which one sees now is a gastric colic which in many cases ends up with rupture of the stomach; secondly, as this type of product is being used more consistently, one encounters considerably more cases of lameness associated with bone changes of the knees as a straightforward carpitis, in some cases also affecting the fetlock region. I would like to know the impression of members of the panel and whether they have made similar associations with the product?

H.F. Hintz: It is rather difficult to draw comparisons. What you call cubes, we call pellets and what we call cubes in the States are about 3" long by 2". We feed a lot of alfalfa hay cubes too, now.

As far as the incidence of colic is concerned, in our clinic, where we have been feeding pellets over the last four years, we have not found any increase in colic but we have encountered many other problems. We had to cover, line or replace everything in the stables with metal as the horses were chewing a lot of wood. They definitely need a roughage factor and we recommend feeding some form of hay to satisfy their appetites even when feeding a complete diet in pellet form. Perhaps Dr. Coffman and Dr. Merritt would care to make some comments.

J.R. Coffman: I was interested in Dr. Azzie's remarks. We are in the centre of the U.S.A., in the middle of the Quarter Horse area. The Quarter Horse industry has moved away from feeding oats and has gone in for feeding cube-sweet feed combinations consisting of oats, milo, cracked corn, regular alfalfa powder and a good deal of molasses. There has been a terrific increase of gastric dilatation. Some of the better horses of the breed have been lost from rupture of the stomach. Pellets do seem to be related to this. According to my impression the incidence of gastric dilatation in relation to a pelleted diet in the U.S.A. is purely a matter of quality control. When using certain products, such as Purina, one does not see this problem but when feeding many of the other pelleted rations gastric dilatation is one of the big hazards.

A.M. Merritt: We are feeding probably as many pellets as they do in the Mid-West. I would like to propose an hypothesis as to the possible pathogenesis of the problem. It is well recognized that both in dogs and cattle, those on a high sugar, and cattle on deep-feeding and a high grain diet, we see an increased incidence of dilatation and torsion in what would be the stomach. We are now in the process of looking at the fermentation products, namely the volatile fatty acids in ruminants and their effects on gastric motility in terms of the electrical events and the mechanical events of gastric atony. We can certainly find, as did Fencham and his Cornell group, that gastric atony can be produced. The mechanism whereby this occurs is still under investigation. We have some ideas that seem to answer my questions about rapid fermentation. Certainly the forestomach is not sterile and the problem is to relate the bacterial fermentation that goes on in there with some specific activity associated with the pellets. They are finely ground products and one could well conceive a process that, with hay and oats will not go on quite as rapidly.

C.V.H. Allin: In our practice in South Africa we certainly have had the suspicion that cube feeding causes gastric tympany, torsion and rupture in both dogs and horses.

The question I have concerns the assertion that has been made that as result of cube feeding on stud farms there has been a higher incidence of retained meconium in foals. Are there any comments on this?

K.J. Barty: I cannot comment speciffically on cubes but most of us in Australia do feel that the consistency of the meconium in the foal is associated with the diet of the mare during perhaps the last five or six months of pregnancy. In marginal areas we have hard weather and have to feed a lot of hay. If we do, we have a lot of retained meconium. The consistency of the individual product may have some effect.

P.A. Boyazoglu: Would this be a problem of the pellet or of the associated roughage, or the long hay, or the absence of green feed? You seem to imply that you then have no green feed at all.

K.J. Barty: I believe it is associated with the dry hay or with dry products. Cubes as a dry feed would probably have the same effect.

J.R. Holmes: I would like, to ask the digestive physiologists a question. Many foals develop a diarrhoea about the tenth day of age. This coincides with the time that the mare comes into season. One can imagine that by the tenth day, the foal having to battle against all the organisms in its environment, the relationship to the mare's oestrus is purely fortuitous. On the other hand, there could be factors in the mare's milk at the time of oestrus which contribute to the diarrhoea. Could the panel comment?

H.F. Hintz: Many people have been looking for something in mare's milk but without success. We tried several years in vain. We took milk from mares at ten days after foaling and fed it to other foals: we had no problem with it. I believe it must be something else.

B. de B. Baker: A question on palatibility of cubes I would like to ask of anybody here. Dr. van der Merwe mentioned that horses tend to regard cubes as 'pudding', I think his words were, *i.e.*, appetizing. I would say this is not so in our experience. If horses are on cubes and become ill for any reason, there is one thing to get them to eat and that is: put them back on to oats or any normal, unprocessed feed, and they will clean the manger up. I would welcome comment.

H.F. Hintz: We have made the same observations. If one has a horse that is ill, give it a coarse quality, rough-cut hay: it is the best thing to get it back on to feed. As far as palatability is concerned, I think it was what Dr. Coffman was talking about: pellets per se are not important; what is in the pellet is what counts. There are people in the States trying to duplicate the size of oats in pellets, thinking probably that the horse keeps separating the oats from the pellets. But I do agree that when a horse is off feed, the best thing to do is to give it some hay to get him back on feed.

P.A. Boyazoglu: We keep on thinking of a pellet as a uniform thing. The potential for variability is so large, in terms of chemical and physical nature, availability, the lot; the range of variation so wide of results obtained, that we should try to avoid speaking about a pellet merely as a pellet.

I do not think Dr. Azzie's second question has been answered, namely the possibility of a correlation between the broad spectrum of bone problems we are getting in our horses here and these formulations.

We have been looking into many problems in this country to try and determine whether there is a genetic or dietetic association. One is the club foot, loosely ascribed to contractedtendons. We like to believe there is no such thing now: the contracted tendon is a secondary complication and not a primary cause. The other is the question of 'open knees', which is a very serious problem that challenges us. Many breeders believe that, if of a mild nature, the animal will grow out of it. Whether this is a good enough reason to forget about it I am not so sure. We are having trouble and comments and opinions are called for regarding any of these conditions which are with us and undoubtedly will be with us for a long time.

C.J. Roberts: We have all been talking about the Ca : P ratio but no one has proposed an actual diet. I want to tell you what happened to me as a breeder. We were told to feed grain to horses; well, it was partly right and partly wrong. Young Charlie – you see that thick-head here to-day – got busy breeding a few horses. All he thought he had to do was feed the horses grain: in New Zealand we have plenty of grass. Daily he chucked some oats into the paddock. By springtime the grass improved and lo! and behold, he had the loveliest crop of epiphysitis he had ever seen.

Hamilton has done some interesting work lately on the Ca levels in grass and has found an increase in Ca in the spring, coindiding with the incidence of epiphysitis, the ratio of Ca to P rising to 3 : 1, whereas during winter and the rest of the year these minerals were present in equal parts. When we were heavily concentrating on feeding horses, only then did we see epiphysitis; we nearly went out of breeding. By feeding bone meal (2 parts) and disodium phosphate (1 part), starting with a level dessertspoonful at weaning and increasing it to a well-heaped dessertspoonful per horse per day at the time of the yearling sales, the problem of epiphysitis was overcome.

As far as open knees are concerned, I think it is entirely an hereditary factor. It only affects the individual and I do not think these open knees are important.

A. Littlejohn: Would Dr. Hintz care to comment on the significance of the saccus caecus of the stomach in relation to the problem of gastric dilatation? We know that it is significant in bacterial fermentation, that takes place in the *saccus caecus* of the stomach, so that in fact the horse has a fermentation chamber at each end of its gastrointestinal tract. This may provide the key to the problem of colic when feeding shall I say 'plastic' rations.

H.F. Hintz: May I first comment on Dr. Roberts' formula. It is very close to what we recommend for epiphysitis: we use dicalcium phosphate rather than bone meal because it is much cheaper. Before doing that, we want to know what the analysis of the feed is. In Arizona and New Mexico they feed a lot of irrigated alfalfa; the phosphorus levels are very low, dropping to less than 0,2 but the alfalfa, which is very digestible, contains considerable quantities of Ca. They were getting a lot of epiphysitis. In other instances, epiphysitis occurred where the diet was low in Ca and high in P. In that case the low Ca was the primary cause. Consequently one has to look at the food analysis before going too far either way.

As regards the question of the rôle of the saccus caecus, I do not really know much about fermentation in the somach. Dr. Alexander pointed out many years ago that much lactic acid is released there. I hope that answers the question.

M. Roberts: May I comment on what Dr. Hintz has said? Certainly Dr. Alexander found that there was no VFA production in the stomach of the horse, but he did find considerable lactic acid production there. He was able to isolate several different types of organisms from the normal horse's stomach. As far as I know, no further work has been done on investigating the bacterial fermentation taking place in the whole equine stomach. Let alone the saccus caecus. I am sorry I cannot really answer Prof. Littlejohn's question.

D.H.G. Irwin: We have found that when some horses develop colic as result of hoven, bloat or tympanites, the problem is not confined to the stomach. We recently had the misfortune to lose a horse and found at autopsy that the stomach and the whole small intestine were vastly distended. There was no mechanical obstruction in the large bowel, or anywhere else, to the passing of flatus, which took place in enormous quantities, together with dung. Nevertheless, the animal literally blew up so rapidly that it died before any treatment could be appplied. The small intestine smelled exactly like what is known in South Africa as 'kaffir beer' (fermented sorghum). I am not sure what VFA was involved. It was a shame that no material was collected, as it could have helped to elucidate the nature of the fermentation. I have an idea that particle size played an important rôle. In the entire gastrointestinal tract, from stomach to rectum, there was no particulate matter that one could recognize as long feed, only a small amount of short feed in fine form. Perhaps the surface area of the particulate ingesta and the presence of some organism has something to do with the rapid fermentation.

As regards calcium metabolism of horses in relation to the causation of bone disease, particularly of the lower limb, I had often hoped that somebody would expatiate upon the rôle of water in terms of its mineral content. Perhaps at some later stage in the Conference someone might have a few words of explanation for us.

J.G. Boswell: During a conversation* yesterday one or two people were very anxious to have opinions from practitioners and scientists as to the interval between feeding a race-horse and the time that horse will be running. If he were to run in a big race, say at 3 o'clock in the afternoon, when should we give him his last feed? Should we increase it, should we decrease it, or starve him? It would be very interesting to have a few opinions on this.

J.D. Steel: It is very common in Australia to either not feed at all on race days, *i.e.*, there would be no morning feed or a very small morning feed if the horse were going to race in the afternoon. I know that some of my New Zealand colleagues have done various kinds of experiments with this. There is one veterinarian that I know, who at one stage, at anv rate, was practising withholding whole food for twenty-four hours prior to racing with the idea of emptying the alimentary canal and lessening the weight the animal had to carry.

W.H.S. Bellinge: I would like to repeat Dr. Boswell's questions: how much and when should a horse be fed before a race? The equine nutritionists and physiologists should be able to answer a simple question of this nature. I would like to ask Dr. Roberts if it would be a logical form of management to give a horse, instead of his breakfast, a decent dollop of glucose on the day of a race. If so, at what time would this be logical? And what should the amount be?

M. Roberts: I have no experience of this. I think it is a most interesting idea. Most certainly it could be done. As regards to what dose and what time: I think one would just have to do it on a traial and error basis and perhaps beforehand run a series of horses, whereby you give one or two grams per kilo and measure the blood glucose level after two hours. I think one would have to give it in the morning if one were not going to incur possible disturbances in motility in the gut of the horse. It is an interesting idea, I have no experience of it, but it is certainly something that could be tried.

* The ensuing part of this discussion, although held on the following day, has been transposed to fall under this heading on account of its relevancy to the preceding part.

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SECOND SESSION : FOAL DISORDERS

Chairman: R. ROUS

HISTOLOGICAL DEVELOPMENT OF THE THYMIC AND INTESTINAL LYMPHOID TISSUE OF THE HORSE

C.D. MACKENZIE*

SUMMARY

The basic components of the immune system, and the defence mechanisms in the gastrointestinal tract, are briefly reviewed. Histological studies in 84 equine foetuses showed that lymphoid cells begin populating the thymic primordium at 11-12 weeks, the mesenteric lymph nodes at 13 weeks, the spleen at 25 weeks and the intestinal *lamina propria* at 13-14 weeks' gestation. Lymphocytes were seen in the intestinal epithelium very early in gestation. Histological signs of response to antigenic stimulation were seen in five foetuses, indicating that the horse is likely to be capable of mounting an immune response *in utero*. The changing status of the foal as it becomes fully immunocompetent is discussed.

INTRODUCTION

Specific immunity is a complex defence system essential for the homeostasis of the normal animal. An understanding of the immune system, and the events taking place in the lymphoid tissues before parturition, is necessary for a subjective approach to diagnostic and prophylactic paediatric medicine. Studies in various species in recent years have shown that immunological competence begins early in an animal's development, birth being a relatively late, but nevertheless important, immunological event.

THE IMMUNE SYSTEM

Immunity is mediated in birds and mammals by two major components: active cells and immunoglobulins. The basic cells involved are the lymphocytes which undergo two separate lines of maturation before becoming either the active lymphocytes, or the plasma cells which produce immunoglobulins. This dual system is described in table 1.

Table 1: MAMMALIAN IMMUNE SYSTEM

	ANTIBODY MEDIATED	CELL MEDIATED
EFFECTOR:	Antibodies (Immunoglobulins)	Lymphocytes + their local products
CELLULAR ORIGIN:	Bonemarrow (?)-derived lymphocytes/ plasma cells	Thymus-derived lymphocytes
SITE IN PERIPHERAL LYMPHOID TISSUES:	Medullary cords	Paracortical regions
FUNCTION:	Humoral defence; *e.g.*, against encapsulated bacteria	Cellular defence reactions *e.g.*, against viral and fungal infections

The origins and differentiation pathways of these two lymphoid cell lines have been the subject of much investigation over the last decade. It is generally accepted that, in mammals, these lines are the thymus-dependent system and the bursal-equivalent or thymus-independent system[9].

In the thymus-dependent system, haemopoietic stem cells, from the yolk sac or foetal liver, enter the thymic rudiment early in foetal life[29]. Here they mature to become cells competent of mediating cellular immune reactions such as delayed allergy, graft rejection and microbial immunity. After it has left the thymus this lymphocyte population is found in many animals to reside in the paracortical regions of the body's lymph nodes and in the periarteriolar lymphocyte sheath areas of the spleen[28].

The site of maturation of the stem cells destined to become the immunoglobulin-producing cells, has been the subject of much controversy for a number of years. In birds, the bursa of Fabricius has been established as the organ involved in the processing of these cells[32]. In mammals, however, the equivalent organ is far from obvious; a number have been proposed, including Peyer's patches, the appendix in rabbits, bone marrow, and recently the foetal liver and spleen have been added to the list of possiblities. The cells of this thymus-independent system, lymphocytes and plasma cells, reside in the medullary cords of lymph nodes and in areas peripheral to the periarteriolar sheaths of the spleen[19].

It can be seen that these two lymphoid populations take separate lines of maturation (Fig. 1) but they combine in a number of ways in their defence functions[18]. The time at which the various lymphoid cell types are found in the differentiation (primary) and the peripheral (secondary), lymphoid organs of the body is an important guide in the assessment of immunocompetence of the developing animal.

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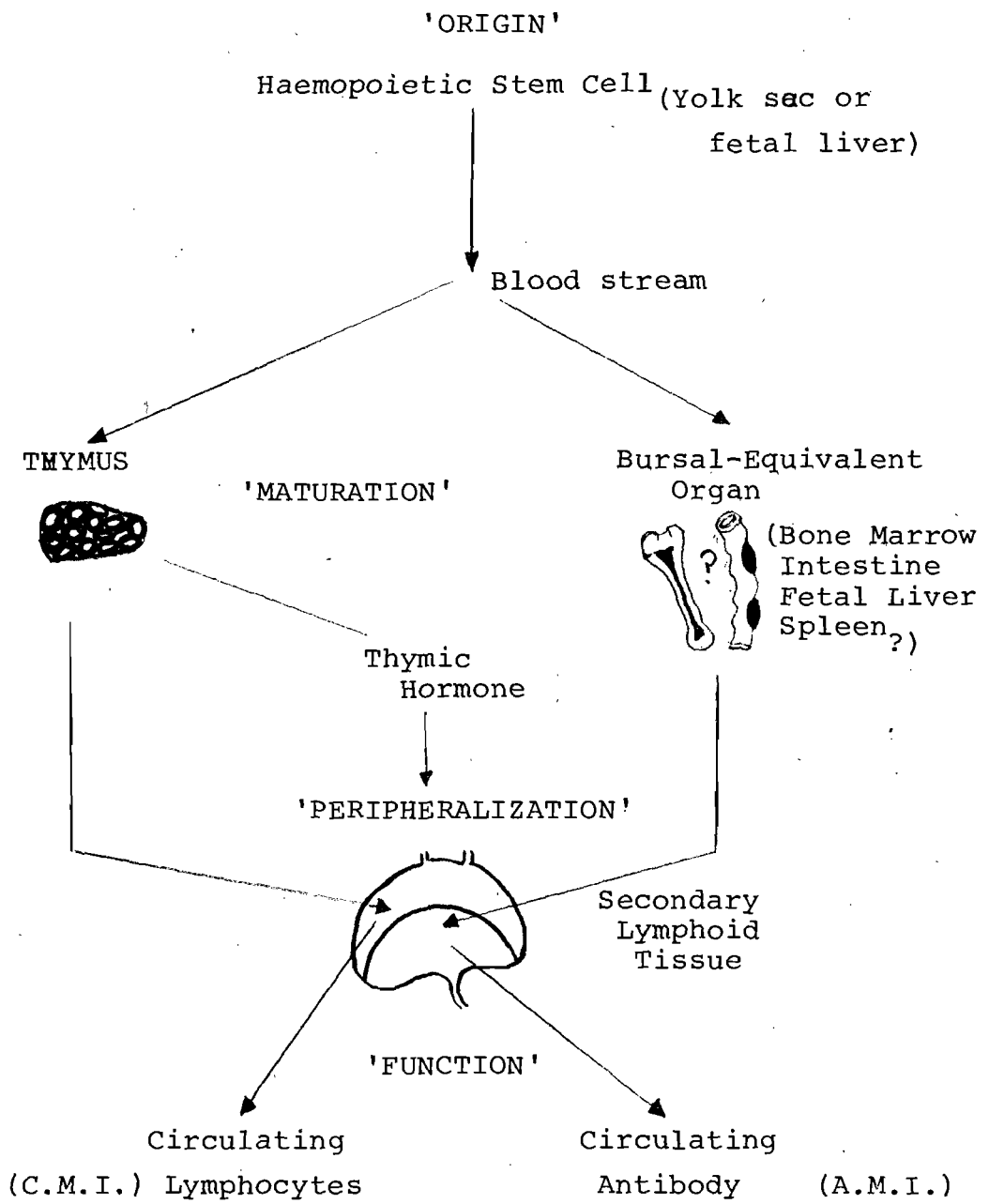

Fig. I. The mammalian lymphocyte maturation pathways.

DEVELOPMENT IN THE HORSE

Characterization of histological and functional parameters of immunocompetence in the developing horse has not been carried out to any great extent. Neither has direct evidence of the existence of thymus-dependent and -independent systems been described in this species. Nevertheless, a disease model similar to that recorded in immunodeficient children has been seen in Arab horses[46]. This condition, together with preliminary studies using *in vitro* lymphocyte techniques specific for thymus-dependent cells[20], indicate the likelihood of a similar dual cell-line system in the horse.

The histology of foetal lymphoid tissues in the horse has not been well reported in the literature. In table 2, figures 2 and 3, the findings on 84 foetuses, aged from six weeks to full term, are described. They were collected from the abattoir, their ages estimated[38] and they were then processed for histological assessment.

Table 2: MASS OF EQUINE THYMUS

AGE (weeks)	%
13	0,05
20	0,06
30	0,15
38	0,25
Full term	0,35

(% of total body mass)

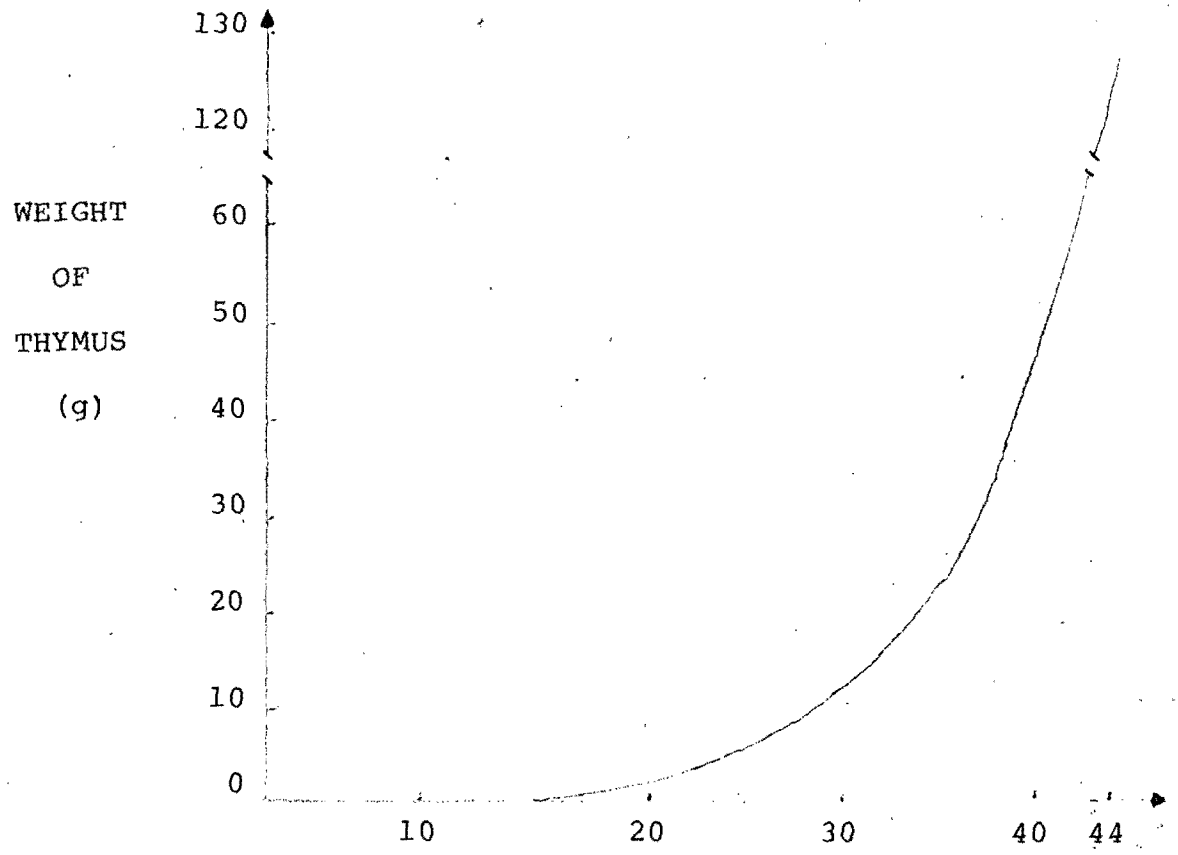

Fig. 2. Development of the equine thymus.

THYMUS, LYMPH NODES AND SPLEEN

The thymus is the organ in which lymphocytes mediating cellular immunity differentiate. It is a multiple organ in the horse, consisting of right and left lobes which continue from the cranial mediastinum into a series of lobules lying along the trachea[45]. Embryonically, the mammalian thymic primordium arises from the third and fourth pharyngeal pouches[54], and is infiltrated with lymphocyte-precursor cells

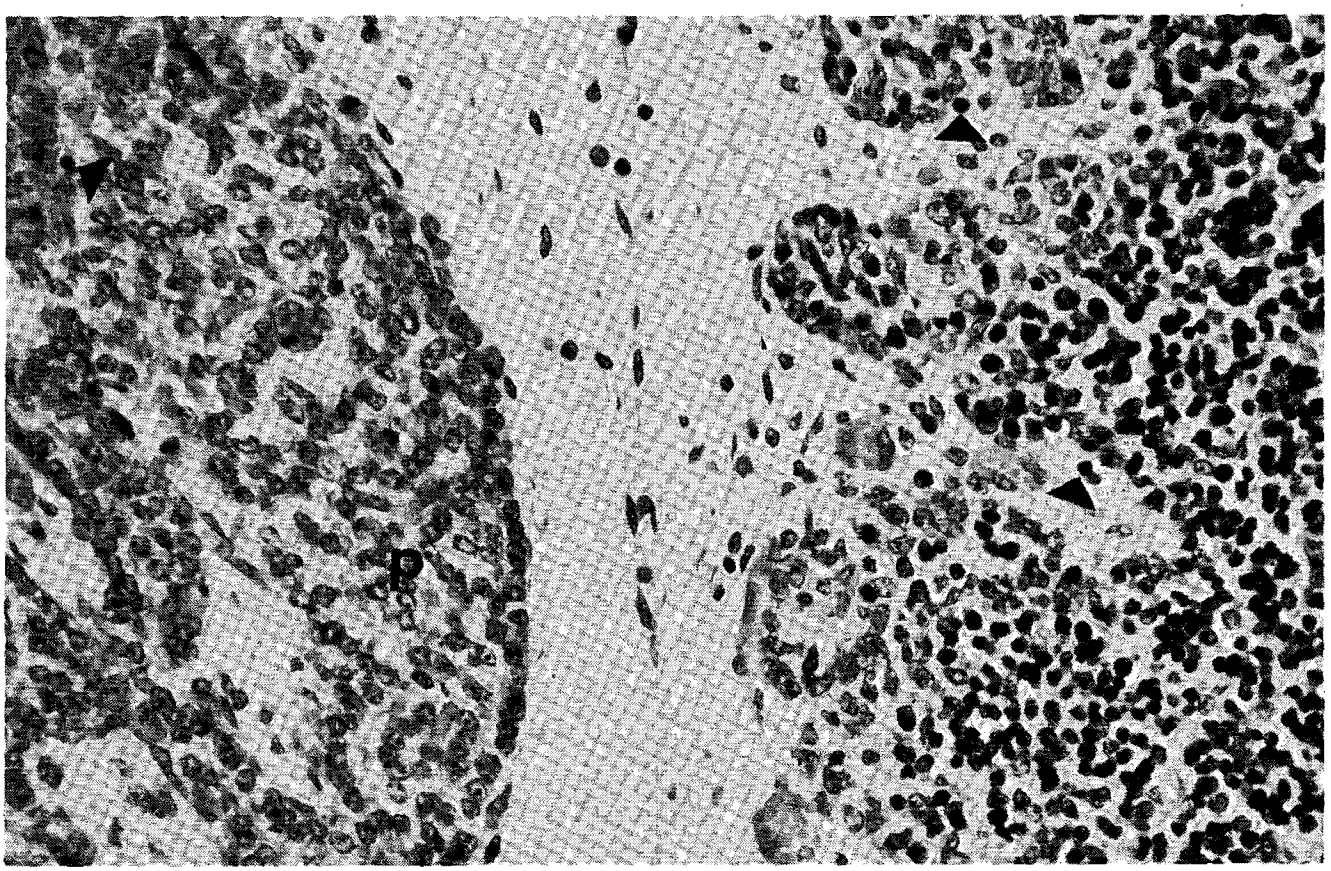

Fig. 3: Thymus of a 12-week-old horse foetus. Lymphocyte (arrow) invading the primordium (p) (x500).

from the yolk sac or foetal liver[37], although in amphibians the lymphocytes may arise from elements of thymic primordium rather than from blood-borne cells[51].

A lymphoid thymus was seen first in foetuses at 11 to 12 weeks of age. The organ increased in size and mass to reach 125 g at 44 weeks' gestation (Fig. 2), at which time it represented about 0,35 per cent of the total body mass (Table 2). The increase in thymic mass, relative to body mass in the second half of gestation, may reflect an increased requirement of thymic lymphoid cells to populate peripheral tissues as the foal prepares itself for the strong antigenic stimulation following birth.

The thymus of the 11- to 12-week foetus consisted of epithelial and mesenchymal cells with lymphocytes beginning to populate individual lobes (Fig. 3). A dense lymphoid cortex developed during the following 12 weeks, at which time the cortical area equalled approximately the less cellular medullary region.

Mature Hassal's corpuscles, medullary epithelial structures which have been implicated in antigen retention[3] and antibody localization[26], were seen in the equine thymus as early as 12 weeks' gestation. This early occurrence may reflect an essential rôle for these structures throughout the functional life of the thymus[4]. Recent *in vitro* evidence in the rabbit[23] supports the idea that epithelial structures in the medulla are responsible for the production of a thymic hormone. This hormone appears to have a possible function in the maintenance of thymic-dependent lymphocytes in the peripheral tissues[8].

A fourth cell-type was observed in the thymic tissue at all stages of its development. Eosinophils were common in the interlobular septa and in the medulla, particularly in association with Hassal's corpuscles. The appearance of mitotic cells, cells of the early myeloid series and eosinophil myelocytes, indicates that eosinophils may originate in this area. In the medulla, however, they appeared more mature, having mainly multi-lobed nuclei. The function of these cells in this particular anatomical situation is unknown[2], although it is interesting to note that circulating eosinophils appear to be thymus-dependent in certain situations in adult mice[10].

The mesenteric lymph nodes were observed macroscopically at 14 weeks as small white foci of 1 mm diameter. Histologically, lymphocytes were seen at 13 weeks in the areas that eventually became primary follicles; this is about two weeks after the lymphoid infiltration of the thymus was seen (Fig. 4). These lymph node lymphocytes generally remained in cortical and paracortical areas until about the 33rd to 35th week, when the medullary cords developed. The appearance of these cells in the medullary region seems to be related to apparent antigenic stimulation of these foetuses, indicated by the presence of large basophilic lymphoblast cells. Germinal centres, structures which develop from primary follicles after antigenic stimulation[31], were seen in foetuses from 40 weeks until birth, and, in occasional instances, slightly earlier. Granulopoiesis was observed in the medullary cords of some foetuses in late gestation. A few mature eosinophils, at varying times in their development, were seen in the paracortical areas of the nodes.

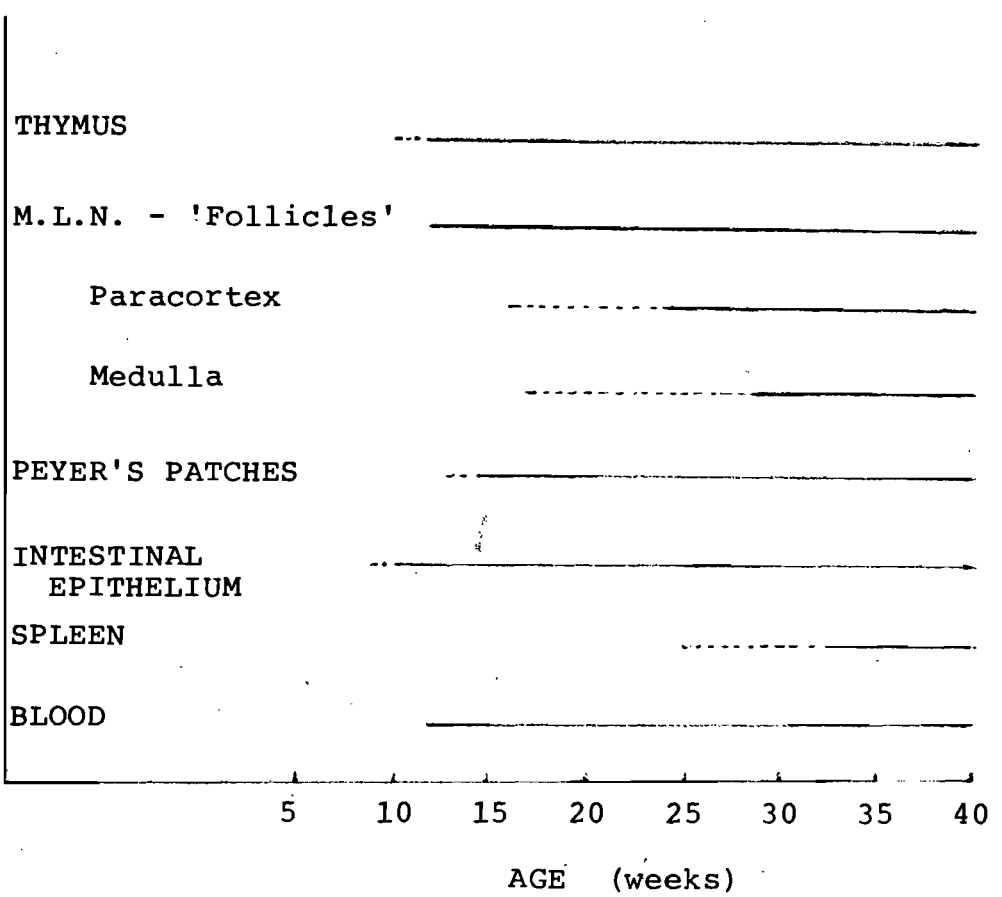

Fig. 4: Invasion of equine foetal tissues by lymphoid cells.

The spleen, a major site of antibody production in the mature animal, did not receive a substantial lymphoid component until the 32nd week, with cellular infiltration of the periarteriolar regions beginning about the 25th week of gestation. Figure 4 shows that this was the last type of lymphoid tissue to begin development. This differs slightly from cattle[41] and dog[7], in which the splenic lymphoid tissues emerge before, or at the same time as, the major lymph nodes. Haemopoietic cells were seen in the spleen at 12 weeks, whereas the liver had haemopoietic foci under the capsule and in the cords from the earliest stage up to the 29th or 30th week.

The earlier blood lymphocytes seen in this series were in foetuses of 12 to 13 weeks. These were both small- and medium-sized cells, presumably circulating to populate the various lymphoid tissues.

THE GASTROINTESTINAL DEFENCE SYSTEM

Various cellular and humoral forms of defence are involved in protecting the animal from invasion by the many antigens that pass through the gastrointestinal tract.

If antigens cross the first defence barrier provided by the intact epithelium, they may be nullified in the *lamina propria* by Immunoglobulin A (IgA), or IgM, molecules originating from the local plasma cells. If IgA itself passes from the villi into the gut lumen, it picks up a protective fragment called 'secretory piece', from the epithelial cells. This additional structure renders the IgA molecule more resistant to degradation in the luminal environment[50], where it can neutralize antigens before they pass through the intestinal epithelium. Other cells involved in gut defence found in the *lamina propria* include macrophages, lymphocytes, eosinophils, and mast cells (Fig. 5).

The population of lymphocytes residing in the epithelial cell layer itself has received attention in recent years. These cells do not appear to be extruded into the lumen but re-enter the *lamina propria* after remaining for a varying period of time in the epithelium. It is likely that they have functional significance[14].

The rôle of eosinophils in the gut probably has an immunoligical basis. Eosinophils are known to have an affinity for, and are capable of phagocytizing, antibody-antigen complexes[15]. They are commonly seen in allergic and parasitic reactions. The histamine-containing cells (mast cells) have been reported to be intimately involved in immunity to parasites. Their number in the *lamina propria* of helminth-infested tracts increases up to the time of expulsion of the parasites[27]. Increased levels of antibody are found in the lumen at this particular time, and the rôle of mast cells in this situation may be that of increasing the mucosal permeability of these immunoglobins[30].

The lymphoid aggregates of the *lamina propria,* the Peyer's patches, respond mainly to gastrointestinal antigens by producing secretory and circulating antibody[34,39], particularly in the secondary immune response[53]. Their origin and basic function, however, have been a controversial topic for a number of years. The weight of evidence at present favours the concept that they function mainly as local lymph nodes, rather than as sole primary bursal-equivalent organs, although the possibility of a partial role in this respect cannot be ruled out a present[12].

The intestinal epithelium immediately above the Peyer's patches has been studied and shown to differ from the remainder of the gut lining in that it has specialized ingestive ability[5]. This property, and the observation that the epithelial lymphocytes in these areas have a longer period of residence than elsewhere in the jejunum[21], may indicate that this particular site has a rôle in antigen uptake from the gut.

Systemic antibody produced elsewhere in the body is the last line of defence against enteric antigens. Excessive IgG antibody in chronic inflammatory bowel conditions, however, can become detrimental rather than protective. Antibody-antigen complexes of IgG

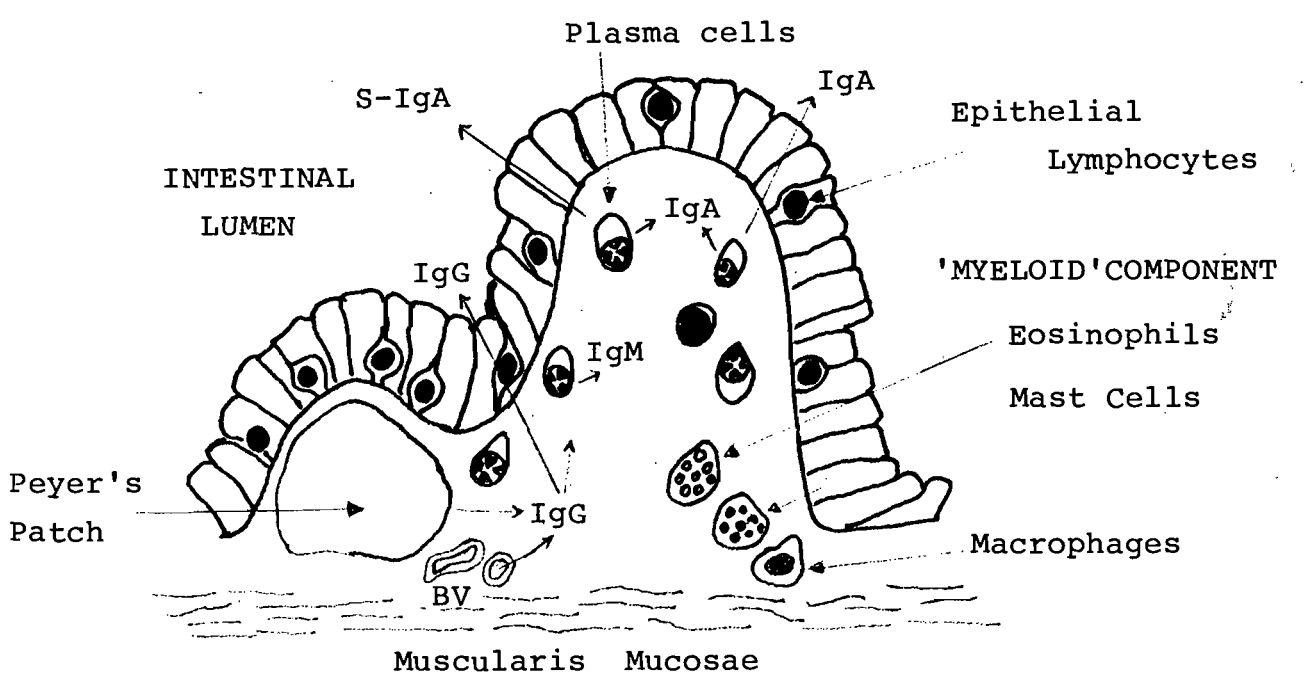

Fig. 5: The immune system of the intestine.

type in this situation, can produce progressive Arthus-type reaction[6], aggravating the initial condition considerably.

An essential rôle of the liver in the absorption of enteric antigens has been suggested by Thomas[49]. Antigens reaching the portal venous system may be combined with antibody entering from the spleen, assisting their removal from the blood stream by the phagocytic cells of the liver.

INTESTINAL LYMPHOID DEVELOPMENT IN THE HORSE

Aggregations of lymphocytes close to the basement membrane (Fig. 6) were seen first in the jejunum at 13 to 14 weeks of gestation. They remained small and elongated until 18 to 20 weeks' gestation (Fig. 7), when they began to develop follicular structures with blastic lymphoid cells accumulating in the deeper areas, and the smaller lymphocytes in the zones near the intestinal epithelium. In the second half of pregnancy, quite a few patches contained large basophilic lymphoblasts but there was no definite time sequence to the appearance of these cells. Macroscopically, these lymph nodules became visible between the 18th and 22nd week in most cases and increased to 1 cm diameter at eight months.

The time of development of these lymphoid aggregates coincides with that of the mesenteric lymph nodes; it is therefore likely that in the horse they act as lymph nodes of the *lamina propria,* rather than having any single special function.

Plasma cells were seen in the intestinal tissues only in one foetus in this series, at 34 weeks' gestation, but generally no cells of this type were present until after birth. Similarly, eosinophils and mast cells were seen infrequently in the *lamina propria* during the period *in utero.*

It is interesting to note that epithelial lymphocytes (Fig. 8) were present as early as 10 weeks. Although they were present in small numbers at this stage, their presence at this time, when the thymic primordium is being infiltrated with lymphocyte stem cells, may support the hypothesis that the intestinal epithelium is a primary lymphoid organ[13]. In later foetuses these lymphocytes were present in larger numbers in the epithelium covering the Peyer's patches.

FOETAL IMMUNOCOMPETENCE

The lymphocytes of many species are capable of forming specific antibodies, and of exhibiting many reactions specific to cell-mediated immunity early in gestation. Rodents appear to develop immunologically comparatively later than the larger mammals; nevertheless, lymphoid precursors taken from embryonic mice before the appearance of a thymic rudiment are capable of producing specific, though limited, antibody responses *in vitro*[52].

The time of first elicitation of responses to antigens appears to be directly related to the particular antigen used. This may be a reflection of the state of maturity of the reticulo-endothelial system rather than a lack of lymphocyte competence.

Direct antigenic stimulation of animal foetuses has been carried out in dogs[16], sheep[44] and cattle[41]. Coliphage T2 antigen injected into equine foetuses at 28 weeks' gestation produced a specific neutralizing antibody which did not originate from their dams' serum [24]. This is, however, the only direct evidence of

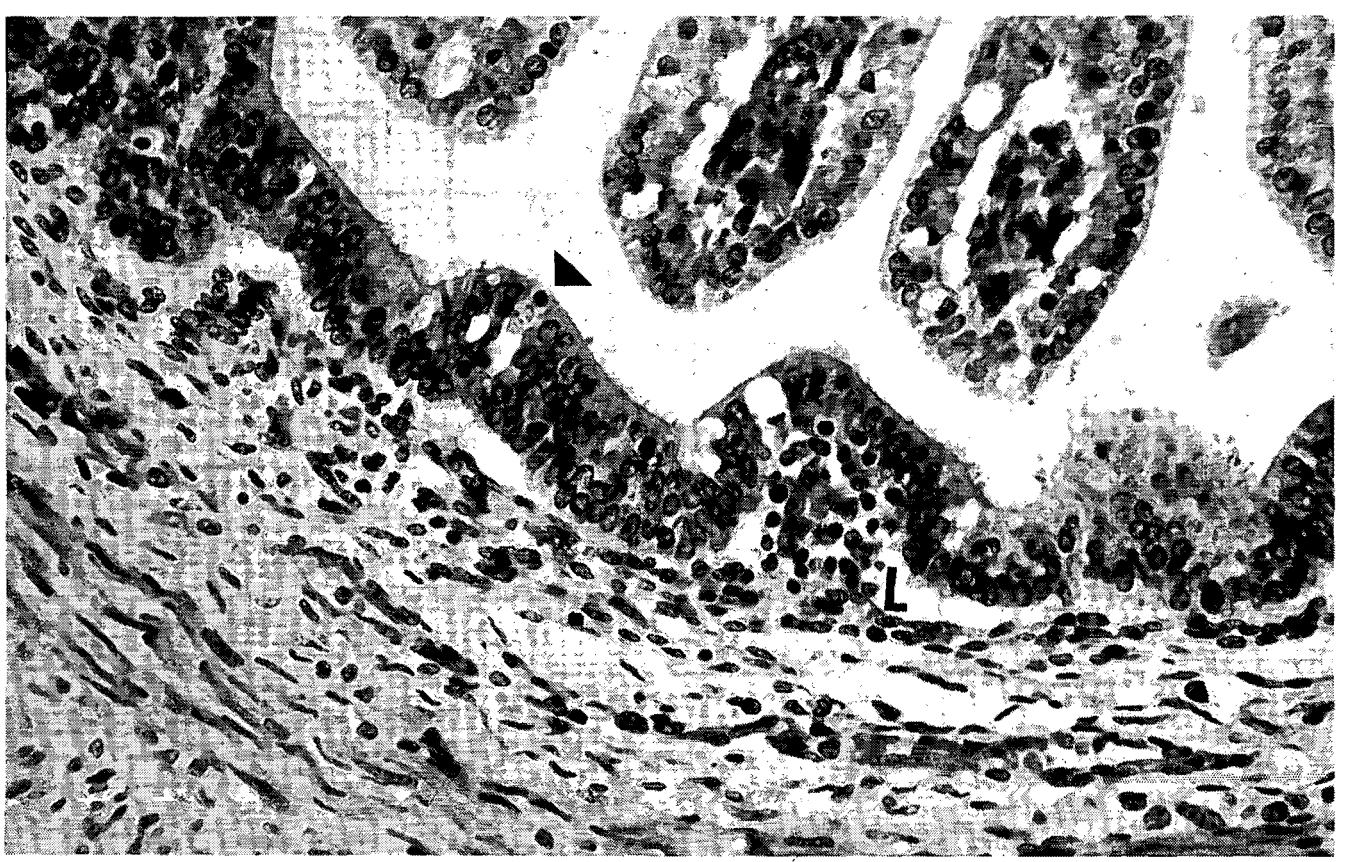

Fig. 6: Early lymphocyte aggregate (L) below gut epithelial layer in a 14-week equine foetus. Epithelial lymphocytes present (arrow) (x500).

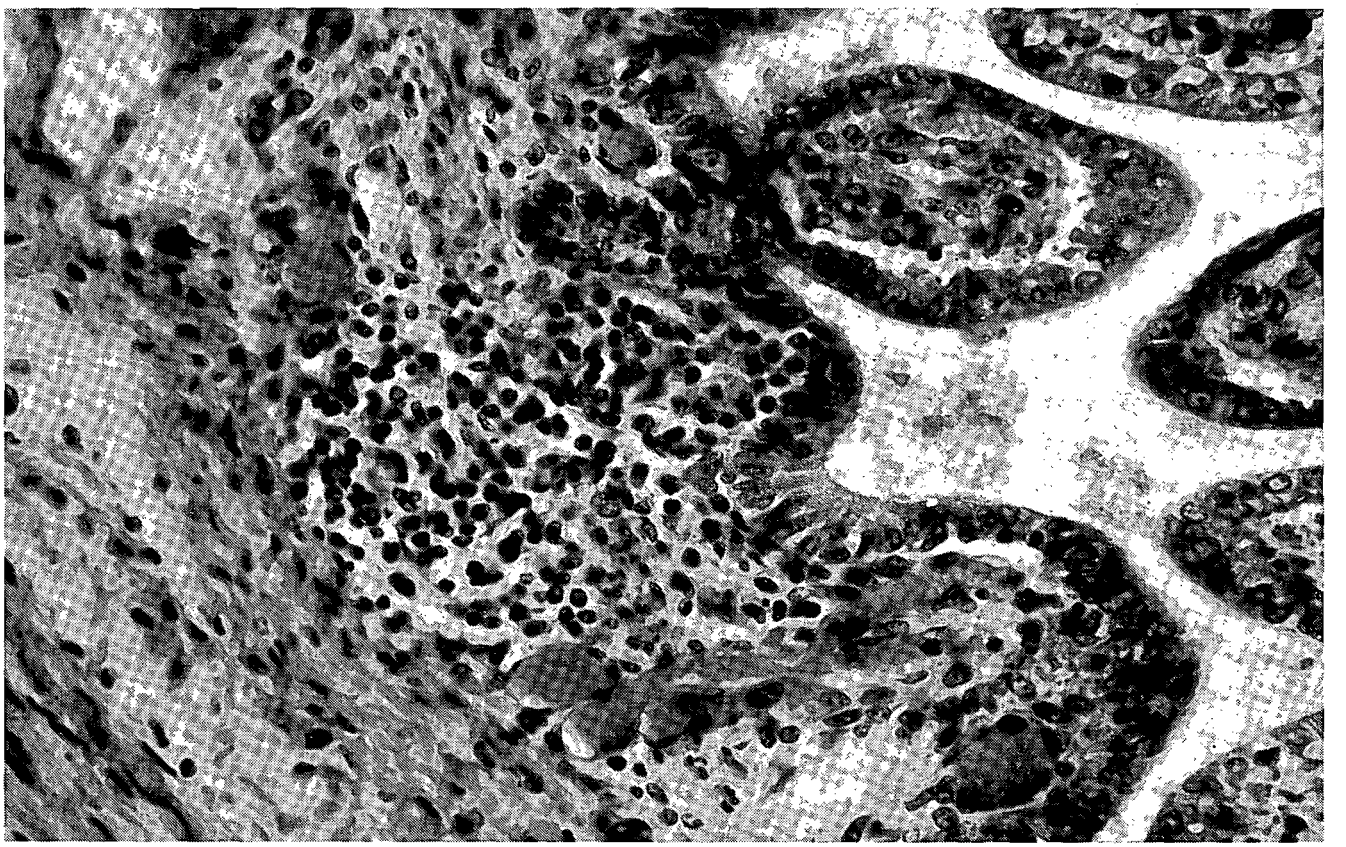

Fig. 7: Lymphocytes aggregating in an area of the *lamina propria* below the basement membrane (x500).

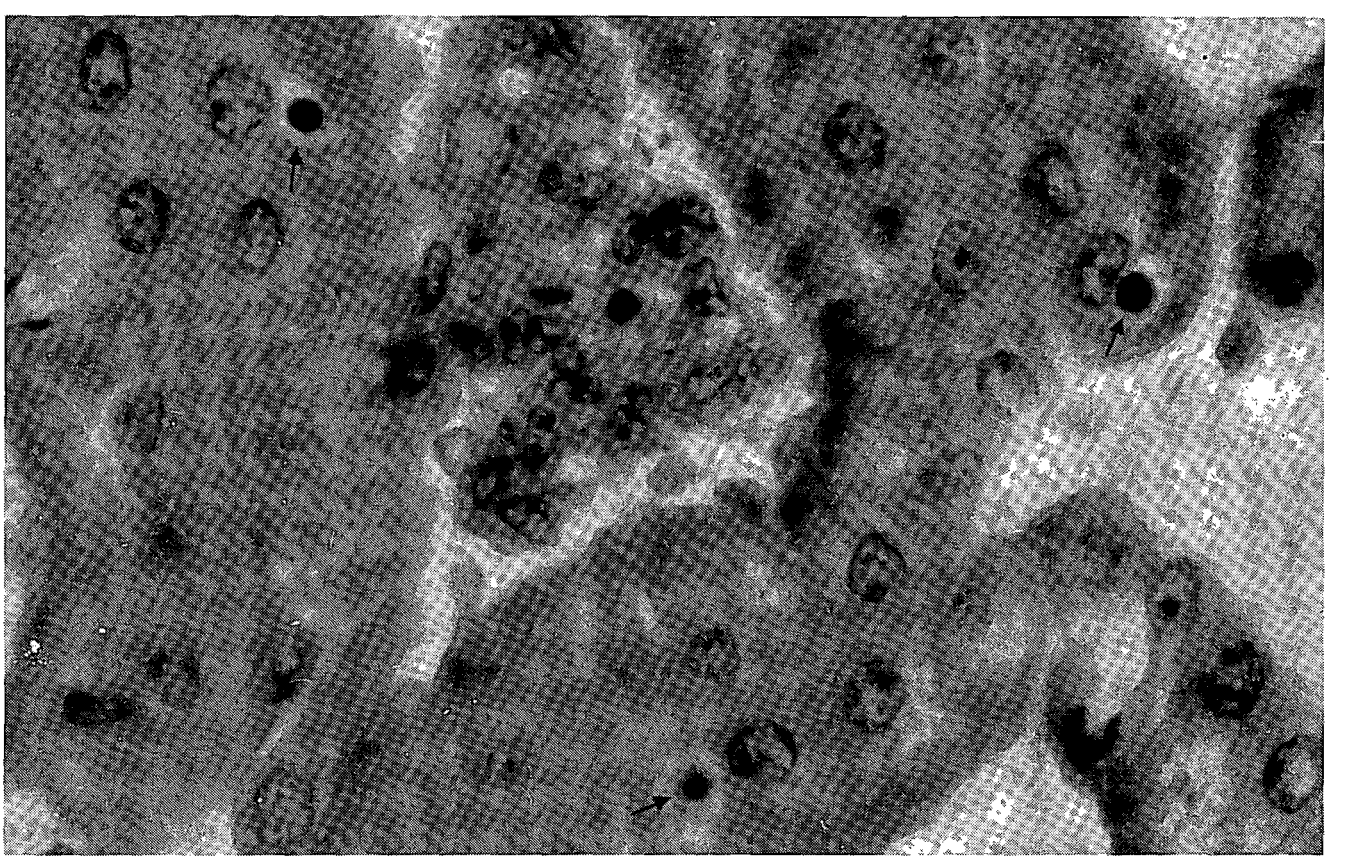

Fig. 8: Epithelial lymphocytes (arrows) above the basement membrane of the intestine of a 13-week equine fetus (x1025).

pre-natal immune function in this species. Phytohaemagglutinin-induced stimulation of foetal lymphocytes, as a cell-mediated immunity parameter [47], has not been carried out in the horse. Lazary *et al* [20], however, tested the leukocytes of 5- to 7-week-old foals and obtained no significant response. This may not correctly reflect the competence of equine foetal lymphocytes, as a reduction in lymphocyte reactivity is sometimes seen in the foetuses of other species at the time of parturition.

In the series of equine foetuses reported here, there were histological indications of antigenic stimulation *in utero*. Large basophilic lymphoblast cells were seen in lymph nodes, spleen and particularly in the Peyer's patches of five foetuses aged from 19 to 34 weeks. This type of cell is seen usually in response to antigenic stimulation[11]. Plasma cells, which were observed in a 34 week-old fetus in this series, arise similarly. It seems likely, therefore, that the horse is similar to other species in being able to respond to antigens relatively early in foetal life.

CLINICAL ASPECTS

The immunological development of the foal can be divided into three phases: *in utero*, the colostrum-protected and the fully-competent phases.

The *in utero* phase, as shown by the survey reported in this paper, is a time of anatomical development of the lymphoid tissues. This physical acquirement appears to be accompanied by the development of an ability to respond immunologically. The capability to mount a substantial response to specific antigens may begin during this phase, but probably matures in the colostrum-protected phase. Significant development of antigenic responsiveness, however, may occur with the entry of particular antigens to the uterus, such as occurs in uterine infections.

The intestinal tract as a site of antigen entry into the foetus is an interesting possibility. Ingestion of amnionic fluid by the foetus, an accepted phenomenon, may be a method of antigen entry to the gut. Petit *et al*[33] found IgM, IgG and IgA to be present in the intestines of human foetuses from 13 – 32 weeks; they interpreted their presence as a response to an intrinsic reaction, rather than to an external stimulus.

The colostrum-protected phase is a period when the foal is quantitatively increasing its already-present capability to respond to the large number of antigens that exist in the environment and now confront the foal in its new post-natal situation. The importance of colostrum in this species, where the neonate enters the world with virtually no circulating antibody, has been well documented[17]. A good colostral protection is aided by a high maternal serum level of antibody to the antigens commonly confronting the newborn foal. The successful use of the IgM-rich fraction of pooled adult serum as a colostrum substitute has been reported in calves[22]; a similar procedure may be of value in foals susceptible to neonatal disease.

An interesting function of colostral antibody has been described in the pig by Anderson who showed that eosinophilic responses were absent in colostrum-deprived animals[1]. This antibody may play an important rôle in early development of the myeloid component of the defence system.

The attainment of full immunocompetence may be influenced by a number of factors. The onset of active responses may vary with different antigens: a variation may also exist between different breeds of a single species. Schultz *et al*[42] showed a difference in the neonatal immune responses of four breeds of cattle. Indirect evidence, showing that Thoroughbred horses are slower than Shetland ponies in reaching the immunocompetent phase, was provided by Rouse[40].

The stage of development of foetal immunity is reflected in the prenates' response to uterine infection. The immunological status of the foetus may profoundly affect the pathogenicity of an infectious agent and the nature of the disease it produces[43]. Lambs, infected with *Brucella ovis* in late gestation, exhibit lesions attributed to the foetus's own now competent immune system, whereas those affected in early gestation show a different pathological picture. The aetiology of the lesions in foetuses aborted from uteri infected with equine herpes virus (EHV1)[48] may be similar.

The syndrome of cerebellar hypoplasia reported in the Arab horse[46] may be an example of a disease process altered by the immune status of the foetus. An immunodeficency disorder has been seen in this breed[25]: a lack of an immune response *in utero* at the time of invasion by a causative agent may be involved in producing the lesions of this disease. The causative agent, which has not been identified, may produce no, or possibly quite different, lesions in normally developing foetuses. The genetic relationship between cases that is documented in this syndrome may be one of a heritable immune deficiency, rather than one of a more specific lesion-producing character.

The early function of the intestinal defence system is important, since the gut is probably a major site of antigen entry in the neonatal foal. This possibility is emphasized by the fact that intestinal disorders are amongst the commonest of neonatal diseases.

Another important area in equine intestinal pathology is that of parasitic infestation. The lack of investigation into the immunological basis of equine parasitic disease is suprising, considering the high incidence of infestation in this species. An investigation into the parasitic status of horses exhibiting immunodeficiency syndromes may be rewarding. Strongyloidiasis has been blamed for causing mortality in immuno-suppressed human beings[40].

Prenatal immunization of lambs in late gestation has been shown by Richardson *et al*[36] to be a possible method of overcoming the non-responsiveness of this species to bacterial antigens. Vaccination studies in foetal and neonatal horses may provide valuable information, aiding the prophylactic treatment of diseases occurring in this early period of life. Early achievement of active immunity against the organisms causing these diseases may be provided by methods that specifically stimulate the gastrointestinal lymphoid system.

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For Discussion see page 67.

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CLINICAL ASPECTS OF PASSIVE IMMUNITY IN FOALS

L.B. JEFFCOTT*

A brief description of some of the factors affecting the immune status of young foals was presented. It was intended to highlight the more important clinical aspects of maternal transfer and to give an estimate of the age at which the foal is fully immunocompetent.

The pathway of transmission of passive immunity involves three main steps, firstly, the secretion of colostrum by the mare, followed by the uptake of immune proteins by the foal's small intestine and finally their transfer to the systemic circulation. Colostrum is made up of immune proteins which are selectively concentrated from the blood by the mammary gland in late gestation. In healthy mares the secretion of colostrum is short-lived and antibody levels fall to insignificant levels by 24 hours postpartum[2]. The premature onset of lactation is relatively common in mares and probably constitutes the most frequent cause of poor immune status in foals[3].

The newly born foal is agammaglobulinaemic at birth and is particularly susceptible to invasive pathogens in its new environment. The absorption of colostral proteins takes place by specialized cells of the lining epithelium of the small intestine. These cells are apparently non-selective in their uptake so that any large molecules present in the bowel will be absorbed whether they have immunological properties or not. Each cell takes up all the protein it can absorb before discharging it out of the base of the cell into the intercellular space. The protein globules then pass into the local lymphatics and finally reach the systemic circulation via the thoracic duct. This complex mechanism of absorption is efficient immediately after birth but declines sharply and by 24 hours it has completely ceased[1,4]. The milk proteins produced by the mammary glad (β-lactoglobulin and α-lactalbumin) are also absorbed in the same way along with the immune proteins. They are of no immunological importance and, being of low molecular weight, are rapidly excreted by the kidney so producing a transient proteinuria. This proteinuria ceases when the intestinal cells no longer absorb the large portein molecules[5].

In healthy foals the period after birth of susceptibility to infection is short-lived as the acquisition of passive antibody is rapidly effected. Levels of γ globulin approaching those of adult horses are attained within 12-18 hours. The duration of passive immunity in foals does not usually extend for more than four to six months of life and, for some period before this, antibody levels are of doubtful protective value. Active immunity can be demonstrated by the ability to sythesize autogenous γ-globulin. This is just detectable after two weeks of life but starts fairly slowly and it is not until about one month of age that they begin to reach useful levels. After this they increase and by three months or so the levels approach those of adult horses.

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VETERINARY CLINICAL DIAGNOSIS

W.R. KELLY

Bailliére Tindall, London: 2nd Edition, 1974.

Pp.VIII + 374; Figs 243; Tabs 18. Publ. price: £7,50 (R15,35)

The second edition of Prof. Kelly's book dealing with the principles and practice of clinical diagnosis in small and large animals is a sound introduction to diagnostics. The text has been revised and slightly reorganized with the addition of some new material and illustrations.

The book is primarily intended for the veterinary student, but would be useful for all practitioners. The stages of clinical diagnosis are set out lucidly and in a logical sequence.

The first six chapters are devoted respectively to general consideration of clinical problems and methods, history, general examination of the patient, temperature, pulse and respiration. In the following nine chapters the individual organ systems are discussed.

The final chapter on diagnostic tests (*e.g.* tuberculin testing, serological diagnosis) and those sections dealing with haematology, bone marrow, urine analysis: blood chemistry and milk samples could well have been omitted. Today's student is expected to have a more intensive knowledge of these subjects than is presented in this book, and numerous text books on clinical pathology are available.

The illustrations of old-fashioned instruments could surely be replaced with others more in keeping with modern equipment.

Some of the parameters given in the book are misleading, *e.g.* cat pulse rate 110 to 130 beats per minute, equine respiratory rate 10 to 14 per minute.

Despite the foregoing minor criticisms the book is recommended as the best text available on this important subject.

C.B.

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INTESTINAL SURGERY IN THE FOAL *

R.C. CROWHURST, D.J. SIMPSON, R.J. McENERY AND R.E.S. GREENWOOD

SUMMARY

Intestinal surgery in the foal is seldom necessary but some conditions in which it is essential are well recognized. The foal is a good surgical subject but success depends mainly on operating promptly before irreversible degenerative changes take place. Exploratory laparotomy is recommended whenever mechanical obstruction is suspected.

Indications fall broadly into two main categories:-

1. Cases arising from congenital defects. These form a miscellaneous group and present considerable difficulties in diagnosis and effective treatment.
2. Cases related to acute onset of obstruction, mainly due to volvulus or torsion. By far the most important condition in this category is volvulus of the ileum close to the ileocaecal valve.

Aetiological factors are discussed, and the value of resection of affected small intestine and creation of a new jejuno-caecal opening is indicated. It is suggested that with improvement of such techniques it will be possible to save a higher proportion of foals suffering from mechanical intestinal obstruction.

INTRODUCTION

Equine Laparotomy is now widely accepted as a perfectly practical and worthwhile procedure in selected cases. This is because it is appreciated that generalized peritonitis is not the hazard once feared, although localized post-operative adhesions often present problems. Also, improved operating conditions and anaesthetic methods have overcome many of the purely practical problems.

A high proportion of laparotomies are undertaken in connection with intestinal defects and several authors have made contributions on the various aspects of this subject. Littlejohn[6] [7] published a comprehensive two-part review concerning intestinal obstruction in particular. Mason, Johnston, Wallace & Christie[8] reported on experience gained in 13 clinical cases at Melbourne and attention is drawn to their paragraph on indications for laparotomy in colic cases. Other papers by Donawick, Christie & Stewart[3] and Huskamp[4] deal particularly with techniques for resection and anastomosis and these are very relevant to problems affecting foals.

In general, authors have concerned themselves mainly with adult horses, but Crowhurst[2] considered wider aspects of abdominal surgery in the foal and made the point that foals are excellent subjects for laparotomy. The problems associated with sheer size and the bulk of abdominal contents in the older animal are clearly much less serious in the foal.

In the authors' practice, which deals almost entirely with Thoroughbreds, bowel surgery in the foal has been carried out since 1948. The purpose of this paper is to present some details of the experience which has been gained from both successful and unsuccessful cases and to indicate how this can be used in the future. A bolder approach has developed over the years and it is now appreciated that major surgery is both feasible and rewarding. The main lesson which has been learnt is that prompt action is essential for success. Nevertheless, it is often difficult to decide whether surgery is necessary in a particular case. Intestinal colic is not common in the foal and when it does occur, it is often only transitory, righting itself without treatment. The most important indication for surgery is severe colic of sudden onset where pain cannot be alleviated.

Surgery is also indicated when unusual clinical signs of intestinal origin persist over a period and for which no account can be given. In the occasional case where gross abdominal tympany is present, the approach should be more guarded, as great difficulty in closing the abdomen can arise.

ANAESTHESIA

This will not be considered in great detail as individual preferences influence choice of method. It is considered that as well as being more suitable for abdominal surgery, the foal is also a better anaesthetic subject than the adult horse.

The cases described in this paper have been anaesthetised as described by Crowhurst[2], *i.e.*, induction with intravenous pentobarbitone sodium†, followed by intubation and maintenance on a halothane†† and oxygen mixture *via* a Fluotec vapourizer†††, calibrated from 0 – 10 per cent. Nitrous oxide at a level of approximately 25 per cent can be included in the gaseous mixture and so reduce halothane requirements. In the case of weak or particularly toxic foals, an open T-piece system is preferred (Fig. 1); this reduces the dead space and resistance to voluntary respiration which is present in the fully closed system with carbon diozide and rebreathing bag.

Pentobarbitone sodium has been found to be very satisfactory for induction, with a wide margin of safety in foals of all ages. It can be used to maintain anaesthesia by repeated intravenous injection if so desired. Langley[5] has described induction with halothane *via* a suitable tube placed into the nostril and this method has also been used in poor risk cases.

Premedication is not usually necessary as most foals presented for intestinal surgery are acute cases which have received sedative and/or analgesic drugs. If not, acetylpromazine in low doses by intramuscular injection is given prior to induction.

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* Presented by D.J. Simpson.
Authors' address: Reynolds House, Newmarket, Suffolk, England.

† SAGATAL: May and Baker, Dagenham, Essex.
†† FLUOTHANE: I.C.I., Macclesfield, Cheshire.
††† Manufactured by Cyprane Ltd., Keighley, Yorkshire.

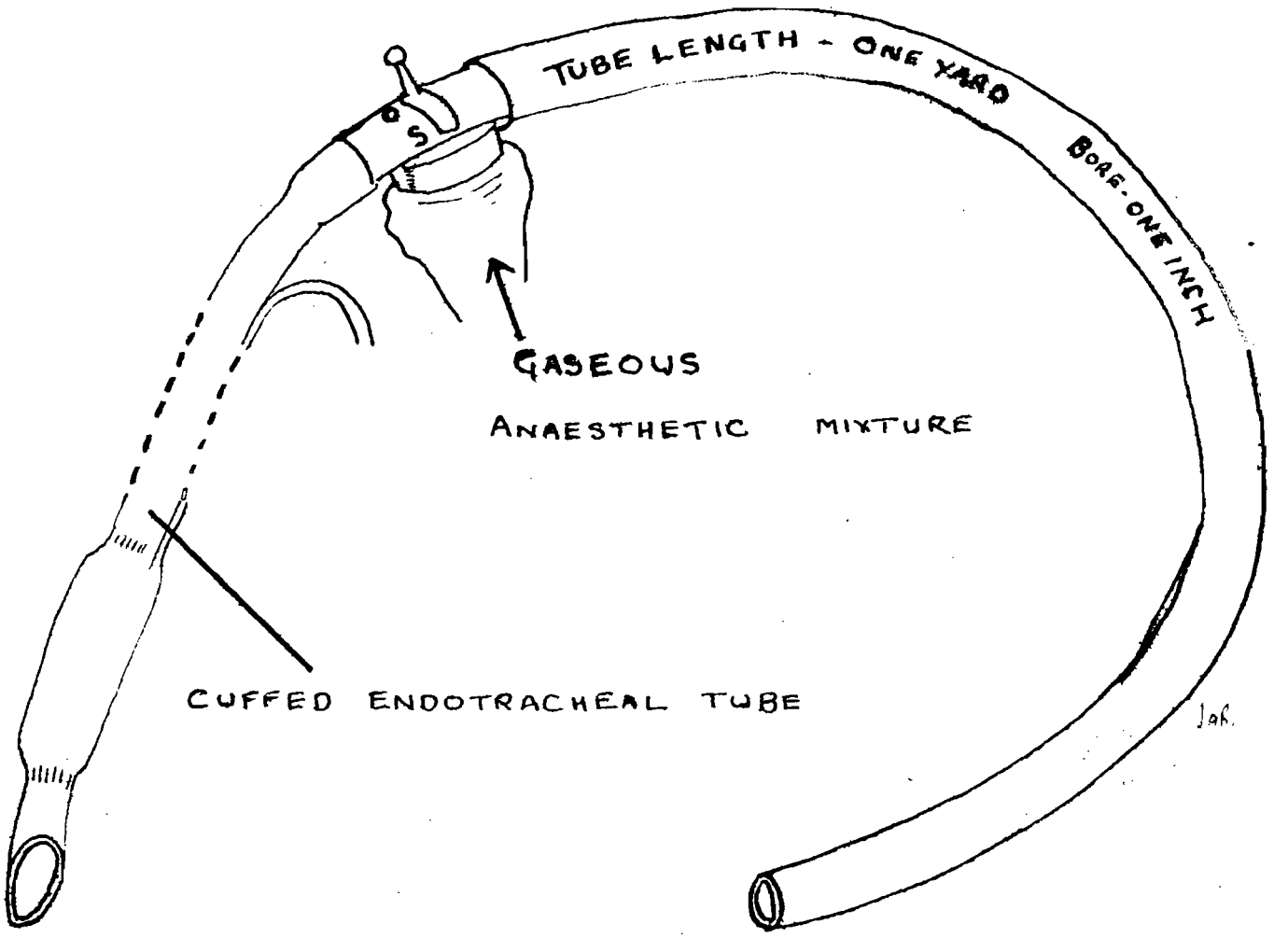

Fig. 1: Endotracheal tube

Supportive thereapy, usually undertaken by the anaesthetist during and immediately after surgery, will be discussed later.

INCIDENCE

The incidence of intestinal lesions requiring surgery is estimated at less than one per cent of the foal crop in any one year, but there are variations from one year to another in the United Kingdom and the possible reasons for this will be indicated.

SPECIFIC SURGICAL CONDITIONS AND THEIR TREATMENT

Conditions encountered in practice and treated surgically will be discussed in anatomical sequence. If a portion of the bowel is not mentioned, it has not been involved in cases to date. Later, some more unusual cases, which were either diagnosed at necropsy or treated conservatively, but which could have benefited from surgery, will be mentioned.

PYLORIC STENOSIS

This is a well recognized and not uncommon condition in human paediatrics. The authors have encountered and treated only one case and consider it to be a rare condition. It was readily recognized on that occasion and it is felt that other cases would not have been missed.

The attendants noticed within a few hours of birth that this foal (a colt) showed signs of pain immediately after sucking. It was otherwise normal and free from obstruction by meconium. The foal then stopped sucking altogether and stomach tube feeding was started, this also caused discomfort and some milk came back up the tube. The stomach tube was then passed right into the stomach and about two pints of milk ran back under pressure.

The foal was by now 48 hours old and it was suspected that obstruction at the pylorus, presumably owing to congenital stenosis, was responsible for the signs.

The consultant surgeon at the local hospital, Mr. R.E.B. Taggart, has always shown an interest in more unusual equine cases and, having experience of the condition in babies, agreed to operate straightaway.

The diagnosis was confirmed at laparotomy and the condition alleviated by Ramstedt's operation (pylomyeloplasty) consisting of longitudinal incisions, about 3 cm. in length, made through the serosa and muscularis layers at the site of the stenosis. Care was taken to avoid penetrating the mucosa but, in fact, this was punctured at one point and required suturing with 2/0 (metric 3) chromic catgut. As an added precaution, a fold of omentum was laid on to the pylorus and sutered in place.

Access to the site was straightforward and ade-

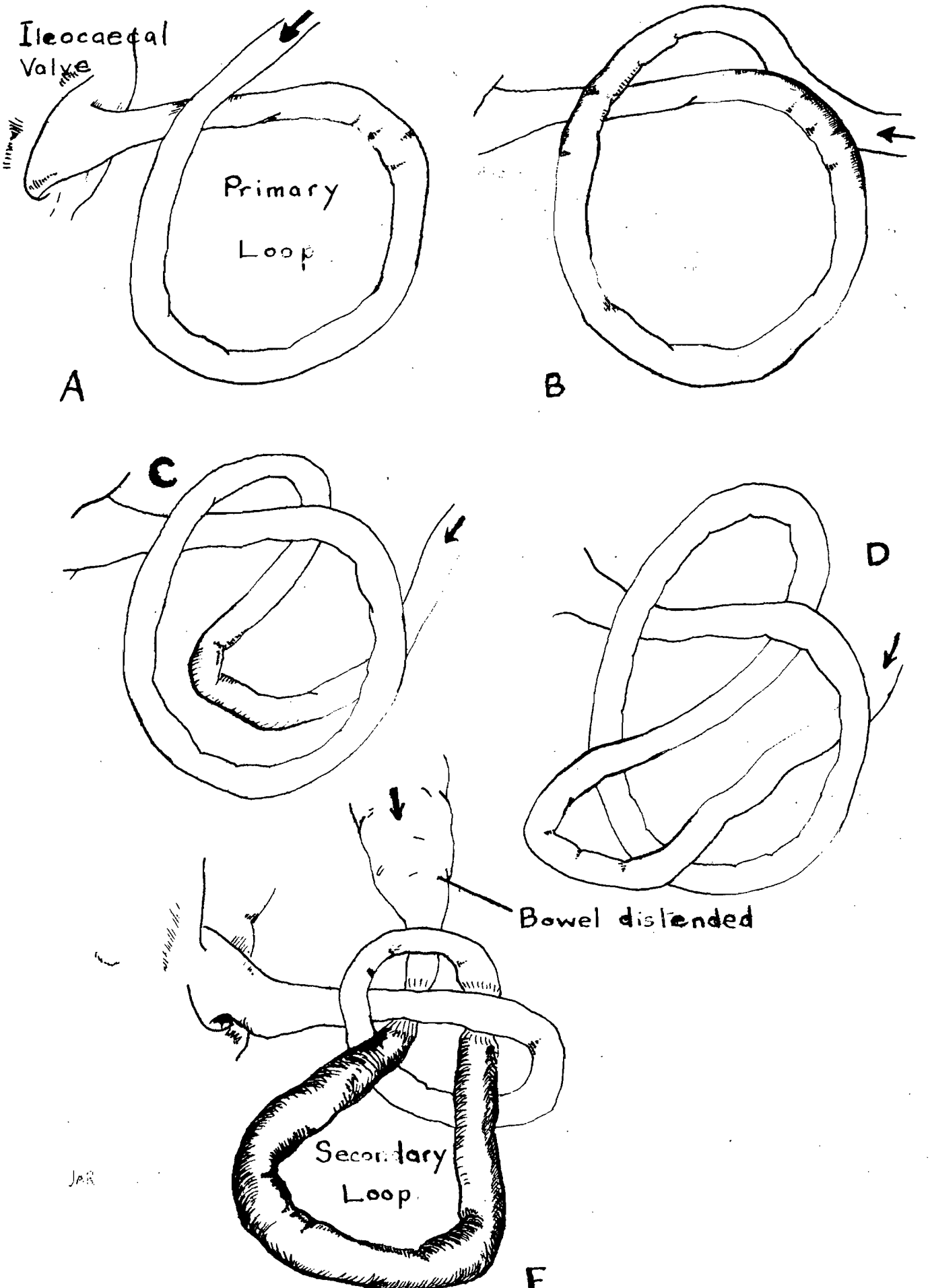

Fig. 2: Schematic representation of formation of volvulus.

(Editors note: This could not be reproduced on cadavers without making a tear in the mesentery. By following a different pattern, however, a "pseudo-knot" could be produced without tearing the mesentery.

quate *via* a longitudinal mid-line anterior abdominal incision which subsequently healed well.

Recovery was rapid and the symptoms disappeared immediately and completely. This foal subsequently developed normally and was put into training in the usual way.

DUODENAL STRICTURE

Again, this is a rare condition and only one case is included in the series. The foal concerned was dull and unthrifty from birth and then at two months of age it ceased to suck and feed and progressively lost weight and strength. No specific diagnosis was made initially but feeding by stomach tube was started and after a few days back pressure from the stomach was evident.

A stenosis was suspected and the likelihood of this was confirmed by giving a barium meal by stomach tube and then taking serial X-rays. Eight days after the foal lost its appetite, an exploratory laparotomy was undertaken but it proved impossible to find the exact site of obstruction until the foal was killed and the abdomen opened completely. A muscular stricture of the duodenum about eight inches (20 cm.) from the pylorus was then found. It was considered that this was congenital; progressive constriction of the bowel lumen had occurred until a critical stage was reached.

In retrospect, it is thought that with a more sophisticated X-ray technique, the lesion could have been identified and located more precisely. Perhaps it could then have been reached and treated surgically by a similar technique to that used in the pyloric stenosis case described above *via* an anterior abdominal incision, either mid-line or para-costal.

VOLVULUS OF THE SMALL INTESTINE

In practice, this is the commonest indication for bowel surgery in the foal; it is known colloquially as 'twisted gut'. The lesion almost invariably involves the terminal portion of the ileum close to the ileocaecal valve. This is of help when exploring the abdomen, as the valve provides a fixed locating point. It may also be important in relation to the manner in which these twists arise.

Aetiology

Twists may be seen at any age but the majority occur in the two- to four-month age group. At this time, foals are becoming less dependent on mare's milk and feeding habits are changing to the adult pattern. This involves corresponding changes in digestive processes and development of the large intestine to cope with bulky contents. These changes often coincide with a plentiful growth of fresh spring grass.

It seems probable that knotting of the small intestine is a chance happening due to hypermotility, and the terminal ileum is a predilection site for anatomical reasons, *i.e.*, the fixed nature of the ileocaecal valve and terminal ileum.

It has been suggested that bowel irritation due to the presence of developing ascarids may be a factor. Large numbers of ascarids have not been noticed in the small intestine during surgery on these cases. Strongyle larvae have also been implicated as possible causal agents by their effect on arterial circulation to the small intestine during the course of larval migration and development. Other possible causes of temporary intestinal irritation by direct action on the mucosa are many and varied.

It is common practice on British stud farms for older foals and their dams to be housed at night and spend a period of six to ten hours at grass during the day. This enforced control of feeding habits could also be important in the aetiology of the condition. Cases tend to be found during the later part of the day while still at grass or after return to the loose box. Sometimes, onset is late in the evening and affected foals are found either *in extremis* or dead next morning.

The length of small intestine involved varies from just a few inches to several feet. The sequence of events thought to be involved in forming a knot is illustrated in Figure 2. For the sake of clarity, the mesentery has not been shown but this usually remains intact and so passes through with the loop involved together with its blood vessels, etc.

The suggested causal factors can therefore be summarized:-

1. Anatomical relationship of terminal ileum and ileocaecal valve.
2. Transition from suckling to herbivorous diet coinciding with:-
3. Plentiful supply of young spring grass.
4. Bowel irritation, *e.g.*, by parasitic action.
5. Management practices affecting natural feeding habits.

A flush of spring grass coinciding with a critical stage in development of the digestive system appears to be the most important factor. This would account for the low incidence during dry seasons, when grass grows slowly and is kept down by grazing.

Clinical Signs and Course of the Condition

The onset of signs is acute, pain quickly becomes severe and is more or less constant. Affected foals will usually spend most of the time down, showing all the signs associated with severe colic.

They will lie still on their sides for a period and then quickly roll right over, then back again and so on. After a while they find that the position affording most relief is that of dorsal recumbency and they usually prop themselves against a wall with the neck curved to one side. This presumably reduces tension on the mesentery. From time to time, the foal will get to its feet and appear more comfortable, perhaps even walk round a little and go to the mare as if wanting to suck, then suddenly and violently it throws itself down again and acts as described previously. Self-inflicted abrasions soon appear, particularly around the eyes.

Naturally, the degree of pain and rate of deterioration depend upon the length of bowel involved and how rapidly the knot tightens, so strangulating the involved loop and eventually producing localized gangrene.

The clinical picture is quite characteristic. Initially, the abdomen is not distended and normal droppings may be passed; on auscultation peristalsis can be heard. This soon ceases and the small intestine anterior to the obstruction fills with gas, producing general abdominal distension. All that can then be heard are occasional hollow splashing sounds of a non-progressive nature.

The pulse becomes faster and weaker, reaching rates of 130/min or more; it may become imperceptible at the point where the facial artery crosses the lower edge of the mandible. The visible mucous membranes become injected and toxic in appearance, progressing to cyanosis in terminal cases. It is worth mentioning that the buccal membrane often looks worse due to trauma and drying out.

Body temperature rises to about 38,3°C (103°F). Respiration becomes rapid and embarrassed owing to pressure on the diaphragm from obdominal distension. Varying degrees of sweating occur.

The haemogram is not very helpful in early diagnosis, as significant variations do not occur until severe degenerative changes are present – a stage which is all too rapidly reached in untreated cases. A rising packed cell volume, certainly if over 55, indicates a poor prognosis, unless surgery is undertaken at once and bowel resection can be carried out, if necessary.

Differential Diagnosis

The clinical signs have been descibed at some length, as they serve as a comparison for other conditions which will be described.

It is sometimes difficult to distinguish an early twist from simple but acute flatulent colic which may be seen in foals of similar age. Such foals will show tympany at an early stage, while other clinical signs remain favourable, but the deciding factor usually is the response to treatment with relaxant, sedative and analgesic drugs. Flatulence cases usually respond rapidly and pass gas freely, becoming more comfortable as they do so. The drugs commonly used are acetylpromazine and Buscopan*. These and similar dugs may afford slight relief in twist cases but the pain and general clinical deterioration continue.

As stated previously, the degree of bowel involvement dictates the severity of signs and this is why difficulties arise in deciding if a case is surgical. Sometimes the worsening of clinical signs is such a gradual process that considerable time passes before it is appreciated that laparotomy is essential. Such delays always reduce chances of recovery.

In any event, preparations are made in good time and suspected cases transferred to the surgery premises at once. Continuing signs with progressive clinical deterioration for more than about four hours is sufficient indication for surgery. A decision to operate has never been regretted, whereas, on several occasions, waiting too long has resulted in loss of patients or jeopardized their chance of recovery.

Another possible differential diagnosis is that of bowel rupture, either of the stomach, small intestine (owing to impaction with ascarids), large colon or caecum. In such cases, the pain is much less severe and the signs are predominantly those of shock and severe toxaemia. Abdominal paracentesis may be used to speed diagnosis.

The chances of successful repair are virtually nil in cases where intestinal rupture has resulted in severe generalized peritonitis and associated shock with toxic changes.

Surgical Technique

The approach is conventional, *via* a simple ventral mid-line incision, long enough to give good exposure of the central abdominal region. On opening the abdomen, gas-filled but otherwise normal coils of small intestine are usually encountered first and, to aid exposure these are gently removed from the abdomen and placed to either side in sterile drapes where they are retained by at least two assistants. They are kept moist with warm normal saline solution.

In the typical case, the loop of ileum involved in the twist can then be located by further exploration and is easily recognized by its purplish congested appearance. The loop may then be partially withdrawn and followed to the point where it passes through the primary constricting loop. This point, close to the ileocaecal valve, cannot usually be exposed very clearly as it is retained at some depth in the abdomen. Gentle traction may elevate it somewhat and a good suction tube is useful to remove surrounding transudative fluid. Adequate assistance, so that plenty of bowel can be exteriorised and held clear, is of great help.

Here, the importance of gentle handling at all stages must be re-emphasized. The bowel becomes progressively more friable as it is devitalized by restriction of blood supply owing to venous congestion. Accidental rupture of the intestine is a very serious complication and tearing of the mesentery also causes delay and introduces further risks.

Reduction of the twist is attempted by passing a finger through the primary loop and opening it a little to allow movement of the secondary loop. By gentle traction from the free side and pressure from the other side the secondary loop may then be eased back. It is necessary to pause from time to time and hold the loop up and milk its fluid contents into the free portion, otherwise the decreasing length of trapped intestine becomes too distended with fluid; this impedes reduction and could lead to rupture.

The ease with which a twist of this nature can be reduced is very variable. Sometimes the knot is very loose and almost frees itself when manipulated. At the other extreme it proves impossible to effect reduction or even to identify the relation of the loops.

Cases have occasionally been encountered with the typical clinical signs of volvulus but in which the problem was rotation of a major part of the small intestine through an arc about its own mesenteric root. Here the congestive changes are less severe but more widespread. The lesion suddenly revolves itself if one is fortunate enough to counter-rotate the involved portion of bowel. Afterwards, it is impossible to say with any certainty what was actually done but all at once everything appears normal again.

After removing the obstruction, some ileal contents are milked through the ileocaecal valve and the involved portion observed for signs of improved circulation and returning peristalsis. The mesentery is checked for accidental tears. After unravelling a typical knot, the plum-coloured, congested section of intestine rapidly regains a normal pink healthy appearance in favourable cases. The points at each end of the secondary loop, where it was actually constricted, have a blanched waisted appearance when first released, but this gradually disappears, especially when peristalsis recommences. Figure 3 illustrates some features of the bowel shortly after release.

If these encouraging signs are not seen, a decision must be made as to whether or not the intestine can return to normal function. Sometimes a necrotic smell can be detected if gangrene has already set in.

At *post-mortem* examination of cases which had died within forty eight hours of operation, it has been seen that, although twists had been completely reduced, irreversible pathological changes were present. Either, circulation has not been restored, or ileus has persisted.

If the surgeon considers that the bowel is not viable, resection is the only alternative. End-to-end

* Buscopan Compositum – Cela. Crown Chemical Co. Ltd., Lamberhurst, Kent, England.

abdominal wound healing. The wound broke down on the fourth day after operation and required resuturing. The anastomosis was inspected on this occasion and the degree of healing was quite remarkable, surrounding reaction being minimal. It was not easy to even find the site of insertion. About six feet (almost two metres) of bowel had been resected. Huskamp has described in detail his technique for jejunocaecostomy[4] and this is recommended.

In one surviving case, it was necessary to re-operate just over three months after the first operation and break down local adhesions which were constricting the lumen of the terminal ileum/jejenum and giving rise to recurrent colic. The adhesions had arisen because the jejunum had been attached with a slight spiral rotation about its longitudinal axis.

In all cases, it is desirable to keep the operating time down to a minimum to avoid surgical shock and also to handle abdominal contents as little as possible in order to reduce the risk of subsequent adhesions. A solution of crystalline penicillin is usually sprayed into the abdominal cavity before closure.

Usually, once a clear passage has been established, any gaseous distension of the small intestine subsides and it can be returned to the abdomen without much difficulty. If tympany is still a problem, the small intestine may be cannulated and decompressed but in the interests of asepsis this is best avoided. If it is carried out, it is essential to use a fairly wide-bore canula through which a rubber or plastic tube with

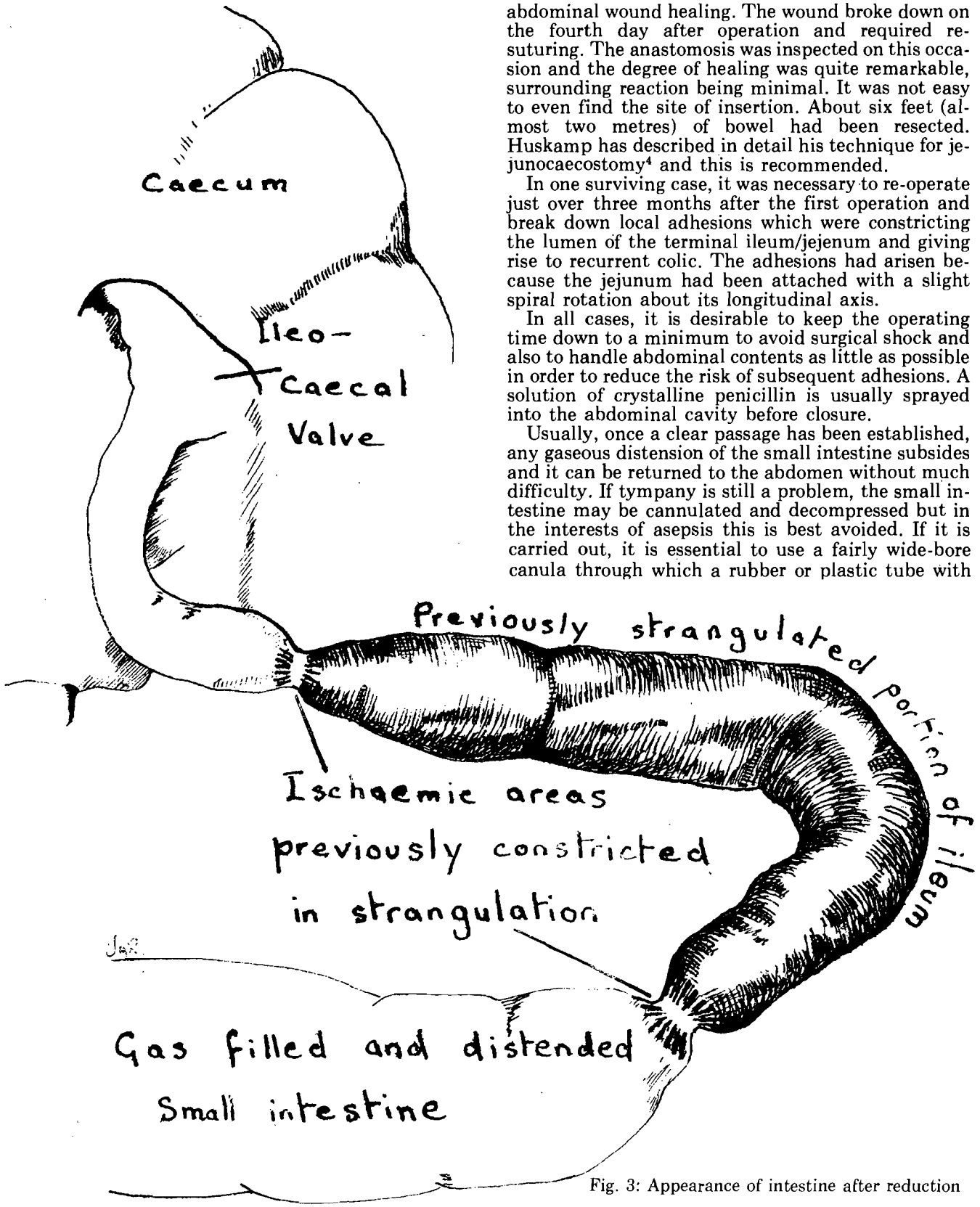

Fig. 3: Appearance of intestine after reduction

anastomosis so close to the ileocaecal valve is difficult. The preferred technique is to close the distal stump and join the proximal end of healthy small intestine directly to the caecum. This by-passes the ileocaecal valve and creates a new ileo- or jejunocaecal connection. This technique was first brought to the authors' attention as a practical proposition by Alexander[1]. To date, little experience of this method has been gained but, in three cases, two have survived for over a year while the other had to be put down two to three months later, owing to serious problems with many perforations can be passed along the intestinal lumen. Small bore needles rapidly become blocked and are not satisfactory for the purpose.

Abdominal Closure

This is carried out in three stages:-

1. Peritoneum and linea alba are closed as one layer with interrupted No. 4. (Metric 7) chromic catgut sutures. Every effort is made to include the peritoneal edges in this layer but not al-

ways with success. The observations of Swanwick and Milne[9] are relevant in this connection. If undue tension is encountered, it helps to draw together the centre of the wound temporarily with surgical tape or some similar strong material while the gut sutures are laid.

2. Subcutaneous connective tissue is co-apted with simple or Matress No. 1 (Metric 4) chromic catgut sutures in order to relieve skin tension and eliminate dead space.
3. The skin is drawn together with simple interrupted sutures using No. 3 (0,3 mm.) or No. 4 (0,4 mm.) Vetafil (Bengen). Overtightening of these sutures is avoided as some post-operative oedema always develops and my lead to cutting in and pulling through the skin sutures.

It will be noticed that interrupted sutures are used throughout which naturally makes abdominal closure a somewhat tedious process. It is believed that, if a knot slips or material breaks at one or two points, the consequences may not be too serious, whereas a similar occurrence with continuous suturing would be disastrous. Varying degrees of post-operative pain persist for up to 24 hours and the foal's response to this imposes strain on the wound repair.

The synthetic absorbable material, Polyglycolic Acid†, has also been used for burried sutures and found satisfactory.

Supportive Therapy

An intravenous fluid drip is set up during surgery, but if cases are tackled promptly, fluid imbalance is not marked. Three to four litres of glucose electrolyte solution is usually given and this provides a vehicle for 5-10 mega-units of crystal-line penicillin and 515 mg of neostigmine†† which is used to stimulate peristalsis once the obstruction has been relieved and while the foal is still under anaesthesia.

During the immediate post-operative period, analgesics are usually necessary to reduce pain which is still present and persists for some hours. Fluid intake by mouth is encouraged, plain water of glucose-saline being offered. Enemata may help to stimulate bowel function and additional doses of neostigmine are given as indicated. Passage of gas and fluid faeces is a very encouraging sign. Conscientious nursing in a well-heated box is essential during the first 24 hours after surgery and this has a great influence on the outcome. it is usual to give antibiotics for 7-10 days and a penicillin and streptomycin combination at high dose level is the usual choice. This may be changed if body temperature rises whilst under treatment. Multivitamins are also given by injection during convalescence.

INTUSSUSCEPTION

This condition can occur acutely and be clinically indistinguishable from volvulus, or, alternatively, may give rise to subacute or chronic signs.

In acute cases, the diagnosis is made at laparotomy; in a limited number of cases it again involved the terminal ileum. Aetiology is thought to be similar to that of volvulus. The lesion is readily recognized. Intussusceptions can usually be reduced by gentle squeezing and traction but, if this fails, or if irreversible changes have occurred, then excision and jejunocaecostomy are indicated.

The subacute form has been seen in younger foals as a sequel to diarrhoea; it presents rather vague signs leading to difficulty in diagnosis. Dull, intermittent pain is the main feature made evident by the usual signs of moderate colic. Grinding of the teeth is often noticed. Affected foals have little appetite and lose condition and become generally unthrifty.

SUBACUTE INTUSSUSCEPTION

This is an uncommon condition and surgical correction has not been undertaken. Two cases have been found at necropsy; they might possibly have been saved by radical surgery. This again underlines the value of exploratory laparotomy if symptoms indicate a possible underlying mechanical cause.

CAECAL TYMPANY

This has been found in two foals of the same age group in which twist cases commonly arise. They showed similar acute clinical signs.

When the abdomen was opened, in contrast to the previously described cases where the large bowel was normal, the apex of the caecum protruded immediately. This organ was distended and had the usual type of discolouration associated with circulatory obstruction, the line of demarcation between healthy and affected areas being quite clear cut. Thromboembolism is presumed to be the cause of the lesion, arising from arterial wall damage caused by migrating strongyle larvae.

In one case, the caecum was punctured to relieve gaseous pressure and then returned to the abdomen, which was closed in the usual way. This filly foal survived to race and win and is now at stud. This treatment was obviously only symptomatic and little credit can be claimed for the result. Partial caecal resection would be considered in future cases in which degenerative changes are present. The other affected foal was destroyed as necrotic changes had already set in.

ATRESIA ANI AND SIMILAR CONGENITAL DEFECTS

Discontinuity of the large intestine at some level is one of the commoner congenital defects seen in Thoroughbred foals. The most readily recognizable cases are those lacking an anal orifice. This has also been seen together with taillessness. Providing the rectum is complete (as is generally the case) it is an easy matter ot make an opening with a simple cruciate incision, under local anaesthesia sultures are not required. Such artificial openings function remarkably well and their size increases in proportion to normal rate of growth.

Other more serious defects are usually first reported as suspected cases of retained meconium but on investigation no meconium can be felt and the rectum has no faecal staining. Diagnosis can be aided by directing a light beam into a suitable test tube inserted into the rectum.

Exploratory laparotomy may reveal a condition which can be overcome surgically. In one foal with a

† Dexon: Cynamid of Great Britain Ltd., Alowych, Lonzon, w.c.z.

†† Prostigmin: Roche (Neostigmine injection B.P. 2,5 mg./ml) Manchester Sq., London, W.I.

blindly ending portion of small colon, it was possible to draw this back through the short, unconnected rectum and suture it to the anal ring. This foal survived for about two months but was eventually put down as it could not defaecate voluntarily: a large pouch had formed in which dung, accumulated until flushed out mechanically. Complete lack of the small colon is a commoner form of the abnormality.

Clearly, any such case must be assessed on its merits but it is unlikely that any major defect can be overcome satisfactorily.

NEONATAL EVISCERATION

Another congenital defect encountered from time to time is herniation of the abdominal contents owing to incomplete closure of the abdominal wall in the umbilical region. This prolapse may occur *pre partum* and lead to foaling difficulties or else it may happen immediately after birth. Obviously, the possibility of successful repair depends upon degree and also the amount of contamination and damage. Other unrelated congenital defects may also be found in such foals and should be looked for when making a decision.

A similar condition may arise within a few days of birth in foals with potential umbilical hernias if the mare licks and chews the umbilical stump so vigorously that she tears it away, causing prolapse.

HERNIAS AND PROLAPSES

Umbilical hernias are the commonest type seen in foals and their treatment, both surgical and non-surgical, is well documented.

Scrotal hernias, single or double, are relatively uncommon in the Thoroughbred but occur more frequently in heavier breeds. The noteworthy thing about them is that spontaneous recovery is common during early life and surgery is rarely necessary.

The authors are not asked to carry out m any foal castrations but in one case a considerable length of small intestineprolapsed through an inguinal ring on the day after apparently straightforward castration. The intestine was so damaged that repair was not attempted. Traumatic ventral ruptures in the lower flank region have been seen following accidents. Usually the history is that the foal had tried to jump a railing fence or gate and got caught on top with the hind legs dangling over one side. This happens more in older foals or weanlings and is readily recognized by palpation and auscultation of the soft subcutaneous swelling which results. Repair is tedious and sometimes difficult owing to the problem of apposing the torn musculature; results have been favourable.

OTHER POSSIBLE INDICATIONS FOR INTESTINAL SURGERY

RETAINED MECONIUM

The authors have not yet found it necessary to operate for this condition but, as mentioned, the possibility of an underlying congenital defect must be considered in atypical cases. The use of sedatives with bowel-relaxant properties is recommended in protracted cases. These relieve discomfort and allow passage of gas past the obstruction and, over a period of time, the impacted meconium will soften sufficiently to be passed or removed by conservative methods. If surgery is undertaken, the first aim should be to massage the meconium through the pelvic canal without enterotomy, but if the bowel has to be opened the sterile nature of the contents is a favourable factor.

One case has been encountered o posterior obstruction in a new-born foal which at *post-mortem* examination was found to be due to herniation of a portion of small colon containing a firm ball of meconium through a hole in the colic mesentery. This defect was considered to be congenital but in other respects the local structures were normal. This particular foal showed all the evidence of meconium retention but pain was more persistent than usual, although the abdomen did not distend with gas. On rectal examination, the lump of meconium could just be felt but only through a layer of bowel. When a test tube was passed and illuminated, slight faecal staining was seen but nothing more. The foal died at four days of age and the lesion was then found at about the level where the small colon merges into the rectum, the section of bowel containing a single lump of meconium having herniated through the mesenteric hole almost adjacent to the bowel.

It would have been difficult to expose this, owing to its position close to the pelvic cavity. It may well have been possible to diagnose and correct by direct palpation *via* a posterior abdominal incision.

TORSION OF THE LARGE COLON

One case has been seen in which torsion of the large colon at the diaphragmatic and sternal flexures led to the death of a foal on the third day of life.

This foal was weak and poorly nourished at birth; on the second day it was treated for apparent impaction. Treatment included gravitation of a large volume of enema fluid into the rectum with the foal held up by the hinds legs. After this, pain became worse and the foal's general clinical condition deteriorated rapidly.

At *post-mortem* examination, the left parts of the large colon were found rotated upon each other anteriorly. The constriction of blood supply was complete, with marked ischaemic changes in the left parts of the large colon. It was not possible to determine whether the twist had been present from birth or had resulted from being held up as described. This particular foal was certainly not a fit subject for surgery but the possibility of such a lesion should be considered in other cases.

GENERAL CONCLUSIONS

Results justify surgical treatment of many cases involving intestinal lesions in the foal. The more important indications arise as emergencies and should

Table : SUMMARIZED RESULTS OF TORSION CASES INVOLVING SMALL INTESTINE (1955 – 1973 INCLUSIVE)

Total number of operations	32
Death within 24 hours	11
Destroyed at surgery	2
Died or destroyed within few months of operation	4
Recovered	15

be treated as such. Reasonable surgical facilities and adequate professional help are essential. If it is necessary to refer such cases elsewhere, this must be done immediately as success depends upon prompt action.

The figures in the table include two cases of mesenteric rotation, both of which made speedy recoveries. Over-all recovery rate in this main group was less than 50 per cent.

The total number of other types of cases treated surgically is lower and consists of a variety of conditions. Statistics based on these results would have no real significance but a similar recovery rate applies.

Exploratory laparotomies were always of considerable help as regards management of the case concerned. Some were destroyed when inoperable lesions were found. In others, no mechanical obstructions were present and the usual treatments to restore normal bowel function could be applied with greater confidence.

ACKNOWLEDGEMENTS

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Mr. J.A. Rodger kindly prepared the illustrations.

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GENERAL DISCUSSION ON IMMUNOCOMPETENCE: PRE- AND PERI-NATAL

CHAIRMAN: R. ROUS

W.J. Donewick: One of the obvious tissues to use in determining whether immunocompetence or hypersensitivity exists, is epithelium. Have you, or has anyone else used skin grafts to determine when the whole system gets started?

C.D. Mackenzie: Yes, this is a good method to use in investigating the onset of immunological competence. The time of development of immunity varies with the particular antigen in question. Immunocompetence to skin grafts would not necessarily show the earliest time a foetus is capable of responding immunologically. With skin grafts I do not think the work has been done in the horse.

L.B. Jeffcott: I should like to confirm that no work with skin grafts on foals or foetuses has yet been performed. Specific antigens have been injected into equine foetuses in late gestation and the immunoglobulin produced in response has been predominantly IgM.

I would like to make a few comments concerning the development of immunocompetance in the foal before birth. This information is based on stimulation of the foal's immunological system by *in utero* challenge with pathogenic fungae. Mycotic placentitis is not uncommonly seen in mares in late gestation and infection usually takes place by an ascending route from the posterior genital tract and there is usually no invasion of the foetus. The fungal lesions are confined to the cervical pole of the allantochorion. However, in about 5% of cases, there is haematogenous spread of infection (as occurs in cattle) and the lesions are found not only at the cerivical pole but distributed all over the placenta. In these cases infection of the foetus does occur and the organism may be cultured from the amniotic fluid and foetal stomach contents. The inflammatory reaction to the fungal invasion is marked in both the foetus and on the allantochorion. The cellular response consists of lymphocytes, giant cells, occasional plasma cells and neutrophils and closely resembles an immunological or rejection-type reaction. Where it has been possible to collect a serum sample from the foetus significant levels of γ globulin (presumably IgM) have been demonstrated. The earliest this has been noted was 33 weeks of gestation; in other words the foetus was immunologically competant at this time.

A.M. Merritt: Three years ago Dr. Fisher published a technique for a qualitative measurement of globulin in immune stats, namely the zinc sulphate turbidity test. Have you applied this at all to foals? Would it be a practical test for the clinician?

L.B. Jeffcott: We have used the Zinc Sulphate Turbidity Test and found it to be extremely useful. It is a quick and simple method for the assessment of immune status in newly born foals. It has the advantage over the more complicated methods for protein estimation in serum and colostrum in that it can be carried out in a practice laboratory or actually on the stud. The results with this test compared favourably with those obtained by fractionation of serum by zone electrophoresis and estimation of total protein concentration in colostrum and milk by a colourimetric method.

R. Rous: Could you tell us what you would recommend in the case of a foal born from an iso-immunized mare?

L.B. Jeffcott: I would recommend that the foal be removed from its dam or muzzled immediately after birth. The most critical time for the foal is in the first few hours of life when the small intestine is most permeable to the iso-antibodies. It should be remembered that even the very smallest quantity of mare's colostrum is sufficient to trigger off intravascular haemagglutination and haemolysis. In addition to muzzling the foal should be given supplementary colostrum from the colostrum bank, fed every 1-3 hours with milk substitute and given parenteral antibiotics and additional cover against infection. The mare's colostrum must be stripped regularly every 1-2 hours and discarded. The foal can be allowed to suckle its dam after 24 hours of life. This is because firstly the specialized cells of the small intestine will no longer be able to absorb large molecules and secondly, by regular stripping of the mammary secretion negligible levels of iso-antibody will be left.

I have occasionally heard of instances where a muzzled foal has developed haemolytic diseases after 4-5 days of age. I am unable to refute this occurrence but would think it is quite possible that such a foal may have suckled even small quantities of colostrum immediately after birth. The other possibility is that the time of closure of the small intestine might occasionally be extended longer than 24 hours. I have not been presented with a case like this myself yet but I recall one case of some interest in a mare which produced a high titre of the most lethal iso-antibody (antiA_1). The foal from this mare showed clinical signs of jaundice, anaemia and haemoglobulinuria within 12 hours of birth. It was given an exchange transfusion of donor blood at 18 hours and returned to suckle the mare at 22 hours old. There were no further clinical complications or recurrence of anaemia; the foal suckled immediately and never looked back. On this evidence it would seem quite safe to return the foal to its dam after 24 hours of life.

R. Rous: Would Dr. Mackenzie care to elaborate on abortion owing to herpesvirus infection?

C.D. Mackenzie: In abortions contributed to infection with equine herpes virus type I, the foetuses that were older than six months had necrotic lesions involving more inflammatory and immune cells than those infected with herpes at an earlier stage. This is probably due to the development of immune competence in the foal foetus.

LA MYXOMATOSE II

L. JOUBERT, E. LEFTHERIOTIS & J. MOUCHET

L'Expansion Scientifique Francaise, 15, rue Saint-Benoit, 735278 Paris. 1973. In French. Pp. 252; Figs 40; Tabs. 29. Publ. Price: 67F.

In recent years a series of monographs dealing with virus diseases of animals has appeared under the direction of Proff. P. Lepine and P. Goret. This book is the second volume covering myxomatosis and is in keeping with the high standard set by these monographs. This volume deals in depth with aspects of the disease including symptomatology, epizootiology and control.

In parts of the world where this disease is not endemic, myxomatosis is associated with classical studies on the host parasite relationship, however this volume serves to remind us of the very serious nature and importance of this disease where rabbits of the genus *Oryctolagus* are exposed to the virus. The authors have successfully incorporated the results of these studies into a handbook which must be regarded as indispensable in any laboratory which is concerned with the diagnosis and control of this disease. At the same time it is also a useful guide in the design of field studies on diseases with a similar epidemiology. It is illustrated by black and white photographs and by some well designed and explicit diagrams. The bibliography is extensive and includes references to literature published until 1971; for the convenience of the reader these references are classified under subject headings and this volume can also therefore be recommended for use by anyone who is seeking further information on this disease.

P.G.H.

BOOK REVIEW BOEKRESENSIE

PESTICIDE RESIDUES IN FOOD

Report of the 1971 Joint Meeting of the F.A.O. Working Party of Experts on Pesticide Residues and the W.H.O. Expert Committee on Pesticide Residues. World Health Organization Technical Report Series No. 502, Geneva, 1972. Pp. 46, Tabs 1, Publ. Price: $1.00.

This is a brief report on the meeting of this body of 20 experts and is one of a series that should be read in conjunction with the others.

An up to date table of 105 pesticides, or groups of pesticides, is included as well the recommended acceptable daily intakes for man, tolerance values and practical limits. It serves as a very valuable reference and sound guide on these matters.

The report is mainly concerned with agricultural products other than those of animal origin. However, where residues in meat, fat, milk and milk products are considered, the residue problems in countries like ours, where for instance acaricides have to be used on a weekly basis to control ticks, have been taken into account. These problems have, however, not necessarily been solved.

It is interesting that residues due to anthelmintics have not been considered as these compounds are not included in their definition of a pesticide.

The need for the Republic of South Africa to investigate the problem of pesticide residues and to determine tolerance values in animal products is emphasized and this report serves as a sound reference on this matter.

T.W.N.

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RADIOGRAPHY OF THE ALIMENTARY CANAL OF THE HORSE

C.F. REID*

INTRODUCTION

Within the last ten years, many significant advances have been made in the development of high powered X-ray equipment for use in veterinary medicine. These developments have enabled the veterinary radiologist to explore the use of radiographic techniques in the examination of the digestive system of the horse. Bargai, in 1972, while working at the University of Pretoria, South Africa, described the technique and equipment necessary to radiograph the horse's normal digestive tract[1].

The term alimentary canal is all-inclusive and deals with the entire digestive tube from the mouth to the anus. This discussion will pay particular attention to (1) the oral cavity, (2) the oesophagus and (3) the stomach. The small and large intestine will be dealt with briefly; the rectum and anal canal will not be discussed.

TECHNIQUE

Radiography is only one modality that can be used to examine the alimentary canal. Before radiographic procedures are employed, a careful physical examination should be completed. Included in such an examination should be rectal palpation and the examination of the oesophagus and possibly the stomach with a fibre-optic 'scope. Following such a preliminary examination, one can proceed with radiography.

The type of radiographic equipment necessary to examine the alimentary canal will depend on the area of the canal to be examined. For example, very adequate diagnostic radiographs can be obtained of the head and neck area of a horse, utilizing portable or mobile radiographic equipment. X-ray equipment with the capability of producing 25 to 300 mA and 85 to 125 kV is sufficient to obtain diagnostic radiographs of the head and neck of horses. Routine flat film radiographs are frequently sufficient for these areas of the horse.

If the thoracic oesophagus, stomach and other areas of the lower digestive tract are to be examined, rather sophisticated high powered X-ray equipment is necessary. Equipment ranging in output from 500 to 1 000 mA and 150 to 200 kv is necessary, if diagnostic radiographs of these areas are to be obtained. In addition, it is extremely helpful, and sometimes necessary, to be able to examine the cervical, thoracic and abdominal oesophagus, in addition to the stomach area, fluoroscopically. In order to carry out this type of examination, sophisticated equiment such as image intensification with either videotape recording or ciné film recording is necessary. In the examination of the oesophagus and stomach, a combination of flat film radiographs and fluoroscopic viewing on a television screen is essential for confirming a suspected problem.

An integral and vital part of such an examination is the use of positive contrast material such as barium sulphate. The suggested consistency of the barium

Fig. 1: A horse standing between the X-ray tube and image intensifier, undergoing fluoroscopic examination of the upper alimentary canal.

Fig. 2: A lateral radiograph of the cranial aspect of the cervical oesophagus, showing a bolus of food obstructing the oesophagus. No artificial contrast material was used.

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sulphate is a one-to-one mixture, half barium and half water. This is administered usually *via* stomach tube and pump. In some cases, it is helpful to mix food with positive contrast material and allow the horse to eat the material. One can then follow the pathway of this material through the oesophagus and into the stomach *via* the image intensifier on a television monitor. Besides the use of positive contrast material, it is sometimes helpful to use negative contrast by pumping air into the oesophagus or stomach area.

The exposure factors utilized to obtain diagnostic radiographs vary considerably in relationship to the size and thickness of the animal radiographed. For this reason, time will not be spent in discussing specific radiographic exposures. When the image intensifier is utilized, there is frequently an automatic brightness control that automatically sets the kilovoltage for an adequate exposure so that the areas of interest can be visualized.

RESULTS

The oral cavity, cervical, thoracic and abdominal oesophagus and stomach can be examined radiographically very satisfactorily by utilizing both flat film techniques and fluoroscopic examination with the aid of an image intensifier and television monitor. These studies are routinely carried out with the aid of both positive (barium sulphate) and negative (air) contrast material.

The radiographic examination of the intestines, including the small intestines (duodenum, jejunum and ileum), large intestines (caecum and colon), rectum and anal canal is not at all satisfactory. In animals of less than 350 kg, diagnostic radiographs of the abdomen can be obtained that may be of help in making a definitive diagnosis of a lower digestive tract problem. Nevertheless, owing to the capacity of the digestive tract in this area and the quantity of retained material, radiography is of questionable value. Numerous technical problems make it extremely difficult adequately to visualize changes in the lower digestive tract, from the intestines to the anal canal. As mentioned previously, the size and quantity of material contained in this area make it difficult to use adequate contrast material for visualization. In addition, the capacity of most X-ray generators is frequently insufficient to penetrate these areas adequately.

Almost all radiographic examinations, carried out on the alimentary canal of the horse, are performed with the animal standing. On infrequent occasions, the animal may be anaesthetized and placed on a radiographic table for examination. This has been done only when areas of the oesophagus have been examined. The most satisfactory radiographic examinations are carried out on the standing, awake animal.

The major lower digestive complaint in horses is

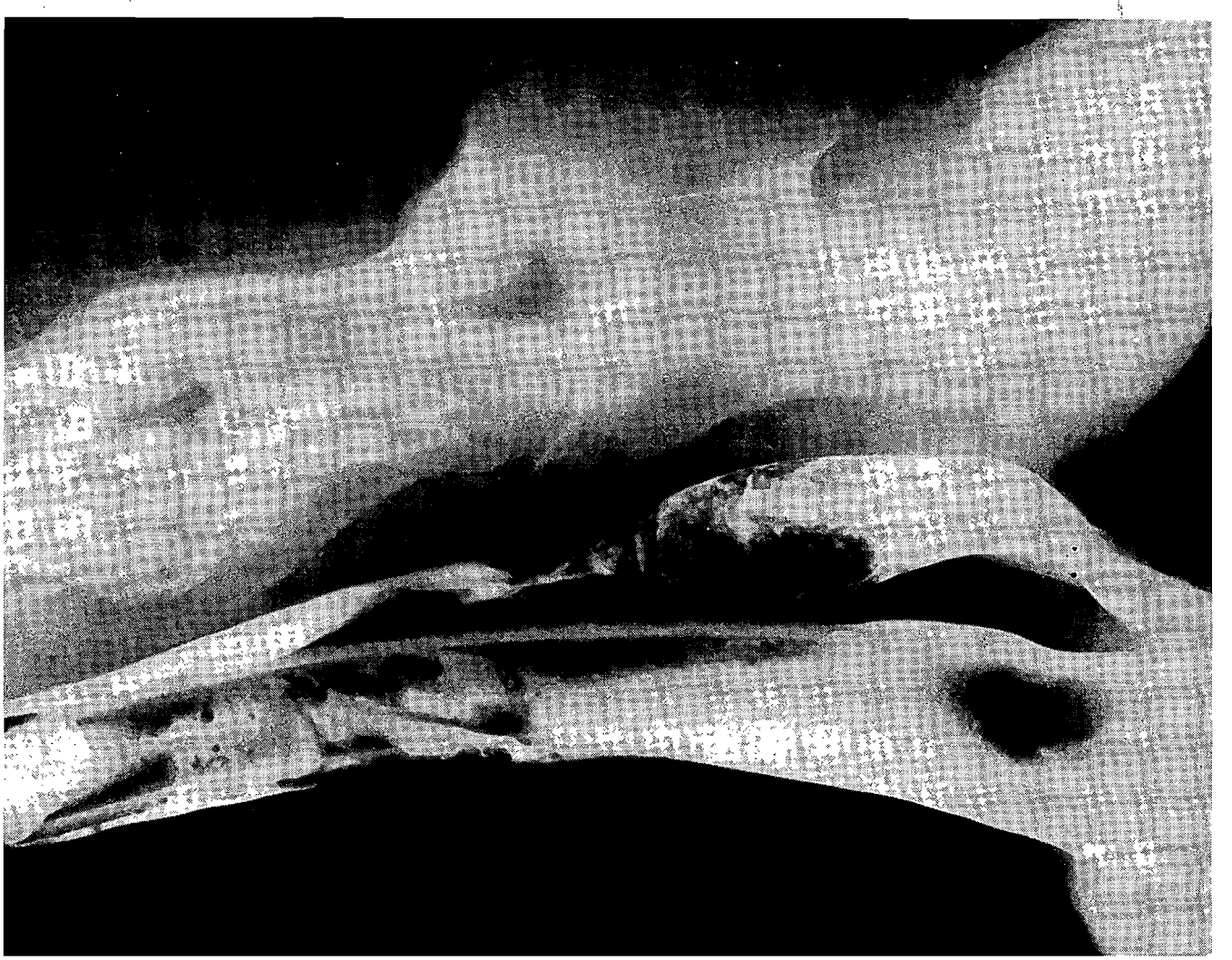

Fig. 3: A lateral radiograph of the cervical oesophagus of a young anaesthetized horse in lateral recumbency, showing a stenosis. Barium sulphate was used as contrast material.

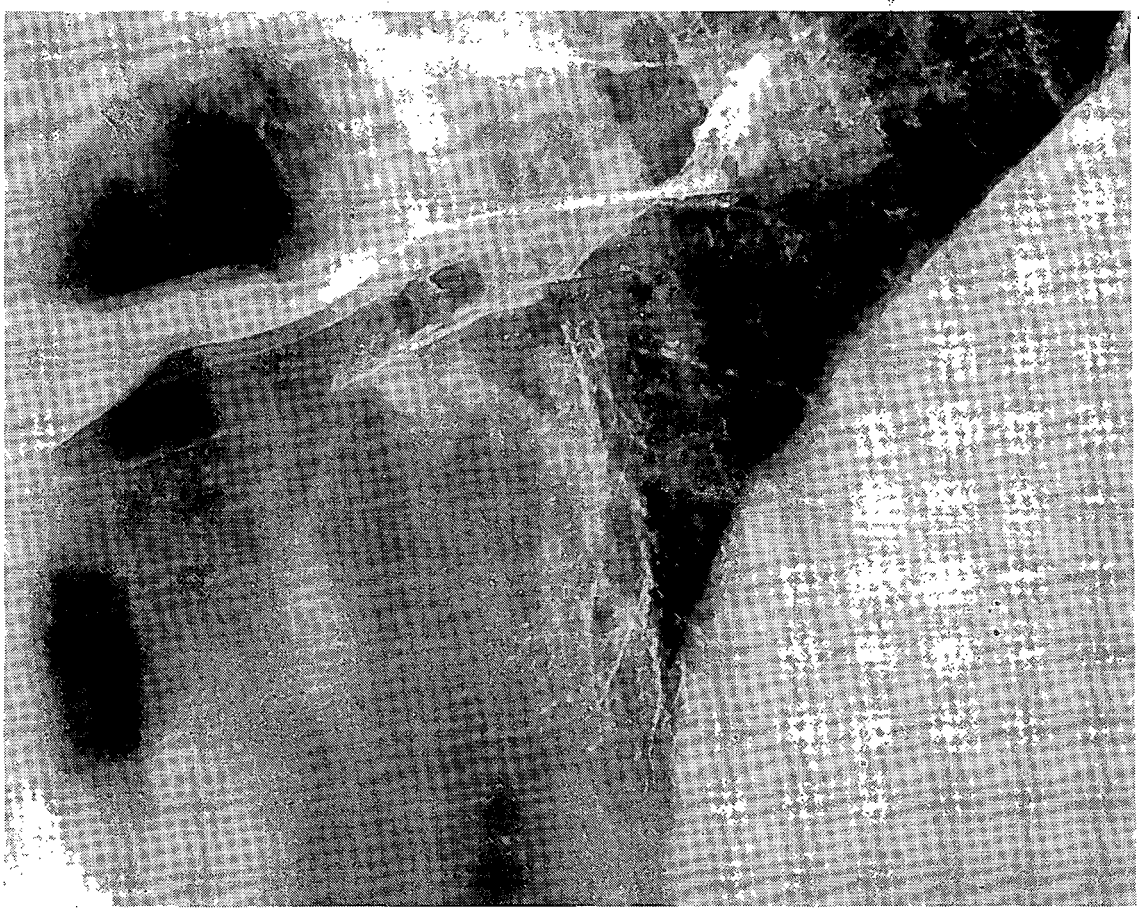

Fig. 4: A lateral radiograph of the thorax of a horse, showing a foreign body obstruction in the mid-thoracic oesophagus. There is dilatation of, and contrast material in, the oesophagus cranial to the obstruction.

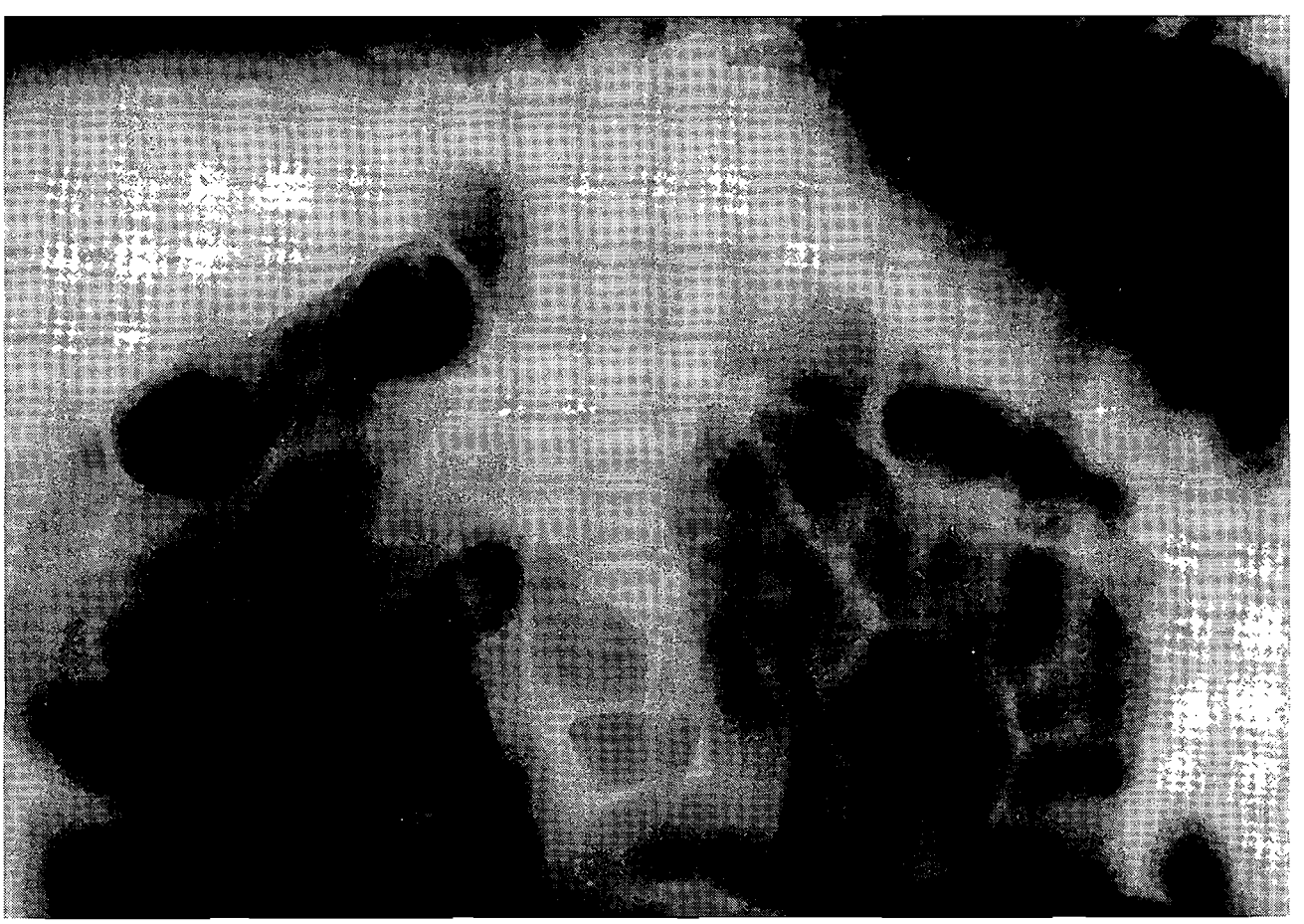

Fig. 5: A lateral radiograph of the abdomen of a young horse, showing marked dilatation of the small bowel as a result of complete obstruction.

colic. Owing to the uneasiness and movement of the animal, in addition to the movement of the intestines (which is not controllable), the radiographs obtained are frequently inadequate. A routine rectal examination frequently gives the clinician sufficient information as to the cause of the digestive upset. In most instances, therefore, a radiographic examination of this area of the digestive tract is not of sufficient value to warrant the procedure.

DISEASES OF THE ALIMENTARY CANAL OF THE HORSE DIAGNOSED RADIOGRAPHICALLY

Oral Cavity

Trauma to the skull, including fractures of the mandible, premaxilla and maxilla, have been diagnosed. Tumours such as adamantinomas and bone cysts can produce problems in the oral cavity. In addition, tooth infections and other secondary infections of the head are common-place.

Oesophagus

The oesophagus consists of cervical, thoracic and abdominal portions. The following conditions have been diagnosed in the cervical and thoracic oesophagus: (1) choke, as a result of some foreign body; (2) stenoses, either congenital or acquired of chemical or traumatic aetiology; (3) traumatic rupture has occurred in the cervical oesophagus; (4) diverticulum as a result of stenosis; (5) acquired achalasia. No abnormalities have been observed in the abdominal oesophagus other than secondary dilatation owing to gastric stenosis.

Stomach

One case of gastric stenosis has been diagnosed[2]. In addition, a penetrating gastric ulcer and a partial torsion of the stomach have been seen. Adhesions of the stomach to the surrounding viscera can produce abnormal reflux of ingesta into the oesophagus.

Intestines

Generalized ileus as a result of either peritonitis or enteritis has been diagnosed. In addition, complete and partial obstructions at the ileocecal valve have been seen. Dilatation of the caecum and areas of the colon have also been observed radiographically as a result of complete or partial obstruction.

Rectum and Anus

No radiographic diagnoses of rectal or anal canal problems have been made.

DISCUSSION

In general, the most satisfactory radiographic examination of the alimentary canal of the horse can be carried out in the oral cavity, oesophagus and stomach. Radiography may be an important aid to diagnosis in examining certain inaccessible parts of the intestinal tract. Most radiographic procedures are carried out on the standing, awake animal. Both positive and negative contrast materials (barium and air) are an essential part of an adequate radiographic examination of the alimentary canal. Numerous diagnoses, including oral cavity, oesophageal, stomach and intestinal problems, have been made, utilizing radiography as the primary diagnostic tool.

When adequately performed with proper equipment and utilizing both flat film and fluoroscopic examination, radiography of the alimentary tract of the horse is an essential aid to diagnosis.

In extremely large horses, certain areas of the small intestine, large intestine, including caecum and colon, and rectum are radiographically inaccessible, the volume of the ingesta and the inadequacy of certain X-ray equipment compounding the problem, Nevertheless, other examination techniques, including rectal examination, are frequently adaquate to make definitive diagnoses.

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DISCUSSION

M.A.J. Azzie: Can you enlighten us about the range of kV and mA.s that one would use for these exposures of the chest areas?

C. Reid: When radiographing horses, especially not so much the chest but the abdomen, the required exposures vary a great deal from horse to horse. For example, to get a good chest radiograph, such as these, we work around 85 or 90 kV at 500 mA, maybe at 1/5 a second, trying to cut down the motion. You could radiograph a horse's chest – and I have done this – with a portable machine, working at 85 kV and going up to 20 mA. If one has a quiet animal that was not moving and one caught it between respirations, one could get a radiograph of the chest at 1 second or 1,5s, maybe even at two seconds. With a mobile machine that can go up to 300mA and 125 kV, operating at 100 kV at 300 mA, exposure of $\frac{1}{3}$ of a second or even less than that, 1/5 of a second, can produce very diagnostic films. It can be done. In fact, the first chest radiograph I took, was of a highly bred, international jumper that had had a very bad accident at a horse show. It had landed on its side; fractured ribs and pulmonary haemohorrage was suspected. This happened in 1964 and the only equipment we had was a small Philips Super practice, that plugged in on 110 V, and operated at a maximum of 85 kV at 25 mA. This was a very quite and co-operative horse: we radiographed him and got some really good films of the fractured ribs and also of the haemmorhage in the lung. The disheartening thing was that, when the horse returned for follow-up films about three or four months later, at a time that our large equipment was operating, I could not get good films; I just was not familiar enough with the large equipment, to get comparably good films.

As far as the abdomen is concerned, operational requirements vary enormously. It it concerns an animal with a complete obstruction, there is going to be a tremendous amount of gas in the abdomen, which is radiolucent, and there is just nothing there to radiograph. On the other hand, one could have a large amount of material in the abdomen, so it really varies. If one is thinking of doing abdominal radiography, one should have a machine that at least goes to the 135-150 kV and capable of operating at a very high amperage, because the trick of the whole thing is getting a short enough exposure during the procedure, during which the viscera are bouncing around. Certain radiographs may be alright for radiologists to read but unless they are of good definition it is very difficult to convince the surgeon to take a knife and open up the abdomen. I can easily say cut him open, but the other guys have to sit up all night, trying to put him together again.

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THIRD SESSION: ADULT DISORDERS, ENTERITIS AND COLITIS

Chairman: G. FAULL

DIFFERENTIAL DIAGNOSIS OF DIARRHOEA IN HORSES OVER SIX MONTHS OF AGE

A.M. MERRITT, J.R. BOLTON AND R. CIMPRICH*

SUMMARY

Pertinent questions regarding the history of a horse with diarrhoea are listed, as are diagnostic procedures that might be included in a complete clinical work-up. For purposes of discussion, diarrhoea is regarded as 'acute' or 'chronic', wherein the former concerns cases where the features of the disease are severe with progressive electrolyte imbalance, dehydration, toxaemia, or other life-threatening manifestations and the latter refers to cases that have been prolonged for a month or more. Patterns of disease, including results of diagnostic techniques, are stressed. In the 'acute' category, salmonellosis, haemorrhagic and oedematous colon (Colitis X), acute peritonitis, and *Corynebacterium equi* infection are discussed in some detail. In the 'chronic' group, strongyle larval migrans, granulomatous enteritis, chronic liver disease, *Corynebacterium equi* infection and chronic salmonellosis are emphasized.

INTRODUCTION

The following discussion is based upon observation and study of horses with diarrhoea seen at the New Bolton Center Hospital, School of Veterinary Medicine, University of Pennsylvania, over the last five years. Only two major categories of diarrhoeal disease will be discussed, namely (1) the acute problem and (2) the chronic syndrome where diarrhoea has persisted for at least one month. It is recognized that 'acute' disease, if uncontrollable, can then become chronic, provided the animal survives the initial onslaught, but why this happens in some cases and not in others is only a matter of speculation at present and not within the scope of this presentation.

Table 1 lists our protocol for examination of horses with diarrhoea. Most of the items on the list can be done by the practitioner or his laboratory; the more esoteric tests are being applied to help us gain a better understanding of the pathophysiology of diarrhoea and will hopefully lead to the development of practical diagnostic methods. Only those items we consider pertinent to the differential diagnosis will be mentioned.

Table 2 lists the parameters of our examination of peritoneal fluid. We routinely perform a surgical scrub on the ventral midline along the most dependent portion of the abdomen and insert needles (18 gauge, 4 cm long) for collection of fluid. Occasionally, longer needles are needed. If any frank blood is encountered, the procedure is stopped and repeated another day.

Table 1: EQUINE DIARRHOEA – CLINICAL WORK-UP

All Cases

1. Physical Examination
2. Rectal Examination
3. Pharyngoscopy
4. Abdominocentesis
5. Complete Blood Count
6. Serum Analysis
   - Albumin/Globulin
   - Electrolytes
7. Examination of Faeces
   - Culture
   - Parasites
   - Protozoa
   - pH
8. Blood Gas

Chronic Cases

1. Serum Analysis
   - Immunoglobulins
   - Electrophoresis
2. D(+)Xylose Absorption
3. Faecal ^{51}Cr-Albumin Excretion
4. Intradermal TB Test
5. Intestinal Biopsy

Table 2: NORMAL EQUINE PERITONEAL FLUID

1. Colour – Clear Light Yellow
2. WBC Count – Less than 10 000/mm^3
3. WBC Differentiol
   - Predominantely Segmenters and Monocytes in Varying Percentages
   - Few Eosinophils
4. Cytology (Sano Stain)
   - Active Phagocytosis, 50-60% of Cells
   - Mitotic Figure Common
   - Look for Neoplastic Cells

HISTORY

The stock questions we ask when a horse is presented with diarrhoea, either acute or chronic, are:

1. Are there other horses in the stable showing similar signs?
2. What is the worming schedule?
3. Was the onset of diarrhoea associated with any stressful event?
   a. some other illness
   b. an injury
   c. heavy antibiotic therapy
   d. marked change in diet
   e. shipping
   f. excessive exercise, especially when combined with any of the above.

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4. Has the horse had recent abdominal surgery, abdomenocentesis or rectal examination?
5. For younger horses, in particular, is *Streptoccus equi* or *Corynebacterium equi* known to be a problem on the farm?
6. What treatment has the animal received so far?

ACUTE DIARRHOEA

The challenge is still before us to develop a comprehensive list of possible causes and differential features of severe acute diarrhoeal disease in the horse which rapidly leads to electrolyte imbalance, dehydration, toxaemia or other life-threatening manifestations.

Only four syndromes will be reviewed here in any detail since they are the most common aetiological diagnoses that we make. As mentioned before, all cases that we see are subjected to as rigorous a work-up as possible, the limitations being the condition of the horse at presentation. Only what we consider to be the essential diagnostic features will be emphasized. The four syndromes are: (1) acute salmonellosis; (2) haemorrhagic oedematous colon, or 'shock colon' ('Colitis X'); (3) acute peritonitis; (4) *Corynebacterium equi* infection. The essential diagnostic features are outlined in table 3.

Table 3: ACUTE EQUINE DIARRHOEA – POSSIBLE DIFFERENTIAL FEATURES

A. ACUTE SALMONELLOSIS

1. History of Recent Abdominal Surgery or Rectal Examination
2. High Fever
3. Profuse Watery Stool – Frank Blood Present?
4. Intitial Degenerative Then Regenerative Left Shift
5. Hyponatraemia with Mild Hypokalaemia
6. Hypoalbuminaemia
7. Mild Metabolic Acidosis
8. Culture Salmonella Organism from Stool

B. HAEMORRHAGIC OEDEMATOUS COLON (COLITIS X)

1. History of Recent Stress
   - Virus Infection Plus Continued Training
   - Massive Antibiotic (Tetracycline?) Therapy
2. Marked Tachycardia (80-100 Beats per Minute)
3. Cold Extremities
4. Copious Explosive Watery Diarrhoea
5. Hyperaemic Mucous Membranes/Slow Capillary Refill
6. Hypothermia
7. Marked Hypovolaemia
8. Very High PCV (60-70%)
9. Severe Metabolic Acidosis

C. ACUTE PERITONITIS

1. History of Recent Abdominal Surgery or Rectal Examination
2. Abdominal Pain
3. Stoal Watery but Not Profuse
4. Fever ?
5. Rectal Examination
   - Tear
   - Mass
   - Adhesions
   - Ingesta on Serosal Surfaces
6. Any Subcutaneous Abscesses?
7. Peritoneal Fluid, WBC Count 10 000, Mainly Neutrophils

D. CORYNEBACTERIUM EQUI

1. Farm History
2. Young Animals
3. Stool Watery but Not Profuse
4. Bronchopneumonia ?
5. Leukocytosis With Moderate Shift
6. Gram Stain Stool – Gram + Pleomorphic Rods
7. Culture *Corynebacterium* From Staol

The most definitive diagnosis of salmonellosis is the culturing of the organism from faeces of affected animals. We usually find *Salmonella typhimurium,* but other types, namely, *S. dublin* and *S. newport,* have been reported.[13] A problem in interpretation of cultures sometimes arises, especially when a commercial Enterotube* is used. Often, we have found an indole positive reaction which codes out to *Edwardsiella.* If swabs containing faecal material are widely streaked on selenite first and **well-isolated** black colonies are picked for the Enterotube*, the indole reaction does not take place and other reactions code to *Salmonella.*

The most suggestive clinical features of acute salmonellosis are the early degenerative left shift with WBC count, the rather rapid onset of hypoalbuminaemia, and the hyponatraemia as opposed to marked hypokalaemia. One can only speculate as to the cause of this latter feature, although the cases we have seen invariably have severe colitis, whereby an active Na pump system[6] might be disrupted. Further, it has recently been shown that *Salmonella typhimurium* causes an active Cl secretion and inhibition of spontaneous Na absorption in the ileum of rabbits[7]. If frank blood is present in the stool of affected horses, the diagnosis of salmonellosis is strengthened.

The history of recent stress, such as continued training while showing signs of respiratory disease or while on high levels of antibiotics, signs of marked circulatory collapse and passage of copious amounts of water from the rectum, strongly suggest the 'shock gut' ('Colitis X') syndrome. Affected animals quickly become hypokalaemic and acidotic; fluid loss through the gut must surely exacerbate the shock state. It is not unusual in such cases to find a packed cell volume of 65-70%, a serum potassium concentration around 1,5 mEq/*l* and an arterial blood pH near 7,0 with a base excess of -15 to -20 mEq/*l*.

Diarrhoea can be caused by acute peritonitis resulting from insults such as contaminated abdominal surgery, manual rupture of the rectum, ruptured intra-abdominal abscess, intestinal leakage or rupture secondary to focal strongyle larval-induced ischaemia, etc. The pathogenesis of this diarrhoea is considered related to altered intestinal motility but this has not yet been proved and no doubt other major intestinal functions – absorption and secretion – may also be disrupted. One of the most effective ways of definitively diagnosing peritonitis is through abdominocentesis; a WBC count of greater than 10 000 per cmm, predominately of neutrophils, is very supportive of this diagnosis.

Young horses infected with *Corynebacterium equi* will sometimes show diarrhoea as the exclusive sign or as part of a pneumonia-enteritis complex. A history of the disease's presence on the farm is helpful in establishing priorities in creating a differential diagnosis, but one of the most specifically diagnostic findings is the presence of large numbers of Gram-positive pleomorphic rods in a smear of the stool. Culture of the organism from the stool is also very helpful since it

* Enterotube: Roche Diagnostics, Division Hoffman-Roche, Inc., Nutley, N. J. 07110.

is not found there in normal horses. The concomitant or subsequent development of signs of bronchopneumonia is the usual course of events with this disease but we have seen a few cases of diarrhoea alone which may become chronic.

There are a number of reports of diarrhoea[2 9] in the horse attributed to *Trichomonas faecalis*. So far, no one has reported transmitting this infection to other horses and the organism's actual pathogenicity is questionable. Protozoa in the stool of diarrhoeic horses will be discussed in more detail in the next section of this paper.

CHRONIC DIARRHOEA

In this discussion, chronic equine diarrhoea will be regarded as that which has persisted for at least one month and which has proved refractory to usual symptomatic therapy. Table 4 reviews the aetiologic/pathologic findings in 28 cases seen at our hospital in the last three years. Twenty-four of these cases were subjected to most or all of the protocol in table 1; the remaining four were less extensively examined. Not mentioned in the list are *Trichomonas faecalis* and viruses. We have not seen a case of chronic diarrhoea in the horse where we could demonstrate, either through microscopic examination or culture, the presence of trichomonads in the faeces, although three animals did have large numbers of ciliate protozoa in the stool. We tend to agree with Manahan[10] that the presence of trichomonads may reflect a disruption in ecology of intestinal microorganisms rather than a pathogenic source. Viral aetiology of chronic equine diarrhoea is under investigation at Purdue in Indiana but at present no definite results of this study have been published.

Table 4: CHRONIC EQUINE DIARRHOEA AETIOLOGIC/PATHOLOGIC FINDINGS IN 28 CASES

CLASSIFICATION	No of CASES
1. Severe Strongyle Larval Migrans	12
A. Definite	9
B. Suspected	3
2. No Lesions/Cause Undetermined	5
3. Granulomatous Enteritis	3
A. Avian tuberculosis	1
B. Aetiology Undetermined	2
4. Oedema, Lamina Propria of Small Bowel	3
5. Chronic ('Cirrhotic') Liver Disease	2
6. Chronic *Corynebacterium equi* Infection	1
7. Salmonellosis	1
8. Gastric Carcinoma	1

From our studies to date, it appears that migrating strongyle larvae may be responsible for causing more chronic diarrhoea in horses than previously expected. Clinical findings suggestive of this problem are reviewed in table 5 and various combinations of the important signs are shown in table 6. So far, D(+)xylose malabsorption has been seen only in those horses where advanced larval migrans was suspected or confirmed. We disagree with Roberts[12] in that we believe D(+)xylose is a more reliable and sensitive marker than glucose for small intestinal carbohydrate absorptive capacity in the horse, particularly where subtle functional disturbances in function are concerned. The pathophysiology of the diarrhoea in these cases is not yet well understood and may be multifactorial, in response to altered circulation and/or local irritation. There appears to be no protein-losing enteropathy and the faeces are usually cow-like and only slightly in excess of normal (Merritt, unpublished observations). The most advanced case we have seen was manifested by a very abnormal D(+)xylose absorption and severe mucosal damage throughout the gut. Good response to a so-called 'larvicidal' dose of thiabendazole, 5 to 10 times the recommended dose orally for two days in a row, may add weight to this differential diagnosis. The

Table 5: CHRONIC EQUINE DIARRHOEA – POSSIBLE DIFFERENTIAL FEATURES

A. STRONGYLE LARVAL MIGRANS

1. History of Recurrent Colic
2. Anterior Mesenteric Arteritis and/or Aneurysm
3. Abdominocentesis
   - Increased Number of Eosinophils
   - Increased Phagocytic Activity
4. Faeces Usually Cow-Like Consistency
5. Dermatosis
   - Patchy Alopecia
   - Severe Scaling
   - General Thinness of Hair
   - Acute Endothelial Proliferation in Dermis
6. Abnormal D(+)Xylose Absorption
7. Eosinophilia
8. Increased Serum Beta Globulin
9. Normal ^{51}Cr Loss in Faeces

B. GRANULOMATOUS ENTERITIS

1. Thin-to-Emaciated Body Condition
2. Rectal Examination
   - Roughened Friable Rectal Mucosa
   - Thick-walled Intestines
   - Prominent Enlarged Mesenteric Lymph Nodes
3. Abdominocentesis
   - Decreased Phagocytic Activity
4. Faeces Very Watery Consistency
5. Marked Hypoalbuminaemia
6. Acid-Fast Organisms in Faeces or Rectal Biopsy
7. Excessive ^{51}Cr Loss in Faeces

C. CHRONIC LIVER DISEASE

1. Thin Body Condition
2. Faeces Soft or Watery, Not Profuse
3. Hypoalbuminaemia
4. Increased Serum Beta Globulin
5. Decreased Bromosulphaphthalein Clearance
6. Increased Prothrombin Time

D. *CORYNEBACTERIUM EQUI* INFECTION

1. Farm History / Young Animals
2. Progressive Weight Loss
3. Significantly Increased Amounts of Watery Faeces
4. Rectal Examination (if possible)
   - Enlarged Soft Mesenteric Lymph Nodes
5. Demonstration of Organisms in Faeces
   - Gram Stain
   - Culture

E. SALMONELLOSIS

1. Farm / Stable History
2. Onset Characterized by Severe Acute Illness
   - Fever
   - WBC Changes
   - Blood in Faeces
3. Rectal Examination Painful
4. Neutrophilia – Left Shift
5. Mild Hypoalbuminaemia
6. Culture Organism in Faeces

Table 6 : CHRONIC EQUINE DIARRHOEA – Cases Possibly Due to Strongyle Larval Migrans

	Anterior Mesenteric Arteritis	Eosinophils in Peritoneal Fluid	Eosinophilia	Larvae in Intestinal Wall	D (+) Xylose Absorption	Response to 5x – 10x TBZ
1.	–	–	+	–	Low Peak	Good
2.	–	+	–	+	Flat	Not Tried
3.	–	–	–	+	Low Peak	No
4.	+	–	+	–	Normal	No
5.	+	–	–	–	–	No
6.	+	–	–	–	Low Peak	Good
7.	–	+	–	–	Low Peak	Transient
8.	+	–	–	–	Normal	Good
9.	–	+	–	–	Low Peak	Transient
10.	+	–	–	+	Low Peak	No
11.	+	–	–	–	Normal	No
12.	+	–	–	–	Normal	Not Tried

TB2 = Thiabendazole

longer the diarrhoea has persisted prior to this therapy, the less likely will there be a satisfactory response.

Granulomatous enteritis (GE) is a pathological description of a chronic enteric disease seen occasionally at our hospital. The pathology is presently being worked out[4]. Only 3 of 10 cases had diarrhoea, though all animals had marked hypoalbuminia. Acid-fast organisms were seen in lesions from one horse; microbiologic examination was done and *Mycobacterium tuberculosis*, avian type, was cultured (by the laboratory of Dr. Richard Merkal, National Animal Disease Laboratories, U.S. Department of Agriculture, Ames, Iowa). The faecal excretion of intravenously administered ^{51}Cr-albumin is markedly increased in animals with this disease, suggesting that it is, among other things, a protein-losing enteropathy, as is a similar pathologic condition in man known as regional enteritis or Crohn's disease. A final diagnostic point that might be helpful is the virtual absence of phagocytic activity in cells collected by abdominocentesis from GE animals.

Fibrotic (cirrhotic) liver disease deserves mention in this group of differentials because it can present as a chronic diarrhoea syndrome and can be missed on a clinical work-up unless some specific tests are performed. Initially, it can look like granulomatous disease, mainly because of the poor body condition and hypoalbuminia, but the faeces are usually not profuse in quantity as has been our experience with GE cases that do have diarrhoea. Interference with bromosulphaphthalein (BSP) clearance from the plasma and a prolonged prothrombin time should point the differential towards liver disease. Icterus index or other measures of bilirubinaemia should not be relied upon; none of the horses we saw was jaundiced.

The one case of *Corynebacterium equi* listed in table 6 was a yearling filly that had diarrhoea for 4-5 months and no signs of respiratory disease. A complete work-up was not possible and diagnosis was made at necropsy when the typical Gram-positive pleomorphic rods were seen in material smeared from enlarged, soft mesenteric lymph nodes.

It is obvious that a definitive aetiologic diagnosis of chronic diarrhoea in the horse due to an infective agent, such as *Corynebacterium* or *Salmonella* depends upon isolation of the organism from stool or affected tissues. No doubt the list of infectious causes, including various viruses, will expand, as will our understanding of inter-relationships between microflora, local immunity and digestive/absorptive processes in the horse bowel.

Any neoplastic disease of the bowel may cause diarrhoea. Lymphosarcoma, adenocarcinoma, gastric squamous cell carcinoma and leiomyoma are the most frequently found neoplasms in the horse gut. The diarrhoea coincident with the squamous cell carcinoma of the stomach listed in table 4 may be unusual, according to published descriptions of this disease. This horse did have some features that were consistent with other cases[11], however, namely, large rough masses palpable rectally near the left kidney and anterior mesenteric root and a marked anaemia (PCV 10%). The finding of neoplastic cells in abdominal fluid is also helpful in establishing a diagnosis of cancer. Unfortunately, the case discussed here had metastatic masses along the ventral floor of the abdomen and we could not obtain any fluid.

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PATHOLOGICAL INFECTION OF THOROUGHBRED HORSES WITH *GASTRODISCUS AEGYPTIACUS*

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SUMMARY

Contrary to common belief, *Gastrodiscus aegyptiacus* can be pathogenic to horses. History of cases of a particular stud, necropsy findings, diagnosis, and successful treatment with hexachlorophene and with dichlorvos are recorded. The presence of eggs can be established by a special flotation technique.

INTRODUCTION

The pathogenicity of this trematode, as quoted from the literature reads: 'Little is known with regard to pathogenicity and it is generally not considered important'. This was not always found to be correct.

CASE REPORT

A four-year-old Thoroughbred mare performed a winning gallop. Two hours later, purging was evident. On arrival of veterinary assistance 25 minutes later, the patient was dead. Necropsy revealed peracute colitis associated with thousands of *Gastrodiscus* immatures. It was difficult to conceive that a trematode parasite would be of pathological significance in a selectively stable-fed horse some three years after grazing was terminated. Investigation into the green feed, drinking water and the use of a paddock with a marsh, were all conducted with inconclusive results.

Subsequently, some yearlings were dewormed with a mixture of piperazine adipate, carbon disulphide and tetrachlorethylene. A report was received that some 'funny bots' were observed in the subsequent droppings. Enquiries revealed that these horses originated from the same stud as the initial case recorded above. *Gastrodiscus* sp. were observed again, following anthelmintic therapy of horses emanating from a neighbouring establishment with access to the same river. Subsequently, parasites were also found on a stud farm in Swaziland.

HISTORY

The most heavily affected establishment had a history of recurrent colics which occurred almost weekly in one of six mares. In spite of adequate feeding of all the horses on the stud, emaciation, or some degree of poor muscular development, was evident in the yearlings, as well as the brood mares. The yearlings were all well grown but did not appear to do well, while others were emaciated. Stock were permitted to drink from the river and grazed in a lush moist pasture nearby. *Bulinus* sp. snails were found on the banks but not near the lush pasture. Although fed ideally when racing, the horses from this stud were constitutionally weak and had to be sparingly raced. In spite of some excellent pedigrees, these progeny raced with only limited success.

NECROPSY FINDINGS

The one case which died from peracute superpurgation had a generalized venous congestion with petechiation, and a severe haemorrhagic and oedematous colitis. *Gastrodiscus* sp. immatures were present in the caecum and large colon, while mature parasites were seen in the small intestines and stomach.

DIAGNOSIS

Faecal examination for larvae or eggs by the rapid flotation test was unsatisfactory. Hence the following procedure was used. Fresh faeces were washed well and dispersed in water. The wash was decanted, strained and allowed to stand in a beaker for four hours. Ninety per cent of the supernatant fluid was drawn off by suction. The residue was then centrifuged for two minutes and then decanted once more. Saturated sodium chloride solution was used to dilute the sediment by 50 per cent. The tubes were filled to the brim when in the centrifuge with saline to form a convex meniscus, to which a cover slip was carefully applied. After two minutes of centrifuging, the cover slips were sharply lifted, applied to a slide and examined microscopically. Parasites were diagnosed by the presence of eggs. This protracted test procedure produced. eggs in specimens up to 24 hours old. Specimen egg counts were inconclusive.

TREATMENT

Initially tetrachlorethylene** was used at the rate of 25 m*l*/500 kg with moderate results. Furthermore, no cases of colitis, dizziness, inco-ordination or abortion were encountered in a stud where 50 brood mares, their foals and yearling progeny were all treated. The dose was repeated after six weeks on the presumption that the life cycle was of a similar duration to that of trematodes like paramphistomes and fasciola.

The use of hexachlorophene*** soon followed. This was administered under close observation in some 20 cases. Doses varied from 20-45 m*l*/500 kg. Effects of prelimimary starvation and feeding were both observed. Ideally, for complete stud control, the drug was administered at therate of 35 m*l*/500 kg without incident to all horses including 60 brood mares, many of which were in foal. No liver function tests were carried out. The parasite evacuation was tremendous: 476 parasites were present in one faecal discharge from a heavily infected horse. The subsequent droppings were of a cow dung consistency for 24 hours after administration. No other side effects were observed and one horse won 11 days after therapy.

* P.O. Box 4024, Alrode, Tvl., South Africa.
** Tetrex – Milborrows, P.O. Box 325, Germiston, Tvl. S.A.
*** Bis 356 – Cooper & Nephew, P.O. Box 677, Kempton Park, Tvl., S.A.

Dichlorvos* administered in the feed to a non-starved horse at the rate of 16,6 gm/500 kg, which dose has been found to be most suitable. It was administered to animals treated 30 days earlier with hexachlorophene in which it produced an ever heavier evacuation of parasites than did the first treatment.

DISCUSSION

The specific life cycle of *Gastrodiscus* sp. is not clearly known but it is believed to be similar to that of the other trematodes. The intermediate host is a snail of the genus *Bulinus*. It is not known if it enters through the unbroken skin, like *Schistosoma* sp. (bilharzia) in man or if it is ingested by drinking. The fact that a horse harboured parasites for three years after being exposed, indicates the need for investigation into the life cycle. The literature does not record this parasite as being significantly pathogenic but these observations clearly indicate the incorrectness hereof. As dichlorvos, like other chemicals in this group, is a cholinesterase inhibitor, the effects on racing or working ability warrants further investigation.

CONCLUSION

Gastrodiscus aegyptiacus can be pathogenic to the horse. It will reduce health and resistance in the horse and may even result in death. Diagnosis can be made by examination of faeces, as indicated above. The exact life cycle warrants investigation. Oral administration of hexachlorophene at the rate of 35m*l*/ 500 kg, or dichlorvos at the rate of 16,6 gm/500 kg is suitable.

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MESENTERIC THROMBOSIS

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SUMMARY

A brief review of mesenteric thrombosis in the horse is given, based in part on the author's experience in equine practice in the Karoo area of South Africa.

INTRODUCTION

In the horse, there is a physiological disposition towards abdominal crisis or colic. This results from this species having a highly motile gut. Indeed, ingested food remains in the small intestine for roughly four hours. This indicates that anything adversely affecting this motility can lead to changes and resultant abdominal crisis. This paper deals with the alteration in gut motility brought about by a mesenteric thrombus and subsequent verminous aneurysm caused by the larvae of *Strongylus vulgaris*.

There is some controversy as to whether the condition is an aneurysm or a thrombus. I believe that initially it is a thrombus, because of the thickened walls of the artery in close proximity to the thrombus. Owing to the narrowing of the lumen of the blood vessel, an aneurysm forms proximally to the thrombus where the walls of the vessel are decidedly thinner than normal[2].

AETIOLOGY

Strongylus vulgaris occurs normally in the large intestine where it lays its eggs which are expelled in the faeces. Under optimal conditions, these eggs hatch and undergo two larval changes before being ingested by the horse as third stage larvae.

Once ingested the subsequent larval migratory routes have been a subject of contention. Basically, there have been two schools of thought:

The first hypothesis is that after changing to fourth stage larvae, the larvae penetrate the intestinal mucosa, enter the circulation and travel to the heart and aorta ending up in the mesenteric arteries from where they return to the intestine [4].

The second theory is similar to the first, except that the parasite takes a more direct route to the site of predilection in the cranial mesenteric artery, *via* the intestinal arteries [3].

Recently, Duncan [3] has shown that after exsheathing, the larvae penetrate the mucosa of the intestine, migrate within the submucosa and moult to the fourth stage, causing oedema and marked dilation of the intestinal arteries and eventually thrombi in the walls of the cranial mesenteric artery.

The actual route taken is of minor importance at this stage. What is of importance, however, is that the parasites arrive at the chosen site, irritate the intima of the cranial mesenteric artery, causing arteritis[7]. A thrombus then forms, enveloping the larvae. As the thrombus grows, an aneurysm forms over and dorsal to the site, the walls of the vessel becoming fibrotic and thickened.

The cranial mesenteric artery divides into three branches, namely [6]:-

(i) The left branch, which subdivides into 15 to 20 small arteries supplying the small intestine. To date, no thrombus has been found in these branches.

(ii) The cranial branch which supplies the right and left dorsal colons and small colon. Thrombi have occasionally been noted affecting this vessel.

(iii) The right branch, which supplies the caecum and right and left ventral colons. This branch is by far the most commonly affected.

These vessels are closely associated with the coeliacomesenteric ganglion[1] and any damage to these vessels can, therefore affect the nervous innervation of the gut[5]. Thus the horse has a highly motile gut whose blood and nerve supply can easily become impaired. The result is:

(1) stasis of the affected segment of the bowel;
(2) ischaemia and
(3) drop in blood pressure.

Owing to the stasis, increased fermentation and excessive gas formation occurs. The walls of affected gut become oedematous owing both to blood seeping into area *via* anastomotic routes and to the drop in blood pressure. This blood is not dispersed, so the area becomes congested and haemorrhagic, with a bloodlike fluid and fibrin deposits being present in the lumen of the intestine. The end result is necrosis, gangrene, shock and finally death.

CLINICAL SIGNS

The signs are those usually seen in most types of colic:

(i) Intense abdominal pain
(ii) Increased heart rate and respiration
(iii) Sweating of varying degree
(iv) Lying down and rolling
(v) Injected mucous membranes
(vi) As only one branch of the cranial mesenteric artery is affected, motility of that part of the gut supplied by the other two branches of the artery is present, and although decreased, may be heard with a stethoscope.
(vii) Heart rate increases and finally a systolic murmur is detectable on the right side. This sign is always present in advanced cases of mesenteric thrombosis and is probably due to gross dilatation of the heart.
(viii) Jugular pulse
(ix) The skin over the loins and hindquarters is cold to the touch.

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TREATMENT

This consists of the following measures:

(1) Reducing the pain as much as possible by using pethedine, chloral hydrate, chlorodyne, etc.
(2) Antihistamines and corticosteroids are administered for shock.
(3) Sodium gluconate (a vasodilator of the splanchnic area) is given intravenously (100 g as a 25% solution). This can be repeated every half hour, up to five treatments. This relieves the congestion and oedema of the affected area, as it partially restores the circulation. Recovery time is dependent on the degree of damage.
(4) Supportive therapy in the form of large quantities of intravenous fluids is given.
(5) Antibiotics.

The above treatment has been very successful in cases of mesenteric thromosis.

INCIDENCE

Most cases of mesenteric thrombosis have been seen in Thoroughbreds. The youngest case was a foal, aged five months, diagnosed at necropsy.

The incidence in American Saddle horses is much less. Amongst farm horses and hacks it is virtually nonexistent. The hygiene practised in the Thoroughbred studs compared to the others is very good and most breeders have a routine dosing programme. Manure in the paddocks is regularly cleared away and clean bedding is used in the stables. Farm horses, on the other hand, are not dosed regularly and do not sleep on straw bedding but on manure. The question arises whether we are perhaps lowering the resistance of the Thoroughbred by dosing too often and not allowing the build-up of an immunity of *Strongylus vulgaris?*

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THE PATHOGENESIS AND CONTROL OF STRONGYLE INFECTION IN THE HORSE

J.L. DUNCAN AND J.D. DARGIE*

SUMMARY

The migratory route of infective *Strongylus vulgaris* larvae was determined: penetration of small and large intestine into the lumina of submucosal arteries, migration up the arterial tree, reaching the cranial mesenteric site by three weeks, where the larvae develop to the mature 4th stage. After 3-4 months they exsheath and the young adults migrate down the arteries towards the intestines, to the limit of arterial narrowing, from where they rupture from nodules into the lumen of the intestine. The prepatent period is about 6 months.

The clinical syndrome was similar to, but less severe than that recorded in the literature. The most significant haematological changes were an early, sharp rise in WBC, increased neutrophil:lymphocyte ratio, and eosinophilia. Marked, progressive increase of total serum proteins as result of betaglobulins occurred. The rate of albumin catabolism was higher in infected horses and red cell survival was reduced.

In field studies, it was shown that foals turned out to grass in spring can be infected both by overwintering larvae and by their untreated dams, the latter being by far the more important source of infection.

INTRODUCTION

All horses reared under natural conditions carry a mixed burden of helminth parasites, the major group of which lives in the large intestine and is collectively known as 'red worms' or 'horse strongyles'. Although these parasites are classified into over 50 different species, an arbitrary division is made according to size and those parasites over 1,5 cm in length are termed 'large strongyles', whereas those under 1,5 cm are referred to as 'small strongyles'. Parasitic disease in the horse is due to the combined effect of the adult worms of these species and hence the control of mixed strongyle infection is an important part of the management of any equine establishment.

For the past four years, a research project on various aspects of strongyle infestation and, in particular, *Strongylus vulgaris* infestation has been in progress in Glasgow, and the purpose of this paper is to present the results of some of these studies.

THE PATHOGENESIS OF EXPERIMENTAL *S. VULGARIS* INFECTION

A considerable diversity of opinion has existed on the migratory route of *S. vulgaris* in the horse[7 8 12]. In an attempt to resolve the controversy, a group of nine worm-free foals was experimentally infected with a pure culture of *S. vulgaris* larvae. The foals were killed at intervals over a period of nine months.

From the results of this experimental work[3], there is little doubt that the migratory route is as follows:

Infective larvae exsheath and penetrate the small and large intestine and, within a few days, the larvae in the submucosa cause oedema and a marked dilatation of arteries, veins and capillaries with local haemorrhage. Approximately one week after infection, 4th stage larvae can be demonstrated within the lumina of submucosal arteries of the intestine and there is then a progressive migration up the arterial tree. Numerous larvae have reached the cranial mesenteric site by three weeks, where they develop to the mature 4th stage. After 3 – 4 months, these larvae exsheath and the young adults migrate down the arteries towards the intestine. Nodules are formed on the serosal surface of the intestine when larvae, owing to their size, can travel no further in the arteries; subsequent rupture of these nodules releases young adults into the lumen of the intestine.

These young adults require a further 6 – 8 week period of development until sexual maturity and the establishment of patency, *i.e.*, there is a prepatent period of approximately six months.

These studies on the life cycle of *S. vulgaris* also provided an opportunity to study the pathogenesis of experimental infection. Previously, two reports on the pathogenesis of experimental *S. vulgaris* infection in worm-free foals dosed with 800 – 8 000 larvae[1 5] had demonstrated that a severe clinical syndrome occurred within three weeks of infection. In these experiments, the majority of the animals died or were killed *in extremis* and gross *post-mortem* changes were evident in the candal small intestine, caecum and colon. The essential lesion was infarction of the intestine and the major clinical signs observed during the acute stage were pyrexia, anorexia and colic.

In our study, using doses of 750 infective larvae[4], the clinical syndrome was similar but less severe. During a period of three to four weeks after infection, a constant finding was a rise in body temperature of the infected foals compared with uninfected controls (Fig. 1). During the pyrexic response, the individual animals were usually anorexic and dull, and intermittent colic occurred in three of the infected foals between the 13th and 17th day of infection. Affected animals showed varying degrees of abdominal pain, evidenced by truning round and looking at the flank, discomfort on lying down, kicking at the abdomen, sweating and rolling. Subsequently, infected animals showed little clinical abnormality other than an occasional moderate temperature rise and general unthriftiness. During the course of these infections, the most significant haematological changes noted in the infected foals were an early sharp rise in the total white blood cell counts (from a mean of 10 000 mm^3 to a mean of 17 000 mm^3) and these counts remained high throughout the prepatent period. A further feature noted in the infected group was an increased neutrophil/lymphocyte ratio and an eosinophilia (Fig. 2). These results are essentially similar to, but less marked than those reported by American workers[1].

*University of Glasgow Veterinary School

INDIVIDUAL RECTAL TEMPERATURES OF FOALS FOLLOWING INFECTION WITH 750 S. vulgaris LARVAE

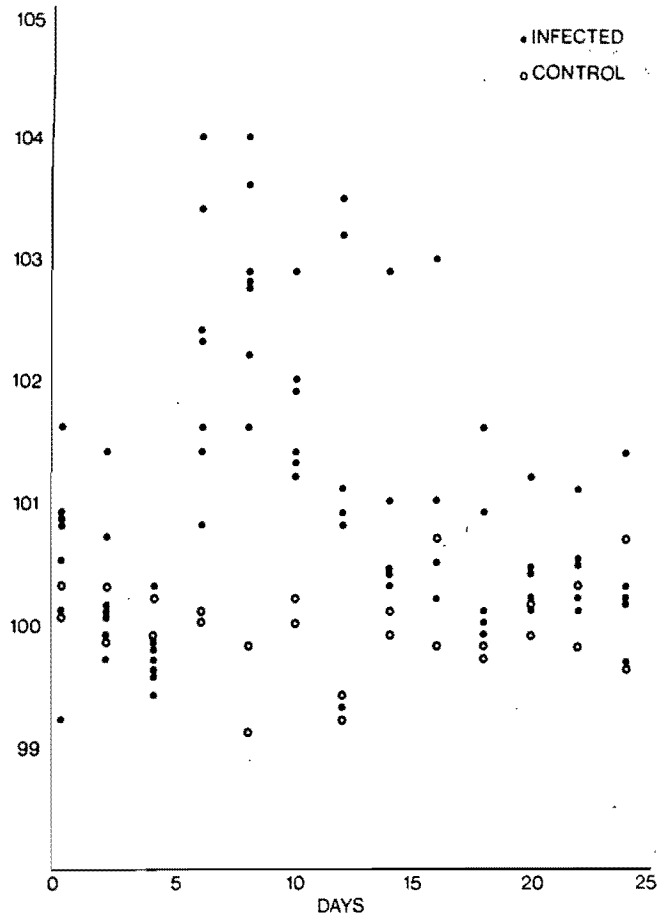

Figure 1

MEAN SERUM PROTEIN CHANGES FOLLOWING INFECTION WITH 750 S. vulgaris LARVAE

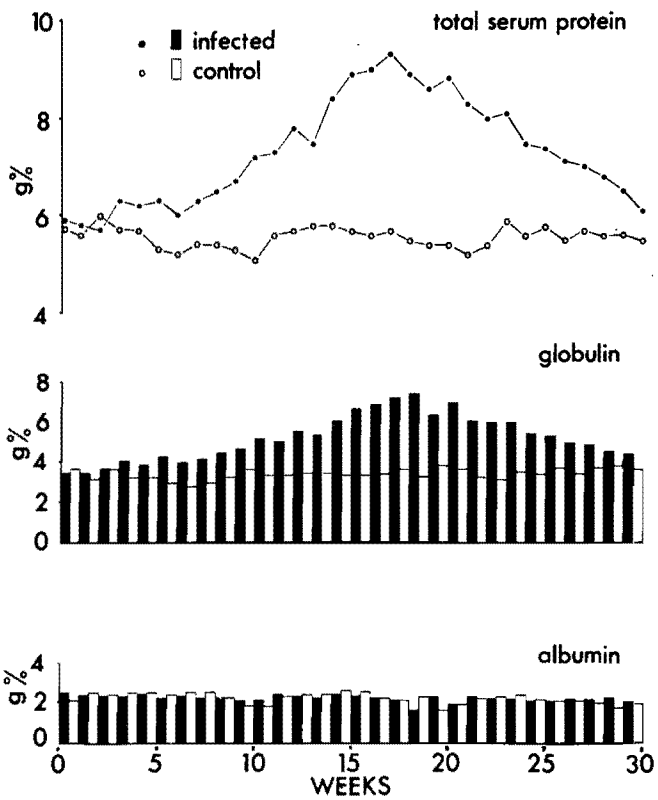

Figure 2

The results of serum protein estimations of the two groups of foals are shown in Figure 3. Marked progressive increases in the levels of total serum proteins in the infected animals are evident and these reached a peak approximately 120 days after infection. Albumin levels of the two groups remained the same throughout the period of observation, the increase in total serum protein being due entirely to elevations in the serum globulins. Electrophoretic analysis of serum on cellulose acetate revealed that the major increase was in the beta globulin fraction. While this may be indicative of an antibody response, as yet no protective effect has been demonstrated.

MEAN WHITE BLOOD CELL CHANGES FOLLOWING INFECTION WITH 750 S. vulgaris LARVAE

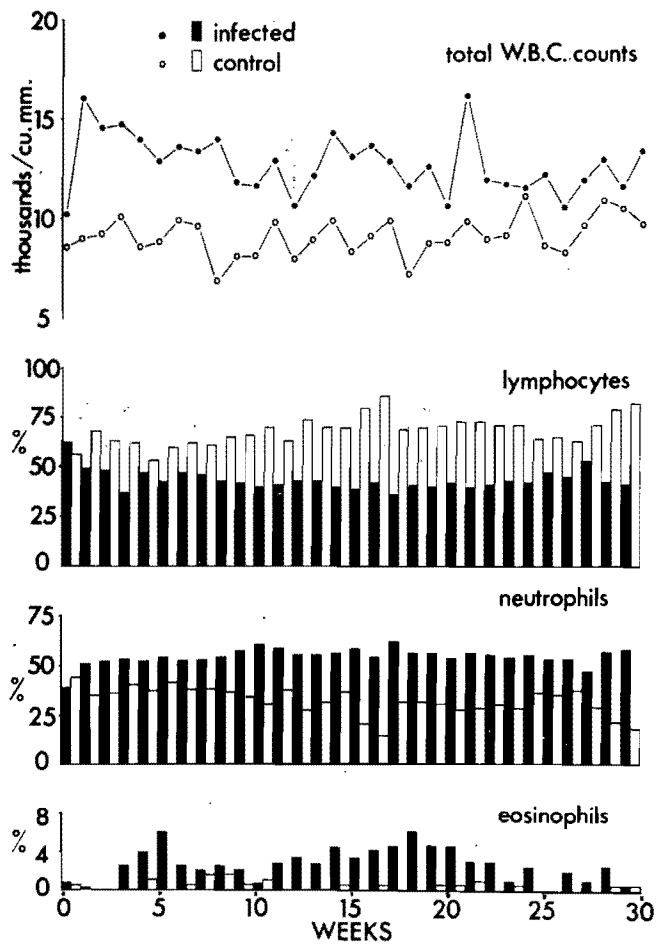

Figure 3

PATHOPHYSIOLOGICAL STUDIES IN PONIES WITH PATENT STRONGYLE INFECTIONS

Detailed information is lacking on the pathogenic effect of adult strongyles in the lumen of the intestine. These parasites are inhabitants of the large intestine and feed by attaching to the glandular epithelium and drawing a plug of mucosa into their buccal capsules. The damage thus caused results in the formation of crater-like ulcers which, in the case of the large strongyles, may extend deeply into the gut wall, and this damage is believed to be the cause of anaemia, unthriftiness and poor performance associated with infection. Since there have been only limited observations on naturally infected animals, an experiment involving the use of radioisotopic tracer techniques was designed to provide more detailed information on the

pathogenic effect of adult parasites in the intestine of both experimentally and naturally infected horses.

Using ^{51}Cr-labelled erythrocytes and ^{125}I-labelled albumin, red cell and plasma protein metabolism were investigated in three groups of animals as follows:

Group 1	Group 2	Group 3
Two worm-free foals	Two foals with patent *S. vulgaris* infections (75 100 adult worms)	Two naturally infected ponies with strongyle faecal egg counts of 100–300 e.p.g.

Each animal received a labelled suspension of its own red cells and ^{125}I-labelled horse albumin by injection. Values obtained for each of the parameters of albumin and red cell metabolism investigated are presented in tables 1 and 2 respectively. In essence, two features distinguished infected and worm-free horses.

First, although serum concentrations and body pools of albumin were similar in all animals, the rate of albumin catabolism was higher in the infected horses (Table 1). This is shown by the shortened apparent 'halflife' and also by the elevated fractional catabolic rates, particularly in the naturally infected group. That this hypercatabolism was due to increased movement of albumin into the alimentary tract is indicated by the higher faecal 'clearance' recorded for each of the parasitised animals (Table 1).

Second, in strongyle-infected horses, red cell survival is reduced as a result of gastrointestinal haemorrhage. This is shown by the increased rate of removal of ^{51}Cr-labelled red cells from the circulation of infected compared with control animals (Table 2) and by the high faecal excretion of isotope expressed as a faecal 'clearance' of both whole blood and red cells. Despite these intestinal red cell losses of up to 30 m*l* daily, anaemia was not clinically detectable.

This experimental work has demonstrated the adverse effect of relatively small numbers of adult parasites in the large intestine; further experiments are

Table 1 : ALBUMIN TURNOVER STUDIES IN INFECTED AND WORM-FREE HORSES
^{125}I-Albumin Results

Group	Animal No.	Serum Albumin	Plasma Volume ml/kg	Albumin Distribution			Albumin Catabolism			Faecal 'Clearance' of Plasma (ml/day)
				CA (g/kg)	EA (g/kg)	TA	Apparent Halflife (days)	K*	Absolute Amount Catabolized (g/kg/day)	
1	1	2,0	42,4	0,85	1,22	2,07	20,9	0,08	0,07	10,3
	2	1,8	51,5	0,93	1,45	2,38	20,9	0,08	0,08	11,4
	Mean	1,9	47,0	0,89	1,34	2,23	20,9	0,08	0,075	10,9
2	1	2,1	47,2	0,99	1,34	2,33	17,9	0,09	0,09	25,1
	2	1,9	49,0	0,93	1,19	2,12	15,7	0,10	0,09	17,7
	Mean	2,0	48,1	0,96	1,27	2,23	16,8	0,095	0,09	21,4
3	1	2,1	38,2	0,80	0,94	1,74	12,5	0,12	0,09	15,7
	2	1,8	38,6	0,69	1,79	2,48	11,4	0,20	0,14	20,0
	Mean	1,95	38,4	0,75	1,37	2,12	12,0	0,16	0,115	17,9

* K – Fractional Catabolic Rate

Table 2 : ^{51}Cr-LABBLED RED BLOOD CELL TURNOVER STUDIES IN INFECTED AND WORM-FREE HORSES

Group	Animal No.	P.C.V.	Circulating RBC Volume (m//kg)	Blood Volume (m//kg)	^{51}Cr RBC T½ (h)	Faecal Clearance (m/ /day)	
						Whole Blood	RBC
1	1	28	13,9	56,3	314	14,9	4,1
	2	25	16,3	67,8	267	18,6	4,9
	Mean	26,5	15,1	62,1	291	16,8	4,5
2	1	27	16,9	64,1	226	34,8	10,0
	2	31	18,0	67,0	232	29,5	9,1
	Mean	29,0	17,5	65,6	229	32,3	9,6
3	1	28	17,0	55,2	215	37,9	10,7
	2	32	13,1	51,7	211	81,6	29,6
	Mean	30,0	15,1	53,5	213	59,8	20,2

planned to examine the situation in naturally infected, heavily parasitized horses.

THE EPIDEMIOLOGY AND CONTROL OF MIXED STRONGYLE INFECTION

The control of mixed strongyle infection in the horse is at present achieved by the use of a variety of anthelmintics under different dosing regimens. Much of the fundamental epidemiological information on which these control measures have been based, has been drawn from field studies on trichostrongylosis of sheep and cattle. Owing to the large number of equine helminths with different developmental cycles, extrapolation of this nature may in fact be misleading.

Some information is available on the incidence and development of various helminths and their prepatent periods[13 14], the lowest number of eggs being passed in the winter, increasing during the spring, to a maximum in summer.

Recent studies on the survival and migration of horse strongyle larvae on herbage plots[10] have demonstrated that in Britain there is a rapid mortality of infective larvae on pasture during the summer but the mortality rate is much lower during the winter months. Thus, a significant proportion of the larvae placed on herbage in late summer and autumn survived until the spring, but at this time there was a rapid disappearance of larvae which coincided with a rise in temperature.

In an attempt to provide epidemiological information on which efficient control measures could be based, field studies were carried out in Scotland over a two-year period using various anthelmintic dosing regimens in mares and foals grazing both infected and clean pastures. Basically, two regimens were employed: one in which untreated mares and foals grazed from the spring on pasture not previously grazed by horses while, in the other, mares regularly treated with anthelmintics and their foals went on to pasture previously grazed by infected horses. These systems were designed to provide two separate sources of infection for the susceptible foals: where mares were treated, the main source of infection was from overwintered larvae; where infected mares went out to clean pasture, infection of the foals could occur only from larvae which had developed from eggs passed in the faeces of the mare. The results of these studies are presented in figures 4 and 5 and table 3 and demonstrate the following important points in the epidemiology of mixed strongyle infection in the horse[2].

Firstly, foals going out to grass in the spring can be infected by both overwintered larvae and by those

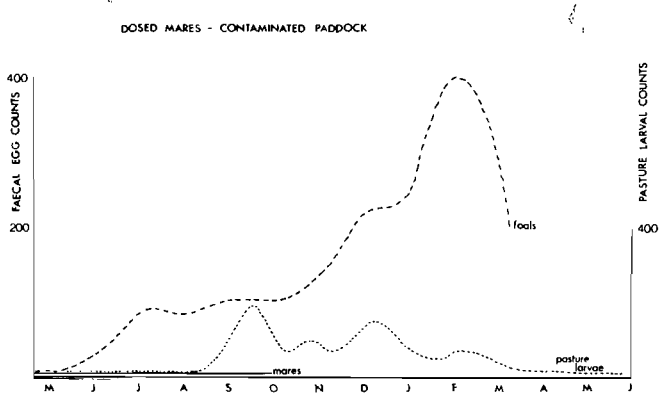

Figure 4

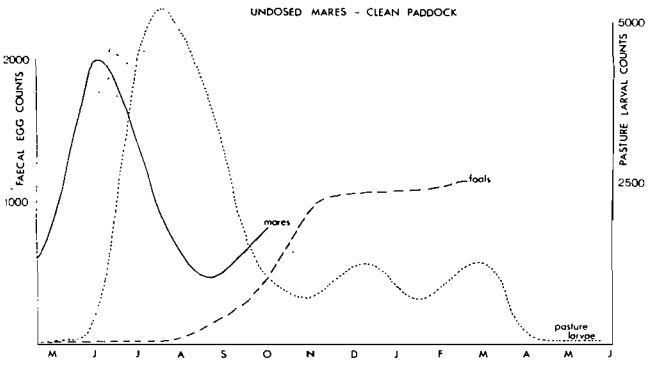

Figure 5

Table 3 : TOTAL STRONGYLE WORM BURDENS IN FOALS

	Foals grazing with dosed mares	Foals grazing with undosed mares
Foal 1	2 159	60 003
Foal 2	3 275	75 397
Foal 3	5 032	36 331
Mean	3 532	57 277

derived from eggs passed in the faeces of their dams, but the latter is by far the more important source of infection.

Secondly, where infected horses received no anthelmintic treatment throughout the grazing season, high levels of third stage strongyle larvae developed on pasture by mid-summer. In contrast, regular anthelmintic treatment of horses at intervals of two to four weeks reduced pasture contamination to a minimum.

Thirdly, although high levels of infective larvae may occur in pasture grazed by infected horses during summer and autumn, these levels fall during the winter and, by the following May, virtually no third stage larvae can be recovered from these pastures.

It is evident from the available epidemiological information that the build-up of parasitic infection can be avoided by the routine use of modern anthelmintics. Where horses are kept in large numbers on limited grazing, a regular dosing programme throughout the grazing season will prevent eggs being deposited on the pasture. This should consist of dosing all animals over two months of age with an efficient broad spectrum anthelmintic every four to six weeks. Foals under two months of age need not be treated, as the minimum prepatent period for small strongyles is approximately eight weeks. If this procedure is adopted and continued for a few years, strongyle populations both in horses and on pasture will fall to an extremely low level. An owner who has only a few horses with ample grazing may increase this dosing interval to two to three months.

When a paddock has been grazed by infected horses for a number of years, ploughing and re-seeding will reduce the pasture larval population to a minimum, but where this is impracticable, the paddock should be rested until the summer months (June) by which time the overwintered larval populations will have largely disappeared. Where possible, clean paddocks

should be provided for nursing mares and foals and it is also important that in-foal and nursing mares should be dosed before going out to grass and thereafter routinely throughout the grazing season in order to minimize contamination of these clean pastures.

Other methods of reducing pasture populations of infective larvae which have been shown to have a beneficial effect are the manual removal of faeces from paadocks once or twice weekly and the practice of mixed grazing.

In conclusion, it should be emphasized that existing management conditions, including the number and value of animals, housing, pasture management and availability of labour, should all be taken into account before recommending any specific parasite control programme.

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TEXTBOOK OF MEAT HYGIENE

H. THORNTON AND J.F. GRACEY

Baillière & Tindall, London, 6th Ed., 1974. Pp. 599, Figs. 219, Colour Plates 4, Public. Price: £8.50.

This is the 6th edition of a now extremely well known "standard" book which was first published in 1949 and has been known to date under the title of "Textbook of Meat Inspection". The new title is welcomed as it more adequately describes the very wide range of related subjects which appear between the covers.

The authors are to be congratulated on the excellence of the manner in which they have revised and updated the previous edition without materially increasing the size and therefore the price of the publication. Newer knowledge, concepts and developments have been expertly woven into the text, and a large number of photographs have been replaced with better ones to bring the reader into touch with modern installations and practice. Each of the four colour plates beautifully depict four types of organ pathology. It is, however, unfortunate that many of these also appear elsewhere in black and white.

A most important change is the regrouping and augmentation of material under a new chapter of nearly 100 pages entitled "Meat Hygiene Practice". This covers important matters such as the transportation of livestock, holding and caring for livestock at the abattoir, ante-mortem inspection, sources of bacterial contamination, food poisoning and bacterial spoilage, sanitation in the abattoir, occupational injuries and infections of abattoir personnel, chemical residues in meat, etc.

Textbooks are invariably deficient in that they cannot include *all* relevant information, and so often they are of necessity out of date in certain respects by the time they are published. Thus recent significant work relative to the life cycles of *Toxoplasma gondii* and *Sarcocystis* spp. is not mentioned in this book. For the South African reader the absence of even brief reference to bovine parafilariasis is also a deficiency. In at least two instances quality of the photographic plates could be better. These criticisms do not, however, in any way detract from the excellence of the work, for which both authors and publishers deserve full credit. The book is indeed an old friend in a new form and will continue to be used on an ever increasing scale by those dealing with a subject of ever increasing importance.

L.W. v.d. H

BOOK REVIEW BOEKRESENSIE

WORLD DIRECTORY OF SCHOOLS FOR ANIMAL HEALTH ASSISTANTS

Published under the auspices of F.A.O. and W.H.O., Geneva, 1974. Pp. 195, Tabs numerous. Price: Swiss Fr.24.

As evidenced in this book, animal health assistants are being trained in numerous parts of the world. Information is presented concerning the administration, conditions of admission, curriculum of the course which frequently extends to four years, examinations, the qualifications given and the conditions under which the successful candidates can apply their knowledge.

There is very considerable diversity concerning each of the requirements under the headings mentioned. Graduates are generally employed to act as the first line of defence against animal disease. In certain instances the courses are directed more at training field assistants while in others there is a greater orientation towards assistance in the diagnostic or research laboratory. In the United Kingdom the training takes the form of qualifying the trainees as animal nurses, a development that is now been followed in various countries and, we hope, in the near future in South Africa. No reference could be found to training of veterinary technologists in South Africa.

This book presents a very useful reference to all those who are engaged in or who contemplate training of personnel beneath the level of full veterinary status. It is particularly interesting to compare the curriculum and general requirements of the course against the background of the animal and human population of the countries concerned and the number of assistants who have obtained their qualifications.

C.F.B.H.

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RUPTURE OF THE CAECUM AT PARTURITION

A. LITTLEJOHN* AND J.D.S. RITCHIE**

SUMMARY

Based on four fatal cases observed in Thoroughbred mares, the signs and *post-mortem* lesions are described. The authors speculate on aetiology, pathogenesis and possible treatment.

INTRODUCTION

This condition, a rapidly fatal rupture of the caecum during the act of parturition in the mare, is well known to veterinarians in Thoroughbred breeding practices, although rarely described in the literature. Voss[3] recorded a case, and Day[1] considered the incidence to be less than 0,1 per cent foaling mares in the Newmarket (England) area.

The following description is based on four cases in Thoroughbred mares.

CLINICAL SIGNS

The mare is apparently normal up to the beginning of parturition. Preparturient behaviour, relaxation of the sacro-iliac ligaments and dropping of wax from teats are normal. Parturition appears to begin normally, at least until rupture of the allanto-chorion and subsequent discharge of watery allantoic fluid from the vulva. It is after this point, during the second stage of labour, that it becomes apparent to the observer that something is wrong. Instead of the normal, powerful straining of thoracic, abdominal and back muscles, straining movements are either weak or absent. Vaginal examination reveals that the foetus is lying in the normal birth position with the forefeet and muzzle within the fully dilated cervix or just posterior to it. If the mare is lying down, the muzzle and forefeet may appear at the vulva. In spite of a fully dilated cervix and a foetus which is normal as regards position and size, the mare appears to be unable to expel it.

Delivery of the foal is accomplished by the veterinarian without difficulty, but subsequent observation of the mare shows that recovery from the stress of foaling does not proceed as usual. Increased patchy sweating, pawing of the ground, looking at the flank, increasing pulse and respiratory rates, and cyanotic mucous membranes, all indicate an acute abdominal catastrophe.

The course is rapidly fatal, and mares succumb approximately four to six hours after delivery of the foal.

PATHOLOGY

At *post-mortem* examination, a tear is found at the lesser curvature of the caecum, 10-15 cm ventral to the ileocaecal orifice. The rupture may be less than 5 cm in length and it is noteworthy that the edges of the tear in the caecal wall curl outwards and not inwards. The extent of macroscopic signs of peritonitis is related to the degree of soiling by caecal contents, but in two of the four cases, it was confined to an area of 2-3 cm in radius around the edges of the tear. Histopathological examination of the caecal wall adjacent to the tear does not necessarily show peritonitis, nor is there any evidence of weakening at the point of rupture. The uterus is normal and shows no lesions of the endometrium, myometrium or peritoneum.

AETIOLOGY

The aetiology of the caecal rupture in so circumscribed an area of the caecal wall remains obscure, but it would appear that at parturition a particular section of the wall of the caecum becomes a target area for the tremendous intra-abdominal pressures of which a straining mare is capable. Hypertrophy of the circular layer of the caecal musculature was recorded by Kalsbeek[2] in his description of a case of caecal rupture and this may well contribute to tearing of the wall.

TREATMENT

No attempt at treatment has been described. Since rupture of viscera other than the caecum have been successfully repaired in horses, it is not unreasonable to propose that repair of the tear in the caecum may be successful in some cases. Unfortunately, access to the lesser curvature of the caecum is extremely difficult even *via* a full-length midline laparotomy incision, owing to the firm attachments of the base of the caecum.

In two experimental Thoroughbred-cross mares, a trans-caecal approach to the area ventral to the ileocaecal and caecocolic orifices was carried out under general anaesthesia. A high right flank incision, 25-30 cm in length, allowed the base of the caecum to be partly exteriorized. The caecum was then incised the full length of the laparotomy incision, whereupon the edge of the caecocolic orifice was firmly grasped and brought out, thus bringing the area of the lesser curvature of the caecum within surgical reach.

DISCUSSION

Since peritonitis in some cases is insignificant, the primary cause of the rapid collapse and death appears to be cardiovascular rather than inflammatory. Neurogenic factors resulting from acute pain may be important in this respect, and prompt recognition of the syndrome followed by general anaesthesia, oxygenation, laparotomy and repair may eventually prove of value in selected cases, where soiling of the visceral peritoneum is minimal.

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THE HORSE'S HEALTH FROM A TO Z

P.D. ROSSDALE AND SUSAN WREFORD

David and Charles Newton Abbot, London and Vancouver, 1974. Pp. 433. Price: £5.25

It is extremely difficult for a veterinarian to assess a book which purports to define for the benefit of the layman, the terms used by veterinarians to describe the ailments of horses: any attempts at objective evaluation are inevitably thwarted by the reviewers own background and veterinary education. However, when the dictionary claims to be indispensable to veterinary students, one feels on surer ground.

Since "The Horse's Health from A to Z" is subtitled "An Equine Veterinary Dictionary" and concerns itself with defining veterinary terms in the English language, one may be forgiven for the hope that it would have served as a model for generations of future veterinarians. Unfortunately, this is not the case. The inconsistencies and basic inaccuracies preclude the use of this as a textbook for veterinary students. Admittedly, some of the inconsistencies are stated (although not justified) in the author's preface which is at pains to point out that dipthongs have been omitted from the centre of words such as anaemia, haemorrhage and haemoglobin, but retained in words in *less common* use such as amoeboid, anaesthesia and caecum. Dipthongs are also retained when they occur at the beginning of words such as oestrus and oedema. Such inconsistencies may be minor sources of irritation only to the purist, but they have no place in a dictionary: nor do misspellings such as carpel (for carpal) and follicile (for follicle).

Some definitons are patently inaccurate, e.g. "Brucella abortus" is defined as "Abortion caused by Brucella genus of bacteria". Under the entry "Helminth" one is advised "See tapeworm". The term colic is defined as "Pain in tubes or ducts of abdomen" — a definition with which not all veterinarians would agree.

In spite of its many inexactitudes: there is little doubt that the book will find a place on the bookshelves of many a horse-owner. One wonders, however, whether such entries as "soap" or "sneeze" or "sound" are strictly necessary for horse-owning laymen.

It is not a book which can be recommended for modern veterinary students.

A.L.

BOOK REVIEW BOEKRESENSIE

HOW TO BREED AND WHELP DOGS

J.S. HANSEN

Charles C. Thomas, Springfield, Illinois, 1972. Pp. 250. Prys: ±R10,00.

Hierdie boek kan sonder huiwering aanbeveel word. In werklikheid behoort elke kleindier-praktisyn 'n paar kopieë aan te koop wat hy op 'n bruik-leen basis aan sy kliënte beskikbaar kan stel.

Pertinente inligting oor die aankoop en versorging van aanteelhonde, Veeartsenykundige ondersoeke van die teef voor en na teling, voorsorgmaatreëls gedurende die dragtigheidsperiode, verloskundige ingrypings, asook inligting met betrekking tot stamboekregistrasies en die administrasie van hondeboerdery in die algemeen word in 'n maklik leesbare vorm weergee.

Alhoewel dr. Hansen ('n kleindier praktisyn) sy boek vir hondetelers aanbeveel kan veral praktisyns wat nog nie die geleentheid gehad het om die "taal" van die hondetelers te bemeester nie, gerus ook van hierdie boek gebruik maak.

Waarskynlik as gevolg van die skrywer se ywer om wetenskaplike feite tot verstaanbare inligting vir die algemene publiek te verwerk word soms dogmatiese en selfs iewat verdraaide stellings gemaak. Hierdie probleem kan egter baie gou reggestel word indien die teler met sy veearts kontak op neem.

A.P.S.

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TREATMENT OF DIARRHOEA IN THE HORSE

A.M. MERRITT*

This presentation crystallizes personal ideas as well as the experiences of the equine group at New Bolton Centre and of other colleagues.

The most important single step in treating acute diarrhoea is administration of intravenous fluids, in the process of which the indwelling jugular catheter is used as a routine. The amount of fluid to be administered depends on the degree of dehydration, based, in the final analysis, on percentage loss of body mass. In clinically evaluating the degree of dehydration skin turgor, PCV and total protein estimations are used. The reliability of the latter two is affected by the occurrence of haemorrhage.

The preferred fluid is a balanced, isotonic solution, prepared at our hospital of the following formulation:

FORMULA

	Grams/Litre
Sodium Chloride (MW 58.44)	4.87
Potassium Chloride (MW 74.56)	0.370
Calcium Chloride ($CaCl_2 \cdot 2H_2O$, MW 147.02)	0.220
Magnesium Chloride ($MgCl_2 \cdot 6H_2O$, MW 203.33)	0.303
Sodium Acetate ($CH_3COONa \cdot 3H_2O$, MW 136.08)	3.672
Sodium Proprionate ($NaC_3H_5O_2$, MW 96.06)	2.208
Sodium Phosphate (Na_2HPO_4, MW 141.96)	0.213
Propyl-hydroxy-benzoate	1.600
Methyl-hydroxy-benzoate	14.400
1.0 N Hydrochloric Acid	8 ml/litre

Cations/Litre		Anions/Litre	
Sodium	137 mEq	Chloride	95 mEq
Potassium	5 mEq	Acetate	27 mEq
Calcium	3 mEq	Propionate	23 mEq
Magnesium	3 mEq	Phosphate	3 mEq

Acetate is used in preference to lactate, as it can be metabolized with greater facility. In contrast to commercial solutions, which also contain gluconate, we use proprionate. Acetate and proprionate are metabolized to yield bicarbonate radicles.

As a routine 8 litres are run in at a definite speed. In acute, and particularly in haemorrhagic colitis, it is run in as fast as possible; We will give at least 16*l* and continue from there depending on the evaluation of the degree of dehydration.

About 99 per cent of all cases of acute diarrhoea have metabolic acidosis, although the very occasional case with alkalosis has been encountered. Hence bicarbonate has to be administered. In the absence of blood gas determinations, an estimated 10% dehydration would indicate roughly a bicarbonate deficiency of at least 10 mEq/*l* of extracellular fluid. The amount of extracellular fluid is over-estimated so that a 500 kg horse would require 500 x 0,4 x 10 = 2 000 mEq total. Fifty grams of commercial chemical grade sodium bicarbonate per litre would yield approximately 600 mEq/*l*, *i.e.*, 200 g would yield 2 400 mEq/*l*, close enough for practical purposes. This is added (50 g/*l*) to the balanced fluid already described so that the horse in the above example would receive extra bicarbonate in the first 4*l* of fluid.

Potassium replacement is essential. The amount to be administered had best be calculated from serum potassium determination and the amount of extracellular fluid, estimated at 0,3 of body mass, according to which a 500 kg horse would have about 150 *l* of extracellular fluid. If the deficiency is 1,5 mEq/*l* (normal level 3,5 mEq/*l* minus serum potassium determined at say 2,0 mEq/*l*), then 1,5 x 150 = 225 mEq has to be added to the base solution. (By way of interjection Dr. Donawick indicated that commercial KCl power usually contains 370 mEq/K in every 30 g.) The addition of potassium and bicarbonate would render the base solution somewhat hypertonic.

Maximally about 100 mEq/*l* K are administered intravenously per hour. If replaced too fast, it can be lost again. It probably could be administered faster without running into cardiac difficulties.

In our experience giving electrolytes orally – essentially Darrow's solution – even combined with dextrose, to horses with acute diarrhoea, exacerbates the condition and all ground gained by intravenous fluid therapy is lost.

The use of oral antibiotics must be avoided assiduously, especially in acute diarrhoea to avoid the risk of upsetting the intestinal flora even more. If absolutely needed, antibiotics should be given parenterally.

The polemic about the value of antibiotics in acute salmonellosis continues. In man, antibiotics like neomycin appear to increase the duration of the 'carrier state' with little evidence of a beneficial effect. Dr. Coffman is invited to comment on pretreatment with chloramphenicol. Once the diarrhoea has manifested itself, the course in most cases is not altered by chloramphenicol treatment.

Concerning peritonitis, which may bring about diarrhoea, it is very much a case of determining the cause. Surgical intervention may be indicated. If the casual organisms can be cultured from paracentetic fluid, antibiograms will give an indication what antibiotics to use.

Acute diarrhoea caused by *Corynebacterium equi* is the one instance where the course of the disease may be influenced favourably by parenteral antibiotic therapy; chloramphenicol at 4g/500 kg *q.i.d.* is the drug of choice, but it is expensive.

In strongyle larvae migrans, ten times the recommended dose of thiabendazole is given – 20 g/50 kg – two days in a row, as suggested originally by Dr Drudge of the University of Kentucky. It has not helped in all cases, perhaps because of incorrect diagnosis. Cases with painful cranial mesenteric arteritis and considerable peri-arteritis have had subsidence of the reaction, regression of the lesion's size and of the diarrhoea. Sometimes this treatment alone is not succesful and iodochlorhydroxyquinoline (Rheaform-Squibb) a protozoocidal drug, given as follow-up, provided the desired result, although the rationale remains unknown. Cases treated initially with Rheaform in vain and subsequently proving to be

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due to migrating strongyle larvae, have responded to the described massive thiabendazole therapy following by Rheaform for three to four weeks.

"Azulfidine" (salicylazosulfapyridine) has been used quite successfully in combination with corticosteroids in regional ileitis of man. It has been used quite empirically on horses. One case did very well; another died very suddenly.

Chronic liver disease is not considered very amenable to therapy.

Many forms of treatment have been advocated for chronic diarrhoea: changing the feed, sour milk, yoghurt, suspending water intake, etc., but generally the only remedy that has had any consistent effect is thiabenzole and Rheaform and even then about half the cases relapsed as soon as the latter drug was withdrawn.

Finally there are cases with 'cow-like' faeces that otherwise appear healthy. If no cause can be ascertained and no other signs or symptoms can be established, they are put back to work.

Relative to Thoroughbreds, most cases of diarrhoea occur in Standardbreds in my experience.

DISCUSSION

J.R. Coffman: Every academic clinic on occasion gets hit with salmonella cases. For us to be forewarned, all of our surgery cases for way over two years, every Monday, Wednesday and Friday undergo routine haematology correlated with daily temperatures. We developed this practice after retrospectively going through previous cases of acute salmonellosis and finding that they all had a precipitous leukopaenia at the onset. Once these horses are clinically ill with diarrhoea, they are, as you are aware, extremely difficult to treat. If one finds an abrupt drop in leukocytes, principally neutrophils and a rise in temperature and one starts right away giving 5 g chloramphenicol intravenously *b.i.d.* the cases will do well, as compared to those on which treatment was started after diarrhoea had developed.

I would agree one hundred per cent that the oral antibiotics are contra-indicated but the interception of diarrhoea by haematology and temperature and early treatment before problems do begin have been quite successful.

Dr. Merrit made the comment about chronic liver disease not being particularly amenable to therapy. I would like to query that. In the chronic liver diseases, that we diagnose on the basis of elevation of LDH, SGOT and sometimes of sorbitol dehydrogenase, and which have been chronically ill with all the typical criteria of chronic liver disease, there is a need for high quality protein and for control of inflammatory cellular changes, as well as for a highly available glucose source. So we just keep these horses on a normal ration and give them a pound of soya bean meal and a pound of table sugar a day, and give them 10 mg dexamethazone a day for the first week. The majority seems to do pretty well, I would be interested in comments on this.

A.M. Merritt: I could not argue against that. I think it is logical. If one has increased enzyme levels, one has a chronic, active disease. I guess the cases that I related were pretty well advanced in all the horses. You do not use a lot of antibiotics, do you, in those instances?

J.R. Coffman: No.

W.J. Donawick: In estimating what the extracellular fluid volume might be, we found through experience that we can safely double or triple the amount. The real purpose is that we are concerned about the potassium ions in the extracellular fluid space and by finding that it is decreased, we assume it is decreased in the cells. Really what we are trying to do, is to get potassium back to the cells, so we try to estimate a dose that will not be lethal to the horse, a safe dose, and certainly that is what is done, just bringing the extracellular fluid volume back up to the normal level, theoretically. But it never does that, so one may double it easily without worry and maybe get a little bit more back to the cells.

L.B. Jeffcott: I just want to ask Dr. Merritt whether, in some of his chronic diarrhoea conditions, he has ever cultured fungi from the faeces. I am not suggesting, and I do not have any evidence for it, that fungi are a primary cause, but from what we have seen over the last few years, one can find fungi and the cases will respond to doses of mycostatin. I would like some comment on that point.

What bothers us in England about the salmonella problem in horses with diarrhoea is, what to do if one cannot treat them. What about the animals in contact with such cases, or is it perfectly alright if one just puts a bucket of disinfectant outside the door and warns everybody that salmonella is around? Is one not potentially infecting the rest of the animals in the stud.

On the question of *Corynebacterium equi,* one aid to diagnosis which we have found particularly useful in these cases which have quite large abscesses or infected lymph nodes with a good deal of purulent material, is the marked rise in the α_2-globulin. We are able to use the level of α_2-globulin as a prognostic factor. If it goes very high then we are going to lose the case, if it does not go too high, then it will recover. I was interested to hear about the chloramphenicol treatment. I want to ask Dr. Merritt whether he has tried high doses of penicillin or penicillin and streptomycin, because in *vitro* sensitivity tests show that the organism is sensitive to penicillin and streptomycin.

A.M. Merritt: Your remarks about *Corynebacterium equi* are very interesting in terms of the α_2 globulin, we should look into this. Concerning the fungi, I suspect you were talking about *Candida albicans*?

L.B. Jeffcott: No. They were of the *Mucor* species.

A.M. Merritt: We have not recognized that. Perhaps we were not looking properly for it. It would be another one on the list about which we could talk and I could learn.

L.B. Jeffcott: It may be purely secondary, I just wondered whether you had recognized it.

A.M. Merritt: No, I have not recognized it.

Concerning salmonellosis and whether in fact one gives antibiotics or not, I recall that in those animals treated systemically, we have had shedding in spite of the fact that they had been under treatment. The remarks that I made regarding the use of antibiotics are based upon papers that came out in the 'Lancet' a few years ago, in which it was indicated that people who had been treated tended to remain shedders longer than those who had not been treated by antibiotics. I think that one cannot depend on therapy to cut down the shedding but I do not think it completely settles the question.

As regards penicillin treatment for *Corynebacterium equi,* we have tried it at times, without much success, at least in our hands, so nowadays I do not take any chances.

C.F.P. Irwin: I would like to pose some questions to Dr. Merritt. Firstly, in Australia we probably see more *Corynebacterium equi* in Melbourne than anywhere else in Australia, or, for that matter, anywhere else in the world. We have found *Corynebacterium equi* is a common form of saprophyte. It can he recovered from the faeces of the clinically normal animal. So I rather wonder whether his *Corynebacterium equi* cases have scoured as a result of that particular infection as such. Certainly in the extension of the lung form which we do get very marked enlargement of the abdominal lymph nodes but no diarrhoea.

Secondly, whilst on this subject, in Australia treatment seems to vary. There is one area where chloramphenicol is widely used, in another area massive doses of streptomycin are combined with penicillin. They are massive doses and,

certainly in my own area, we have found this to be far more effective than chloramphenicol.

Coming to thiabendazole treatment, I myself, and other practitioners, have noticed there is a quite marked resistance to thiabendazole developing in the strongyles and I would like to know if they have also noticed this in the U.S.A.

Fourthly, I think he referred to the alopecia in severe strongyle infestation. I wonder if this is not adrenal exhaustion, which can be observed in the race-horse, which starts with lesions around the eyes, and in which it has been found that it can be controlled with weekly injections of dexamethazone.

Lastly, I would like to ask if you have tried intravenous formalin for controlling scoures.

A.M. Merrit: We have not tried intravenous formalin for controlling scours. We do not routinely culture *Corynebacterium equi* from the faeces of horses. We have done numerous cultures, which I am sure you have too. I am quite sure that in a number of cases, particularly those that died, that we had been dealing with *C. equi* diarrhoea, because we had the typical lesins of it, which included multiple ulcerations, from which you could culture the organism and lymph node involvement as well. I do not think this is 100 per cent effective as a diagnostic tool and I certainly stand by that. All I can say is, when we are presented with a foal of six months of age with an onset of acute diarrhoea, it is one of our problems of differential diagnosis. We go through a list of differential diagnoses that I went through here and we try to look for organisms in the stool. If I see them there, and particularly in the Gram Stain, I feel quite confident that that is probably what we are dealing with. I cannot comment on your success with penicillin, I can only say our results have been better with chloramphenicol, but perhaps we had not given large enough doses of penicillin.

Thiabendazole resistance we have seen too. The routine now in the States is to alternate between thiabendazole,perhaps mebendazole and dichlorvos on a yearly programme and not just to stick to thiabendazole. Thiabendazole was selected in the first instance because it is relatively non-toxic and because we had had indications that it is effective against larvae migrans, in certain instances at least. I do find, when we give animals ten times the dose, they go off feed for a day or two – a day at least – but I have never seen any other untoward effect of this therapy.

With regard to the dermatosis, it is pretty well established that the gut can reflect skin problems and the skin can reflect gut problems, at least in man. There are numerous diseases in which this combination is apparent. We have tried dexamethazone on a couple of the cases without good results but in one case that looked similar we got excellent results. That animal finally went to autopsy and no lesions of any importance affecting the gastrointestinal tract could be found. I am not sure what you mean by adrenal exhaustion, unless it has been demonstrated that such cases were low in circulating corticosteroid levels or that adrenocortical loss was demonstrated at autopsy. We did not do any adrenal studies. But if the question is well taken, I tend to associate it in a few of these cases with the strongyle problem and assume that there is some association between gut and skin; I have not got any further than that with it. I would hate to have it as a solace. Again, when considering a differential diagnosis, one has to take note of this aspect.

H.C. Kalsbeek: I would like to ask Dr. Merritt if he has seen ulcers in the stomach in the course of common diarrhoea. We have seen it sometimes, but have not been able to diagnose it. I should like to know if he could, and in which way. That is the first question.

Dr. Merritt also mentioned a tear in the rectum with subsequent acute peritonitis as the cause of diarrhoea. I cannot imagine that to be the case.

The third question is: have you used an agent that decreases bowel movements in attempting to relieve diarrhoea? We, at times, use tincture of opium and perhaps add kaolin, sometimes with indifferent results, but sometimes it helps. I should like to know if you have used any of these drugs.

A.M. Merritt: We have not seen any ulcers in the stomach in adult animals to which alone I could attribute the cause of diarrhoea. Maybe Dr. Coffman has and could make a statement in this regard. I am sure it could occur; we just have not seen it. We have seen ulcers in foals and they just present acute peritonitis and not ulcers with a diarrhoea. Again, I am not trying to suggest that every case of acute peritonitis has diarrhoea or that it always results from a tear of the rectum. These are just possibilities when considering the differential diagnosis. I cannot recall a particular case of acute peritonitis owing to a rectal tear to cause diarrhoea, although I know cases of acute peritonitis where we have seen diarrhoea as a manifestation. It is only if you are suspecting it then you should be looking for causes of the peritonitis, and that would be one of the possible causes. As far as the opiates are concerned, no, I have not had any experience with them. All that I know is that there are people in this audience, like yourself and the Newmarket people, who might consider these drugs helpful while the animal is on them. Probably, once you take them off, they get diarrhoea again. This is our experience with a number of drugs, even with Rheaform. I could hardly stand here as an expert on treatment on chronic diarrhoea, because I do not know much about it either. Some of the topics I dealt with were possibilities which have to be taken into account. Yes, I could have used the opiates but we do not use them. We have sometimes slowed down the signs with atropine but again as soon as treatment is stopped, the animal starts to scour again. Kaolin and pectin we feel have not been helpful in the management of these cases. That is a personal opinion.

C.J. Roberts: I have not had the experience of *C. equi* that the Australians have had, but it does surprise me that you associate it with scouring. In the few cases that I have seen, it tended to be the opposite of what the Australians have stated.

A.M. Merritt: I should say that most of our cases are also respiratory. All I am saying is that, in a foal of that age, with an acute onset of diarrhoea, one ought to consider *C. equi* as part of the differential diagnosis. There is no question that the majority of cases that we have seen, certainly have bronchopneumonia with it or as a primary sign. I would not argue against that for a minute. It has to be considered in the differential diagnosis of acute diarrhoea in horses of that age.

C.J. Roberts: Yes, I will admit that my experience with it is fairly limited. What I actually got up to talk about, was salmonellosis. We get a lot of this in New Zealand. I am surprised also to find that the drug Trivetrine by Burroughs Wellcome, also put up as Galben by Hoechst, has not been mentioned. I have found it to be highly effective: it is a wonder drug to us. You did not mention the prevention of salmonellosis. We in New Zealand vaccinate the mares a month before foaling: it gives a passive immunity for one month and we then inject the foal at a month old.It is very efficacious. In fact, I have been using the vaccine myself and as a result have found no sign of salmonella. As to the spread of infection, I can tell you that the first thing you want to do is get that infected animal out of the way, because if you have got him amongst young susceptible animals, it will go through them like wildfire. As a clinical aside, is anyone aware that one can get salmonella abscesses, from which one can obtain salmonella in pure culture, and that from abscesses in the pleural cavity.

A.M. Merritt: I cannot deny anything that you have said. We have not seen any publications regarding the efficacy of vaccination. In studying the literature we found very controversial opinions and not much at all about the real efficacy of vaccination. It is certainly an advantage that you can do it. With what you have said about the control of salmonellosis, I heartily agree. I have had no experience of the drugs which you have mentioned and would accept your word for it.

C.J. Roberts: The drugs Trivetrin and Galben, as far as I know, are identical. They are sulpha drugs.

A.M. Merritt: I have not had any experience with the sulpha drugs.

L.B. Jeffcott: In England I have tried Trivetrin in cases of salmonellosis and it has not cleared them up at all. There is another point I would like to make regarding *C. equi*. Having had a number of these, I have found that, although the primary symptoms are respiratory, one will also find some form of alimentary lesion, however small, even if it is only in one mesenteric lymph node. This is in accord with the epidemiology. We know it is a soil organism and that foals will pick it up in the alimentary tract; then there is haema-

togenous and/or lymphogenous spread from the lymphoid follicles of the gut wall and mesenteric lymph nodes to the lungs and bronchial lymph nodes. One thing about salmonella abscesses: Salmonella is a great organism for producing pus and certainly chronic salmonella infection can produce abscesses up to a degree where Salmonella can stimulate *C. equi* infection in the lungs, with full-blown abscesses spread throughout these organs.

GENERAL DISCUSSION ON ENTERITIS AND COLITIS

(Dr. Merritt's *ex tempore* presentation on 'Treatment of Diarrhoea' and ensuing discussion – see page 89 originally formed part of this General Discussion.)

Chairman: G. FAULL

Dr. L.J. Loots: I would like to confirm the occurence of *Gastrodiscus*. We did an anthelmintic trial on 22 donkeys coming from north-western parts of the Transvaal. They were stabled the whole time we had them. They were fed teff hay during the trial but we do not know where it came from. One donkey passed *Gastrodiscus* after treatment but at autopsy no other specimens of *Gastrodiscus* were found.

A point about the safety of dichlorvos.

In a critical trial put up specifically to test the remedy of registration purposes, two horses, one of either sex, were given ten times the recommended dose, and two donkeys, again of either sex, received twenty times the normal dose. Close monitoring of blood cholinesterase levels did not indicate any deviations beyond tolerable limits. The only signs occurred in one donkey: the first day it was off colour and had 'cow's stools but otherwise remained healthy. Administering dichlorvos at such high dosages creates quite a problem. It is put up in plastic coated form and does not dissolve. Giving it by stomach tube means virtually stuffing it down trocarwise with a wire and washing it down with such copious quantities of water, the animal almost drowns.

H.O. Flanagan: This is just a comment. I was interested to hear Dr. Azzie say that he found these parasites in the gastric area. This form of parasitism is quite a problem in Rhodesia; of all the autopsies I have done on horses, and in which I have found *Gastrodiscus*, it was never found in the stomach at all: it has always been in the caecum or the large colon.

M.A.J. Azzie: In reply to Dr. Flanagan's comment, we did observe, in animals that had been dead for more than twelve hours, that all the larvae and adults had migrated into the lower part of the intestine. It might be a question of how long the animals had been dead before they were autopsied.

J.G. Gaenssler: I have been informed that, as far as treatment against *Gastrodiscus* is concerned, resorantel (Terenol) at the rate of 65 mg/kg gives reasonably reliable results.

W.H.S. Bellinge: We are beginning to get quite a problem with *Habronema musca* infestation in Western Australia and I would like to know if either Dr. Duncan or any other helminthologist could prescribe a good treatment, because our cases do not seem to respond to any of the treatments we are using.

J.L. Duncan: The reason that they do not respond well to treatment is that *Habronema* produces a thick layer of mucus protecting it within the stomach wall. There are some reports on the use of gastric lavage before treatment with the prospect of removing the mucus and that the method has had some success. I agree, it is very difficult to treat and usually unsuccessful.

M.A.J. Azzie: I would like to ask Dr. Duncan one question with regard to migration of strongyle larvae. He has indicated in his paper they are associated with the cranial mesenteric artery. In the literature there is mention of migration to different areas in the body. My specific question is, has he observed any tracts of the strongyle larvae in the region of the aortic/iliac bifurcation?

J.L. Duncan: A quick answer is, yes. We observed migration tracts beyond the cranial mesenteric artery. If we had had better light in the foal that was killed at three weeks after infection, such tracts would have been seen quite easily. These larvae come up more like an overflow and migrate backwards and forwards. The difficulty that has been experienced in the past is that so many people had been looking at naturally infected horses: they would not be able to discern what the uncomplicated infection site and migration route would be, whether it has been altered in some way and has become unbound when grazing animals are subjected to succeeding waves of infection, perhaps a low grade infection, rather than a simple experimental infection. But certainly, in naturally infected horses we find lesions which could be attributed to *Strongylus vulgaris* as far forward as the aortic valve, and extending into the iliac and renal arteries.

K.J. Barty: Has Prof. Littlejohn given consideration to the possibility of the injury to the caecum being caused by the hoof of the foal. It is very similar to the appearance of a wound in the rectum, when, say, one gets a hoof going through the rectal wall. This happens while the hoof is still covered by the vaginal wall. One gets this round-edged wound in the rectum which has the same appearance as the round-edged wound one encounters in caecal rupture.

A. Littlejohn: Yes, we have given thought to this. It is quite possible that the hoof of the foal at parturition does play a rôle and I would not deny it. The only thing that is very puzzling is that one does not find any evidence of trauma in the uterus at all. At least, I have not, perhaps other people have, but it certainly is a very difficult thing to explain. Also, the fact that the tear is invariably in very much the same area, is rather puzzling: one would hate to imagine that a whole series of foals would have such a good aim. Yes, we certainly have given this matter thought and I am not denying that the hind hoof of the foal might be an important factor, but how this all works and ties together, I really do not know. I would be glad if anyone could comment on this.

C.J. Roberts: We have had a considerable number of cases of this. We believe that it is due to *Anoplocephala perfoliata*, otherwise known as *A. magna*. It has been reported, by the way, as being the cause of caecal rupture. Our problem, when we looked at the ruptured site, was very often we could not demonstrate any *Anoplocephala* there. But if we put our arm in the ruptured site, and went in up to our elbows, there they were, like a lovely little nest of bees, handfuls and handfuls of them. Now let us look at the situation where one has a lot of *Anoplocephala* at the base of the caecum and through some reason or other they have weakened the bowel structure and have caused it to rupture. It is reasonable to assume that these parasites are good examples of living organisms and that they are capable of an avoidance reaction. They do not want to be shed out in to the peritoneal cavity. I believe, if one goes into their structure, one will find that they are capable of moving. I think what happens is that all these parasites 'decide they do not want to be in the peritoneal cavity' which is not their environment and by the time you open the horse up and look in, these parasites have already migrated up the bowel. I have known this to happen twice to me at home, where veterinarians in our practice have encountered colic, suspected *Anopolcephala* because there was a caecal rupture, but could find no *Anoplocephala* at autopsy. In both instances I put my hand into the lesion, well up to the base of the caecum, and pulled out a handful of these parasites.

Another point is we do not only get it in foaling mares. I will give you a case history. One morning, whilst doing an ovarian palpation, I looked across the paddock and there was one of my own mares, just starting to scratch the ground. I thought: "She is going to foal, but she has not made much of it. She is two weeks away and does not look like foaling." By the time I had completed the palpation she started to turn around, with all the signs of colic and went down onto the ground. I thought she was going to foal, anyway. I put my hand in: there was no relaxed cervix, nothing to indicate onset of parturition. On the basis I suspected caecal rupture, which suspicion proved to be correct. That mare died two weeks later. I had the same thing with another mare.

This always seems to happen in our State, in Auckland, where I come from, and not further south in New Zealand, where it is colder. The reason we believe is there is an oribatid mite that is part of the lifecycle. We believe the population of oribatid mites flourish just under those conditions of our spring. We get them in September, early October, in the damp season.

Caecal tears are not always associated with foaling. I presume the tapeworms weaken the bowel and, with the pressure during foaling, the caecum ruptures.

A. Littlejohn: Thank you, Charlie Roberts, those are most interesting observations as far as I am concerned. I must admit our pathologists in Edinburgh did not find any *Anoplocephala;* I certainly have not myself, I am not sure if there is a problem in the United Kingdom. The cases that I have seen, have only been associated with foaling, but obviously there is a spectrum of this condition, which is not necessarily associated with parturition. Perhaps one of our parasitologists would care to comment.

J.L. Duncan: This is venturing into the tapeworm field. In horses which are autopsied for other reasons, we do find *Anoplocephala perfoliata.* We found numerous specimens all over the place that were apparently causing no damage at all. This is the only comment I can make. It is an oribatid mite which is the intermediate host. It is very difficult when one is dealing with a parasitized horse, to assign a particular pathological state to a particular parasite. There are so many parasites and the finding of bots or *Anoplocephala* or what have you, is often dramatic, but not necessarily the aetiological answer. It may in fact not be the cause of the trouble. Did you look in these animals with *Anoplocephala* to find out if there was any other reason, for example, cranial mesenteric arteritis? Did you examine anything else, or was it simply a matter of finding *Anoplocephala?*

C.J. Roberts: No, but one thing that I did neglect to say, was this. We have treated the horses for *Anoplocephala* using a cestocide. (I have not used Banminth but they say it is very effective.) The one point is that we have been encountering sporadic cases of caecal rupture, we have three cases in one year, but whenever we have treated mares before foaling we have not seen it.

J.L. Duncan: The trouble here with *Anoplocephala,* the two species, namely *A. perfoliata* and *A. magna,* is that there has been virtually no research work done. We do not even know the complete life cycle of these parasites. We do not know their pathogenic effect; we only know they are there. Most people have assumed because of their presence in large numbers in clinically normal horses, that they do not have a very marked pathogenic effect, but you should put it down on paper.

C.J. Roberts: Would you agree though, that their normal site is the base of the caecum? That is where you will find them in any horse that has not had caecal rupture.

J.L. Duncan: You will find a ring of *Anoplocephala perfoliata* around the ileocaecal orifice. You will find them like a rosette and free within the caecum.

C.J. Roberts: Right, and where are they when the bowel ruptures? They are not there at all, they are where they are normally not, an arm's length away, because under those conditions they would be washed out, so they go up.

J.L. Duncan: I do not quite understand, because we have autopsied horses and immediately after they are put down, we open the caecum and the position of *Anoplocephala* is around the ileocaecal orifice; most of them are there. There are a few free specimens within the lumen.

C.J. Roberts: That is where these ruptures occur; they will also occur at the base of the caecum.

J.L. Duncan: It depends what you mean, the area or the ileocaecal orifice.

C.J. Roberts: Yes, that is at the base of the caecum, isn't it? My point is, they are where the rupture would occur. Once that happens, they move away.

J.L. Duncan: The area mentioned by Professor Littlejohn is certainly not the area where one finds *Anoplocepnala perfoliata.*

D.H.G. Irwin: Like everybody else who has succeeded in rupturing a rectum, I would like to mention the fact that it is not necessarily rectal palpation which is the primary or only factor concerned: *Gastrophilus* larvae occasionally weaken the rectum within an arm's length of the anus and it is then remarkably easy in some instances to see how anyone could rupture the rectum by hand. It can help to explain this mishap to oneself and to any irate owner.

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FOURTH SESSION: IMPACTION AND COLICS

Chairman: C.F.B. HOFMEYR

CAUSES OF COLIC AND TYPES REQUIRING SURGICAL INTERVENTION

J.D. WHEAT*

SUMMARY

The migration of strongyle larvae is the most common or basic underlying cause of colic in the horse. Disease conditions producing symptoms of colic occur in all sections of the intestinal tract and consist of impactions, torsions, herniations and foreign bodies. Colic also occurs as a result of pre- and post-partum diseases such as torsion of the uterus, haemorrhage, rupture and inversion of the uterus. In general, lesions resulting in circulatory obstruction are the types requiring surgical intervention. There are six general types of small intestine obstruction that lend themselves to surgical treatment; namely, volvulus, herniations, intussusceptions, stenosis of the lumen of the bowel by external bands or by foreign bodies and chronic inflammatory lesions.

The large intestine is not subject to the variety of obstructive lesions found in the small intestine. Impactions count for a large percentage of the obstructions seen. A standing laparotomy for diagnostic purposes may be indicated. Small colon impactions are readily treated by standing laparotomy. Enteroliths are of very common occurrence in some areas of the country; they often result in rupture of the colon. Torsions of the colon produce septic shock very rapidly. The left dorsal colon moving medially or laterally and ventrally initiates the torsion. Clockwise rotation is most common.

Massive intravenous therapy is needed to maintain hydration. Ventral midline laparotomy gives best access. Surgery must be performed very early to avoid massive tissue necrosis. Survival rate is 30% or less. The small colon is also capable of rotation and volvulus, and of strangulation in the umbilical or inguinal ring.

CAUSES OF COLIC

The immediate visible diseases or lesions that produce clinical signs of colic are well described and documented.

The question that has not been well answered is: What, if any, underlying condition causes a horse to develop volvulus, enteroliths, rotations of the colon, etc.?

I believe that the strongyle intestinal parasite plays the major rôle as a basic underlying cause of colic. Based on necropsy and exploratory laparotomy, our hospital records show the diagnosis of thromo–embolic colic in 2,1 percent of our colic cases. Others report figures from 1,2 to 5 percent. Such data may lead us to conclude that emboli are not a large factor in production of the disease. What we often do not realize is that many of the transient spasmodic colics may be initiated by a sudden obstruction of blood flow which, in turn, produces a section of bowel that becomes paralyzed and distended with gas and ingesta. This puts tension on the mesentery which serves as an axis for loops of bowel in peristalsis to slide over and start the formation of a volvulus or torsion.

Feeding practices are also incriminated as a cause of colic. Such factors as over–eating and abnormal or spoiled feed do cause severe intestinal problems. On the other hand, horses which, because of changes in feeding practices, are subject to colic, may develop the condition because of previous thrombosis of vessels and failure to develop an adequate vascular anastomosis. This, in turn, results in reduced blood supply to a section of intestine which cannot respond to the demands of an increased blood supply necessary to digest and handle unsuitable feeds or altered feeding practices.

Excessive stress from exhausting work or disease results also in severe intestinal disorders. Whether this is due directly to the stress itself, with altered electrolyte balance and the production of endotoxins in the bowel, or to the bowel circulation being compromised by previous thrombi and an inability to function properly because of lowered blood pressure and reduced blood volume and flow is, as yet, an unanswered question.

It has been my experience that very little, if any intestinal disease is encountered in stables that maintain a rigid parasite control programme and use good quality feed in adequate amounts. The majority of our colic cases come from stables and backyard horse operations where no regular system of parasite control is practised.

Contrary to popular belief, there are many parasitized horses at the race–track. Some horses bring their parasite load with them from the farm; others become infected at the race–track.

DISEASE CONDITIONS LEADING TO DEVELOPMENT OF SIGNS OF 'COLIC'.

I. STOMACH
- A. Rupture
  - 1. Primary
  - 2. Secondary
- B. Impaction
- C. Dilatation
- D. Carcinoma
- E. Pyloric Obstruction – Bots

II. SMALL INTESTINE
- A. Intussusception
- B. Volvulus
- C. Pedunculated Lipoma
- D. Adhesions – Primary and Secondary
- E. Herniation through Body Openings
  - 1. Mesenteric
  - 2. Foraminal

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3. Diaphragmatic
4. Umbilical
5. Inguinal

F. Impaction – Plant Material, Ascarids
G. Stricture
H. Meckel's Diverticulum
I. Functional Ileus

III. CAECUM
A. Torsion
B. Impaction
C. Rupture – Primary and Secondary
D. Tympany

IV. LARGE COLON
A. Torison
B. Impaction
C. Rupture
1. Tympany
2. Enteroliths

D. Tympany

V. SMALL COLON
A. Impaction
1. Faecolith
2. Stone (calculus)
3. Other Foreign Bodies

B. Torsion

VI. OTHERS
A. Sand Colic
B. Recurrent (of unknown aetiology)
C. Thrombo–embolic
1. Parasitic
2. Other

D. Meconium Impaction
E. Atresia Coli/Ani
F. Spasmodic
G. Abdominal Pain
1. Aetiology Unknown
a. Physical examination inadequate
b. Undiagnosed, in spite of physical and rectal examinations

H. Hernia Complications
I. Pre– and Post–Partum Diseases
1. Torsion of Uterus
2. Uterine Artery Haemorrhage and Haematoma
3. Inversion of Uterine Horn
4. Rupture of Uterus

TYPES OF COLIC REQUIRING SURGICAL INTERVENTION

In general, lesions causing or resulting in circulatory obstruction require surgical intervention. The types of small intestinal obstructions that lend themselves to surgical treatment are as follows:

Volvulus involving the ileum causes one of the most common acute episodes. It is very easily detected when the abdominal cavity is opened. The distended and discoloured loops of bowel bulge out of the incision and, as one explores the root of the mesentery, a taut rotated mass can be felt.

Unless operated on very early in the course of the disease, it is seldom possible to correct a volvulus because of the massive vascular thrombosis and inability to expose the mesenteric root.

Herniations: In the stallion and gelding, the internal inguinal rings should always be palpated. Once the entrapped intestine starts to swell, it cannot be reduced by traction but must be reduced by incision over the external inguinal ring and the external tunic of the spermatic cord. It is then necessary to stretch or enlarge the inguinal ring, reduce the hernia and then perform a covered castration, or reconstruct the enlarged inguinal ring and incised tunics.

Diaphragmatic hernias of congenital origin are rare. Acquired diaphragmatic hernias are more common. I have never attempted to repair one, so cannot offer any comments.

The epiploic foramen lying on the visceral surface of the liver near the portal fissure provides an opening for entrapment. As horses grow older, the right lobe of the liver atrophies and the foramen enlarges, allowing the small intestine to enter. Seven years is the youngest age of horse I have seen with this obstruction. With this type of hernia, exposure and reduction is a problem. Usually, a large volume of bowel is involved and the resulting distension makes reduction very difficult.

The nephrosplenic ligament, passing from the spleen to the left kidney, provides a space or cleft on the left side through which either small intestine or small colon can pass and become entrapped. The intestine always passes through from left to right and in a forward direction. This type of lesion is more easily reduced than is herniation through the epiploic foramen and merits a good prognosis.

The great omentum can tear and form a thick, ring–like orifice which can and may admit intestine and cause strangulation.

Ligamentous hernias, caused by a congenital defect or acquired tear in a normal ligament, can become strangulated, the most common example being the mesenteric hernia. Herniations through the broad ligament of the uterus also occur.

Intussusceptions are uncommon and involve usually the terminal ileum or the ileocaecal valve. They do not always 'shut down' as a sudden acute obstruction. Some horses have shown colic for as long as two weeks before complete occlusion occurred, resulting in the terminal acute clinical signs. Owing to lack of viable intestine at the termination of the ileum, it is often necessary to anastomose the ileum to the caecum and obliterate the natural opening.

Another type of obstruction results from *stenosis* or occlusion of the bowel lumen owing to external adhesions or bands, the most common example being strangulation of the small intestine by a pedunculated lipoma. This is a disease of older horses, 12 years of age being the youngest in our records.

The small intestine is rarely obstructed by foreign bodies, but ascarid impaction following worming sometimes occurs.

Cronic inflammatory lesions, resulting from possible strongyle infection and migration, may cause adhesions and stenosis. The chronic case of bowel stenosis often causes marked dilatation of the intestine proximal to the obstruction and results in chronic diarrhoea.

The vast majority of acute small intestine obstructions require resection of the bowel. It is seldom that a horse is presented to our clinic prior to development of irreversible vascular thrombosis.

TYPES OF LARGE INTESTINE DISEASE REQUIRING SURGICAL INTERVENTION

The large intestine is not subject to the variety of

obstructive diseases of the small intestine. Impactions account for a large percentage of large intestinal obstruction and are commonly thought to be related to water intake, quality of feed, dental disease and lack of exercise. With the exception of torsions, colic involving the large intestine tends to be less acute and less severe than is the case with small intestinal diseases.

Impaction of the Caecum

This is an uncommon condition, with a slow, gradual onset. If treatment with softening agents, fluids and small doses of neostigmine are ineffective, exploratory laparotomy through the right flank, with the horse in a standing position, is indicated. By this approach, it is possible to break down the obstruction manually and also direct the injection of softening agents into the impacted mass.

Impactions of the Large and Small Colon

If medical treatment fails to relieve the impaction, surgical exploration must be considered.

Impaction of the small colon or pelvic flexure can be treated by a standing laparotomy through the left flank. Often, massage of the mass plus injection directly into it with softening agents such as dioctyl–sodium–sulphosuccinate are sufficient to disintegrate the mass and allow it to move on.

If the impaction involves the large ventral colon, it is difficult to gain access to it through the flank, the mass being too voluminous for easy manipulation. So, it is much better to use a ventral midline approach.

If possible, one should avoid evacuating a large colon impaction *via* enterotomy. The volume of ingesta is so large that it is very difficult to exteriorize and evacuate the large colon without causing gross contamination

Enteroliths are of very common occurrence in some areas of the U.S.A. They can be single or multiple. Clinical signs are rather vague and mild until the onset of acute obstruction which occurs usually when the enterolith passes from the large to the small colon. When obstruction occurs, the signs are more acute and severe than in the case of impaction by food material. If they are not promptly removed, they often cause rupture of the colon. Because of the size of the mass and the difficulty of manipulation, a ventral laparotomy is often preferable. The enteroliths which we have had analysed are of magnesium ammonium phosphate. Often, there is a small nidus of metal in the centre of the enterolith.

Sand Impaction of the large colon is seen frequently in areas where the soil is sandy and where horses are fed hay on the ground, or maintained extensively on pastures with short grass and inadequate feed.

Many horses with sand impaction respond to medical treatment. If surgery is considered because of the massive size of the impaction and failure of the sand mass to respond to softening agents, a ventral midline approach is needed and the colon exteriorized as much as possible. The washing out and evacuation of a sand–impacted colon is a long procedure and unless the gut is carefully packed off and protected, a great risk of contamination is present.

Torsion of the Large Colon

Torsion of the large colon will cause obstruction to the passage of ingesta and if the vascular supply is occluded, severe septic shock will develop rapidly. The left ventral and left dorsal segments of the large colon are the most freely movable parts.

It has been suggested that the left dorsal colon moving medially or laterally and downward, initiates the torsion.

Rotations of 90° or less, probably occur frequently and go unnoticed because they cause little or no discomfort. Torsions of 180° to 270° will stop the flow of ingesta and will compromise the blood supply to some extent. Torsions of 360° or greater will occlude the vasculature and ischaemic necrosis of the affected part will occur rapidly.

Torsion may be in either a clockwise (most common) or counterclockwise direction when viewed from the posterior standing position. The torsion may involve only the pelvic flexure, the left dorsal and ventral colons, almost the entire large colon or almost the entire large colon and part of the caecum. Torsion of the large colon may occur spontaneously or may result from the horse rolling or struggling to rise when cast. It has been suggested that horses rolling from milder types of colic, such as spasmodic or impaction colic, may cause this disease. A disproportionate amount of gas and ingesta in various parts of the colon may also predispose to its occurrence. One case of torsion of the large colon seen at the Veterinary Medical Teaching Hospital, University of California at Davis, had a sand impaction of the pelvic flexure as well as the torsion. Presumably, the solid mass of sand in the pelvic flexure had caused the colic originally but as the horse rolled, it sustained a 360° torsion of the left dorsal and ventral colon. The sand impaction had sufficient weight to influence the torsion.

If the torsion has occluded the passage of ingesta but has not compromised the blood supply, the signs will be similar to impaction of the large colon. Moderate, dull pain, getting up and down, occasional rolling, reduction or absence of intestinal sounds and anorexia are signs.

On rectal palpation, the large colon may be distended with gas but perhaps only to a limited extent. There will be a mass of ingesta proximal to the obstruction that will feel firm, like an impaction. Usually, the twisted section of the colon cannot be palpated rectally. The signs will progress and the case will appear to be an unresponsive impaction of the colon.

One filly, suffering from a 180° torsion of the large colon, did not become violent but remained uncomfortable for 36 hours before the diagnosis was made at surgery. It appeared that she was not responding to conservative treatment for impaction. Her large colon did not become distended with gas and she did not show any signs of impending shock. She seemed most comfortable in dorsal recumbency and would roll up on her back and remain there for long periods of time, if not forced to rise.

When the torsion is complete and the blood supply is stopped to a section of the large colon, the horse will become extremely sick in a very short period of time. Within a couple of hours or less, such a horse can be going into shock with a pulse rate ranging up to 150 and very cyanotic mucous membranes. It will be

in extreme pain and will throw itself about violently. Profuse sweating is evident and abdominal distension is very rapid.

On rectal palpation, the large colon is forced back into the pelvic cavity and is grossly distended with gas. The distension may have reached the point where it is not possible to pass the hand beyond the pelvis. Haemoconcentration occurs rapidly, the animal going into shock a very few hours after the onset of distress. Paracentesis of the abdomen produces large amounts of blood-tinged fluid which may have a high white cell count, depending on the duration of the condition.

Surgical reduction of the torsion is the only effective treatment. It is essential that this be done as early as possible because the horse's general condition will deteriorate very rapidly. As the ischaemic necrosis of the affected section of the colon progresses, the horse will become more toxic.

Massive intravenous fluid therapy will be required to maintain adequate hydration. Affected horses usually are in such acute pain that they cannot be kept on their feet for very long, so hydration may have to be done during surgery rather than before. It is necessary to utilize a ventral midline approach and the incision needs to be long enough to allow for exteriorization of the affected portion.

The extreme tension within the abdomen will cause respiratory difficulty, so respiratory assistance may be needed. Because of the gross distension of the abdomen and its effect on normal ventilation, it is necessary to open the abdomen as rapidly as possible after the horse is anaesthetised. When the peritoneum is opened, much of the affected portion of the large colon, being severely distended with gas, will protrude. The gas is removed by means of a large hypodermic needle or a bleeding trocar attached to a rubber tube.

It is not possible to adequately explore and reduce these lesions unless the affected portion is exteriorized. After the torsion has been reduced, the horse may show more severe signs of shock as a result of all the toxic products that have entered the circulation. In some cases, the organ is too distended with ingesta and fluid to allow for reduction of the torsion without evacuation of the contents.

Unless the horse goes to surgery very early, the colon will be so devitalized that its function will be impaired. It is, therefore, beneficial to evacuate the colon so that its recovery is not complicated by excess distension with fluid and ingesta. This can be a very difficult procedure and often necessitates the exteriorization of the left large colon. The walls of the colon are often very friable so that surgical manipulations must be done carefully or rupture will occur.

If the torsion has been present for sufficient time to cause severe necrosis of the tissue and thrombosis of the vessels has occured, normal circulation will not be restored. Observation of colour change of the affected part following reduction of the torsion is an indication of response. If the affected part of the colon remains dark and does not bleed on a cut surface, irreparable damage has been done. When the necrotic areas involve the pelvic flexure it may be resected. Resection of large portions of the large colon has not been done frequently enough to evaluate the technique.

Because of the large mass of tissue that is involved and the severe toxaemia and shock that can occur, intensive post-operative care is needed.

Torsions that produce obstruction without vascular occlusion are reduced through a ventral midline incision. Since the tissue is viable and peristalsis will return more rapidly in these cases, the prognosis is better. If there is a large accumulation of ingesta proximal to the torsion, enterotomy and evacuation will aid the recovery. When a small amount of ingesta is present, it may be treated as an impaction following reduction of the torsion.

Torsion of the large colon with tissue necrosis is a severe disease. In unselected cases at this time, surgical treatment may result in a 30 percent or less survival rate. With surgical intervention earlier in the course of the disease, improved anaesthesia and improved intensive care, more of these cases can be saved. Torsion of the caecum has been reported in the horse, but it is not common.

Rotation and Volvulus of the Small Colon

This condition occurs in the horse but is frequently associated with malpositioning of other organs or lesions such as adhesions and abscesses. If uncomplicated, volvulus will cause marked signs of colic depending on the extent of the twist. The affected section of mesentery might be picked up on rectal examination, depending on the extent of the distension in the large colon. Surgical correction is indicated. If the involved section is gangrenous, it should be resected. The ingesta in the proximal small colon and large colon may have become dry and firm as a result of the obstruction and dehydration. The horse should be well hydrated and this material softened by oral or direct medication.

Strangulated Hernia

Occasionally, a loop of small colon will become strangulated in an umbilical or inguinal hernia. The signs are those of an acute progressive colic. Careful physical and rectal examination will usually provide a diagnosis of strangulated hernia. Whether it is small intestine or small colon may not be apparent until it is examined surgically. If the case is seen early, reduction of the hernia will suffice. If the involved section is necrotic, it will have to be resected.

Adhesions, fibrous bands and abscesses may obstruct a part of the large intestine. Colic from these causes cannot be differentiated clinically unless they can be felt on rectal palpation. These lesions can easily be differentiated by a standing exploratory laparotomy. This is a safe procedure that can be done with a minimum of equipment and facilities.

Congenital Atresia of the Colon

This condition occurs in foals and is usually recognized shortly after birth. No faeces is passed and the foal will develop a distended abdomen and signs of colic. Examination and attempts at administering an enema may provide a diagnosis. Upon surgical exploration, it may be found that the colon is not patent. Surgical correction may be very difficult and other congenital abnormalities frequently are present.

Primary neoplasia of the large intestine occurs but is not common. Polyps and leiomyomas have been seen in the rectum and small colon. Polyps are often on a small stalk and can be surgically removed. If the base of the stalk is removed, they have little tendency to recur.

C.J. Roberts: I would like to ask Dr. Wheat if he has tried using massive doses of thiabendazole to try and get some of these parasitisms and infarcts to resolve.

J.D. Wheat: We use massive doses of thiabendazole in horses that we feel have problems with migrating strongle larvae in cases of the thrombo-embolic colic. The horse with the small infarct of the bowel will make a spontaneous recovery without resection but it has to be a very small segment in order for this to happen, otherwise necrosis and sloughing of the bowel itself will supervene. It is rather difficult to evaluate as to just how much involvement is present. In such cases I would rather do and exploratory laparotomy and evaluate the amount of bowel that is involved. More than a segment of 2 to 5 cm I would expect to necrose and end up with a fatal result, so that we have no alternative in such cases. If the condition of the animal was deteriorating I would do a resection and follow these cases up after recovery, if they do recover, with the massive doses of thiabendazole. In some cases heparin has been used after surgery with the idea of trying to lessen the amount of infarction and possibly improve the circulation in these areas that are partially occluded or have an insufficient blood supply. I do not know if that really answers the question.

If there has been a strongyle problem and aneurisms with thrombo-embolic colic occur we do give massive doses and then recommend the owner from now on that this horse should be dewormed at frequent intervals, at least four times a year, with the normal therapeutic dose.

C.J. Roberts: We have had problems with this. I would never have thought that a yearling, say 9 to 10 months old, that had extreme congestion of the mucous membranes, a pulse rate of about 100 and flatus in the intestinal tract, of which one would say it was going to die, could recover. Nowadays, when we get a colic case, believing it to be parasitic, the first thing that we do is give massive doses of thiabendazole. Initially, of course, from previous experience we expected that we were very likely to end up with a dead horse, because in every case they had gone through the phase of congestion of mucous membranes, abdominal enlargement and we have had them die. We then initiated massive thiabendazole therapy. In spite of a pulse rate of above 100 and all the cardinal signs one gets in a horse that is going to die, the three cases on which we had done this, have made quite miraculous recoveries. They have taken a couple of days. We even had one case with its feet up the wall and had already decided to operate. We had given it thiabendazole initially into the muscle. As we were going through the surgery, we reckoned it was a bit better. We left it until the next morning, when we could see the congestion of the mucous membranes clearing up. Slowly it recovered and was sold at the last sales in New Zealand for $5 000.

LABORATORY TECHNIQUES IN RABIES

EDITED BY M.M. KAPLAN AND H. KOPROWSKI

W.H.O., 3rd edition, 1973. Price: £6.00.

The two earlier editions of this work (in 1954 and 1966) although published by the W.H.O. with Drs Kaplan and Koprowski as contributors (amongst others) had no specific editors and, although an extremely useful and essential book, it was simply a laboratory manual. This new edition under its dynamic and talented editors has in one great leap doubled its size and become a veritable encyclopaedia on rabies embracing all the latest research and diagnostic developments in impressive detail.

It is obvious that a drastic change like this does not come about within a few years merely by revising the earlier work, regardless of the pace of progress, and what has in fact caused it is the wise recognition that specific details of advanced techniques, such as, for example, tissue culture rabies vaccines and fractionation of the rabies virion for antigenic enhancement, are difficult for overseas workers to come by and thus limit their application. In this new edition the details are all there, clearly described in some 20 new chapters covering tissue culture, complement fixation, haemagglutination, production and testing of newer vaccines and of animal and human immuno globulin, and concentration and purification of the rabies virus. Only budgetary and human obstacles need seemingly now prevent any country developing the most up to date methods of diagnosis and serum and vaccine treatment.

In the thorough-going review of those portions of the earlier editions that have been retained new and additional illustrations have been incorporated into the text. Thus despite the technical nature of its subject the book remains lucid and readable.

Without question no laboratory involved in rabies in any way can operate without this book. The wealth of information it contains should ensure it a place in the reference literature of any virological laboratory as well. The World Health Organization is to be congratulated on this fine piece of work.

C.D.M.

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INDICATIONS FOR SURGICAL INTERVENTION IN EQUINE COLIC

H.C. KALSBEEK*

SUMMARY

The indications for laparotomy are reviewed and some background information is supplied.

In this discussion of the diagnostic criteria we use in Holland to make the decision to perform a laparotomy in a horse with colic, I will talk only about horses with what is called 'true' colic. This means that the cause of the colic lies in the digestive tract.

After a clinical examination it is often impossible to reach an exact diagnosis, so it is advisable to list the types of colic in which surgical intervention is necessary:-

1. Patients in which the exact cause of the colic can be diagnosed and which require surgical intervention.
2. Patients without an exact diagnosis, but in which there is sufficient evidence that surgical treatment is probably the only means of saving the animal.
3. Patients with recurrent or persistent colic in which adhesions are anticipated. In such cases, exploratory laparotomy is advisable. These cases are self-evident and will not be discussed in greater detail. All cases in the first two categories may roughly be defined as cases of ileus or subileus. They include patients with displacement of the intestines and obstruction of the circulation in the bowel wall, impaction of the small colon and pelvic flexure in foals and obstipation of the small intestine.

In the first group the diagnosis does not present any problems; it includes (a) animals with incarcerated scrotal hernia, or (b) patients in which rectal palpation makes it possible to detect the abnormality, for instance strangulation by a lipoma, an adhesive portion of the intestine or an incarcerated portion of intestine with its markedly thickened and oedematous wall. In all these cases, surgical treatment is definitely indicated, assuming that the condition of the patient has not deteriorated too much.

The second group presents more difficulties. In these patients an exact diagnosis has not been established. Actually, this situation is frequently encountered and two variations exist: first, patients in such severe distress that surgery is undoubtedly indicated, but in which an exact diagnosis is replaced by a less accurate one, for example, 'displacement' of the jejunum, which may mean volvulus, strangulation, incarceration, etc. In these cases the part of the intestine involved is known but not the nature of the lesion. The second variation is that the patient is presented in an early stage of disease and rectal examination reveals an alteration from the normal situation, such as distended loops of the small intestine. The rest of the examination, however, fails to reveal other serious changes so that only a tentative diagnosis can be established. In such a case, one will hesitate to perform laparotomy. On one hand, the diagnosis is uncertain, on the other, there is the knowledge, referred to by many authors, that any delay in operating will reduce the likelihood of a satisfactory result. Although we agree that this is so, one should be careful not to operate on horses needlessly. When we started to perform laparotomy in horses some years ago, we supposed that the operation itself did not involve any particular risk. Since then, we have seen a number of horses in which this proved to be untrue: adhesions, peritonitis and other post-operative disorders were occasionally observed. In these cases, the remedy may have been worse than the disease. Therefore, a middle course between waiting too long and acting too quickly has to be found. In Holland, we say: 'Vind de gulden middenweg' (Find the golden mean). As you all know, gold is not so easy to find.

Nevertheless, when confronted with a patient, we have to start the examination. It is good to remember the words of Guido Gezelle, the Belgian poet 'Think before you start to work and, while at work, keep thinking.'

Let us first consider which abnormalities can be found in a horse in the final stages of colic caused, for instance, by a volvulus. Such a horse is no longer violent; on the contrary, it is dull, keeps its head low, has a more-or-less distended abdomen, wide nostrils and a staggering gait. The pulse is very weak, its rate is high. The ears, lips and extremities are cold. The mucous membranes are bluish-red and pressure on the gingiva reveals poor circulation. The elasticity of the skin is reduced. On auscultation, the abdomen is completely silent. On rectal examination, distended loops of the jejunum can be palpated. If the stomach has not already ruptured, large amounts of stomach content can be siphoned off *via* a nasal tube. Sometimes, some greenish discharge is evident at the nostrils. The blood appears dark and sticky because of the high cell content. Laboratory analysis reveals a high packed cell volume, (P.C.V.) sometimes leukopenia, metabolic acidosis, high lactate content and high glucose level. The combined clinical and laboratory findings point to a state of severe shock.

During our examination, therefore, we must look not merely for the direct cause of the colic, but also for the reactions of the whole body and the degree of disintegration of homeostasis. This disintegration is caused by the loss of fluid from the anterior portion of the intestine on one hand, and the absorbtion of toxic material from the altered, more distal, intestine on the other.

With these two points in mind, one can appreciate that horses in which the cause of the colic gives rise to obstruction, not only of the intestine but also of the circulation in the bowel wall, will die sooner than those patients in which the circulation in the bowel

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wall is not seriously impaired. For instance, the condition of a patient with obstipation of the ileum or small colon will deteriorate less rapidly than that of a patient with strangulation of the analogous part of the intestine.

Moreover, one will appreciate that there will usually be a relationship between the rapidity of deterioration of a patient with completely obstructed circulation in the bowel wall and the size of the affected part of the intestine. This is true only if there is an adequate contact between the altered part of the intestine and the peritoneum. Intussusceptions therefore run a much slower course because there is less opportunity for toxins to be resorbed.

Returning to the examination of the patient, the following points should be checked:

1. THE CASE HISTORY

(a) Time of onset of colic.
(b) Severity of abdominal pain.
(c) Defaecation and micturition pattern.
(d) The ration fed to the animal.
(e) Type of work prior to the onset.
(f) Changes in the abdominal size.
(g) Degree of sweating.
(h) Is the patient willing to drink?
(i) Has the patient had any previous attacks of colic?
(j) In mares, the pregnancy or delivery status should be determined.

2. BEHAVIOUR OF THE PATIENT

Behaviour provides some clues to the diagnosis. In general, it can be said that the more severe the pain, the greater the likelihood that the patient is suffering from a serious type of colic, but this is not always so. There are some rather specific signs, however, for instance, (a) a 'stretched' horse indicates a probable impaction of the colon; (b) the horse which according to the owner 'presses on the urine' almost always indicates impaction of the left colon and (c) the young horse lying frequently on its back, almost without moving, is likely to be a case of infarction of the bowel wall. These signs should be confirmed by further examination.

The response to noramidopyrine methane sulfonate sodium (50% solution)† can be used as a diagnostic aid. Failure to respond, or a brief response, indicates usually a more serious cause. Other therapeutic agents that act in the same way can be used, but one should always use the same agent and, by so doing, learn about its activity in cases of colic with different causes.

One should never use phenothiazine derivates because they lower blood pressure too much, which is unacceptable, since such patients are apt to go into shock.

When the animal becomes dull and sweats profusely after having shown intense pain, the prognosis is very bad, but merely from this fact one cannot arrive at a precise diagnosis.

A greenish nasal discharge is a sign of severe stomach distension; a nasal tube should therefore be inserted without delay.

† Novalgin: Farbwerke Hoechst A.G., Frankfurt (Main).

3. HEART RATE

Determination of the heart rate is one of the most important parts of the examination.

The heart rate is influenced by (a) pain and movements as a consequence of pain, (b) circulating toxins and (c) diminished venous return caused mechanically or by loss of fluid. The speed of movements induced by colic pain is rather slow and as a consequence has only a minor influence on the heart rate. The influence of toxins is very difficult to quantitate. Toxins do, however, play a rôle in inducing and maintaining shock. Loss of fluid as a cause of diminished venous return, needs no further comment. A mechanical basis for a decreased return of blood to the heart can be created by severe tympany of the caecum or large colon or both. After trocharizing these portions of the intestine, the heart rate frequently decreases by about 20 beats per minute. From these facts it is clear that there will be a close relationship between the number of heart beats and (a) the duration of the colic, (b) the magnitude of the altered portion of the bowel and (c) the possibility of contact of the altered bowel wall with functional peritoneum.

When these theoretical factors are taken into account, about 12 hours after the onset of colic, a heart rate of less than 60 indicates a minor cause; 60-80 indicates a possibly serious cause and a rate above 80 indicates a very serious cause (provided tympany is not marked). Naturally, exceptions will occur in every group.

4. BODY TEMPERATURE

Experience has shown that the temperature is not a reliable guide in establishing a diagnosis. Only when a high temperature is repeatedly recorded, is it evidence of an inflammatory cause of colic. A normal body temperature does not rule out this possibility.

5. SKIN

The skin is examined for two reasons: first, the temperature of the ears, lips and legs can be estimated. Second, the elasticity of the skin in the neck region can be determined. A decreased temperature of the extremities is indicative of dimenished circulation and a certain stage of shock, while decreased skin elasticity indicates dehydration.

6. MUCOUS MEMBRANES

The reddened mucous membranes reflect rather well the degree of haemoconcentration. At a later stage when the patient is in shock, vasodilation may add its part to the reddening process. Also, some degree of cyanosis may be evident.

The gingiva can be used for testing the capillary refilling time. Pressure with a finger gives, as in the human nail bed, an anaemic spot which has to be refilled with blood within a second after relief of the pressure. An increase in refilling time is another proof of a deficient circulation.

7. SIZE AND TENSION OF THE ABDOMEN

In most patients with displacement of the intestine, the size and tension of the abdomen increases. When this clinical sign is absent, the colic is less likely to be due to displacement. The presence of muscular rigidity is an unfavourable sign.

When the abdomen is distended in the small area

between the last rib and tuber coxae, gaseous distension of the left colon or caecum is probably responsible. Tympany of the right colon, but not of the caecum, forces the abdominal wall to become prominent behind the curvature of the right rib, a little lower and more anterior than is the case when the caecum is involved.

8. BOWEL SOUNDS

Repeated auscultation of the abdomen is of great diagnostic and prognostic value, especially in advanced cases. A completely silent abdomen is a bad sign, in which case the disease process is very severe and the prognosis bad, even if surgery is successful. In almost every case, such patients develop paralytic ileus and die.

During operation, an injection of acetylcholine (25 mg.) into the subserosa of the bowel wall may be of prognostic value. When contraction of the musculature around the injection site was not seen, the animal died, all such cases having developed paralytic ileus. When an animal, in which no bowel sounds can be heard, is also in very poor general condition, *i.e.*, it has a very rapid heart rate, very high P.C.V., very low pH and possibly leukopenia, we believe euthanasia is indicated. On the other hand, when in a prolonged case normal bowel sounds are still discernible, ileus is less likely.

Moreover, one can do some tests to distinguish between a distended small or large intestine, particularly in the foal with tympany. The first is the steelband test, which is used as one of the diagnostic aids in cattle with abomasal displacement. If this is positive, the superfluous gas is in the large intestine. The second test is to push the abdomen while at the same time the ear is held close to the abdominal wall. A clear, splashing sound will be audible if the gas is in the large intestine.

9. RECTAL EXAMINATION

This, of course, is a very important part of the examination. Sometimes it is possible to palpate the abnormal site completely, for example an incarceration of the small intestine in the inguinal canal. In many cases, however, it can be determined only that parts of the intestine are distended. Then it is usually possible to recognize the part of the intestine involved, either by its localization, or shape, or both. When one can palpate a distended portion of the small intestine, one should be very apprehensive.

Little if any abnormality is palpable when the colic is due to a local inflammatory lesion other than a verminous aneurysm. Laparotomy is rarely performed when, on rectal exploration, we do not obtain a clear indication as to which portion of the bowel is affected. This does not mean that the entire abdomen has to be filled with markedly distended intestinal loops; a slightly dilated loop of the jejunum may be sufficient to cause colic.

It is of little value to discuss all the possible deviations that can be palpated on rectal examination, since most of them can be found in the literature, but two useful hints may be given. When a strand, the course of which is from high caudal right to low cranial left, is palpable in the abdomen, it causes the erroneous impression that one is dealing with a strangulation. This strand is almost invariably a taenia of the caecum. To solve the problem is rather easy. A strand can be passed by on both sides, the taenia of the caecum only on the left side. The second is overcoming the problem of distinguishing between an incomplete torsion of the left colon and tympany of this portion of the bowel. When there is a torsion, the lymph nodes of the colon will become oedematous and can then be palpated, which is not the case when tympany alone is present.

10. USE OF A NASAL TUBE

In almost every case of colic, a nasal tube should be employed. The tube should not only have an opening at the end but also two side holes near the end, otherwise, when one tries to siphon fluid contents out of the stomach, the end will be sucked against the stomach wall and the opening will therefore be closed.

When large quantities of fluid can be siphoned from the stomach in patients in which dilated loops of the jejunum are palpable on rectal examination, ileus is usually indicated, in which situation laparotomy should not be postponed. This naturally presupposes that the general condition of the patient has not deteriorated too far. Yet there can be other exceptions; to one of them I will return at the end of this paper.

11. HAEMATOLOGICAL EXAMINATION

When an adequately equipped laboratory is at hand, it is very useful to determine at least the P.C.V., the venous pH, base excess (B.E.), PCO_2 and the leukocyte count. The determination of glucose and lactate concentrations can be helpful to estimate the severity of the condition.

(a) Packed Cell Volume

Tables 1 and 2 were constructed as follows:- The P.C.V. of 123 horses, of which 59 died, was measured and then the percentage by which it had increased was calculated, starting from the es-

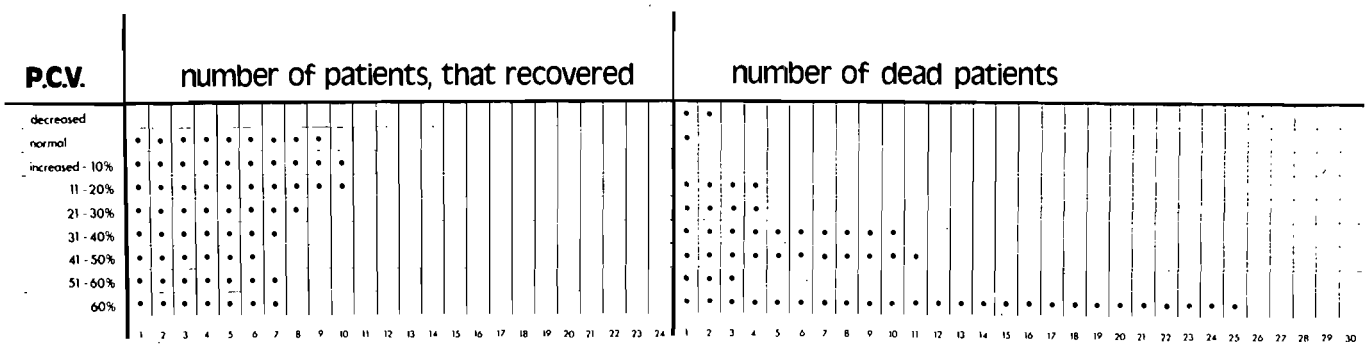

Fig. 1: Relation of P.C.V. to recovery and death rate.

timated normal value. The results are shown in figure 1. From this figure it can be seen that there is nothing 'black and white'. It is all a matter of chance. With a more marked increase in P.C.V., the chance of a more serious cause is increased. When there is no increase in P.C.V. and when haemorrhage can be ruled out, this, in conjunction with other parameters, may be a reason for postponing laparotomy.

(b) *pH, B.E. and* PCO_2

Most horses with colic develop metabolic acidoses, which in many patients is partly compensated by a decrease of the PCO_2. The likelihood of a serious cause increases with an increasing acidosis Figure 2. When the pH drops below 7 in an adult horse, laparotomy is useless; this probably also is the case when pH levels vary from 7,0 to 7,1. When the pH shows no decrease or only a slight one (for instance $\geq$ 7,280) this, in conjunction with other parameters, may be a reason for postponing laparotomy. Table 1 shows the prognostic value of the pH. Table 2 shows the prognostic value of combining the data of P.C.V. and pH.

(c) *Leukocyte Count*

Leukocyte counts are also made, although they do not give any clue to the diagnosis in most horses with colic. Marked leukopenia, however, is indicative of severe irritation of the peritoneum. When leukopenia is present, paracentesis is performed. When this produces a very evil-smelling fluid containing a large number of vegetable particles, euthanasia is performed.

Table 1 : PROGNOSTIC VALUE OF BLOOD pH IN CASES OF EQUINE COLIC

Blood pH Level	Died No. (%)	Recovered No. (%)
pH< 7,250	25 84%	5 16%
pH< 7,200	15 94%	1 6%
pH< 7,100	6 100%	0 0%

Table 2 : PROGNOSTIC VALUE OF BLOOD pH ALONE AND COMBINED WITH PCV LEVELS IN CASES OF EQUINE COLIC

	Died No. (%)	Recovered No. (%)
PCV rise > 40	38 65,5%	20 34,5%
pH< 7,250 PCV rise > 40	18 86%	3 14%

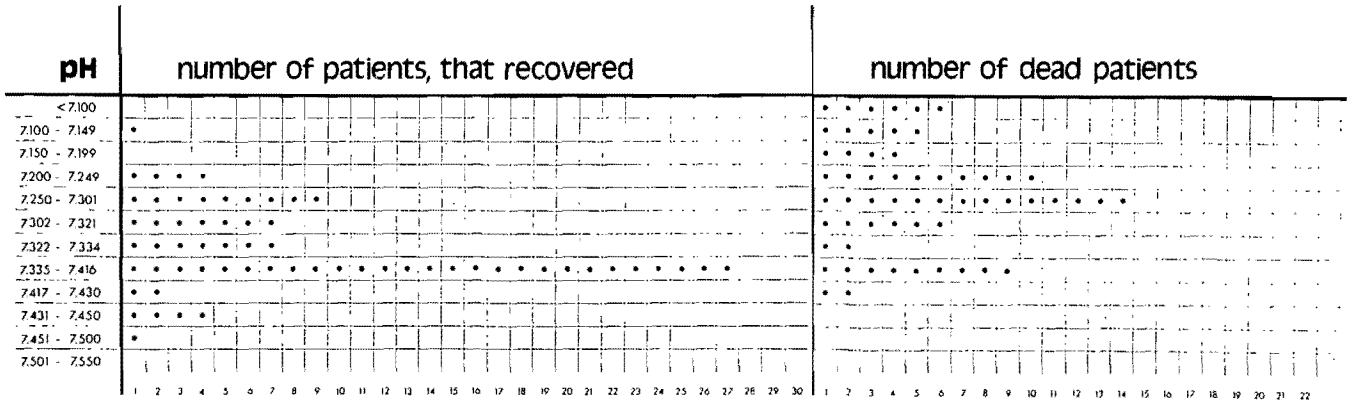

Fig. 2: Relation of pH to recovery and death rate.

(d) Glucose Concentration

The level of the glucose concentration also shows a certain correlation with the severity of the disease. When the glucose level is 200 mg/100 m*l* or more, the colic is due to a serious cause. Only 20 per cent of these patients survived (Figure 3).

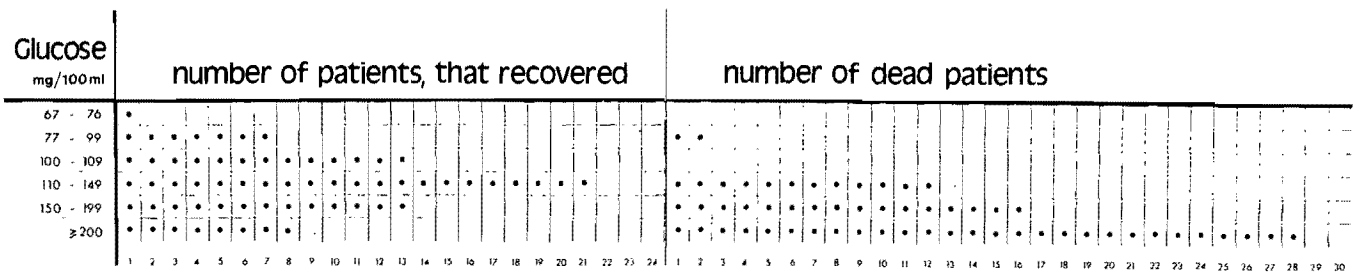

Fig. 3: Relation of blood glucose level to recovery and death rate.

(e) *Lactate Concentration*

A rise of the lactate concentration is the main reason for the development of metabolic acidosis.

In countries where the practitioner already has learned to give a colicky horse a bicarbonate solution intravenously before sending the horse to a clinic for laparotomy, measuring the pH will not give sufficient information about the condition of the patient. In such cases the measuring of the lactate concentration gives a better clue.

Figure 4 shows the lactate levels of 30 horses of which 17 died. Again, it can be seen that the difference is not unequivocal. Therefore it is not enough only to measure the lactate concentration. Additional parameters have to be checked.

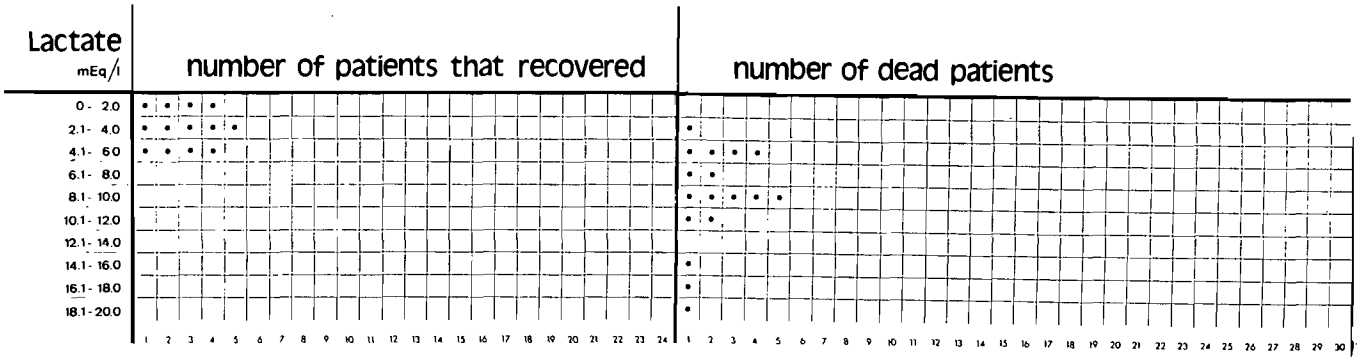

Fig. 4: Relation of blood lactate level to recovery and death rate.

Depending on all the results obtained from the examination, a decision is then made whether or not to operate, or to postpone surgery until there are more cogent reasons for surgical intervention. These reasons will become apparent only after repeated examinations at short intervals (maximally, two hours) and reconsideration of the findings. It will be obvious that, in considering these findings, the interpretation will be more or less subjective. The experience of the investigator plays an important rôle, particularly in rectal examination. Also, the disadvantages of laparotomy will carry more weight with one veterinary surgeon than they will with another; the former will delay the operation for as long as possible and of course sometimes for too long. On the contrary, there are cases in which it is advisable to postpone laparotomy. I will end with the description of such a patient.

It concerns an Arab mare of 15 years which was 10 months pregnant. The horse had colic for nine hours. Pain was moderate, but sometimes the mare rolled over onto her back and could not be kept on her feet. Favourable reaction to Novalgin was minimal. The animal had sweated, but not profusely. The frequency of the bowel sounds had decreased. Rectal examination by the practitioner revealed no abnormalities. The foal was still living. One litre of liquid paraffin was given. Two hours before entering the clinic, the mare produced a small amount of faeces of normal consistency. On examination in the clinic, the patient was rather quiet. She tried to lie down and roll if given the opportunity. Again, the reaction to an intravenous Novalgin injection was poor. The mare sweated a little. She was still alert. Respiration rate was 25, heart rate 72 and temperature 37,4°C. The ears and legs were abnormally cold, the lips still warm. The elasticity of the skin was slightly decreased. The mucous membranes were red. The capillary refilling time was slightly increased. The abdominal wall was supple. The bowel sounds were occasionally audible on both sides but were not high-pitched. During rectal examination some slightly distended loops of the small intestine could be palpated lying on the large uterus which contained a live foal. Just underneath the cranial mesenteric root one hard loop could be touched by the fingertips. This loop was painful on palpation at which time the horse almost went down. By means of a nasal tube, eight litres of sour-smelling fluid were siphoned from the stomach. Examination of the venous blood gave the following results:

P.C.V.	50%
pH	7,425
B9E.	-1,6 mEq/*l*
PCO_2	40,7 x 10^2 Pa (30,5 mm Hg)
Leukocyte count	9 800/mm^3
Glucose	118 mg/100 m*l*
Lactate	3,3 mEq/q/*l*

After that, we were in doubt as to what to do. On the one hand, the possibility existed of serious trouble in the small intestine (pulse 72; strongly decreased frequency of bowel sounds; distended small intestine with a painful portion and an already overloaded stomach; insufficient reaction to Novalgin; P.C.V. 50) which almost demanded a laparotomy. On the other hand, a horse that was still alert after 10 hours of colic, with warm lips, with an almost normal acid-base status and a normal leukocyte count and a 10-mont-old foetus was not a good reason to perform laparotomy. We decided to force the situation somewhat by giving another two litres of liquid parraffin by nasal tube, watching the mare carefully during the next hour and re-examining her at intervals of two hours.

For the first two hours the situation had not changed markedly, but after the second it had grown much better. The horse was quiet, with a heart rate of 72, but there were more bowel sounds, there were no stomach contents to siphon and rectally the distended loops of the small intestine were no longer discernible. Obviously, the diagnosis now was obstipation of the ileum. The horse recovered uneventfully and gave birth to a sound foal one month later.

VETERINARY MEDICINE

D.C. BLOOD AND J.A. HENDERSON

Baillière Tindall, London: 4th Edition, 1974. Pp.X+ 964, Tabs 22, Publ. Price: £8.00

It is a pleasure to welcome a new edition of what must be the best known textbook in English dealing with the medical diseases of large animals, and designated by the authors as ..."a core book for large animal practitioners and students with inclinations in that direction..." That it certainly is.

Despite the 6 years which have elapsed since the appearance of the third edition, the authors are to be congratulated on requiring only a further 34 pages to include their selection of the mass of published material which has appeared since 1968.

In their preface to this edition the authors give a broad indication of the changes made. Under General Medicine the sections on equine colic and diseases of the ruminant forestomach required significant additions. In Special Medicine much detail has been added, particularly in the sections relating to mastitis, colibacillosis and salmonellosis, viral diseases, parasitic diseases, the "metabolic profile" system of diagnosis and vitamin E — selenium deficiency. In a special note of local interest, the development of iso-immune haemolytic anaemia in calves whose dams have been vaccinated against protozoal diseases is recorded. There is even a brief reference to the disease of that newly discovered domestic animal species, *Brunus edwardii* (n.s.), now familiar to regular readers of the Veterinary Record.

This book is beautifully produced and most reasonably priced in these inflation-ridden times. It is a veritable mine of information for all who deal with farm animal medicine.

R.K.L.

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METABOLIC MANAGEMENT OF THE HORSE WITH AN ACUTE ABDOMINAL CRISIS

W.J. DONAWICK*

SUMMARY

The horse with an abdominal crisis caused by acute gastro-intestinal tract obstruction develops hypovolaemia, haemoconcentration, electrolyte depletion, metabolic acidosis and shock. During preparation for operation, treatment with fluids, antibiotics and bicarbonate will impede metabolic imbalance. Stomach decompression may slow the passage of sodium, water and potassium to the gut lumen, reduce pain and minimize the risk of stomach rupture. Selected laboratory determinations and the monitoring of arterial and venous pressures will provide a measure of security, and serve as a guide to replacement therapy. In the post-surgical period, vigilance must be directed towards potassium and bicarbonate imbalance and adequate hydration.

INTRODUCTION

Modern principles of surgery place high priority on provision for the metabolic needs of the patient as an adjunct to surgical management. Few dispute the benefits derived from attention to the patient's needs for water, electrolytes, acid-base balance and energy. Yet, there remain those who shun its use altogether or, equally disturbing, reserve such support for the critically ill patient in which the need for surgery is far beyond debate.

Since there is no reliable means of differentiating the horse with simple spasmodic colic or impaction from one with life-threatening intestinal obstruction, all must remain suspect. Therefore, beginning with the first medical assistance and continuing well into the post-opeative period, if a laporotomy be necessary, treatment must be directed to provide the best possible metabolic care. To do so may require frequent re-evaluation of even the most routine procedures in light of ever-changing knowledge.

Though plagued with pitfalls, the basis for most metabolic care is the result of research in species other than the horse and clinical experience gained during the treatment of man. Without conclusive evidence for modes of treatment, differences of opinion in extrapolation to the horse are inevitable and understandable. Fortunately, the principles of metabolic care appear to hold true among differing species and the application of those that are well-founded in the horse has resulted in a gratifying improvement in survival.

INITIAL MEDICAL ASSISTANCE

In the earliest hours of colic, the principles of metabolic care may be conveniently described as 'do's' and 'don'ts'. They apply to cases in which intra-abdominal digestive system involvement is highly proable but where a specific cause has not been determined.

There are five 'do's.'

Do relieve distenion of the stomach.

Shield [11] showed that obstructed bowel begins to secrete, rather than absorb, water and electrolytes within 12 hours of the onset of intestinal obstruction and the rate of secretion increases with time. Passing a stomach tube to remove accumulated fluid may slow the passage of extracellular water and electrolytes to the gut lumen, relieve gastric and intestinal distension, reduce pain and minimize the risk of stomach rupture. Passage of a stomach tube also serves as an aid to diagnosis. The ability to repeatedly drain fluid from the stomach or to recover medicaments hours after placement, signals intestinal occlusion.

– Do give sufficient analgesics to suppress pain.

The intense abdominal pain of colic appears to be a function of spasms of the jejunum [9]. These spasms serve no useful purpose and may be detrimental if, in response to pain, the horse rolls and causes torsion or displacement of the bowel. Xylazine– and pentazocine–– seem superior to the other commonly-used narcotic and non-narcotic analgesic drugs and have the advantage of causing minimal depression of gut motility or arterial blood pressure.

Do give a broad spectrum antibiotic.

The experimental studies of Fine [3] eloquently described the value of broad spectrum antibiotics in preventing death of dogs from sepsis, if given before and continued for four days after the onset of haemorrhagic shock. Survival after shock was attributed to control of growth of Gram-negative bacteria contained within the gut lumen until recovery of natural antibacterial defences. Administration of an antibiotic effective against a broad range of Gram-negative bacteria, such as tetracycline or chloramphenicol, if begun in the early hours of colic, may delay onset of septic shock and improve survival.

— Do give a balanced polyionic solution intravenously.

Cessation of oral intake of water and food during colic, conbined with water and electrolyte loss in the urine, in sweat, from the respiratory system and by insorption to the gut lumen, all contribute to rapid dehydration, haemoconcentration, hypovolaemia and shock. If the disease is allowed to progress unimpeded, tissues of vital organs go unnourished, total body oxygen deficit develops, lactic, pyruvic and other organic acids accumulate, lowering arterial

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blood pH and any remaining intestinal function fades because of its particular vulnerability to anoxia [1]. Administration of intravenous fluids to replenish extracellular water and electrolytes will delay but, in the horse, with current technology, not prevent the manifestations of shock.

– Do give bicarbonate or a bicarbonate precursor intravenously.

Buffering of extracellular and intravascular fluids will assure continued function of vital organs receiving sufficient nourishment and will improve function of tissues experiencing reduced perfusion. One cannot assume that all horses, even those with severe signs of acute intestinal obstruction, will develop metabolic acidosis. Some have no change in their acid-base status and others, particularly those with obstruction of the large bowel, may have metabolic alkalosis. Nevertheless, the incidence of metabolic acidosis is high and, lacking acid-base analysis, the administration of 50 g (596 mEq) of sodium bicarbonate empirically in one litre of sterile, distilled water will temporarily buffer a moderate acidaemia and will not be deleterious to a horse in alkalosis.

The 'don'ts' number but four.

– Don't give drugs which cause peripheral vasodilatation unless assured of adequate intravascular fluid volume.

Of particular concern is the use by the wary of one of the promazine tranquilizers for its sedative action without consideration of its peripheral vasodilatory effects. Normally, the contracting blood volume of shock causes vasoconstriction which maintains arterial blood pressure and assures, within limits, perfusion of vital organs, though at the expense of hypoxia of peripheral tissues. Use of promazine blocks this life-saving response.

– Don't give drugs per os which will cause fluid to pool in the intestine.

As stated previously, obstruction of the gastrointestinal tract cause secretion of sodium, water and potassium into the intestine [11]. Mistreatment may occur if medications which increase this insorption of water are used. Often, magnesium sulphate is given in the treatment of impaction. If acute intestinal obstruction is the true cause of the signs, rather than impaction, its use may lead to irreversible shock from extracellular fluid loss. Unless impaction with no complications can be diagnosed with confidence, the oral administration of osmotic drugs should be avoided for fear of intensifying extracellular fluid loss and accentuating shock.

– Don't give gastrointestinal stimulants.

Intense contractions of the intestine occur following the administration of parasympathomimetic drugs, an example of which is neostigmine methylsulphate. Its use may aggravate pain and, in addition, it has the potential of causing rupture of the distended, devitalized gastrointestinal tract.

– Don't delay the decision to perform a laparotomy.

A decision to operate must be based on a philosophy that it is better to err and operate when the diagnosis is less than certain than to delay and end with catastrophe [12]. Confidence in the ability to perform a laparotomy in the horse without untoward complication has spread around the world. The veterinary surgeon should accept the fact that he will inevitably make some misjudgments but they will be far less costly in terms of patients lost if he decides to operate early.

PRE-OPERATIVE METABOLIC CARE

The methods of metabolic care outlined will sufice for only a few hours. As severity of the disease quickens, accurate clinical assessment becomes increasingly difficult and proper metabolic care will become more dependent on measurements of arterial blood and central venous pressures [5], urine output and the results of laboratory determinations of packed cell volume, total protein, blood gases and acid-base balance.

Ideally, no horse should undergo laparotomy unless all parameters are within normal limits and the gastrointestinal tract is decompressed. In actuality, the surgeon will have to accept less but, in the interim during preparation for operation, every effort should be directed towards maintaining or improving the patient's condition through the administration of additional fluids, plasma and bicarbonate, therapy being based on assessment of clinical and laboratory findings.

BLOOD GAS MONITORING DURING ANESTHESIA

An integral part of metabolic care is careful attention to methods of administration of anaesthesia. General anaesthesia with the horse in lateral or dorsal recumbency compromises the adequacy of ventilation. During spontaneous ventilation, arterial blood O_2 tensions may dip dangerously low and high arterial blood CO_2 tensions may lower blood pH below acceptable levels. Controlled positive pressure ventilation will, in most instances, assure adequate ventilatory exchange. Nevertheless, injudicious use of unnecessarily high inspiratory pressure and rapid cycling of the respirator may be harmful. The resulting overventilation may cause rupture of alveoli, obstruct venous return by compression of the venae cavae, lower cardiac output, increase venous pressure and cause pulmonary oedema.

FLUID REPLACEMENT DURING OPERATION

Guided by trends in arterial blood and central venous pressures, rapid fluid replacement can be given with safety and accuracy. A low (less than 80 mm Hg – 106 X 10^2 Pa) mean arterial blood pressure and a low (less than 6 cm H_2O – 5,8 X 10^2 Pa– during spontaneous respiration) central venous pressure indicate a need for additional intravenous fluids. Rapid infusion of fluids (20 litres or more per hour) is possible without fear for complication if the administration is made through a large-bore catheter placed well into the anterior vena cava to prevent damage to the intimal surface of the vein. The rate of flow of fluids is adjusted to maintain the central venous pressure in the range of 6 to 12 cm H_2O. Use of sympathomimetic drugs has no place in the maintenance of arterial

pressure in hypovolaemic shock, with the possible exception of cardiac failure, which can be diagnosed when an increase in central venous pressure occurs simultaneously with a decrease in arterial blood pressure.

SELECTION OF FLUIDS FOR REPLACEMENT THERAPY

The fluid used for intravenous administration should be chosen carefully. Examination of biochemical parameters of horses in acute abdominal crisis [6 8] has revealed – a slight elevation of plasma sodium; a slight to moderate decrease in plasma potassium; a slight to moderate but, on occasion, marked decrease in plasma chloride; normal plasma proteins; increased blood lactate; and a high plasma glucose concentration. Thus, the hypovolaemia accompanying an abdominal crisis in the horse is not a water-loss dehydration but rather a proportional – or nearly so – loss of water, electrolytes and protein to the lumen of the gastriontestinal tract and the peritoneal cavity. A polyionic solution approximating plasma in milliequivalent content per litre of sodium and chloride is, therefore, superior to physiologic saline, which contains excesses of these ions. Anion balance of the solution is best achieved by the addition of 40 to 50 mEq/*l* of bicarbonate or a bicarbonate precursor such as acetate or gluconate. Lactate commonly formulated as Ringer's lactate solution should not be used because of the delay in its conversion to bicarbonate as evidenced by the elevated blood lactate so commonly found. The concentration of potassium per litre may be increased cautiously to 6 to 8 mEq/*l* but levels approximating normal plasma concentrations have proved adequate [2].

Administration of fluids containing dextrose, advocated by Lucke [7], has no apparent rationale since free water is not the prime need; the plasma glucose levels are without exception normal or elevated, and the additional dextrose will lead to an osmotic diuresis with unwanted water loss from the kidneys.

The degree of bicarbonate deficit can be measured by any of the many reliable pH and blood gas electrodes now commercially available. From the determinations of arterial blood pH and carbon dioxide tensions, the amount of bicarbonate needed to correct the deficit temporarily can be calculated. In general, the use of fluids containing bicarbonate precursors, even at the rate of 50 mEq/*l*, have proved insufficient because of the technical limits to the rate of their infusion. Empirically, we have found that for the purpose of calculating the quantity of bicarbonate needed, it is best to estimate the extracellular fluid volume at 40 per cent of body mass. The product of extracellular fluid volume (in litres) X the bicarbonate deficit (in mEq/*l*) derived by nomogram will result in an approximate dosage of bicarbonate needed for replacement.

HYPOPROTEINAEMIA ACCOMPANYING RAPID FLUID REPLACEMENT

Of recent concern to us has been the potentially dangerous decrease in total protein which occurs often after the administration of many litres of polyionic fluids. The resulting decrease in osmolality may cause pulmonary and peripheral tissue oedema. Such hypoproteinaemia can be avoided if a portion of the replacement fluid is plasma or concentrated equine albumin. With no commercial ready-to-use, inexpensive supply of horse plasma or concentrated albumin and frustrated in our own attempts to prepare it, we have resorted to the frequent measurement of total protein and when the level dips below 5 mg%, the rate of fluid administration is slowed.

DECOMPRESSION OF THE GASTROINTESTINAL TRACT

Inability to properly decompress the gastrointestinal tract before, during or after surgery remains a perplexing problem which violates well-established principles of gastrointestinal surgery[10]. Continued distension contributes to further sodium, water and potassium loss, difficulty in closing the laparotomy incision, and post-surgical pain and ileus. An important improvement in the metabolic care of the horse with gastrointestinal obstruction will be development of a reliable method of intestinal decompression.

POSTOPERATIVE CARE

Early in the post-surgical period, maintenance of adequate oxygenation is of critical importance. Room air may not provide sufficient oxygen. If the horse has been positioned in dorsal or lateral recumbency for surgery, changing the position to remove weight and pressure on the diaphragmatic lung lobes while continuing controlled positive pressure ventilation, or manually hyperinflating the lungs, will open collapsed airways and improve ventilatory-perfusion efficiency. While breathing room air, the percentage of inspired oxygen to the horse can be easily increased by administering 5 to 8 litres of oxygen per minute *via* a catheter placed well back in the pharynx and sutured to a naris, through a catheter placed in the endotracheal tube if it has not been removed, or through a percutaneous catheter placed in the lower trachea. By whatever means, every effort must be made to maintain an arterial blood oxygen tension of a least 70 mm Hg ($93x10^2$ Pa). The supplemental oxygen must be continued, except for brief periods of blood sampling, until, while breathing room air, the minimal oxygen tension is maintained.

Following relief of experimental small bowel obstruction in the dog, the ileum, and undoubtedly other affected bowel, continue to secrete water and electrolytes for at least 48 hours[4]. This finding is compatible with the observation in clinical cases of small bowel obstruction in the horse where the need for water and electrolytes, especially during the first 24 hours after surgery, exceeds normal maintenance. Increased packed cell volume and a low serum potassium are common. A 450 kg horse needs 27 litres of water per day for maintenance alone. To this must be added sufficient sodium, water and potassium to compensate for continued loss into the gut lumen and for repair of extracellular and intracellular fluid deficits. In the early post-operative period, we allow the horse water *ad lib* to which bicarbonate and potassium salts have been added. If there is no voluntary consumption, the water and electrolytes are given intravenously or by stomach tube.

The 'dos' and 'don'ts' of pre-operative management apply equally well to the post-surgical period. Attention to them and periodic measurement of packed cell volume, serum potassium and estimation of acid-base balance have resulted in our saving horses consistently lost in years past.

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MONITORING AND EVALUATING THE PHYSIOLOGICAL CHANGES IN THE HORSE WITH ACUTE ABDOMINAL DISEASE

J. R. COFFMAN*

SUMMARY

Initial examination and therapy, and the avoidance of maltreatment are emphasized. Gastric decompression is of prime importance, after which no compound should be administered *via* stomach tube.

Where large amounts of high starch grains are fed, primary acute gastric dilatation must be differentiated from that secondary to small bowel dilatation, by immediate gastric intubation and irrigation of the cardia with lidocaine. If cessation of pain and improvement of peristalsis and general attitude follow, the former state may be assumed. If pain persists and peristalsis does not improve markedly, one should assume small bowel displacement.

Rectal examination is helpful in initial evaluation: impactions, inguinal herniation and ileocaecal intussusception may be diagnosed and small bowel displacement suspected. Palpation of one or more distended loops of bowel in the ventral middle third of the abdomen indicates small bowel displacement or ileus and flaccid distension. Distinction by rectal palpation alone is difficult. Palpation of the gas-distended apex of the caecum in the middle third of the abdomen is virtually pathognomonic for 180° rotation of the large bowel.

Abdominal paracentesis yielding true sanguineous effusion indicates a necrotizing segment of the bowel. If negative, such a segment is absent, or there is an infarcted segment, not yet damaged to the point of leaching whole blood, or the necrotizing segment is outside the peritoneal cavity, *i.e.*, in the thorax, intussuscepted into the caecum, or herniated into the inguinal canal.

Recurrent colics frequently may be due to verminous arteritis but the relationship to diet should be investigated. Recurrent colics after grain ingestion with occult blood in the faeces may be due to ulcers; such cases respond well to grain withdrawal.

The advantages and disadvantages of phenothiazine-derived tranquillizers are discussed. They are contra-indicated if there is any evidence of circulating volume insufficiency but are benefical in many instances through improved peripheral perfusion of organs provided circulating volume is adequate, *i.e.*, early in acute abdominal disease prior to development of circulatory insufficiency. They should not be administered if immediate surgery is contemplated because of hypotensive effects.

The administration of oral antibiotics (Neomycin) early in the course of the disease is encouraged. This is contra-indicated if the horse is already toxic, when it should receive parenteral antibiotics, preferably chloromycetin. Tetracyclines may predispose to the later development of salmonella diarrhoea.

Absolute analgesia should be provided; our preference is the magnesium sulphate-chloral hydrate solutions. Administration of mineral oil is desirable in initiation of peristalsis, depression of Gram-negative overgrowth and softening of impactioning obstructions but nothing should be administered per os if the stomach has required decompression.

INTRODUCTION

This paper deals with differentiation of acute abdominal diseases and evaluation of the patient at or soon after time of onset, *i.e.*, when first observed by the veterinary clinician. Principles associated with diagnosis, evaluation and monitoring of physiologic al parameters are applicable to the ongoing management of the case (medical or surgical). Systematic, objective methodology should always be observed[1 2 7 13 14]

Principles of diagnosis and evaluation will, for purposes of discussion, be divided into two basic parts:

1. Morphologic and functional status of the gastrointestinal tract.
2. Status of the cardiopulmonary system

MORPHOLOGIC AND FUNCTIONAL STATUS OF THE GASTROINTESTINAL TRACT

Gastrointestinal crises, as manifest by colicky pain, may be associated with normal, insufficient, or increased intestinal motility. Pragmatically, the clinician must assume a correlation between intestinal sounds ascultated through the abdominal wall and intestinal motility. Normal or increased peristaltic sounds usually indicate minor indigestion. Exceptions include intestinal or gastric ulceration, transient cellular acidosis during partial or complete vascular occlusion, or 180 degree rotation of the large bowel on its mesenteric attachment.

Thus, one must focus on abdominal pain associated with depressed or inaudible peristaltic sounds, *i.e.*, insufficient intestinal motility (ileus). It is impossible to know whether a silent abdomen is caused by stasis owing to intestinal spasm, or flaccid paralysis. Distended segments of bowel become progressively paralyzed by local neuromuscular pressure and feed-back inhibition of the vagus[11].

During periods of small bowel immotility, retrograde filling of the stomach with fluid and gas will occur. In such instances the duodenum is also under pressure, and the stomach will reflexly fail to empty. Less commonly, gastric dilatation may accompany distension of the large bowel, more frequently after more than 48 hours of large bowel distension with refractory impactions.

The first step in diagnosis is to determine whether the disease process involves principally small or large bowel. Gastric dilatation must be differentiated from gastric distension secondary to small bowel displacement. Only the large colon and caecum have the physical capacity to distend the barrel of the abdomen, whereas generalized stasis and distension of small bowel with retrograde filling of the stomach will not seriously distend the abdomen. Nevertheless, gastric distention may produce diaphragmatic pressure resulting in difficult respiration. Any time gastroin-

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testinal distension is sufficient to distend the abdomen or displace the diaphragn, pressure on the caudal vena cava may impede venous return from the mesenteric circulation.

When physical examination and auscultation are not definitive in revealing the segment of the gastrointestinal tract involved, intubation of the stomach and rectal examination in concert usually furnish this information.

Use of the Stomach Tube

The most important move early in the course of any acute abdomen is intubation of the stomach. The importance of immediate gastric decompression cannot be overemphasized. Sustained gastroduodenal distension contributes to progressive loss of isotonic fluids into the lumen of the bowel and refractory ileus. If dyspnoea is observed, one may assume that the diaphragm is displaced by gastric distension (particularly when the abdomen is not distended) but the stomach may be under considerable pressure without enough diaphragmatic displacement to result in dyspnoea.

Evacuation of the stomach is difficult and failure to produce a reflux of gas and fluid by intubation alone is not adequate. Our efforts toward gastric evacuation have been improved by irrigating the cardia with 6 to 8 m*l* of 2% lidocaine. Because reflux of fluid may wash the lidocaine back up the tube, the procedure should be repeated when regurgitation stops. Obviously, such decompression measures are essential in either acute gastric dilatation or in gastric dilatation secondary to small bowel displacement. Mineral oil and other laxatives should never be pumped into a stomach that requires decompression. If the administration of mineral oil or other compounds *via* stomach tube results in attempted retching, decompression should again be attempted.

Response to gastric decompression is helpful in differentiating between acute gastric dilatation and gastric distension secondary to small bowel displacement. If the small bowel has been displaced long enough for the stomach to be filled secondarily, one should observe significant signs of intoxication, *i.e.*, prolonged capillary refill time, rapid, poor quality pulse, and congestion of the palpebral conjunctiva. In contrast, acute gastric dilatation is associated usually with quasinormal membrane colour and capillary refill time even though pain may be intense. If the stomach is under pressure secondary to small bowel displacement, decompression causes usually only a partial amelioration of pain and little or no improvement in signs of intoxication. Furthermore, decompression in such instances does not improve peristalsis, whereas thorough decompression of the stomach in acute gastric dilatation without small bowel displacement results usually in marked relief of pain and aid improved peristalsis.

Rectal Examination.

Meaningful rectal examination must frequently be preceded by administration of sedative and analgesic compounds and may be enhanced in sensitive individuals by irrigating the rectum with lidocaine.

Distended loops of small bowel may be palpated to the left of the midline in the ventral middle third of the abdomen. In the author's opinion, it is not possible to differentiate rectally between small bowel displacement and small bowel ileus such as may be associated with acute gastric dilatation. In stallions, even without a history of recent service, one must routinely sweep the pelvic brim to detect inguinal hernia. In the first few hours of inguinal hernia it is usually possible to correct the displacement during rectal examination.

Rectal examination may be helpful to establish that the large bowel is distended. Also, one may be able to palpate an impaction. In our experience the pelvic flexure, right dorsal colon and small colon are the most common sites of impaction, the right dorsal colon being most frequently involved. We have observed that the majority of right dorsal colon impactions are associated with a 'door-knob-shaped-' verminous aneurysm along the course of the ileocaecalcolic artery approximately 15 cm distal from the cranial mesenteric artery. This lesion may be too distal to palpate per rectum but is readily palpable during standing right flank exploratory surgery. We believe that large bowel impactions should not be allowed to remain refractory for more than 72 hours because the mucosa of the bowel by that time may be too seriously damaged, although signs of advanced intoxication do not usually occur if the horse has received proper supportive therapy. We therefore prefer to explore large bowel cases relatively early through a standing right flank exploratory laparotomy. Massage at the most caudal point is usually sufficient to move the mass if mineral oil and a surface tension reducing agent have been administered previously.

Palpation of the gas-distended apex of the cacum in the upper middle third of the abdomen indicates that the large bowel has turned 180^0 on its mesenteric attachment. Rectal palpation of the cranial mesenteric artery is helpful and necessary during examination of recurrent colics, particularly those associated with progressive debilitation. A wide diversity of findings may be noted but increased size, fibrosis, nodularity, fremitus and pain on digital pressure are characteristic. Vascular response to larvacidal doses of Thiabendazole† is routinely rewarding.

Abdominal Paracentesis

During abdominal crisis, paracentesis may be helpful in determining the morphologic status of the bowel[1 2 14]. If the effusion is sanguineous, there is a necrotizing segment of the bowel; if not, several possibilities must be considered:

1. There is no strangulated segment of bowel.
2. There is a strangulated segment of bowel but irreversible damage has not yet occurred.
3. There is a strangulated, infarcted segment of bowel which is not situated in the peritoneal cavity, *i.e.*, it may be in the inguinal canal, the thoracic cavity or intussuscepted into the caecum[3].

Occult Blood

Examination for faecal occult blood should be routinely performed on horses affected by recurring colics. This is particularly true of recurrent colics associated with gastric dilatation, in horses with colics associated with progressive debilitation, and when recurring colics follow ingestion of grain. Recurrent colics associated with a positive test for occult blood in faeces should be related to gastric ulcers. The horse may benefit markedly by being switched from a

† Merck and Co., Rahway, New Jersey, U.S.A.

high grain diet to a total hay diet. As a note of interest, protein-losing enteropathy may be associated with gastric ulcers, although migration of strongyle larvae has been suggested as the most common cause [10].

EVALUATION OF CIRCULATORY CAPACITY

The circulatory capacity of the horse is routinely evaluated by the rate and quality of the peripheral pulse (compared with auscultated heart rate), capillary refill time assessed by compression of the gingival mucosa, membrane colour (palpebral conjunctiva and gingival mucosa), packed cell volume and total serum or plasma protein. Measurement of venous or arterial blood pH, arterial PO_2 and PCO_2, arterial blood pressure, central venous pressure, and urine output bring greater resolution to circulatory capacity and should be performed when possible. Evaluation may be further enhanced by measurement of plasma water volume and subsequent computation of total blood volume by the Evans blue method[5].

Meaningful interpretation of these findings must be related to basic mechanisms of septic shock[6 9 12]. Fundamentally, stasis and degeneration of the gut wall leads to increased endotoxin absorption. Increased endotoxin levels are associated with increased catecholamine levels producing an increase in peripheral resistance and secondary capillary pooling. Prolonged capillary pooling results in cellular hypoxic changes and in loss of capillary integrity leading to movement of plasma water into the interstitial space. Resulting haemoconcentration increases the viscosity of the blood, contributing further to decreased capillary flow.

For purposes of discussion methods of evaluating circulatory capacity may be organized as follows:

Venous Return, Cardiac Output and Arterial Blood Pressure

Normal homeostatic mechanisms will support central circulating volume in an effort to maintain perfusion of vital organs. Cardiac output and arterial blood pressure, therefore may be maintained within reasonably normal limits even though some tissues (including the capillary beds in the wall of the bowel) may not be perfused adequately. Thus, if cardiac output and arterial blood pressure are decreased (as evidenced by a rapid pulse with qualitative pulse deficit) it is a virtual certainty that all vital organs are poorly perfused. Decline of central venous pressure indicates that an insufficient volume of blood is being returned to the right atrium. This may be due to either a declining plasma water volume or to sequestration of blood volume in the splanchnic circulation. Decreased venous return will lead to decreased ventricular filling, decreased cardiac output and subsequently decreased arterial blood pressure. It is possible, therefore, to have a decrease in arterial blood pressure even in the face of profound peripheral vasoconstriction. Rising central venous pressure (above 15 cm H_2O – 17 x 10^2 Pa –) indicates cardiac failure. The clinical observation of normal quality pulse and a heart rate in the near normal range suggests adequate central volume and adequate peripheral arterial pressure, particularly if the horse voids urine. It is desirable to maintain a central venous pressure of 6 to 10 cm of water 6-10 x 10^2 Pa) and a mean arterial blood pressure approximating 80mm of mercury (106 x 10^2 Pa). Normal systolic values will range around 110mm of mercury and normal diastolic values will be in the area of 60 to 70mm of mercury (80-90 x 10^2 Pa). In a hospital environment it is simple and practical to monitor central venous pressure with a water manometer and to monitor arterial blood pressure by the Doppler method[8].

Hypotensive or haemoconcentrated individuals should not receive vasodilatory drugs but normotensive, normovolaemic individuals may benefit from the judicious administration of vasodilatory compounds such as promazine-derived tranquillizers which increase tissue perfusion through vasodilation. We have observed that administration of promazine HCl to such individuals with colic results in a precipitous drop in blood pressure with recovery into the low normal range within 30 minutes.

Capillary Perfusion

Capillary refill time as measured by compression of the gingival mucosa is an index of tissue perfusion. Normal capillary refill time approximates two seconds. Prolonged capillary refill time is associated with vascular constriction, constricted blood volume or capillary and venous pooling.

Blood Volume and Viscosity

Blood volume may be estimated and viscosity monitored by measuring the amount of solids remaining in the circulating blood. Packed cell volume and total protein are commonly used. Haemoglobin determinations can also be used since three times the haemoglobin value approximates the packed cell volume. Disadvantages of packed cell volume include a wide range of normal and spontaneous elevation by contraction and evacuation of the spleen during periods of excitement. Total protein has the advantage of a narrow normal range (6 to 7,5 g% for serum) and is unaffected by excitement. One may be led astray, however, during periods of plasmaphoresis associated with peritonitis. In such instances, large volumes of plasma protein are lost into the peritoneal cavity and serum protein values may remain normal or subnormal even as haemoconcentration occurs. It is therefore obvious that a combination of packed cell volume and total protein is far more reliable than either of the two used independently.

Haemoconcentration, as reflected by increase in packed cell volume and/or total protein, indicates a contracting total blood volume. It should be emphasized that vasodilatory compounds are patently contraindicated when blood volume is compromised. These compounds should be used only when blood volume is known to be adequate.

Plasma water volume may be quasi-definitively measured and total blood volume subsequently computed by the Evans blue method if a spectrophotometer is available for reading the results. If the body weight of the horse is known, these data can be generated on a useful basis within one hour[5].

Acid-Base Balance

Increased respiratory rate suggests systemic acidosis. Acidosis may be associated with arterial venous shunting in the lung with subsequent CO_2 retention or with systemic lactic acidosis resulting from decreased arterial perfusion of the periphery with hypoxic cellular changes. Certainly, cyanosis and increased respiratory rate suggest hypoxia and a ten-

dency toward acidosis. Venous pH is more readily monitored than arterial pH and is helpful as a screening practice in measuring or detecting systemic acidosis. If the venous pH is abnormal, arterial PO_2 and PCO_2 are definitive methods of characterizing the cause. Regardless of the cause of acidosis, the administrations of bicarbonate intravenously and oxygen *via* a patent airway are obvious remedies.

Discussion of hypoxia as a common complication in abdominal crises raises an important point concerning forced exercise. Forced exercise is a valid first-aid measure to prevent self-trauma. In present-day pharmacologic efficacy, however, one should be able to provide absolute analgesia and, if necessary, anaesthesia to the extent that pain is not a persistent problem, except for short, mild episodes. Forced exercise is contra-indicated as a substitute for pharmacologic modes of providing analgesia. During hypoxia, cells are unable to utilize energy sources in the ongoing generation of available ATP. Also, catecholamine and steroid output associated with pain, fear and anxiety contributes to reverse glycolysis, further diminishing effective energy consumption. Thus, what energy sources are available should be preserved since forced exercise causes undue consumption of a failing supply of ATP.

INITIAL THERAPY

Based on the above discussion, several guide-lines may be suggested for initiating treatment.

1. Gastric intubation is the first move toward diagnosis and therapy.
2. Mineral oil should be administered to the majority of horses suffering from abdominal crisis but is contra-indicated when gastric decompression is necessary.
3. Oral antibiotics (neomycin sulphate$_4$) should be administered with mineral oil unless the horse is toxic, in which case broad spectrum antibiotics should be given parenterally. Tetracyclines should be avoided because they have been incriminated in causing acute colitis[4]. Chloramphenicol is preferred in our practice.
4. Sedation and analgesia are essential. Our preference is intravenous chloral hydrate and magnesium sulphate in combination because they can be given to effect. If surgery is not contemplated, chloral hydrate, magnesium sulphate and pentabarbitol combinations are desirable. These compunds may be efficaciously combined with promazine-derived tranquillizers if blood volume is adequate and surgery is not contemplated.
5. Smooth musle stimulants and relaxants may be beneficial when used judiciously but should not be substituted for decompression and analgesia. These compunds are much more effective in treatment of the large bowel. Cholinergic comounds may even be contra-indicated in treatment of the small bowel because of increasing the rate of fluid dump into the lumen.

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SURGICAL TECHNIQUES IN EQUINE COLIC

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SUMMARY

Emergency equine abdominal surgery is easiest and most efficiently carried out with a team of surgeons. The surgical site should be as protected as possible by the use of sterile drapes and wound protectors. A ventral midline laparotomy incision has been found to be the most convenient approach to most equine intestinal obstructions. A standing laparotomy through the left paralumbar fossa gives adequate exposure for exploration of the abdomen and is, therefore, useful as a diagnostic tool. Horses tolerate having both ventral midline and left paralumbar laparotomy incisions well.

If the cause of the intestinal obstruction is not readily apparent upon opening the abdominal cavity, a thorough systematic exploration of the abdominal cavity is necessary. If the problem cannot be found with the bowel *in situ*, intestine must be exteriorized for examination.

The decision as to the extent of adequate bowel resection often depends on a subjective assessment of bowel function. In equivocal cases, the surgeon should choose to resect some normal bowel rather than taking a chance on leaving compromised bowel in place.

Incarcerations are frequent causes of small intestinal obstructions. The small intestine may become incarcerated in the epiploic foramen, the inguinal canal or in an umbilical hernia. Thromboembolic compromise to intestinal vessels results in the longest lengths of embarrassed bowel requiring resection. Impactions are the most common obstructions associated with the caecum.

Large colon torsions of 270° or less may be corrected by surgical manipulation; with 360° torsions of the large colon, however, vascular compromise is usually sufficient to devitalize this organ. Enterotomy of the large colon allows retrieval of most enteroliths from its lumen. Enterotomy of the right dorsal colon is also useful for removal of foreign bodies which cause obstruction of the most proximal portion of the small colon.

In our Clinic a two-layer end to end anastomosis is usually utilized. Recently introduced automated stapling and ligating instruments have been useful in decreasing surgical time.

Antibiotics, usually furacin and sodium or potassium penicillin in 2 litres of Normasol-R, are placed in the peritoneal cavity before closure of the abdomen. A Penrose drain is commonly placed into the abdominal cavity to provide drainage of the peritoneal cavity after surgery. The peritoneum is sutured with No 0 chromic gut in a simple continuous pattern. A second Penrose drain may be placed between peritoneum and ventral body wall, with its ends retracted through stab incisions in the skin. The linea alba is closed with simple interrupted sutures of stainless steel wire or No. 3 chromic gut.

Employing the above described principles and techniques has increased the success of abdominal surgery in our Clinic.

GENERAL PRINCIPLES

Many factors have contributed to the increasing survival rate of equine surgical colic patients within recent years, including a better understanding and management of the associated physiological disturbances, greater confidence in our ability to make an early diagnosis, improvement in aneasthetic management and refinement of surgical techniques employed in the equine abdomen. This discussion is concerned with some techniques of abdominal surgery frequently employed at the Large Animal Clinic at the University of Pennsylvania School of Veterinary Medicine.

Since most abdominal operations are lengthy enough to tire one surgeon physically and mentally a team of three or four surgeons is preferable. Many procedures require at least two surgeons and a third assistant may decrease surgical time significantly. If possible, a surgeon other than the one primarily responsible for the intestinal surgery should be available to suture the ventral abdominal incision, thus ensuring that a dependable closure is performed as rapidly as possible.

Adequate draping to protect the surgical field is a necessity. We use at least six 1,5 x 2m drapes to cover the abdomen, the legs and the areas of the surgery table in close proximity to the surgical field. On top of our regular drapes we use a large laparotomy drape with the sides folded to form a sling for support of exteriorized loops of bowel. Frequently, the surgical field becomes quite moist and, without proper protection, may become contaminated by wick action of soaked cloth drapes in contact with the aseptic field, as well as with surgically unprepared areas of the horse. Large sheets of Vi-Drape† surgical film were considered unsatisfactory because motion at the incision and moisture often resulted in their displacement. Rumenotomy-style drapes††, which are self-retaining in the incision, have been useful as barriers to intraperitoneal and incisional contamination.

We routinely use a ventral midline approach with the dorsally recumbent horse under general inhalation anaesthesia. This approach gives the greatest single incision exposure of the peritoneal cavity and is the quickest approach with the least haemorrhage. The unitial, approximately 20 cm incision anterior to the umbilicus can be extended from the xiphoid to the pubis of the female and, with surgical reflection of the prepuce, to the pubis of the male. A flank approach through the left paralumbar fossa may sometimes be employed if simple exploration with minimal visualization or mobilization of the bowel is required. Occasionally, a standing laparotomy, performed as a diagnostic tool, may demonstrate that a ventral midline approach is necessary for correction of the gastrointestinal condition. Horses readily tolerate both types of laparotomy.

Immediately after entering the peritoneal cavity, the presence of extraluminal faecal material, the character of peritoneal fluid, and discoloured,

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† Vi-Drape Regular Surgical Film: Parke, Davis & Co., Detroit, Michigan.

†† Vi-Drape Wound Protector, 36" x 36" with 11" Ring: Parke, Davis & Co., Detroit, Michigan.

thickened, or distended loops of intestine should be noted. If numerous loops of gas or fluid-distended intestine are ballooning out of the incision, decompression should be employed immediately to facilitate abdominal exploration. Decompression of gas is accomplished by penetrating the bowel with a 14-gauge needle attached to a vacuum line. The site of needle penetration is closed with one simple Lembert or infolding purse string suture. If a signifficant amount of fluid is present, a larger bore instrument is required, such as 10 mm internal diameter rubber tubing attached by a vacuum line to a sediment receptacle. A purse string suture is placed around the penetration site and tightened as the tubing is removed from the intestine. The ideal site for decompression enterotomy is controversial. Some prefer the intestinal taenia for suction decompression as it is avascular[4]. As haemorrhage is seldom a problem when a suture is placed across the penetration site, non-taenia areas of the intestine are acceptable and may actually be easier to close with a suture than the taenia in slightly friable intestine. When enterotomy is indicated, the involved bowel should be exteriorized and packed off from the surgical field.

If the obstructive lesion is not readily discernible upon entering the abdominal cavity, a thorough exploration of the abdomen is indicated. Moistened, shoulder-length rubber obstetrical gloves should be worn by the surgeons to explore the abdominal cavity since the moist sleeves of the surgeon's gown in contact with the surgeon's arm may act as a wick and the cloth gown may abraid serosal surfaces. A systematic method of abdominal exploration is mandatory. The simplest, most efficient method is to divide the abdomen anatomically into quadrants. Gentle sweeping or ballottment through the intestines in each quadrant usually reveals any abnormality present. Organs such as liver, spleen, bladder and associated reproductive organs must be examined. It is important to remember the presence of the spleen and pelvic flexure in the left posterior quadrant. The area of the gastrosplenic omentum and splenic suspensory ligament must be explored because intestinal displacement to this area may result in strangulation of the bowel. Any potential spaces such as the epiploic foramen or inguinal rings, capable of incarcerating intestine must be palpated. The epiploic foramen is most effectively examined in the right anterior quadrant. Palpation is aimed at the detection of distended, thickened, impacted, displaced or strangulated bowel. Abrupt changes in the direction of intestine, tight mesenteric bands around or across intestine, or immobility of normally mobile portions of intestine indicate an area of involvement. The relationship of specific organs may help to indicate the cause of the obstruction; for example, an anterior displacement of the spleen is suggestive of a torsion of the left large colon. To routinely and effectively recognize intra-abdominal abnormalities, the surgeon must have a sound understanding of the anatomy of the normal abdomen.

If palpation fails to demonstrate the causative obstruction, the bowel must be exteriorized to facilitate visual examination for discoloration, distension and motility. The strongly attached stomach cannot be exteriorized through the incision. Normal small intestine can be easily exteriorized except for the most proximal three or four feet and the terminal six to eight inches. The apex and three-fourths of the body of the caecum can be exteriorized without difficulty. The left large colon, portions of the sternal and diaphragmatic flexures, and portions of the right large colon may also be exteriorized. The proximal 50-70 cm and the distal 30 cm of the small colon are not easily exteriorized. The small intestine and the small colon should be examined in a continuous manner either in a retrograde or antegrade direction. The ileocaecal fold will aid in identifying the terminal part of the small intestine, or the apex of the caecum can be lifted out and used as a reference to find the ileum. The large colon is identified after locating the pelvic flexure.

Any exposed intestine must be kept moist and warm, preferably by using a warm polyionic solution†††. Isotonic saline has been shown to be as detrimental to the mesothelia serosal cell as is the drying out of serosal surfaces [3]. Apparently, damaged mesothelial cells allow clotting blood to adhere to the injured surfaces. Blood clots apparently stimulate growth of granulation tissue responsible for mature adhesions. It is hoped that the polyionic solution more closely simulates peritoneal fluid than the saline previously used. The best means of preventing the trauma of exposure is to leave as much uninvolved intestine as possible in the abdominal cavity and to return normal intestine to the abdominal cavity as soon as possible after examination.

The major objective in the surgical management of obstruction, *i.e.*, removal of the obstruction, so that a patent, functional intestinal tract remains, may require the simple repositioning of a displaced portion of intestine or the tedious resection of 3 m or more of the bowel. Intestine which appears abnormal because of vascular embarrassment, scarring or other significant lesion should be removed, if feasible. The decision as to the extent of adequate resection often depends on a subjective assessment of bowel function. Any intestine of abnormal colour, indicating vascular embarrassment, decreased motility or thickening or distension, must be considered suspect. After correction of lesions causing secondary vascular embarrassment, observation of the bowel for return of colour, peristalsis and pulsation of vessels will aid in deciding for or against resection. It may be very difficult to discern the junction between viable and compromised bowel, especially after a displacement has been corrected or decompression performed and significant improvement in the gross appearance of the bowel has taken place. Making a small enterotomy in questionable bowel to determine how easily it bleeds, as well as the appearance of the mucosa, may aid in making a decision. In equivocal cases, a liberal decision is best; the surgeon should choose to resect some normal bowel rather than taking a chance on leaving compromised bowel in place. To this end, one segment of decidedly normal bowel, supplied by a functional mesenteric artery, should be resected at each end of the portion to be removed. One significant cause of surgical failure in initial colic operations was failure to recognize and resect all diseased bowel. The surgeon's adage, 'when in doubt, take it out,' applies here.

Surgical management of specific obstructive entities observed in our clinic will be discussed.

††† Normosal-R: Abbott Laboratories, North Chicago, IL.

SMALL INTESTINE

Incarcerations are frequent causes of obstruction of the small intestine. The small intestine can become incarcerated in the epiploic foramen, the inguinal rings, patent umbilicus or mesenteric rents. Bowel incarcerated in the epiploic foramen becomes firm, thickened and distended. Often, if a small segment is involved, a definite loop can be palpated with both ends disappearing into the foramen. Most incarcerations within the epiploic foramen involve gut moving from the right to the left side of the abdomen. The simplest method of reduction is traction on the bowel leading into the foramen with simultaneous digital manipulation of the incarcerated bowel to ease it gently through the restrictive tissue. The epiploic foramen may be slightly enlarged digitally, to further aid in reduction. If this approach is unsuccessful, the bowel should be resected and oversewn either distally or proximally to the area of the involvement and this segment drawn through to the side of the incarcerated loop. This manoeuvre should allow easier reduction which can be accomplished by bringing a single piece of bowel, rather than a loop, through the epiploic foramen. Significantly embarrassed intestine is reseacted and the normal ends are anastomosed.

If a diagnosis of inguinal incarceration is made preoperatively, two surgical approaches should be considered.

(1). With the horse in lateral recumbency, an incision is made over the inguinal ring. After opening the parietal vaginal tunic, the inguinal ring is enlarged and the incarcerated bowel exteriorized further. It is then possible to do resection and anastomosis of the bowel outside the inguinal ring without actually invading the abdomen. If the testicle is not removed, the size of the inguinal ring is reduced and the incision in athe parietal vaginal tunic sutured. If the testicle is removed, the internal inguinal ring is closed. There is usually a great deal of post-operative scrotal swelling if castration is not performed, but horses treated in such a manner have become fertile sires. If the animal is not to be used as a sire, castration simplifies the surgery. The testicle should be removed if there is any compromise of its vascular supply.

(2). Through a ventral midline laparotomy, reduction of the inguinal incarceration is accomplished by gentle traction with manipulation of the spermatic cord externally. If necessary, an additional incision is made over the inguinal ring and the inguinal ring enlarged. The testicle can be salvaged with this approach, also.

Usually, small intestine is found in umbilical incarcerations. If a chronic inflammatory process has occurred in the umbilical area, however, other portions of the intestinal tract may be adherent to the umbilicus. Some subcutaneous oedema is usually associated with an umbilical incarceration, making dissection difficult. An incision is made to expose, the hernial ring which is extended longitudially until the bowel can be exteriorized for careful examination. If necessary, resection and anastomosis are carried out. After reduction of a hernia, the linea alba is sutured, usually with a Mayo overlap pattern.

Intussusceptions most commonly involve the small intestine, usually with the proximal ileum intussuscepted into a more distal portion of the ileum, or the distal ileum intussuscepted into the caecum. Donawick described a technique of joining the small intestine to the caecum by an end-to-side anastomosis which allows for creation of a new ileocaecal junction in situations requiring resection of the terminal ileum[1]. The terminal ileum is resected as close to the caecum as possible leaving sufficient length to accommodate an infolding closure of the terminal ileum, thus creating an ileal stump on the caecum. Any proximally involved small intestine is resected. The small intestine is sutured to the caecum distal to the original ileocaecal junction and on one side of the ileocaecal fold. This fold traverses the caecum from its apex to the antimesenteric surface of the terminal 50-70 cm of the ileum. The use of this band as a landmark precludes improper placement of the new ileocaecal junction in relation to the caecum. If difficulty is encountered in reducing the ileocaecal intussusception by traction on the proximal portion and manipulation of the intussuscepted bowel through the caecal wall, a caecotomy may be necessary to make reduction possible. A hand is placed into the caecal lumen to force the intussuscepted intestine in retrograde fashion through the ileocaecal valve. We have been able to reduce all ileocaecal intussusceptions by one of the above methods, but surgeons have stated that in cases where the intussusception was not reducible, they closed the ileal stump on the caecum, leaving the intussuscepted portion of intestine within the lumen of the caecum and then performed an end-to-side anastomosis, creating a new ileocaecal junction.

Thrombo-embolic vascular compromise results probably in the longest lengths of embarrassed intestine requiring resection. Close to 9m of small intestine has been removed successfully. While resecting large portions of intestine, it is easy to create a 360° twist in the intestine prior to anastomosis. By noting the relationship of the intestinal clamps to each other and to the projected anastomosis and maintaining this relationship, the possibility of a twist is eliminated. When large areas of necrotic intestine are to be resected, chromic catgut is used for vessel ligation as opposed to silk. It is thought that non-absorbable ligatures may serve as a nidus for mesenteric abscessation.

CAECUM

The primary obstruction associated with the caecum is impaction, comprised usually of dry fibrous ingesta or, occasionally, sand. The caecum may be quite friable if the impaction has been present several days; therefore, care must be taken in exteriorizing it, which is best done by mobilizing the apex first. If a significant amount of the impacted material is sand or gravel, the treatment of choice is caecotomy and manual evacuation of the material. Rumenotomy drapes serve as excellent protectors to minimize contamination of the surgical field. The hoop of the drape is situated over the caecum and the drape spread over the surgical field. The caecotomy may be performed through a taenia or in the non-taenia portion. If the impaction is of a dry fibrous nature, it may respond well to massage and the injection of 5% dioctyl sodium sulphosuccinate (D.S.S.)† through the intestinal wall into the mass. As much as two litres of

† Permeatrate: Haver Lockhart, Laboratories P.O. Box 390, Shawnee Mission, Kanasas, 66201.

5% D.S.S. have been injected into impacted masses. Because D.S.S. can be irritating to serosal surfaces, care must be taken not to spill the solution on to the surgical field, and to oversew the injection sites. The intraluminal injection of D.S.S. is indicated for dry fibrous ingesta impactions in any portion of the intestinal tract.

LARGE COLON

Torsions of the large colon can be most difficult to correct physically. Torsions of the colon through 360°; extending to the root of its mesenteric attachment, are often associated with tremendous gas distension and a gangrenous colon. Decompression and evacuation are usually necessary before the colon can be exteriorized sufficiently to determine the character and direction of the torsion. A large colon with a 360° torsion is frequently so necrotic and friable that it ruptures before it can be exteriorized sufficiently. Lesser torsions, or torsions other than those extending to the root of the mesentery, are often surgically correctable. In such cases, the torsion is most easily corrected if the pelvic flexure and as much associated colon as possible are exteriorized. Once the direction of the torsion is determined, the exposed bowel is twisted in the opposite direction and the correcting twist transmitted to the intra-abdominal portions of the colon. The exteriorized intestine is used as a mechanical lever. If the diagnosis is made early and a torsion of 270° or less is present, vascular embarrassment may not be sufficient to require colonic resection. To replace the large colon into the abdominal cavity, the pelvic flexure is elevated and the weight of the ingestra is allowed to aid in returning the viscus to the abdomen. The sternal and diaphragmatic flexures are positioned anteriorly just below the body wall and the left colon laid along the left abdominal wall. The pelvic flexure is placed as far caudally in the left flank as possible. If large portions of the large colon are removed, the remaining large colon increases greatly in diameter.

Enteroliths causing impaction of the large colon are most commonly found in the left dorsal colon, just distal to the pelvic flexure. If the obstructed area can be exteriorized, a simple enterotomy will allow removal of the enterolith. If the obstruction cannot be exteriorized, an enterotomy has to be performed proximally as close to the obstruction as possible to allow manual retraction of the enterolith.

SMALL COLON

Foreign body obstructions caused by fibrous threads and rubber from rubber fencing have recently been found in the small colon and unexteriorizable transverse small colon. In such cases, an enterotomy is performed over that portion of the gut which can be approached surgically and removal of the foreign body attempted by that route. If the intestine is plicated around the foreign body and the latter cannot be removed from the unexteriorized bowel, consideration should be given to creating a by-pass around that segment of intestine by anastomosing distal, uninvolved small colon to the pelvic flexure and oversewing the proximal small colon stump.

RECTUM

It is difficult to repair rectal tears surgically, adequate exposure of the rectal defect being almost impossible. We have been developing a procedure whereby a temporary colostomy in the left flank would allow sufficient time for the rectal defect to heal. The small colon anterior to the rectal tear and distal to the colostomy is oversewn. After approximately 30 days, the intestinal continuity is re-established through a ventral midline laparotomy. The small colon is re-established with a side-to-side anastomosis.

INTESTINAL ANASTOMOSES

In end-to-end anastomosis two layers are used for closure in our clinic. Stay sutures are first placed in the mucosa at the mesenteric and antemesenteric edges. These stay sutures are held apart by an assistant to prevent narrowing of the lumen during the rest of the anastomosing procedure. The mucosa is then approximated by a continuous simple suture pattern with 00 chromic catgut. The submucosa, muscular and serosal layers are apposed with silk or catgut, so that one-half of the circumference is closed with interrupted sutures to permit later expansion of the anastomosis. The remainder is closed by simple continuous suture. The simple interrupted crushing suture described in the horse by Herthel [2] is occasionally used, especially for end-to-end anastomosis of the small intestine or small colon in foals, as it forms less immediate stricture of the lumen at the anastomotic site.

We have been using automated stapling * and ligating ** instruments for about one year, for closure of enterotomies, end-to-end anastomoses of the small intestine or small colon, and end-to-side, small intestine to caecum anastomoses. The instruments used for the intestinal anastomoses produce a linear double-staggered row of stainless steel staples. End-to-end anastomoses are done in three sided or traingular fashion with the first two sides stapled serosa to serosa and the remaining edges stapled mucosa to mucosa. This pattern creates inversion of one-third and eversion of two-thirds of the anastomotic circumference. Another instrument ligates the mesenteric vessels with a compressed staple on either side of the simultaneously divided blood vessels. These instruments significantly decrease the time required for mesenteric or omental vessel ligation and division, and for intestinal anastomoses. The decreased anaesthesia and surgery time required may offset the cost of using this equipment as measured by the increased survival owing to a shorter surgical procedure.

FINAL PROCEDURES

After anastomosis or closure of an enterotomy, the involved area of bowel is flushed with a polyionic (Normosol-R) and antibiotic solution. After completion of intestinal manipulation and just before closure of the abdominal incision, a warm antibiotic solution is dispersed throughout the abdominal cavity *via* a flexible tube. The antibiotic solution consists of 40 ml nitrofurazone *** and 20 million units of potassium **** or sodium penicillin G † in a litre of Normosol-R. Two litres of this solution is instilled into the

* Model TA55 and TA90, Auto Suture, United States Surgical Corporation, 845 Third Avenue, New York, NY 10022.
** LDS, United States Surgical Corporation, 919 Third Avenue, New York, NY 10022.

*** Nifurone: Diamond Laboratories, Des Monies, Iowa 50304.
**** Potassium Pencillin G: E.R. Squibb & Sons, Inc., New York, NY 10022.
† Buffered Sodium Penicillin G: E.R. Squibb & Sons, Inc., New York NY 10022

abdominal cavity. Some clinicians prefer only one litre of Normosol-R containing 4g of neomycin sulphate †† and 40 million units of potassium or sodium penicillin G.

A Penrose drain ††† is placed into the peritoneal cavity and its ends are retracted through muscle, subcutaneous tissue and stab incisions in the skin just to the side of either end of the surgical wound. When the peritoneum is strong enough to hold sutures well, it is closed with No. 0 chromic catgut in a simple continuous pattern. A second Penrose drain is placed between the peritoneum and ventral body wall and its ends are retracted through stab incisions in the skin on the opposite side of the surgical wound from the first drain. If contamination of the incision may have occurred, non-absorbable suture material (with the exception of stainless steel) should not be used to close the body wall. We have not found a completely ideal suture material for closure of the linea alba. At present, we are most often using stainless steel or No. 3 chromic gut. The use of autoclaved synthetic non-absorbable suture material †††† has been incriminated as a cause of some post-operative incision complications, such as fistula formation. The linea alba is closed with simple interrupted sutures. If necessary, Backhaus towel clamps can be used to hold the edges at the linea albea together to decrease the tension on the suture line and facilitate closure. The exposed portions of the drains are sutured to the skin to prevent retraction into the incision during recovery and post-operatively. After clossure of the subcutaneous layer with No. 0 chromic catgut in a continuous pattern, the skin is sutured with non-absorbable material in interrupted fashion.

CONCLUSIONS

The above-described principles and techniques have favourably influenced the outcome of abdominal surgery in our clinic. Successful surgical management of the acute abdomen is not unique; it is based on aseptic technique, atraumatic organ manipulation, reconstruction of any organ damage found or created; appreciation of the normal or abnormal states likely to be present; and sufficient imagination to devise viable means of surgical repair.

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† Biosol: Upjohn Company, Kalamazoo, Michigan 49001.
††† X-ray Opaque Amber Latex Penrose Drain: Davol, Inc., Providence, Rhode Island 02901.
†††† Vetamid, Size 3 extra heavy, Lentz, Philadelphia, PA.

THE LABORATORY ANIMAL — PRINCIPLES AND PRACTICE

W. LANE-PETTER AND A.E.G. PEARSON

Academic Press, London and New York, 1971. Pp. XI+ 293, Figs. 28, Tabs 9. Publ. Price: £4.00.

This book contains a wealth of valuable information and will repay intensive study by anybody either directly or indirectly concerned with the use of animals for experimental work. Scientists, legislators, animal welfare societies, animal lovers, educationists and conservationists will find guidance on problems encountered or expected. A balanced and practical view of the issues involved in the use of animals for experiments is presented. The technical information is excellent.

The book begins with a review of the animals used in experiments, the numbers of each species used, who uses them and for what purpose. Sources of supply, matching supply and demand, production and disposal of surplus animals are considered. There is a list of safety precautions to take when stray dogs or cats are presented for use in experiments. Trends in the use of animals in experiments are considered. There is a section on breeding, genetics and the causes, manifestations, prevention and elimination of ill health. Animal houses, equipment, cages, the requirements of each type of animal and control of the environment to suit their needs are dealt with. Consideration is given to feeding requirements, diets, feed storage, sterilization of feed and the provision of disease free, suitable water. The organization of the administration and husbandry of the enterprise including the qualities required in staff, their selection, responsibilities, work, training both on the job and in formal course with a syllabus for such courses are set out.

An account is given of financial management, costs, budgeting, requirements of production, type and size of colony required, possible need of expansion, breeding systems, size of breeding units, control of breeding including optimum breeding age and gestation periods, and factors which could result in introduction of disease. Means of transport, travelling cages and the best means of ensuring that experimental animals travel safely and arrive at their correct destination are discussed. Finally, organisations in the United Kingdom and elsewhere concerned with the welfare of animals are listed and legal requirements set out.

Explanatory diagrams and tables are included in the text. The printing is clear, well spaced and easy to read. There is a list of references, bibliography, author index and subject index.

G.D.S.

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TEMPORARY COLOSTOMY IN THE MANAGEMENT OF RECTAL TEARS IN THE HORSE*

M.A.J. AZZIE**

SUMMARY

Basing his experiences on two cases, one with a fatal complication and one with a successful outcome, the author describes the technique of temporary colostomy and subsequent treatment in the management of rectal tears.

INTRODUCTION

Accidental rupture of the rectum during rectal examination of abdominal organs of the horse is an ever-present, potential disaster that may befall the practitioner. The professional embarrassment is profound and the financial loss usually considerable. The reactions of the clinician vary from impetuous destruction of the horse to dishonest silence, thus casting a doubt on the integrity of the profession. Undoubtedly, such an accident probably will not be ascribed legally to negligence, provided that proper care, handling, restraint and lubrication are practised, but that in itself is a poor solace.

The circumstances that may lead to such mishaps are: improper management, such as examination in the open with only a head stall to control the horse; resistance of the animal to the discomfort of the examination or to the excruciating pain when a pathological lesion or an organ in a particular physiological state is manipulated, such as a grossly enlarged ovarian follicle or a hypersensitive post-ovulatory follicle; imperfect state of health of the rectum, as occurs in ischaemia caused by, *inter alia,* aorto-iliac thrombosis. The temperament of the patient, but most of all that of the attending veterinarian, is important.

Should such a disaster be suspected, a careful, complete examination must be conducted: an immediate and accurate diagnosis is of vital importance for the eventual survival of the horse. A small tear will become gross if left unattended and death will follow in spite of the sparing effect of the ensuing intestinal atony. Suturing the rectal tear may sometimes be accomplished successfully. In the event of the tear being a major one, however, with probable intracoelomic involvement, colostomy is indicated.

Two cases are reported, one with a fatal complication and one with a sucessful outcome.

CASE HISTORIES

Case 1: A Six-year-old stallion. The rectum was torn when being examined during a violent spasmodic colic, under circumstances of tremendous resistance. The tear was retroperitoneal.

Case 2: A maiden brood mare which resisted ovarian examination.
The resulting tear was intracoelomic.

SYMPTOMATOLOGY AND COURSE

The clinical signs a few hours after occurrence of the tear were: evidence of severe pain, particularly when she was forced to move, but without violence or attempts to roll or lie down; muscular spasms over the dorsal flank region; accelerated pulse; congestion and petechiae of the conjunctivae; a mild respiratory grunt; and tremendous resistance to rectal examination. Haematological examination revealed an elevated venous heamatocrit, a retarded ESR and an elevated WBC.

Since both cases were referred, there was some delay before surgery was commenced. Case 1 was operated on 48 hours after occurrence; it made excellent progress for five weeks, when it died of acute haemorrhage resulting from a mesenteric tear associated with an everted prolapse of the colon through the colostomy wound a day before closure and rectocolic anastomosis were programmed.

In Case 2 surgical intervention took place within six hours. This was successful and the rectal tear healed within four weeks, making closure of the colostomy feasible. The mare was discharged fourteen days after repair of the colostomy and performance of bowel anastomosis.

SURGICAL TECHNIQUE

Acetyl promazine malleate [1] tranquilization was followed by thiopentone sodium [2] induction and halothane [3] as maintenance anaesthesia. Glyceryl guiacolate aether provided suitable relaxation.

An oblique incision large enough to admit the hand and forearm, was made in the left iliac region, 10 cm cranial to the level of the umbilicus, extending caudoventrally to the level of the mesogastrium. This site was selected because of the proximity of the small colon and because evacuated faeces would clear the body with a minimum of contamination. Furthermore, by having this site well cranialward, the colostomy could be performed at that level of the small colon where the faeces had not yet undergone such a degree of dehydration that impaction at the colostomy opening would be likely to occur. In retrospect it was considered that too large an incision could facilitate or predispose to eversion and prolapse.

An assessment of the damage was made by inta-abdominal manual examination. The omentum was found to have localized the trauma in the distal small colon. It was left undisturbed and only infiltrated

* The subject matter of this paper was reported upon at the New Bolton Centre in December, 1972. The paper was presented at the SAVA Congress in Pretoria in October, 1973, but never submitted for publication. It is included here on account of its relevancy and with reference to the General Discussion which follows later.

** P.O. Box 4024, Alrode, Transvaal.

1 Acetylpromazine: Boots Co., Nottingham, England.
2 Intraval: Maybaker, Port Elizabeth, South Africa.
3 Fluothane: Imperial Chemical Industries, Johannesburg.

with antibiotic solution. Two incisions of 1 cm long were made midventrally in the abdominal wall and a 20 cm Penrose drain was inserted and fixed to the skin externally to provide drainage for the septic peritoneal exudate.

Colostomy Procedure

The distal end of the small colon, about one metre from the rectal junction, was used for performing the colostomy.

It was everted into abdominal wound where it was fixed with three layers of interrupted matress sutures of braided silk. The first series of sutures attached the visceral to the parietal peritoneum. The second series fixed the visceral peritoneum *(tunica serosa)* and the *tunica muscularis* of the colon to the surrounding musculo-aponeurotic layers of the abdominal wall. The colon was then incised – a 10 cm opening was found to be adequate – and the third layer of sutures was applied through the epidermis and through the wall of the colon into the lumen. The sutures were tied on the epithelial surface.

The distal part of the colon was flushed with water from a low pressure hose. The rectum was flushed without pressure near the tear by means of an antiseptic solution [4]. A polythene tube was inserted into the rectum to bypass the tear and left in situ for daily flushing of the distal colon. Nylon tape tension sutures were then applied to the skin across the colostomy site to prevent prolapsing of the proximal part of the colon, in view of the untoward occurrence in Case 1.

After-care

Rectal and reverse colonic irrigation were carried out daily. Rectal stricture was prevented by gentle manual tearing of adhesions. Antibiotic therapy in the form of procaine penicillin and dihydrostrepto mycin [5] was applied daily by intramuscular and intraperitoneal administration.

Post-operative Course

The rectal tear healed adequately within four weeks. In the surviving case the original surgical adhesion of the colon to the abdominal wound was incised, a loop of small colon exteriorized, the damaged section of the bowel clamped off, and an end-to-side anastomosis of the healthy portions of the small colon was performed satisfactorily. Bowel lubricants were administered, as well as physostigmine [6] orally at the rate of 5 mg t.i.d. The first faeces were evacuated 24 hours after closure of the colostomy.

CONCULSION

Surgical intervention to effect a temporary colostomy as an emergency aid to horses that have suffered rectal damage has been performed successfully and is to be recommended. The rectal tear can heal within four weeks when completely rested. Subsequent repair of the colostomy and bowel anastomosis re-instate normal digestive and eliminative function.

4 Betadine: Saphar Laboratories, Aeroton, South Africa.
5 Pen-strep: Millborrows, Pietermaritzburg, South Africa.
6 Prostigmine: Roche Laboratories, Isando, South Africa.

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POST-OPERATIVE MANAGEMENT OF EQUINE ABDOMINAL PATIENTS

C. BOLES*

SUMMARY

Adequate post-operative management of equine abdominal patients is as necessary to patient survival as the most heroic corrective surgery. Post-operative management must begin during the anaesthesia recovery phase to insure adequate oxygen supply, ventilation, and minimize any abdominal discomfort. The animal's physiological status must be constantly monitored to detect and determine the degree of abnormalities concerning serum electrolytes, fluid balance, and acid-base abnormalities. The most commonly observed serum electrolyte imbalance is hypokalaemia. Replacement potassium is usually supplied intravenously. If the dificit is slight, oral replacement may be possible. Fluid balance is usually maintained via intravenous fluid therapy as determined by monitoring PCV and plasma protein. Metabolic acidosis is treated with 5% sodium bicarbonate administered intravenously. Broad spectrum antibiotics are usually given intravenously but may be administered intra-peritoneally. To facilitate the intravenous therapy, an indwelling silastic catheter is often employed to minimize jugular thrombosis.

Successful post-operative management of equine abdominal patients depends upon adequate intensive care as guided by close monitoring of physiologic parameters including acid-base, electrolyte and body fluid balance. Good nursing care must include adequate antibiotic therapy and management of the surgical wound. This discussion is based on a chronological description of the routine handling of post-operative patients at the Large Animal Clinic of the University of Pennsylvania, and includes some practical aspects of fluid and electrolyte replacement therapy.

Post-operatively, the clinician's first concern is the horse's recovery from anaesthesia. When 100% oxygen and positive pressure ventilation are withdrawn and horses must breathe room air, they may experience a drop in arterial oxygen levels, sometimes to as low as 40 mm Hg (53 x 10^2 Pa). Since arterial oxygen tension can be elevated significantly with oxygen therapy, in the recovery stall eight to ten liters of oxygen per minute are bubbled through water and administered through the endotracheal tube. When the swallowing reflex returns and the endotracheal tube must be removed, oxygen may be administered through the nose if necessary until the animals stand. Pulmonary function in animals in lateral recumbency while recovering from anaesthesia is probably similar to that in the anaesthetized, laterally recumbent horses described by Gillespie *et al* in 1969 [4]. Their work indicated that a marked decrease in the arterial oxygen tension may be correlated with pulmonary arterial venous shunts, passive congestion and mechanical hindrance to efficient expansion of the dependent lung. Therefore, although post-operative patients are not encouraged to stand in the recovery stall until they are willing to do so, they are assisted to the sternal position as soon as possible to maximize pulmonary efficiency.

During the first two hours of recovery, haemoconcentration may occur. At the conclusion of the surgery, most animals have a PCV in the high thirties or low forties, with plasma protein levels which are variable but within normal limits. During the initial recovery phase, the PCV can increase to the high forties or low fifties with slight, if any, alteration of the concentration of plasma proteins. This absolute haemoconcentration is thought to be the result of splenic contraction under the influence of circulating epinephrine during the stress and struggling of recovery. It is usually not necessary to give intravenous fluids to such animals, as experience has shown that, within a few hours after standing, the PCV will begin to approach a normal level in uncomplicated cases. When the PCV and plasma protein levels are both rising significantly, however, appropriate intravenous fluid therapy should be initiated as such findings indicate major body fluid shifts.

As soon as horses are able to stand in the recovery stall, they may exhibit signs of distress such as shaking, trembling, sweating, musculoskeletal stiffness, and vague abdominal discomfort, especially if there has been extensive surgical manipulation of the gastrointestinal tract. Clinically, it appears that horses have a smoother and easier recovery if they receive 4 mg/kg body mass of phenylbutazone [1†] intravenously, immediately prior to the operation.

Three or four hours post-operatively, if the horse is comfortable, it is offered one to two gallons of warm water. If this amount is tolerated, the horse is allowed to slowly satisfy its thirst over a one-half-hour period and may be given a small amount of hay or a bran mash, but usually is not fed for at least 12-24 hours. Interest in eating, drinking, return of auscultatable gastrointestinal sounds, and ability to defaecate and urinate indicate returning gastrointestinal function and are prognostically good signs. Animals responding in such a fashion are closely observed throughout the first 24 hours and PCV, plasma protein concentration and TPR are frequently determined. A routine CBC is usually obtained during this period. If no further complications develop, daily physical examination, plasma protein concentration, and PCV determinations are sufficient. Attention should be paid to the presence and amount of faeces passed daily.

Occasionally, animals will exhibit signs of colic immediately upon recovery from anaesthesia. A nasogastric tube should be passed in such animals immediately. If the reason for the discomfort is distension of the stomach, gas and sometimes as much as 10 litres of fluid can be recovered under pressure with some manipulation of the tube. Some horses will not appear uncomfortable until they have consumed a small amount of water, free choice. These animals commonly paw, play in their water bucket, and may have ele-

1† Butazolidin: Jensen-Salsbery Laboratories, Kansas City, MO 64141.

vated heart and respiratory rates. As much of the gastric fluid as possible should be removed from such individuals and no consideration should be given to allowing them to consume food or water for 24 hours.

Even though their oral intake is completely restricted, some horses will continue to accumulate fluid in the stomach and evidence signs of distress. The stomach accumulates reflux secretions from the small intestine, secondarily to absence of gastrointestinal motility (ileus) and impaired gastrointestinal function. When ileus occurs, any portion of the bowel may sequester large amounts of fluid, which deplete the extracellular fluid compartment and may result in dehydration and massive electrolyte shifts. In addition, continued necessary evacuation from the stomach of the reflux fluid rich in electrolytes, HCO_3- and H+ may further exacerbate dehydration, acidosis, hypokalaemia, alkalosis, or other abnormal metabolical states. Animals with post-operative ileus should have blood gas, electrolyte and multiple PCV and plasma protein concentration determinations, for adequate management. There are no reliable clinical observations which will enable the clinician to predict the metabolic state of the patient accurately. According to laboratory values obtained, appropriate supportive intravenous therapy should be initiated immediately.

The aetiology of ileus in the horse has not been elucidated; therefore, specific therapy is empirical. The cardinal principle of ileus management is removal of the cause and appropriate supportive therapy. Post-operative ileus may be the result of a mechanical obstruction preventing the movement of ingesta. The surgery may have been unsuccessful in correcting the original problem or an obstruction may have been induced at the time of surgery by malpositioning of intestines or by intestinal oedema. Vascular insufficiency or ischaemia can produce a nonfunctional intestine. Failure to resect any intestine with vascular compromise, or the creation of ischaemic intestine during surgery, can be responsible for the ileus. Surgically manipulated or traumatized intestine may be slow to regain normal function. Severe metabolic disturbances are candidates as the causes or potentiators of intestinal dysfunction necessitating the continual monitoring, reevaluation and maintenance of the animal's physiological state. If a second laparotomy is indicated, it should be performed as soon as any significant metabolic disturbances can be corrected. Pain may play a significant rôle in decreasing intestinal motility. Phenylbutazone is effective in the control of musculoskeletal pain and clinically it appears as though it may have some visceral analgesic properties. Dipyrone [2†] is often quite useful in the control os post-operative abdominal pain with clinically variable effects on intestinal motility. One major post-operative disadvantage is its antipyretic effect which decreases the usefulness of body temperature as an aid in determining the animal's well-being. Pentazoncine [3†] and meperidine hydrochloride [4†] are also employed as post-operative analgesics.

The administration of 0,55 mg/kg of meperidine hydrochloride intramuscularly to animals with post-operative abdominal discomfort has occasionally been associate with an increase in auscultatable intestinal sounds and clinical regression of ileus. Tranquilization with agents which may have adrenergic blocking activity probably should be avoided immediately post-operatively. The possibly hypotension produced by these agents may be deleterious to animals so recently having undergone lengthy anaesthesia and surgery, and to animals which may soon require additional surgery. Consideration should be given to the use of agents which stimulate gastrointestinal motility. Parasympathomimetic agents such as neostigmine methylsuphate [5†] (0,005 – 0,007 mg/kg subcutaneously) may be of benefit in some cases. Sympatholytic agents such as chlorpromazine are used in man for the relief of paralytic ileus [6†] and may be useful in the horse. To our knowledge, the drug has not been tested adequately in horses: one normal animal, given the drug at our clinic, became extremely excited. In our experience, gastrointestinal stimulants have been of questionable value in the treatment of ileus.

Light, controlled exercise in the form of hand walking may be of value in the initiation and maintenance of intestinal motility in some animals, especially those which were reasonably fit and active prior to surgery. Lower bowel motility may be stimulated with enemas.

The remaining portion of the discussion will be directed toward consideration of some practical aspects of post-operative therapy.

For the intravenous management of metabolic acidosis, we use a 5% solution of sodium bicarbonate. This solution is commercially available or can be prepared by the addition of 50 grams of sodium bicarbonate to a litre of saline or water. In determining the amount of bicarbonate to be given, we use the arterial blood bicarbonate ion concentration, obtained from blood gas determinations. If 25 mEq/*l* is the normal level and the blood bicarbonate ion concentration is reported to be 15 mEq/*l*, a deficit of 10 mEq/*l* exists. If the functional extracellular fluid volume is estimated to be 40 per cent of the body mass[3], a 450 kg horse would have 180 *l* of extracellular volume. Multiplication of the deficit (10 mEq/*l*) by the extracellular fluid space volume (180 litres) gives the total calculated extracellular deficit (1800 mEq) or 150 grams (3 litres of 5% bicarbonate).

Post-operatively, we must replace body fluids which have been lost or are sequestered and are therefore unavailable to the extracellular space. For intravenous fluid therapy, we almost always use a polyionic solution [6] which is also isotonic and contains the equivalent of 50 mEq/*l* of bicarbonate in the form of acetate and gluconate. These precursors are metabolized by extrahepatic tissues, the former very rapidly and the latter slowly; hence, the mixture provides bicarbonate over a long period [5]. Normosol-R is superior to lactated Ringer's solution which has 28 mEq/*l* bicarbonate available as lactate. Lactate must be converted to bicarbonate in the liver. If the liver is already overburdened with lactate from anaerobic metabolism, the lactate in the Ringer's solution will not be as effeciently or readily converted to bicarbonate.

Hypokalaemia is one of the more commonly observed post-operative electrolyte disturbances. Pre-

2† Dipyrone C: Hart-Delta, Inc., 5055 Choctaw Drive, Baton Rouge, LA.
3† Talwin: Winthrop Laboratories, New York, NY.
4† Demerol: Winthrop Laboratories, New York, NY.
5† Neostigmine: Elkins-Sinn, Inc., Cherry Hill, NJ.
6† Normosol-R: Abbott Laboratories, North Chicato, Ill.

surgically, most untreated severe colic patients are in a state of metabolic acidosis. With the increasing blood hydrogen ion concentration, there is a shift of the hydrogen ions into the cells and a reciprocal loss of intracellular potassium ions into the extracellular space. Post-operatively, after the acid-base balance has been restored, the reverse shift occurs. Hydrogen ions leave the cells and potassium ions enter the intracellular spaces resulting possibly in hypokalaemia, especially in inappetent animals. It is essential that any significant hypokalaemia be corrected.

In this discussion, calculation of potassium deficit for replacement therapy serves as a model for determining deficits of other ions. The serum potassium level is determined. The same formulation is used to determine the amount of potassium to be given as was used for the bicarbonate ion, with the exception that some clinicians consider the functional extracellular fluid volume to be 30 per cent of body mass rather than 40 per cent when calculating electrolyte deficits. The calculated amount of potassium is added to the polyionic replacement fluid. The post-operative colic patient's potassium is most efficiently replaced intravenously and the ion probably should be administered at a rate no greater than 100 mEq per hour. If the horse does not have post-operative ileus and is not sequestering fluid into the stomach during the first post-operative day, some clinicians prefer to administer the potassium orally by giving 30 g of KCl (404 mEq) in 8 litres of water by stomach tube, two or three times a day, depending on the severity of the hypokalaemia[2]. Some animals will consume enough electrolyte solution, free choice, in addition to their usual water intake, to partially correct their hypokalaemia. Such an electrolyte solution is prepared by adding 30g of a mixture of 117 g NaCl, 150 g KCl, 168 g $NaHCO_3$, and 135 g of K_2HPO_4 or $KHCO_3$ to 250 g of dextrose, with enough water to make four litres. Replacement of calculated deficits, which are only rough estimates of actual loss, seldom immediately corrects metabolic imbalance; therefore, continued monitoring of PCV and plasma protein to assess dehydration, blood gases to assess acid-base disturbance and serum electrolyte values is essential to appreciate the dynamic nature of each parameter. Continued therapy should always be guided by response to treatment.

The antibiotic chosen for therapy of the surgical colic patient should be of broad spectrum. There is no agreement among the clinicians in our clinic on the most effective antibiotic. One successful antibiotic regime consists of the administration of a loading dose of 9-13 mg/kg of oxytetracycline hydrochloride [7]† intravenously presurgically, 7 mg/kg intravenously *t.i.d.* for 48 hours post-operatively, and maintenance of the animal on 7 mg/kg intravenously *b.i.d.* for 3 or 4 days. If there is any evidence of developing infection such as an elevation in temperature or leukocytosis, 7 mg/kg neomycin sulphate [8]† intravenously *t.i.d.* can be added to this regime. Other clinicians routinely use the combination of oxytetracycline and neomycin throughout the period of antibiotic therapy at the level of 7 mg/kg oxytetracycline intravenously *b.i.d.* and 7 mg/kg neomycin sulphate intravenously *t.i.d*; or 7 mg/kg neomycin sulphate intravenously *t.i.d.* alone as their antibiotic therapy.[3] It is worth mentioning that in some animals a low-grade fever will develop after six or seven days of oxytetracycline therapy. If there is no associated change in the CBC suggestive of an infectious process, the antibiotic may be withdrawn and the fever usually decreases. Long term oxytetracycline therapy may also be associated with inappetence, loose stools, and colitis [1].

Horses developing infectious post-operative peritonitis are most frequently those which have undergone enterotomy. Such patients become febrile, inappetent, frequently have a painful abdomen and show typical leukocytosis with a left shift. Intraperitoneal antibiotics can be useful in the management of acute peritonitis. The most frequently employed therapy is 40 m*l* furacin [9]† and 20 x 10^6 units of potassium [10]† or sodium penicillin G [11]† placed into the peritoneal cavity through each paralumbar fossa, twice daily. If surgical drains are in place when peritonitis develops, they provide an egress for fluid pooling in the dependent portion of the abdomen. In this fashion, limited peritoneal lavage can be effected.

Careful management of the post-operative incision will decrease the likelihood of draining fistulas and ventral hernia formation. Drains surgically placed in the incision should be carefully cleaned twice daily with a soapy surgical scrub and an antibiotic spray applied around the drain sites. The drains should be removed when they cease to function or at the latest by the fourth post-operative day. If the surgical incision begins to suppurate, any suture material in the infected area should be removed, if feasible, and ventral drainage established. The area should be flushed with appropriate antibiotics.

Post-operative intravenous therapy is essential in the survival of some horses. Commonly, horses with severe physiological disturbances tend to develop intravascular coagulopathies with associated jugular vein thrombosis. The use of a non-irritating silastic catheter, placed surgically into the jugular vein, facilitates long-term intravenous catheterization and obviates the need for multiple venepunctures[2]. With the use of this catheter we have seen a significant reduction in the incidence of post-operative jugular thrombosis.

The increased survival rate of equine colic patients within recent years can be attributed surely to a better understanding of the physiological changes of these patients as well as to the refinement of surgical techniques. No matter how heroic the surgery, survival often depends on observation of clinical abnormalities, laboratory evaluation of metabolic imbalances and institution of appropriate supportive medical therapy.

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7† Liquamycin: PFIZER, Inc., New York, New York.
8† Biosol: UpJohn Company, Kalamazoo, Michigan.
9† Nifurone solution: Diamond Laboratories, Des Moines, Iowa.
10† Potassium Penicillin G: E.R. Squibb & Sons, New York, NY.
11† Buffered Sodium Penicillin G: E.R. Squibb & Sons, New York, Ny.

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THE DIAGNOSTIC AND PROGNOSTIC VALUE OF LACTATE DETERMINATIONS IN HORSES WITH ACUTE ABDOMINAL CRISIS

W.J. DONAWICK, C.F. RAMBERG, S.R. PAUL AND MARGARET A. HIZA*

Blood lactate increases during shock owing to poor perfusion of tissues and anaerobic metabolism. Others have shown that blood lactate, in the critically ill patient, is a sensitive biochemical indicator of oxygen deficit, severity of disease and prognosis. In retrospective studies, we examined the value of blood lactate as a measure of the need for surgery and as a means to forecast survival in horses with an acute abdomen. The blood lactate was measured** in clinically normal horses, normal horses under anaesthesia and horses admitted with signs of abdominal pain. The mean blood lactate in the conscious horses was 0,81 mmol/*l*, and 0,68 mmol/l in anaesthetized horses. Of 18 horses presented with acute abdominal distress, 12 had gastrointestinal lesions that would have proven fatal if left unattended. The mean blood lactate of this group was 4,84 mmol/l. In the remaining 6 horses, which recovered without surgery, the mean blood lactate was 1,12 mmol/*l*. The difference in blood lactate between these two groups was highly significant and with 95% confidence we can state that a horse with a blood lactate greater than 4,7 mmol/*l* is in need of surgery, whereas a horse with a blood lactate of less than 1 mmol/*l* may be treated conservatively. The horses were also divided into four groups based on assessment of clinical signs. We found a highly significant difference in blood lactate between horses with severe or moderate signs and those with mild or no signs. It was possible to predict the value of blood lactate in horses with mild or no signs, but impossible to predict the blood lactate in horses with moderate to severe signs. All horses in our study with a blood lactate less than 3,59 mmol/*l* lived, whereas if the lactate exceeded 7,0 mmol/*l* all died. The probability of survival for 48 hours as judged by blood lactate levels, decreased from 100% to 0% as the blood lactate increased from 0,52 mmol/*l* to 11,05 mmol/*l*.

* New Bolton Center, Kennett Sq RD7, Pennsylvania 19348.
** Calbiochem Rapid Lactate Stat-Pack, 10933 N. Terrey Pines Rd., La Jolla, Calif. 92037.

GENERAL DISCUSSION ON IMPACTION AND COLICS

Chairman: C.F.B. HOFMEYR

M.A.J. Azzie: I would like to compliment Dr. Boles, and Dr. Evans in his absence, for so ably following up my indications for performing temporary colostomy in the management of rectal tears, offered at an informal lecture at New Bolton Centre in December, 1972.

Regarding the problems that he mentioned, faecal impaction at the colostomy site for the first three or four days can be overcome by making the incision about 30 cm more craniolaterally than described by him, then the faecal material in the small colon will not be so dehydrated.

I see no reason for making a midventral incision for repair of the temporary colostomy and establishment of the end-to-side anatomosis. It is simpler to dehisce the original colostomy site from the abdominal wall, clamp off that part of the bowel affected by the colostomy, exteriorize sufficient of the small colon, perform the anastomosis and close up the wound.

Do you, Dr Boles, have, or has anyone with much experience of abdominal surgery in the horse, any idea of the number of cases that have come back with abdominal problems a year or even two years after original abdominal surgery had been performed. South African colleagues have intimated that after major abdominal surgery they tend to have problems 18 months or 2 years later.

C. Boles: I am sorry I do not have any specific numbers but we certainly are not free of this problem, we probably have one or two cases a year from previous abdominal surgery. Usually the cause is an adhesion. I think the least traumatic and the more sophisticated we can get in the handling and the manipulation of the bowel, perhaps the less the adhesion, that probably we will have. Nevertheless, problems we will have as far as animals returning to us with colic following abdominal surgery are concerned.

A. Littlejohn: I just want to make one point with regard to the administration of bicarbonate, which reinforces Dr. Donawick's remarks, and that is the very dramatic improvement in oxygen transport which occurs when one administers quantities of bicarbonate. If one looks at the oxygen disassociation curve of the horse, one finds it is approximately the same shape and position as that of man, and, as in man, the position of the curve is affected by the pH of the blood. This is known as the 'Bohr' effect. A decrease in the pH obviously shifts the curve to the right and if the pH drops to 7,2 or lower, this makes a significant difference to the amount of oxygen carried by the blood. The majority of horses with intestinal obstruction are both hypoxic and acidotic and have a 25% or 30% drop in arterial oxygen tension. It is not uncommon, if one gives sufficient bicarbonate to bring the pH back to around 7,35 or 7,4 to find an increase in oxygen carrying capacity by perhaps as much as 10%. In my opinion, the administration of bicarbonate should be given before surgery, in order to get full advantage of this effect. Regarding the other point which Dr. Donawick made about post-anaesthetic hypoxia, we found at the Royal Dick School in Edinburgh that keeping a horse in sternal recumbency without administration of oxygen was sufficient to bring the arterial oxygen tension back to 70m*l* of mercury or more. We also got the impression that the severity of post-anaesthetic hypoxia was related to the length of time that the horse spent in dorsal recumbency. I wonder if Dr. Donawick has made any correlation in relation to this time factor.

W.J. Donawick: I cannot do anything but agree with you. We certainly try to put our horses in sternal recumbency as soon as possible. We prop them up if we have to, but there are times that even by doing that with very sick horses, it is insufficient: the PO_2 does not rise sufficiently and we still have to administer bicarbonate. So it is just one of those things that is done routinely. It is not going to hurt anything for the first hour or two to administer it and it is better than not having the equipment ready.

O. Knudsen: I would like to put a question to Dr. Donawick. Is the administration of 5 litres of homogenized, sour skim-milk administered by stomach tube to be looked upon as contributing to the pooling in the proximal part of the intestine in cases of colic, as stated by him under his list of "Don'ts"?

I will give a background to this question. During the last years we in Sweden have rather frequently encountered a type of enteritis affecting horses in good condition and hard training. It is characterized by a marked increase of *Clostridium perfringens* in the intestinal microflora and, in severe cases, also by bacteraemia (sepsis). Clinically the horses have a heart rate of approximately 130/s, a body temperature of around 41°C and they become toxic very soon. In the peracute form they die within half a day of onset of signs, without developing diarrhoea. If they survive the peracute stage, diarrhoea will appear. Sometimes the disease starts with signs of colic.

A very effective *basic* treatment is to acidify the gastric contents with 5 to 8 litres of sour skim-milk given by stomach tube. After we started to treat this type of enteritis with sour skim-milk, it has become a standard method in all cases of enteritis and also a basic treatment for different types of colic. It will stop the production of gas and affords one some time to arrive at a diagnosis and – if necessary – for applying other treatment.

The horses stand the sour skim-milk surprisingly well and today several of our best trotters with chronic intestinal trouble have consumed one to two litres of sour milk for many months or even years.

W.J. Donawick: Enteritis poses a special problem of its own but in colic I should, hesitate to use it as a standard method.

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BROWN SNAKE BITE IN HORSES IN SOUTH-EASTERN QUEENSLAND*

R.R. PASCOE**

INTRODUCTION

Altogether there are less than 140 species of Australian snakes recorded [1]. Twenty-one of them belong to the family *Typhlopidae* – blind snakes – which are harmless. Ten species are non-venomous constrictors of the family *Boidae* – pythons – which do not possess venom glands. A further twelve species belong to the family *Colubridae* which is the dominant family both in world distribution and species number. This family is very poorly represented in Australia and is mostly restricted to the northern tropics. Furthermore, these snakes have either solid fangs or have poison fangs set at the rear of the upper jaw, so that it is almost impossible for them to inflict a dangerous bite. The rest of the Australian species, about 70 percent of the total number, belongs to another series called the *Proteroglypha.* All possess venom glands on their grooved or channelled fangs, which are situated at the front end of their upper jaws. Twenty-four of these are sea snakes of the family *Hydrophiidae.* This then leaves about 70 species of proteroglyphid snakes, all of which belong to the one family, the *Elapidae.* Only twenty of these can be considered as dangerous to man, and, of these, only ten present any serious hazard to man and animals in Australia.

Kellaway [2] has outlined the pharmacological action of Australian snake venom as being predominately neurotoxic on both central and peripheral nerves. There is a curare-like action on the motor nerve endings and a further direct action on the muscle itself. The phrenic end-plates in the diaphragm are particularly sensitive and this partial curarization of the diaphragm plays an important part in the failure of respiration, which is the commonest cause of death after the bites of these snakes.

Motor paralytic effects include paralysis of the ciliary muscles causing fixed dilated pupils, – paralysis of the soft plate, paresis of the tongue with alteration in whinney and difficulty in swallowing. Terminal convulsive movements are more probably related to low grade asphyxia or directly to the action of the venom.

The second group of actions is linked with their haemolytic and cytolytic power. This eventally leads to histamine liberation which gives rise to symptoms of cardiovascular failure which follows the bite of the common Australian snakes, the most potent being those of the black snake *(Pseudechis porphyriacus)* and the copper-head *(Denisonia superba.)*

The third action is coagulant and depends on the conversion of prothrombin to thrombin. The venoms of the death adder and copperhead lack this property but it is strongly present in the venom of the tiger-snake *(Notechis scutatus),* and brown snake *(Demansia textilis)* and the black snake *(P. prophyriacus)* and also in the taipan *(Oxyuranus scutellatus).*

* This presentation was illustrated by a film.

** P.O. Box 2, Oakly, Queensland 4401, Australia

CLINICAL CASES

While all ages of horses are susceptible to snakebite, there have been two distinct age groups involved:

Group 1: Foals from 3 days old to 50 days
Group 2: Yearlings and adults

Group 1: The average age of this group was 6 weeks, and with one exception of a three-day-old foal, most cases occurred in foals between 29 and 49 days old.

With the initial observation, the foals appeared to be slow moving with a goose-stepping type of leg action and frequent rests when suckling. Once the syndrome became recognized it was noted that the earliest diagnostic sign was the mare with an udder full or partly full of milk and with what on first visual examination appeared to be a normal foal. Closer examination showed drowsiness, drooping of the upper eyelids, partly to complete paralysis of the tongue muscles and, occasionally, paralysis of the lower lip as well. The foal's gait became more stilted in the front legs, and if driven, the foal literally collapsed to the ground. Abdominal respiratory efforts were barely evident in the early cases, constipation became evident at this stage and urination occurred as retention with overflow. As the effects of the venom became more pronounced, muscle twitching increased, efforts to remain standing became more pronounced, respiration became abdominal and more laboured, heart rate quickly returned to normal once the foal became recumbent. Some foals showed dilated pupils. As envenomation developed, so it became more difficult for the foal to hold its head upright and respiration became completely abdominal. Sweating was marked in all foals when recumbent. Foals lost their ability to whinney and to suck but could still chew if grass was placed between the molars. Swallowing was not possible once the tongue became paralysed, and often milk, when suckled, ran back out the foal's nose when the head was lowered. The foals eventually became laterally recumbent, respirations became very laboured and they died within a short period.

Group 2: Adult horses showed salivation, inability to swallow, inco-ordination of gait, muscle twitching and trembling, foul-smelling breath from unswallowed food present in the mouth and pharynx, continuous attempts to drink water, heavy, laboured abdominal respiration, slight elevation of temperature and heart rate between 40-60. Mucous membranes were usually injected and this became more marked with time. Most horses sweated profusely. Some horses showed sleepiness with droopy eyelids and lower lip paresis. Terminally all horses showed extremely laboured abdominal respirations, staggery gait, recumbency and death.

TREATMENT

Before a diagnosis of snake-bite was confirmed, horses affected with this syndrome were suspected of

having botulism and were treated for this disease but after the horses had been vaccinated and deaths still occurred, the diagnosis was reconsidered.

Supportive treatment consisted of the use of intravenous glucose saline with either whole milk or Denkavite plus 6 egg yolks per day, given three or four times daily by stomach tube. (Most cases bled from the nose after drenching in this way.)

Many cases also received oxytetracycline (10mg/kg) intravenously daily for three days. Foals were also given ½ pint paraffin oil *per os* in an endeavour to relieve constipation. Some cases also received physostigmine (12,5 mg/500 kg) intravenously in 1 litre of saline several times daily. This often caused defeacation and allowed the horses to stand and walk normally for short periods. Antivenene has been used as shown in table 1.

Trinca[3] also stated the brown snake bite is ineffective and this could appear likely to be due to the short fangs of this snake. When disturbed, however, this snake is a vigorous and fearless attacker and can inflict numerous bites very rapidly. In the case of foals with short, soft hair and soft skin, the bite would be more likely to penetrate the skin than in an adult horse.

It has been noted that most cases have appeared on the day following rain. This could be due to two factors. Firstly, in black soil country, large cracks appear in a dry period. Snakes are often found in these locations and are hunted out by the heavier rain storms. Secondly, mares are ground fed on lucerne hay and snakes have been observed to shelter in these lucerne sheaves after such rain. This may be the contributing factor to the large number of horses bitten

Table 1 : RESULTS OF TREATMENT WITH SNAKE ANTIVENENE USED IN 12 HORSES

LIVED						DIED						REMARKS
Treatment						Treatment						
Immediate			Delayed			Immediate			Delayed			
BSAV	TSAV	BOTH	BSAV	TSAV	BOTH	BSAV	TSAV	BOTH	BSAV	TSAV	BOTH	
1	1											Goose stepping, lame only
	1											Mare unable to swallow sweating
			1									Third day after symptoms appeared
						1						Lived 5 days after treatment
								1 *				Found down, died 4 hours later
									1 *			Found down, lived 3 more days
									1			Treated 24 hours after first symptoms
										2	1	Treated 48 hours after first symptoms
								1				Died with pneumonia 10 days after being bitten, was recovering.

BSAV = 500 i.u. brown snake antivenene (Commonwealth Serum Laboratories, Parkville, Victoria)
TSAV = 3 000 i.u. tiger snake antivenene (Commonwealth Serum Laboratories, Parkville, Victoria)
BOTH = One ampoule of each of the above
* 2 doses of antivenene

DISCUSSION

Kellaway[2] stated that in humans there was a period of freedom from symptoms from brown snake bites as long as 24 hours and then death owing to respiratory failure as result of paralysis of the diaphragm could occur 36-48 hours after the bite. This would appear to occur in horses also, as symptoms have appeared when the horse could not have been bitten during the previous 10-12 hours.

If antivenene is given within an hour, signs and symptoms are rapidly reversed[3]. This has been amply borne out in this series of cases. Two foals and one mare, treated when the first stiffness of gait was noted, recovered rapidly. Others, which received either larger doses of venom or were affected longer, did not recover in spite of higher doses of either or both tiger-snake antivenene and brown snake antivenene.

on this particular stud.

The loss of foals treated with both tiger-snake and brown snake antivenenes can only be related to the long period between bite and treatment. One foal, which was treated with both antivenenes and showed a slow but gradual return to normal after being recumbent for 8 days, died suddenly in four hours from peracute pneumonia.

Horses which were confined to stables and foals which were transported by float from the paddock showed less severe symptoms and recovered with antivenene much more quickly than those which were driven.

Observations made in this series of cases would indicate that brown snake venom produced symptoms of drowsiness, stiffness, inability to swallow, paralysis of the tongue, sweating, respiratory distress and

muscular twitchings followed by paresis. No haemoglobinurea was observed.

CONCLUSIONS

Brown snake venom can produce a fatal respiratory and general paralysis of the head and limbs of the horse.

Tiger-snake and brown snake antivenene appeared to be equally effective if administered as early as possible after envenomation.

Supportive treatment with saline transfusions, antibiotic therapy and complete rest in stables are necessary to increase the percentage of cases recovering from snake-bite.

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TITLES AND ABSTRACTS OF UNSCHEDULED PRESENTATIONS*

EPIGLOTTIC ENTRAPMENT: A SELDOM DIAGNOSED UPPER AIRWAY OBSTRUCTION IN THE HORSE

J.D. WHEAT AND C. BOLES

This paper was kindly presented by Dr. C. Boles in place of the scheduled paper by Dr. D.W. Milne on 'Azoturia and Intracellular Potassium Levels in Equine Rhabdomyosis'. As Drs. Wheat and Boles intend publishing their paper in full detail in the Journal of the American Veterinary Medical Association, no further details will be published in the present proceedings. That part of the discussion, however, having no bearing on the above theme, although related thereto, is reproduced here.

DISCUSSION

C. Button: Could you tell us more about follicular pharyngitis and any idea you may have on the aetiology?

C. Boles: I do not have any idea on the aetiology. I have spent probably the better part of four years with quite a bit of interest in this area but I have to admit quite honestly that I know very little more about it than I did initially, other than it seems to be becoming more common. Perhaps Dr Wheat has some idea but I do not.

J.D. Wheat: In biopsy specimens it is just solid lymphoid tissue packed with lymphocytes. Conservative treatment, such as irrigation or use of certain steroids and the like has been rather useless in my experience. By allowing a long rest of say six months, the horse will get over it. In extensive cases cautery is advisable. I do not like chemical cautery. We tried that for a short period of time but we cannot control the application to the exact area of involvement. I use general thermal cautery by means of a regular bovine surgical cauterizer. What works best is a long section of insulated copper wire, about a number 9-12, with the insulation at the tip removed and the tip itself flattened and the wire put back into the instrument. Under visual control of a fibroptic scope, the individual areas are cauterized. It has worked very well for destroying these very large follicular areas.

M.A.J. Azzie: When these cases are treated by cautery, have you not had any cases of chronic fistulas or anything like that developing?

J.D. Wheat: No, we do not burn deep enough to cause complications: we go on the surface till we just see a white cauterized area. We certainly do not want to burn deeply through the entire wall of the pharynx but just on the surface till we destroy the follicles. There was the problem of finding an instrument long enough to reach back into the pharynx going through the nostril, but the wire has worked very well. There is no more suitable equipment I have found today for cauterizing the pharynx.

A CASE OF AORTIC-ILIAC THROMBOSIS IN THE HORSE

M.A.J. AZZIE

This case report was based on a film showing symptomatology and pathology. A table was shown relating saphenous vein filling time to incidence of the condition.

OPERATION TABLE FOR EQUINE SURGERY

A. LINDHOLM

The operation table designed by Professor Obel of Stockholm was explained. The total cost of table and accessories was estimated to be in the region of R150 000, that of the table only at R15 000 to R20 000.

* These presentations were volunteered "off the cuff" during the proceedings.

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RADIOGRAPHIC EXAMINATION OF THE EQUINE BACK CASE

L.B. JEFFCOTT

Chronic low back pain is a perennial problem in all types of working horses, especially showjumpers, dressage, steeplechasers and three-day eventers. The problem is further complicated by the difficulties of making an exact diagnosis and so most cases must be treated empirically. This paper was presented as a preliminary communication and was intended to illustrate the importance of radiology as an aid to diagnosis in these cases.

The technique for radiography of the horse's back in the standing position was described and a series of X-rays of clinically normal horses was shown. A Siemens Triplex Optimatic 1023 machine with a 200 kV, 1000 mA tube was used and exposures ranged from 75-120 kV and 100 – 250 mAs according to the thickness of the part being X-rayed. Fast film and intesifying screens were used together with a cross hatch grid where appropriate. A further improvement in quality was obtained by filtering (dodging) part of the primary beam to help compensate for the marked variation in thickness of the horse's spine.

The most important causes of back conditions in horses are associated with soft tissue injuries, particularly muscle strain of the *m. longissimus dorsi* and sublumbar groups of muscles. These cases normally respond to rest and physiotherapy and generally carry a good prognosis. Other conditions are usually of a more chronic nature and include:-

(1) *Overriding of the dorsal spinous processes.* The presence of impingement of the summits of the dorsal spines beneath the saddle area ($T_{12} - T_{18}$) appears to predispose to low back pain in some horses. The presence and location of pressure points between adjacent spines with overriding local periosteal reaction, small bony cysts and false joint formation were described. No evidence of actual fusion of any of the spines has yet been seen. Radiographic lesions of this type have also been seen in animals not suffering from back pain, although there is usually a lower incidence and lesions are less severe. A useful aid in the diagnosis of this condition is provided by the injection of local anaesthetic into the affected interspinous spaces. Treatment in persistent cases by resection of one or more of the summits to relieve the crowding of the spines is usually very successful.

(2) *Ossifying spondylosis* affecting the mid- to posterior thoracic region occurs uncommonly in working horses. When it does occur there is usually little that can be done in the way of treatment to keep the animals in work. Osteoarthritic lesions of the transverse and articular process of the lumbar vertebrae are much more common, especially in older horses but they are often of doubtful clinical significance as this part of the spine is kept particularly rigid even when the horse is jumping.

(3) *Tying-up* (setfast) of the back muscles is a condition occasionally referred as a back problem. These cases can be diagnosed fairly simply by estimation of the muscle enzymes before and after exercise when marked elevation of CPK and usually GOT will be seen within 24 hours.

(4) *Fracture of the dorsal spinous processes* occurs infrequently and usually as a result of a fall. Diagnosis is confirmed by radiography.

(5) *Undue curvature of the spine.* Congenital scoliosis of the thoracic spine occurs not uncommonly in foals and arises as a deformity *in utero.* The extent of the malformation is best assessed by radiographic examination. Acquired kyphosis and lordosis are occasionally seen and will, of course, predispose to back weakness.

(6) *Sacroiliac strain.* It is not possible to take useful radiographs of the sacroiliac region in the standing animal. The technique of general anaesthesia and radiography of the pelvic and sacral regions with the animal in dorsal recumbency was described. Even in very large horses, of over 700 kg, it has not been found necessary to use exposures above 150 kV although the larger the mAs the better quality of the X-rays and up to 400 mAs has been used. In order to limit scattered radiation it was necessary to use crossed high-ratio grids and to put additional lead on the back of the cassette to prevent back scatter.

Acute and severe sacroiliac strain usually occurs following a bad fall or similar traumatic incident and is not usually difficult to diagnose clinically. It is the more chronic, low grade situation that may present itself as a typical back case. The only radiographic findings are increased joint space of the sacroiliac articulations sometimes with associated slight rotation of the pelvis. Providing these cases are diagnosed fairly early and the animal rested long enough for complete healing of the damaged ligaments they will usually come right completely.

DISCUSSION

W.H.S. Bellinge: Following fractures, long rests and clinical evidence of good union how long afterwards do you expect to find complete calcification of the actual fracture on radiography? I found even after twelve months we sometimes observe good healing, soundness is complete and yet we see a very marked gap between the two ends of the fracture.

L.B. Jeffcott: I think that last slide demonstrates it very nicely. That mare was very nearly sound. In fact she had a big gap between the fracture ends and there was considerable overriding. One tuber coxae coult not be palpated at all, yet she was in good condition. She was practically sound and was going to be put back to work. I had her in for X-ray to see if one would be able to breed from her as we could see no reason why the pelvic canal would be reduced. But ones other than that which we have seen so far have, in fact, healed up pretty well. The problem that we have seen, particularly those with hip fractures, is that one can get an arthritis of the hip; then one often loses them from an osteoarthritis and not from malunion.

Discussion continued on Page 136.

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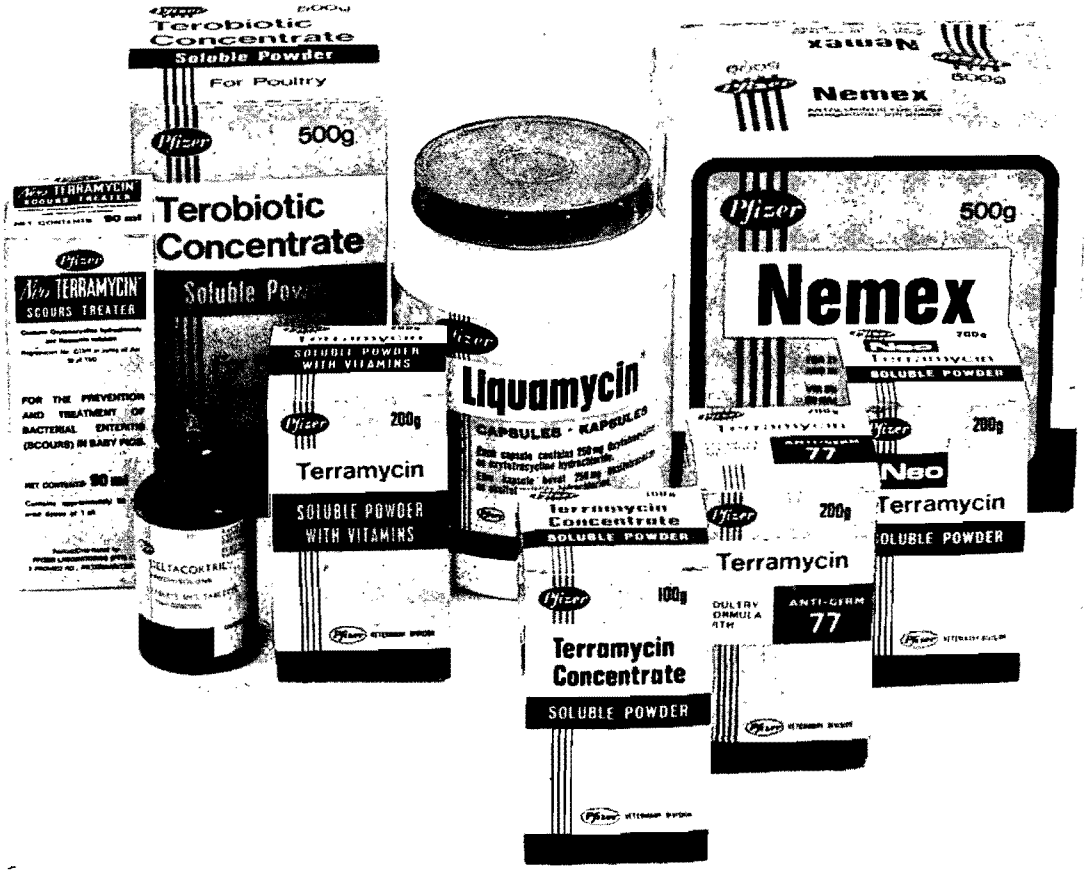

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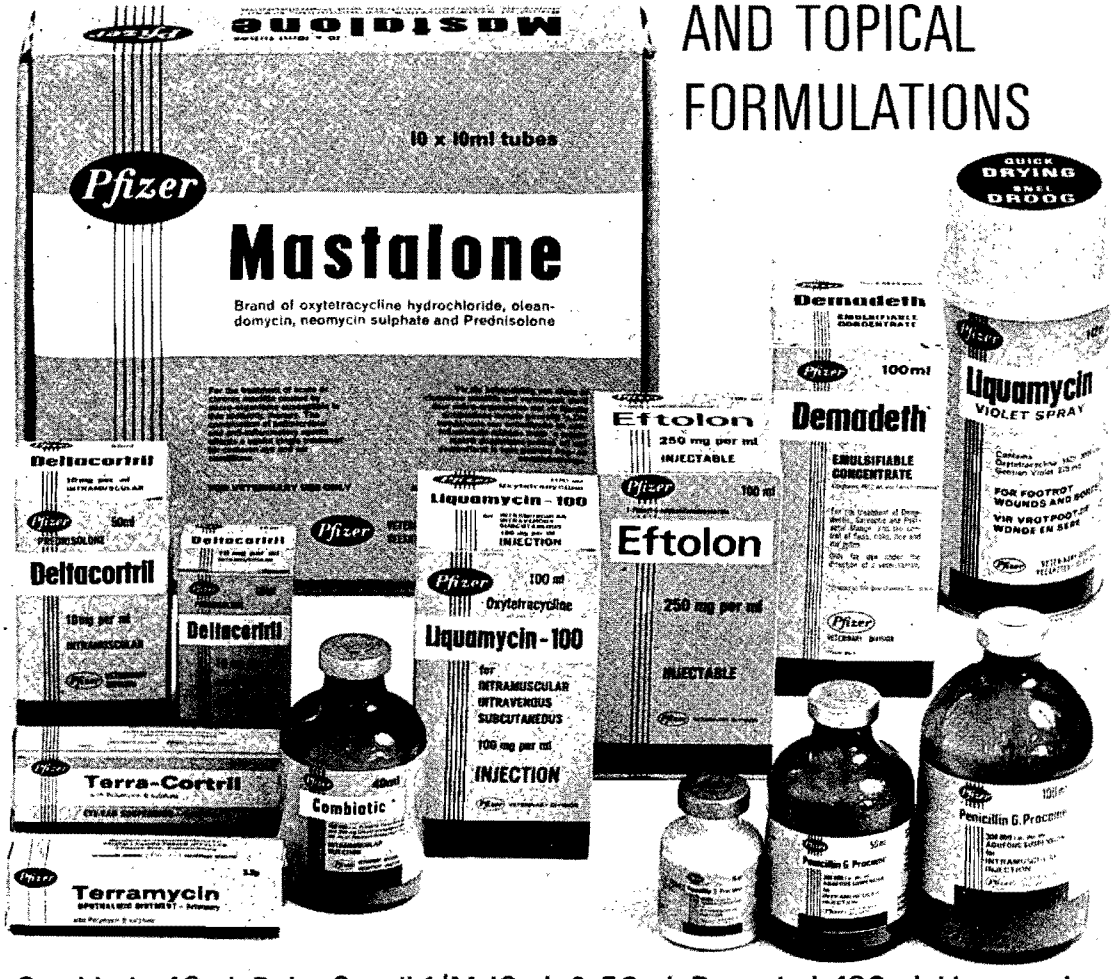

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F. Milne: Yes, but many people bring the fracture case back for you to look at and ask if it is alright to put the horse to work as they behave quite normally. The horse is not sound yet in terms of the radiograph, yet it goes quite well. I am not talking about the pelvic type of fracture, that is difficult to X-ray. I am referring to more simple radiographs generally. It can be very difficult on radiography in all fairness to say, look, put it back to work or not. The only criterion seems to be soundness.

L.B. Jeffcott: I would prefer it if Dr. Reid could answer that. I am sorry, I thought you were talking about pelvic fractures.

C. Reid: At that point of time you tell the people to start using the horse to stimulate the callus to form more completely. If, after twelve months, one has some evidence of a fracture line, the chances are that after a further six to 12 months one still may have some evidence of that line. By exercising some caution one should start using him a little bit to try and stimulate the fracture to ossify completely or one may end up with a partially fibrous union. We followed some of these cases, such as fractures of the cannon bone, for a period of time and sometimes after twelve months, if there still was some radiolucency, we felt that we should start the horse doing light exercise to try and stimulate the fracture to heal more completely. If it did not and yet the horse got fitter and fitter and ready to race, we considered we might as well go on.

As for radiographing the dorsal spinous processes, one can get diagnostic films especially of the withers and saddle area that he spoke of, maybe not as good as Dr. Jeffcott's, by using a portable 85kV machine. If one tranquillizes the horse and takes a half second to one second exposure, one can often see these impingements. We radiographed a number of backs and found impingements up and down them. The most difficult thing to do was to decide which lesion was producing the supposed back problems. Dr. Jeffcott pointed out a very important aspect: it is the clinical examination that frequently helps one to decide which areas are the problem areas. Even though one has these impingements, even though one has pathological changes, they may not be producing the problem; it could be elsewhere in the back. It is not infrequent to radiograph a horse 8 to 10 years of age and find rather significant radiographic changes in that horse's back and yet those particular areas, certainly at that point in time, are not causing a problem.

F. Milne: Two questions I would like to pose to Dr. Jeffcott. The first is, are you equating the condition you described as straining of the sacroiliac joint the same as subluxation of the sacroiliac joint described by Dr. Adams on the one hand and Dr. Rooney and Dr. Delaney on the other? Is that basically the same condition? You did not mention any of the so-called clinical signs of the 'Hunter's bumps,' which the later discribed, where there is a true displacement.

L.B. Jeffcott: I am talking tonight about the back case. I am not talking about the frank, obvious pelvic case, where one has complete subluxation.

F. Milne: Thank you. The second question concerns the over-riding of spinous processes. I was present when the TV demonstration was given in Philadelphia some years ago and yet I have been very frustrated trying to replicate the X-rays which you have obviously improved upon. Our radiographic technologists said it was impossible to get the pictures that I wanted with our equipment. At autopsy, however, we have seen several of those over-riding lesions which were asymptomatic. I know you had intended to prove your point by injecting local anaesthetic into the area and, if relief was obtained, by operating on it. The point that I wanted to make is this: some years ago, in 1956 or 1957 to be exact, we were all hood-winked by a situation called the 'fractured fibula' syndrome in horses. Somebody else in the audience looks embarrassed, too. I can see that from the response. I also feel embarrassed when I look back and think what we considered 'fractured' was really not. In man, many people have radiographically obvious productive or destructive lesions of various parts of the body, such as the hip. There is one standing in front of you now, and yet they are entirely asymptomatic. Do those lesions which you see on the X-rays, when you can do an X-ray, really prove positively that that is the cause of the trouble? I think that is what Dr. Reid was trying to point out just a moment ago.

L.B. Jeffcott: I obviously did not make myself very clear. I agree with you entirely that there is no 'black and white' in this and I am not trying to put into your minds that this condition is as absolute as shown up on the X-ray. I think more important than the radiological examination is, first of all, the clinical examination. Once having decided that it is a back case one has to eliminate all the other radiological possibilities which I have tried to show you tonight. This work is reasonably new to myself, and I feel, and you will obviously appreciate, that there are horses which show over-riding without any clincal signs. Horses, I feel, probably have a conformational predisposition to this condition, as in horses with short backs, or slightly sloping backs, where the spines are going to come together. Of the two-year-olds I have done X-rays on, I have seen two horses with over-riding of the spinous processes having no back trouble at all. I am not, at this point, trying to be dogmatic. I would suggest that a horse that does have this radiographic problem is more prone to back trouble than a horse that looks like the first one that I showed you. If one can prove it clinically by injecting local anaesthetic and eliminate all the other causes of back trouble, then I think one stands a good chance of being correct. That is the way we do it. I also do not want to give you the impression that we operate on all these cases; we certainly do not. We operate only on very chronic back cases. Tonight I am talking of radiography, so I have not gone into the operating technique or the clinical side so much. It was just to throw it out as an idea. We like to rest these horses, because the primary problem is a back condition, be it conformational, or as a result of bad training, over-weight riding, you name it. Though the spines rub together, one gets a secondary muscular spasm of the longissimus dorsi muscles, and it is the muscle spasm that gives the horse the back pain. These horses are not necessarily in great, acute pain, they just do not act or perform well, they do not jump well and they know if they are going to do any particular manoeuvre, those spines are going to rub together, they are going to get back pain, so they do not do it and do not perform well. They save themselves, they keep their backs flat, they jump flat, they do not use their hind legs properly, they have sort of a 'pottery' action. If one can relax those muscles, so much the better. This is how manipulation, rest, and physiotherapy work in so many cases that are not even the same as this condition. But I do assert, if this is a genuine case then it will probably recur because it has the predisposition to it. I am not a great believer in manipulations, but can see that if one has two spines that are very close together and are over-riding and causing the animal a considerable amount of secondary muscular pain, and if one could come along with magic hands and bring them apart just enough to get those muscles to relax, the animal would be better.

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