All members of the camel family (Camelidae) belong to the order Artiodactyla (even-toed ungulates) and suborder:Tylopoda (pad-footed). To detect abnormality in camels, the veterinarian must be familiar with the normal health state. A healthy camel is one, which has an erect head, clear eyes, prickled ears with a bright manner, normal appetite, pulse rate, and temperature. The hump often gives a good indication of health and should be rounded, plump and vertical or slightly inclined. Some smaller breeds of desert camels have small humps, which should not be confused with the flabby, shrunken hump of deprived, aged or sick camel (HMSO, 1956). Other signs of disease in camels are similar to other species. The description of camels' disease, its detection and prevention is described in this book.

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Camel and Its Disease

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INTRODUCTION

All members of the camel family (Camelidae) belong to the order Artiodactyla (even-toed ungulates) and suborder:Tylopoda (pad-footed). The old-world camels belong to the genus Camelus with two species: C. bactrianus (two humped camels) and C. dromedarius (one-humped camels). The New World camels belong to the genus Lama with three species, L. guanicoe (the guanaco), L. peruana (the llama) and L. pacos (the alpaca), and to the genus Vicugna with only one species, the vicuna. The present distribution of camels is the result of man's activities, in which domestication, extirpation, transference, and artificial dispersal have played important roles.

Camels originated in North America when the landmasses were still joined (Zeuner, 1963). These animals were no larger than hares. They remained in this area from the Eocene Epoch period throughout the Tertiary period, into the Pleistocene epoch, a period of 40 million years. From North America the animals migrated to other parts of the world, finally disappearing from their original habitat entirely adapting to the various areas to which they migrated. The camel evolved into the different species of the camel family, as we know them today. It is thought that the single-humped dromedary evolved from the two-humped Bactrian. It has been reported that at an early stage of embryogenesis two rudimentary humps are present in the fetal dromedary as well as in the Bactrian and in hybrid crosses (Leese, 1927).

Dromedaries were originally domesticated in Central and Southern Arabia (Zeuner, 1963). From here they gradually dispersed to North Africa and eastwards to the deserts and semi deserts of the Middle East. The Romans used camels in many parts of their empire thus accounting for the presence of camels in parts of Europe and Asia (Ripinsky, 1983). The spread of Islam also contributed to the dispersal of camels throughout the Islamic Empire. The name dromedary, for the one-humped camel, is derived from the Greek word 'dromeus', a runner, or 'dromas', running. This name originally designated only for the swift Arabian camels renowned for their fleetness of foot. Later, it applied to all one-humped camels. The Bactrian camel was named after the area of Bactriana in Central Asia.

In the past camel was primarily used for the transport of goods and passengers in the desert and semi-desert areas. Wool, milk, skin, and meat were the by-products (Williamson and Pyane, 1978). In the modern times, camel is a neglected species due to rapid replacement by other alternatives such as more rapid means of communication and transport, for example, automobiles. To the Bedouin, however, the camel is more than 'the ship of the desert'. It is woven into his religion and folklore as the special gift from God. In the Koran the camels' importance to the people is described as, 'Lo this is the camel of Allah, a token unto you; so let her feed in Allah's earth, and touch her not with hurt lest painful torment seize you' (Koran: Sura 7-73). ‘And (remember) when we

prepared for Abraham the place of the (holy) House of Allah and proclaim unto mankind the pilgrimage. They will come unto thee on foot and on every lean camel' (Surah XXII 26-28 The Pilgrimage). In Surah XXVI it is mentioned: 'He said: (behold) this she camel. She hath the right to drink (at the well) and ye have the right to drink (each) on an appointed day; and touch her not with ill lest there come on you retribution. But they hamstrung her, and then were penitent'. Muhammed (peace be upon him) used to love and praise the camel. Caliph, Umar stated 'the Arab prospers only where the camel prospers'. Carrying soldiers into war on camel back was an accepted practice in Biblical times and is still done until today. Most armies in the Middle East and some countries of Asia (Pakistan and India) and North Africa still have camel corps in their modern mechanized units.

The world stock of dromedaries is 17 million and Bactrians 2 million. Fifteen million dromedaries are present in Africa and Middle East and 2 million in India and Pakistan (Shwartz, 1992). It has been estimated that there are approximately 14,000 racing and 40,000 breeding camels in the United Arab Emirates alone (Higgins et al., 1992). Peak milk yield of 20-40 liter per day has been recorded for camel (Knoess, 1979; Quereshi, 1986; Khanna and Rai, 1993). Camels have a lactation period that lasts throughout or most of the year, where as local cattle kept in the desert of East Africa produce milk only during the rainy season for approximately two months (Kohler-Rollefson, 1994). It appears that the home consumption of camel meat is rare among the dromedary owning nomadic tribes, however, camel meat is popular in many towns in North Africa and Arabia (Dorman, 1986). Daily weight gain of 1.5 kg by males and 1.0 kg by females on low cost diet indicates that camel is an efficient meat producer (Qureshi, 1986).

The camel's body has a large body mass, which heats up slowly than a small body mass when exposed to sun. The characteristic hump of the camel is a store of subcutaneous fat joined together by fibrous tissue. On the other parts of the body the subcutis is almost fat free so that it can dissipate heat more efficiently (Clooudsley-Thompson, 1969). The dromedary camel is much taller than the two-humped camel. An adult full-grown camel weighs between 450-600 kg and its shoulder height is about 2.2 meters.

The colors of camels vary from deep brown to dusty Grey. White camels are rare, while black camels are considered as a bad omen by some tribes. The hair are longer and thicker on the top of the head, under the throat, on the dorsum of the neck, shoulders, and hump. The coat reaches its maximum length by early winter. The thick winter coat is normally shed by mid-summer. The lips of the camel are extremely sensitive and enable the animal to avoid being pricked by thorns when browsing thorny bushes. The nostrils are almost horizontal slits that virtually meet in the front, and contain numerous hair, which prevent sand entering the nostrils. The camel can therefore continue breathing normally even in the most severe storm. The eyes are large and prominent. The eyelashes are long and heavy. There is a thick nictitating membrane, third eye lid, with well-developed glands. This third eye lid protects the cornea from all foreign materials including sand. There are thick extruding eyebrows, which shield the eyes from the sun glare.

The digital bones are wide and flattened and are embedded in a broad cutaneous pad, which forms the sole of the foot. The two protruding digits are spread almost flat on the ground and terminate in short, broad and slightly curved claws or nails. The second and fifth toes are not present. The camel treads on its toes or on the cushioned pad that underlies them. The pads expand with the pressure each time the foot is placed on the ground. This provides the traction in the sandy area. The ability to walk across the sand dunes gives the camel its name 'the ship of the desert'.

Under the chest and on the knees and hocks of all four legs, are thick, horny keratin pads. The pads are already present in the fetus of 5-6 months. These pads prevent abrasions when the animal kneels and allow him to squat on the hot sand by slightly elevating the body above the hot surface.

In short the camel posses certain physiological features that enables him to thrive in extremely arid environments (Shwartz, 1992) and the necessity of man to exploit the useful traits, which may ultimately guarantee the camel's survival as a domestic animal. The mystery of water deprivation is almost solved now. About 12% of the total body-water is contained in the alimentary tract of fully hydrated camel, which is about the same as in cattle. The camels, however, are able to recycle the water to conserve it. This is achieved by a large number of endocrine cells in the epithelium of fore stomachs. The cells control the passage of water, electrolytes and urea in and out of the fore stomachs. About 50% of water absorbed in the omasum is excreted in the abomasum. It helps to dilute ingesta to easily be delivered to the intestinal tract for absorption. Water and urea are continuously recycled from the blood into the stomachs. Water deprived/dehydrated camels lose much less plasma volume than other animals, which is due to continuous absorption of water from the intestines.

During the last 10 years a more formal structure to racing has been established in the Gulf countries. The racing season starts in October and extends to mid-April. Races are held over distances up to 20 km.

Traditionally, camel races were held over a straight track, however, circular tracks of up to 10 km have now been built. Camel racing is a serious sport, which maintains cultural heritage and has significant socio-economic implications.

The maintenance of good health and productivity (whether fitness for racing or fitness for milk, meat and hair production) is of vital importance to the owner.

In general, most camel diseases must be considered as herd problems. It is often impractical for a herd to remain in one place for any length of time. As a result, the attending veterinarian may have the opportunity of only a single visit to a 'patient' before the whole herd moves to new grazing area. In these circumstances, the importance of clinical examination in reaching a correct diagnosis is self-evident (Higgins and Kock, 1986).

To detect abnormality in camels, the veterinarian must be familiar with the normal health state. A healthy camel is one, which has an erect head, clear eyes, prickled ears with a bright manner, normal appetite, pulse rate, and temperature. The hump often gives a good indication of health and should be rounded, plump and vertical or slightly inclined. Some smaller breeds of desert camels have small humps, which should not be confused with the

flabby, shrunken hump of deprived, aged or sick camel (HMSO, 1956). Other signs of disease in camels are similar to other species.

It has been said that the camel suffers from few diseases of importance. However, it is apparent that we know too little about the diseases from which it does suffer from (Higgins 1983). Some are well known such as mange and trypnosomiasis; others are just beginning to come to the light. Trypanosomiasis is probably the most serious infectious disease of camels and is widespread throughout camel-rearing areas.

Although most camels are placid by nature, the degree of pliability reflects the degree of human handling. Particular care must be taken when examining the female camels with calves at foot or male camels during the breeding season. Rectal examinations can be particularly tiring. It is worth remembering that even the most docile camel can inflict damage on the unwary.

Physiologically, camels adapt well to high temperatures. Blood volume is maintained partly by water diversion from the skin to other body tissues and organs. Studies in the Sahara have shown that, when the ambient temperature exceeds 40^{o}C, as much as 40% bodyweight may be lost and several drinking sessions may be required before full weight is regained (Gauthier-Pilters and Dagg 1981). Water conservation is assisted by highly efficient renal mechanisms (perhaps related to the long nephrons found in camels), nitrogen retention and re-use, production of dry feces and a flexible diurnal body temperature which can vary by up to 6^{o}C in 24 h period. Additionally, water is recirculated continuously in the gut from the duodenum and colon into the fore stomachs via the blood.

Many of the above physiological characteristics of the camel play a role in the way in which drugs are metabolized by this species. The veterinarian must be aware of such metabolic variables when treating camels and also when using tranquilizer and anaesthetic drugs for examination and surgical procedures.

Having relatively long neck and legs and, at times, aggressive, the camel suffers from a spectrum of traumatic lesions that require surgical repair. Surgical procedures to such cases will not only allow salvage of the patient but full return to rigorous performance levels as required in racing camels.

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DIGESTIVE SYSTEM

THE LIPS

The lips of the camels are extremely mobile and sensitive. The upper lip is livid and overlaps the lower lip, which helps in oral recovery of moisture from the nose. It also helps avoiding thorns, to select small portions of plant material as food, and to pick small particles of food from ground when required. The lips are covered by soft hair of varying sizes, some of which are tactile (Manefield and Tinson, 1996).

ORAL CAVITY

The oral cavity of the camels is typical of a ruminant and is designed for the efficient handling of large volumes of plant roughage. Both upper and lower lips are extremely mobile, and are covered by soft tactile hair. The upper lip is bifid, which enables the camel in selecting food and avoiding thorns. *Labial* glands are numerous and open on the mucous membrane. The buccal vestibule contains large conical buccal papillae, which are cornified and are directed towards the pharynx. The hard palate is narrow rostrally with more or less parallel lateral borders. The hard palate widens in the caudal half of the molar region. An extensive venous plexus lies between the mucous membrane and the bone. A dental pad is present and is covered by a thick, heavily cornified epithelium. Unlike the other ruminants, a pair of incisor teeth are present. In the deciduous set incisor N 3 does erupt, although it is not very well developed. Centrally along the caudal border of the dental pad the incisive papilla occurs with no detectable openings of the incisive ducts. The palatine ridges are V-shaped rostrally but become irregular transverse ridges caudally. The caudal region which is bordered by the molar teeth is devoid of ridges and is concave, to accommodate the torus of the tongue. The tongue fills the long narrow oral cavity when upper and lower teeth are in contact. The tongue is spatula-shaped in appearance. The mucous membrane is thin and delicate along ventral and lower lateral surfaces. It abruptly goes over into thick, cornified mucosa which covers borders and dorsum of the organ, and contains numerous papillae. The filiform, conical, and lentiform papillae have mechanical functions, while the fungiform and vallate papillae contain taste buds, and serve mainly a gustatory function. Foliate papillae are absent. Approximately 20 mm rostrally to the frenulum are the openings of the mandibular salivary duct.

There are no detectable openings of the orobasal organ. The lateral sublingual recesses contain the openings of the sublingual salivary glands. The important salivary glands are the parotid, mandibular, and buccal glands and, to a lesser extent the sublingual gland. The secretion of the parotid and ventral buccal glands is serous in character; the dorsal and middle buccal as well as the sublingual have purely mucous acini while of the mandibular gland is mixed. The sublingual gland consists of a series of pale yellow lobules, along the lateral sides of the base of the tongue. The monostomatic sublingual gland is absent. Numerous small mucous glands are present in the mucosa of the cheeks, around the tonsils, and in the peculiar diverticulum of the soft palate (Smuts and Bezuidenhout, 1987).

SOFT PALATE

Of all the domestic animals the camel's soft palate is the longest, with an average length of 160 mm in the adult. It stretches from the end of the hard palate caudally over the epiglottis to the level of the arytenoid cartilages (Smuts and Bezuidenhout, 1987).

PALATINE DIVERTICULUM (DULLA)

A peculiar expandable diverticulum occurs on the ventral median aspect of the soft palate, close to its origin. It is more developed in males than in the female. This palatine diverticulum is popularly referred to as 'DULLA', an Arab word for the balloon-like structure which is seen to be inflated from within and projected from the buccal cavity of males during the breeding season. This act is accompanied by gurgling sounds. The diverticulum consists of loose connective tissue and mucous glands covered by mucosa, which may be partly pigmented. A fold of mucous membrane from each side of the root of the tongue runs dorsally and caudally to form a semi-lunar fold on each side of the dorsal midline. The two folds meet dorsally and continue caudally to form a roughly triangular enlargement of the soft palate, with the apex directed caudally and with a semi-lunar fossa formed on each side of it. The most logical explanation about the mechanism of distension of the 'DULLA' seems to be the following: eructed gas from the stomach is forced into the lung, from where air is forced into the oral pharynx while the soft palate is raised and the entrance of the nasopharynx is closed. The distension of the 'Dulla' has been achieved artificially by blowing air through the trachea with the nares tightly closed (Arnautovic and Magid 1974)

PHARYNX

Contrary to the other ruminants, the pharynx is relatively long and extends as far caudally as the first cervical vertebra. The nasopharynx is part of the respiratory canal. It lies dorsally to the oropharynx and stretches from the choanae to the intra-pharyngeal opening. The mucous membrane may be partly pigmented. The roof, Fornix pharyngis, is concave. It is divided by a prominent transverse crest into two dome-shaped compartments. The pharyngeal openings of the auditory tubes are in the dorsolateral aspect of the wall of the rostral compartment, directly behind the choanae. Each opening consists of a semi-lunar slit which is partly covered by a low cushion of mucous membrane. Caudoventrally, the nasopharynx communicates with the oro-and laryngopharynx through the intrapharyngeal opening. The oropharynx extends from the palatoglossal arches to the base of the epiglottis. Its roof is formed by the soft palate and 'DULLA'. The lateral walls contain the palatine tonsils (Smuts and Bezuidenhoat, 1987).

THE TEETH

The formula for the permanent dentition is as follows:

$$2(I^{1}/_{3}C^{1}/_{1}P^{3}/_{2}M^{3}/_{3}) = 34$$

The characteristic features of camel dentition are the presence of a pair of incisors in the upper jaw and canine teeth in both jaws. This is a deviation from the ruminant pattern.

The single incisor in the upper jaw represents incisor 3. It is strong and tusk-like in the male; in the female it is smaller and may be absent. It reaches its greatest length at 9 years of age (Leese 1927). The incisors of the lower jaw are haplodont, with crowns which are compressed rostrocaudally and become wedge-shaped towards the neck. In the young adult the incisors project almost horizontally from the jaw, and with advancing years they become upright.

The tusk-like canines are placed caudally, but fairly close to incisor 3. When the jaws are closed the lower incisors are placed in front of the larger upper ones. They

are considerably smaller in the female. A rudimentary second incisor (Di2) may occur in the gum but it never erupts. The tusk-like first premolar is not replaced.

ESOPHAGUS

The esophagus starts caudally to the esophageal vestibulum and the V-shaped dorsal fold, which marks the termination of the palato pharyngeal arches. The rostral end of the esophagus lies on the cricoid cartilage and the dorsal cricoarytenoid muscle. Caudally to the larynx, it is related to the longus colli muscle dorsally, the trachea ventrally, and the common carotid artery and vagosympathetic trunk on its ventro-lateral aspect. It is contained in the gutter like channel, which occurs ventrally to the vertebral bodies and medially to the vertically placed transverse processes. Towards the thoracic inlet the esophagus lies on the left dorsolateral aspect of trachea. The position of the esophagus in the cranial mediastinum is dorsal to the trachea. It continues in the caudal mediastinum towards the esophageal hiatus of the diaphragm. In this region it is bordered dorsally by the caudal mediastinal lymph nodes.

Esophagial function in camels, like in ruminants, is much more diverse than in monogastric animals. Large quantities of saliva and sometimes very thorny roughages have to be swollowed, to reach the foregut system. Eructation of gas needs to be performed according to microbial gas production. Regurgitation of partly digested forestomach contents for rumination is certainly the most complex task of the esophagus. The circular and longitudinal muscles of the esophagus in camels like in ruminants are made up of striated muscles. Muscularis mucosae is rudimentary in camels and glandulae oesophageae can be seen over the whole length of esophagus (Lechner-Doll and Hoffrogge, 1994).

STOMACH

Although Camels are ruminating animals they are not classified as *Rumiantia*. They differ from true ruminants in having a completely different stomach system. The same general characteristics of rumination and microbial digestion of fibrous feeds in a large and compartmented stomach have developed independently in camelids and ruminants and at different geological times. This independent development resulted in marked differences in morphology, histology and motility of the stomach system.

The stomach of the camel is divided into compartments, namely a rumen (compartment 1), reticulum (compartment 2), omasum, and abomasum (omasum and abomasum are designated as compartment 3). These compartments differ greatly in shape and structure from the typical design found in the ox and sheep.

The rumen has a capacity of approximately 100 liters. It occupies the major portion of the abdominal cavity. It extends from the diaphragm to the pelvic inlet, filling the left half of the abdominal cavity. Its caudal aspect extends well over the median plane into the right half of the abdominal cavity. In the suckling animal, it is relatively smaller. The camel rumen is not divided in the same way as that of the ox and sheep. It is subdivided by a strong muscular ridge into cranial and a caudal portion. The ventral parts of the rumen are made up of series of glandular sacs. These glands resemble cardiac glands in other species of animals. These glands are distributed in four regions: The luminal surface epithelium covering the ridges between different foveolae; the foveolar epithelium; the isthmic epithelium; and the epithelium of the end-piece (Cummings et al., 1972). The luminal surface epithelium is composed of tall, laterally compressed cells containing mucous granules, whose cell numbers increase close to the foveolar orifice and reach a maximum in the foveolar epithelium. Many endocrine cells are also present in this area (Luciano and Engelhardt, 1980). The columnar cells at the surface contain several apical microvilli and many mitochondria. These cells are of special interest with respect to their absorptive ability (Cummings et al., 1972; Luciano and Engelhardt, 1980). The dorsal

part of the rumen is lined by smooth stratified squamous epithelium without papillae (Engelhardt et al., 1992).

The reticulum is situated to the right and cranioventrally to the cardia. Its initial part lies directly cranially to the glandular sac of the caudodorsal compartment. It is bean shaped, with its greater curvature directed ventrally. The interior of the reticulum is characterized by a powerful and complex system of radiating pillars which are interconnected by lattice-work of slender pillars (Smuts and Bezuidenhout, 1987). The structure of the mucous membrane of the reticulum resembles the adjoining glandular sac (Engelhardt et al., 1988).

The omasum is long and sausage-shaped. It lies cranially below the cardia on the dorsal surface of the cranioventral sac. It has an initial dilatation which becomes constricted a few centimeters further by an incomplete muscular band in its ventrocranial aspect. This is followed by marked enlargement of its lumen as it approaches the left body wall. The diameter of the omasum decreases as it bends ventrally and to the right. When it approaches the ventral aspect of the reticulum it becomes constricted ventrally before going over into the abomasum (Smuts and Bezuidenhout, 1987). The omasum is lined internally by glandular mucosa arranged in longitudinal folds (Engelhardt et al., 1992).

The primary functions of the alimentary tract are the prehension, digestion and absorption of food and water, and the maintenance of the internal environment by modification of the amount and nature of the materials absorbed.

The rumen, reticulum, and omasum, in camels, are collectively termed forestomach, which is comparatively shorter relative to body weight (8.9 ± 0.32 and $11.3 + 0.31$ liters/100 kg during the green and the dry season, respectively) than the other ruminants. Motility of the forestomach in camels results in an effective mixture of fluid and feed particles. Rumination reaches a peak in the early morning. In contrast to domestic ruminants, rumination activity in camels occurs independently of feeding time (Kaske et al., 1989). This maximum activity in the early morning may be a mechanism to achieve a prolonged retention time of particles in the forestomach. Particles have to be reduced in size before they can pass into omasum (Lechner-Doll and Engelhardt, 1989). If rumination begins after a long period of non-rumination, feed particles remain for a long time in the forestomach and cellulose digestibility may be improved. Feed particles are retained selectively in the forestomach of camels and ruminants.

The hydrochloric acid producing abomasum (hind stomach) is a relatively short compartment. The transition between omasum and abomasum is not obvious externally. The greater curvature lies ventrally. It expands to form a prominent convexity towards the pylorus, which is placed dorsally, below the tenth costochondral junction. The lesser curvature forms a marked constriction before expanding into a bulbous pyloric part (Smuts and Bezuidenhout, 1987). A pertinent point is that the contents in the abomasum become diluted. The estimated secretion of water into the abomasum totals 150% of the amount of water absorbed in the omasum. Together with water that enters the rumen, there is a net transfer of water from the blood into the stomachs. The fairly dry ingesta that passes into the abomasum are therefore diluted before passing quickly into the intestines where water is reabsorbed back into the blood.

The most striking feature differentiating camel stomach from the other ruminants is the presence of glandular sacs.

Stomach motility also differs strongly between ruminants and camelids. In the former the total ingesta in the reticulo-rumen are mixed and transported within the organ some hours after feed intake rather homogeneously. In the latter, particles and fluids are separated in a suction pressure rhythum during the motility cycle, whereby fluids and solutes are pressed into the glandular sacs for potential absorption. Thus larger feed particles are selectively retained in the forestomach for prolonged microbial degradation.

Contents in the forestomach of camels are comparatively solid. Motility leads to a slow rotation of the fibrous mass (Hoffrogge et al., 1994). Two basic forestomach contraction sequences (A-contraction and B-contraction) have been observed (Heller et al., 1986). A-sequence starts with a contraction of reticulum followed by a contraction of caudal rumen about 4 seconds later. B-sequence begins with a contraction of cranial rumen followed by reticulum and caudal rumen. B-sequence lasts for about 9 seconds. The flow of digesta through the canal between reticulum and omasum occurs during contraction of reticulum. During eating and rumination, forestomach motility is frequent (about 100 A- and B-sequences/h). During resting periods long pauses in motility (upto 20 mins) occur. Motility of the forestomach in camels results in an effective mixture of fluid and particles.

Fluid mean retention time (MRT) in the forestomach is substantially longer in cattle (28 h) and in camels (25 h) than in sheep and goats (20 h).

In contrast to most ruminant species, camels are not restricted to one feeding strategy. They can, if necessary, use low quality, fibre rich diets. But if presented with a choice, they select the most nutritive parts of plant. When only poor-quality, high fiber diets are available, camels are classified as bulk and roughage eaters. The digestibility of fibrous feed in the forestomach of camels can be more efficient as compared to most ruminants due to a long retention time of the diet (Engelhardt, 1994). On the basis of feeding behavior, camels foraging on a thorn bush 'Savannah pasture' are classified as selective browsers (Engelhardt et al., 1992).

When low protein feed with sufficient available energy for microbial growth is fed to camels, recycling and use of endogenous urea nitrogen is high and renal urea excretion is low. Ninety to 96% of total urea turn over is recycled into the gastrointestinal tract, compared with 47% under control condition (Engelhardt et al., 1978; Mousa et al., 1983).

The return of urea from blood into the forestomach is beneficial only under conditions of nitrogen shortage. If the recycled urea nitrogen cannot be incorporated into the microbial protein, ammonia absorbs back into the circulation and resynthesizes to urea (Engelhardt et al., 1978).

Fermentation of feed is basically similar in camels and ruminants. Short chain fatty acids (SCFA), being major product of microbial carbohydrate digestion are the most important energy source for the camel. These are absorbed from the forestomach into the blood in large quantities (Dycker et al., 1994). The concentration of SCFA is 40-90 % higher in camels than the ruminants (Lechner-Doll et al., 1991). The mechanism of SCFA-absorption may be different between ruminants and camels because of the the differences in the, histological structure of the forestomach. There is some evidence that the magnitude of SCFA absorption in the camels may vary in a wide range, depending on feeding situation (Holler et al., 1989). Dycker et al. (1994) found that SCFA absorption depends on the pH, Na+ absorption and SCFA concentration.

SMALL INTESTINES

The total length of the small intestine averages 40 m while the large intestine is 19.5 m. The duodenum begins at the pylorus, which is situated below the tenth costochondral junction. The Pars cranialis lies against the visceral surface of the liver. It consists of a prominent Ampulla duodeni followed by a slender Ansa sigmidea, which is directed craniodorsally. At the Flexure duodeni cranialis the strongly convoluted "Duodenum descendens" commences, passing in a caudal direction. It is suspended by a short mesoduodenum, which contains the right lobe of the pancreas. Interiorly, at the junction of the ampulla and the sigmoid portion, a shelf-like partition projects into the lumen and the walls of the two portions are attached to each other. Directly caudally to the cranial flexure, the hepato-duodenal duct emerges from the pancreas to enter the duodenal wall. The intramural course of the duct is 45-60 mm. It opens through the major duodenal papilla, which is situated between the free and

attached borders of the dorsal aspect of the duodenal wall. Ventrally to the caudal pole of the right kidney, the duodenum turns medially as the Pars transversa. It attaches to the last part of the ascending colon, and passes cranially between the latter and the angle formed by the transverse and descending colon to bend ventrally at the Flexura duodenojejunalis (Smuts and Bezuidenhout, 1987).

The convoluted jejunum is placed mainly on the right flank, in abdominal region, and on the sternum in the medial plane. Its suspensory fold, the mesojejunum, is fused with the mesocolon. Its terminal portion is placed on the left of the median plane. A short peritoneal fold, the Plica ileocecalis, attaches it to the lesser curvature of the cecum and marks the beginning of the ileum. The short ileum ends at the ileal orifice which demarcates the cecocolic level of the caudal pole of the midline, on the level of the caudal pole of the left kidney.

LARGE INTESTINE

The cecum and the initial part of the colon is larger in diameter than the small intestine. The ascending colon is arranged in a flat spiral, the transverse colon is short, and the descending colon has a sigmoid flexure in its initial part.

The cecum of camel resembles that of other domestic ruminants. It is a slightly S-shaped blind tube which is situated chiefly in the right flank. It arises on the level of the fifth lumber vertebra, medially to the left kidney. The body is directed towards the right and caudally, while the apex points cranioventrally. The ileum lies medially to it.

The colon arises at the ileal opening. The initial part of the ascending colon passes cranially along the ventral surface of the left kidney and to the right of the rumen. It then turns ventrally and caudally into the first centripetal coil of the spiral consisting of five and a half centripetal coils. At the middle of the second spiral the diameter decreases considerably. The Flexura centralis marks the beginning of the centrifugal coils which are also five and a half in number. The last loop of the ascending colon runs caudally and to the right, before passing cranially and to the left around the cranial mesenteric vessels as the Colon transversum. The latter is a short segment and bends caudally to mark the beginning of the Colon descendens. A sigmoid flexure occurs in its initial part. It then continues along the dorsal body wall suspended by a short mesocolon to enter the pelvic inlet and to be continued by the rectum.

The rectum is covered by peritoneum as far as the level of the second caudal vertebra. The retroperitoneal portion is therefore short. Longitudinal folds of mucous membrane (Columnae rectales) end abruptly at the anorectal line. The Zona cutanea is relatively smooth and is continued at the anocutaneous line by normal skin containing hair follicles as the external covering of the anus. The anal opening is situated below the third or fourth caudal vertebra.

LIVER

The camel liver is dark brown in color when in fresh state. Its weight in adult animals varies from 6.5- 10 kg. It is only partly lobulated. The gall bladder is absent. The absence of a gall bladder, landmark between the quadrate and right lobes, poses some difficulty in determining the boundary between these lobes in the camel. To the left of an imaginary line connecting the esophageal notch with the notch for the round ligament is the left lobe, Lobus hepatis sinister. It is subdivided into L. hepatis sinister medialis and lateralis. To the right of the notch for the round ligament is the quadrate lobe, L. quadratus. An indistinct incisure marks the limit of the quadrate lobe. To the right of an imaginary line from this notch to where the caudal vena cava crosses the dorsal border of the liver is the undivided right lobe, L. hepatis dexter. The caudate lobe, L. caudatus, which is situated dorsally to the porta, has a well developed flap-like Proc. papillaris and a pointed Proc. caudatus on the right. The dorsal border of the liver is thick and includes the concave renal impression, Impressio renalis, the groove

for the caudal vena cava and the esophageal notch. The remaining borders are sharp and are characterized by the presence of numerous irregular fissures. They are continued as a network of grooves along the diaphragmatic and visceral surfaces for a variable distance, giving these areas a cobbled appearance. The lobulation is distinct. Every lobule is surrounded by connective tissue (Smuts and Bezuidenhout, 1987).

The two hepatic ducts unite to form the common hepatic duct, which unites with the pancreatic duct before opening into the duodenum. The distance between the origin of the common hepatic duct and its opening into the duodenum is 100 - 110 mm, and there is no sphincter at the terminal end of the duct (Radmanesh, 1974).

The liver lies mainly on the right side of the midline, under cover of the ribs. It extends from the 5th to the 12th rib. The left lateral lobe lies on the left of the median plane, at the caudal border of the 5th rib. In the newborn animal the entire left lobe lies on the left side. The diaphragmatic surface lies against the diaphragm. The visceral surface is in contact with the different compartments of the stomach, the duodenum, colon, and pancreas.

In the neonates, the liver is relatively larger and the borders are more rounded. The papillary process is more prominent, and both left and right lobes protrude from the rib cage and are in contact with the abdominal wall.

PANCREAS

The pancreas of camel consists of a narrow body, and right and left lobes, of which the left is larger and longer. At its ventral surface is the 'Incisura pancreatis', which is occupied by the portal vein. The left lobe of the pancreas is inserted between the left crus of the diaphragm and the caudodorsal sac of the rumen. Caudally, it is related to the ventral surface of the left kidney and the mediocaudal part of the spleen. The right lobe is situated in the mesoduodenum. Caudally, it is associated with the transverse and descending colon. Cranially, it is attached to the ampulla of the duodenum. At the level of the cranial duodenal flexure the hepatoduodenal duct emerges from the pancreas to enter the duodenal wall and opens on the major duodenal papilla. Its weight, in adult animals, is approximately 0.5 kg (Smuts and Bezuidenhout, 1987).

ALIMENTARY TRACT DYSFUNCTIONS

Stepankina and Tashenov (1958) carried out duodenal function studies using a permanent fistula and found that in the camel there was a normal volume of chyme of 86 liters. The volume declined to 60 liters after 15 days of dehydration. If we assume that the camels weighed 400 kg each and that they lost an average of 25% of their body weight, the above mentioned data show that the losses of body water come from the intestines. Studies using duodenal fistula also showed that when the animals were dehydrated the chyme became much more diluted. It was assumed that the cause of this was the salivary secretion. However other research using a parotid as well as duodenal fistula showed that there was a great reduction in parotid secretion, from 20 liters to a day to about 1 liter (Macfarlane et al., 1963). It is pertinent that the saliva contained more urea which would take water with it, thereby counteracting the drying effect of the absorption of sodium bicarbonate and water. It is the secretion of water into the abomasum which dilutes the chyme.

The advantage that the camel has over other mammals living in the same environment is that there are large amounts of sodium and bicarbonate not only in the stomachs but also in the intestines (Maloiy and Clemens, 1980). As water absorption from the intestines is generally associated with the absorption of salt, the camel has a large potential water source in its intestines. The colon has an extremely effective water-absorbing capability (Maloiy and Clemens, 1980). The low water turnover of the dehydrated camel enable utilization of this reservoir over a far longer period than any other animal (Macfarlane et al., 1963; Etzion et al., 1984). The regulation of

water content could be important for conserving body water as the renal water-conserving mechanism (Farid et al., 1979).

The extracellular fluid compartment losses during water deprivation are almost entirely from the alimentary canal (Hecker et al., 1964; Thornton and Yates, 1968). This accounts for the fact that there are minimal losses of plasma volume in the dehydrated camel (Macfarelane, 1977). Besides water absorption from the intestines, the quantity of feces excreted is determined by the quantity, quality and digestibility of the fodder. When food has more roughage, more feces are produced. On good pastures camels defecate twice an hour (Gauthier-Pilters and Dagg, 1981), producing 200g dry fecal weight. On poor pastures defication occurs only once an hour producing 100 g dry feces. As a rule the production of feces totalled 370-500 g dry weight per 100 kg body weight (Maloiy, 1972; Schmidt-Nielsen et al., 1956). The colonic contents in camel contain the lowest concentration of potassium. The advantage of this fact is that under the influence of aldosterone sodium will be absorbed and potassium secreted into the intestines.

The primary functions of the alimentary tract are prehension, digestion and absorption of food and water and regulate the amount and nature of the materials to be absorbed.

There are four major modes of alimentary dysfunction. These are either abnormality of motility, secretion, digestion or absorption. The procedure in diagnosis should be to determine which mode or modes of function is or are disturbed before the determination of the site, nature of the lesion and of the specific cause.

Abnormal motor function may take the form of increased or decreased motility. Peristalsis and segmental movements are usually affected equally and in the same manner. Motility depends upon stimulation via the sympathetic and parasympathetic nervous systems and is thus dependent upon the activity of the central and peripheral parts of these systems, and the intestinal musculature and its intrinsic nervous plexus. Autonomic imbalance, resulting in a relative dominance of one or other systems, is manifested by hypermotility or hypomotility, and can arise as a result of stimulation or destruction of hypothalamic centres, the ganglia, or the efferent or afferent peripheral branches of the system. Debility, accompanied by weakness of the musculature, or severe inflammation, as occurs in acute peritonitis or trauma, results in atony of gut wall. Less severe inflammation, as occurs in mild gastritis and enteritis, causes an increase in muscular activity. Increased motility causes diarrhea, decreased motility cause constipation, and both have deleterious effects on digestion and absorption.

Irritability at a particular segment increases its activity, and disturbs the normal downward gradient activity, which ensures that the ingesta is passed from the esophagus to the rectum. The gradient towards the rectum if made steeper, there is an increase in the rate of passage of ingesta in that direction. If increased potential activity of an irritated segment is sufficiently high it may produce a reverse gradient to the oral segments. Thus the direction of the peristaltic waves is reversed. It is by this means that vomiting occurs and intestinal contents, even feces, are returned to the stomach and vomited.

One of the major results of abnormality of motility is distension of the tract. It occurs in a number of disturbances, including the rapid accumulation or insufficient expulsion of gases, complete occlusion of the lumen by intestinal accident, pyloric or ileocecal valve obstructions, and engorgement of solid or liquid foods. Fluids and to lesser extent gas, accumulate because of their failure to pass along the tract. Much of the accumulated fluid represents saliva and gastric and intestinal juices secreted during normal digestion. Distension causes pain and, reflexly, increased spasm and motility of adjoining gut segments. Distension also stimulates further secreation of fluid into the lumen of the gut and this exaggerates the distension. When the distension passes a critical point, the ability of the musculature of the wall to respond diminishes, the

initial pain disappears, and a state of paralysis develops in which all muscle tone is lost.

An immediate effect of distension of the stomach or small intestine by the accumulation of saliva and normal gastric and intestinal secretions stimulates further secretion of fluid and electrolytes in the oral segments. The stimulation is self-perpetuating and creates a vicious cycle resulting in loss of fluid and electrolytes to the point where fatal dehydration can occur. The dehydration is accompanied by acidosis or alkalosis depending on whether the obstruction is in the intestine and accompanied by loss of alkali, or in the stomach and accompanied by heavy loss of acid radicals. The net effect is the same whether the fluid is lost by vomiting or is retained in the gut. The same cycle of events occurs in camels which engorge on grains but here the precipitating mechanism is not distension but a gross increase in osmotic pressure of the ingesta due to the accumulation of lactic acid. Dehydration is also of major importance in diarrhea irrespective of the cause. An important additional factor in the production of shock, when there is distension of alimentary segments, is a marked reflex depression of vasomotor, cardio-vascular and respiratory function. In calf diarrhea where there is neither septicemia nor toxemia caused by bacteria, the end point in the phase of dehydration can be cardiac failure due to severe metabolic acidosis. Renal ischemia leading to Uremia may result from decreased circulating blood volume and also contribute to a fatal outcome.

In a number of circumstances, the activity of the flora in the stomach of camels can be modified so that digestion is abnormal or ceases. Failure to provide the correct diet, prolonged starvation or inappetence, and hyperacidity which occurs in engorgement on grains, all result in impairment of microbial digestion. The bacteria, yeasts and protozoa may also be adversely affected by the oral administration of antibiotics and sulphonamides, or drugs which drastically alter the pH of the rumen contents.

EXAMINATION OF THE DIGESTIVE SYSTEM

Radiological examination is difficult in camels. Imaging by ultrasound is mainly practiced technique for pregnancy diagnosis and the detection of certain reproductive lesions.

External palpation of the abdomen is of limited value in camels and is replaced by rectal palpation and auscultation. Attempts to pass a stomach tube may detect complete or partial obstruction of the esophagus. Fiber optic gastroendoscopy may be of value in diagnosing stomach lesions.

Examination of the feces may provide valuable information on the digestive and motor functions of the tract. They should be examined for volume, consistency, form, color, covering, odor and composition. Note should be made of the frequency and the time taken for material to pass through the tract. Laboratory examination may be advisable to detect the presence of helminthic eggs, occult blood, bile pigments, pathogenic bacteria or protozoa. It is usually sufficient to say that the volume is scanty, normal or copious but, in special circumstances, it may be advisable to weigh the daily output. There is an increased bulk when much fiber is fed or during attacks of diarrhea. The stool of the dromedary camel is passed in irregularly shaped pellets. A medium-sized female may void pellets that range is size from 15 x 15 x 15 to 20 x 25 x 25 mm. A large male may pass pellets as large as 25 x 30 x 35 mm (Fowler, 1991). There is considerable variation in the consistency of feces, depending upon the nature of feed, weather and water intake. The old world camels have the capacity to pass totally dessicated feces when water intake is restricted. In the Arabian desert during the summer, freshly passed feces can be used for fuel. Variation in consistency not explainable by changes in the character of the feed may indicate abnormalities of any of the functions of the tract. The consistency is more fluid in diarrhea and less fluid than normal in constipation. The consistency and form of the feces may provide some

indication of the location of the dysfunction of the gastrointestinal tract. In, general, large quantities of fluid feces suggest a dysfunction of the small intestine where normally most of the fluid is absorbed. If the feces contain large quantities of undigested feed, this suggests overfeeding, incomplete mastication, and digestive enzyme deficiency or an acute disorder of the small intestine or stomach. Large quantities of soft feces which contain well digested ingesta suggest a dysfunction of the large intestine. However, these are guidelines and are subject to error.

The color of the feces also varies widely with the color of the food, but feces of a lighter color than normal may be caused by insufficient secretion of bile or by simple dilution of the pigments as occur in diarrhea. Discoloration due to colored medicaments should also be taken into consideration when the animal is under treatment. Hemorrhage into the stomach results in the formation of acid hematin which gives vomitus a dark brown color and feces a black or very dark brown, or tarry appearance. The change in the appearance of the feces caused by hemorrhage into the bowel varies with the level at which the hemorrhage occurs. If the blood originates in the small intestine, the feces may be brown-black, but if it is in the colon or cecum, the blood is unchanged and gives the feces an even red color. Hemorrhage into the lower colon and rectum may cause voiding of stools containing or consisting of entirely clots of whole blood. Hemorrhages in the pharynx is unusual, but when it occurs the blood may be swallowed and appear in the vomits or feces. In case of doubt regarding the presence or absence of hemorrhage the laboratory should be consulted.

Odor also depends largely on the nature of the feed eaten by the camel, but in severe enteritis the odor is characteristic of putrefaction. In the camels feces there is always a proportion of undigested fiber but excessive amount of it suggests, incomplete digestion due to, for example bad teeth or faulty mastication. Excessively pasty feces are usually associated with a prolonged sojourn in the tract such as occurs in vagal indigestion or abomasal displacement. Forgein material of diagnostic significance includes particles of sand or gravel, wood, and shreds of mucosa. Mucous is a normal constituent of feces, but if in excessive amount, indicates chronic inflammation (when it is associated with fluid and copious feces), or constipation (when the feces are small in volume and hard). Mucosal shreds or casts always indicate inflammation.

Frequency of defecation and the length of sojourn are usually closely allied. An increase in frequency and decreased sojourn occur in diarrhea and reverse in constipation.

Observation of other acts associated with the function of the alimentary tract may provide information of diagnostic value. Prehension, mastication, swallowing, vomiting and defecation should be observed and attempts should be made to analyze the behavior of the camel when there is evidence of abdominal pain.

Collection of a sample of peritoneal fluid is the most useful aid in the diagnosis of diseases of peritoneum and the abdominal segment of the alimentary tract.

Normal peritoneal fluid is a transudate and has functions similar to those of other tissue fluids. It contains mesothelial cells, lymphocytes, neutrophils, a few erythrocytes and occasionally monocytes and eosinophils. The peritoneal fluid should be examined for its physical characteristics (especially color, translucence, specific gravity and clotting time), biochemical composition, and cellular constituents.

The examination of the fluid may help in determining: peritonitis (chemical or infectious), infarction of a segment of gut wall, perforation of the alimentary tract wall, rupture of the urinary bladder, leakage from the biliary system, intraperitoneal hemorrhage and peritoneal neoplasia.

The inflammatory reaction of the peritoneum varies with time and a single examination can be dangerously misleading. A series of examinations may be necessary, in acute cases at intervals as short as an hour. A significant reaction in a

peritoneal cavity may be quite localized and a sample of fluid collected at one point in the cavity may not be representative of the entire cavity.

Normal peritoneal fluid is crystal clear and straw to yellow in color. Turbidity indicates the presence of increased leukocytes and protein, which may include fine strands of fibrin. A green color of the peritoneal fluid suggests leakage of food material, intense orange-green color indicates rupture of biliary system. Pink to red color indicates presence of hemoglobin, degenerated or intact erythrocytes, entire and damaged vascular system by infarction, perforation or insrease in hydrostatic pressure. Red brown color indicates the late stages of necrosis of the gut wall and the presence of degenerated blood and damage to gut wall. The blood is likely to be concentrated if there has been sufficient time for fluid resorption from the peritoneum. Spleenic blood has a higher packed cell volume also, but there is no erythrophagocytosis. A packed cell volume of less than 5 % in peritoneal fluid suggests extravasation of blood from an infarcted or inflammed gut; one of greater than 20 % suggests a significant hemorrhage.

A high specific gravity and increase in protein content of peritoneal fluid are indicative of vascular damage and leakage of plasma protein as seen in peritonitis or mural infarction.

The presence of whole blood, or clear peritoneal fluid streaked with blood, or heavily blood stained fluid indicate that the sample has been collected from the spleen, a blood vessel, or that there is hemoperitoneum due to rupture of uterus or bladder, or from dicoumarol poisoning.

An increase in total white cell count of the fluid including a disproportionate number of polymorphonuclear cells indicate the presence of acute inflammation of infectious origin, or it can be sterile. An increase in mononuclear phagocytes in the peritoneal fluid is an indication of chronic peritonitis. Degenerative changes in the neutrophils gives estimation of the probability of infection. An increase in the mesothelial cells with the distinctive presence of actively dividing mitotic figures suggests neoplasia. Bacteria found as phagocytosed inclusions in the leukocytes or by culture of fluid indicate infective peritonitis which may arise by hematogenous spread in which case the infection is likely to be a specific one. If there has been leakage from peritoneal abscess the same comment applies, but if there is leakage through a segment of devitalized wall or a perforated wall there is likely to be a mixed infection.

A dark green sample, containing motile protozoa, with very few leukocytes and no mesothelial cells indicates that the sample has been collected from the gut lumen.

DISEASES OF THE BUCCAL CAVITY AND ASSOCIATED ORGANS

DISEASES OF THE LIPS

The lesions of contagious ecthyma and pox, on the lips, are common in camels (Ali et al. 1991). It was believed that vesicles of ecthyma were not contagious and occurred due to damage done by thorny plants (Borisovich and Orekhov, 1966). Later it was realized that the thorny plants damaged the lips, allowing transmission of the parapoxvirus (Buchnev et al., 1987). Cases of papillomatosis of the muzzle in dromedary camel have also been reported (Wernery, 1995). Moussa et al. (1987) in Egypt have shown that dromedaries are susceptible to foot and mouth disease and the authors described ruptured vesicles ulceration on the upper lips. There is no report regarding the presence of lesions of blue tongue, rinderpest, malignant catarrhal fever, and mucosal disease in dromedary camels.

In older animals, the lower lip often sags away from the mandible when not involved in prehension. It flaps rhythmically as the animal runs. At times, it may fold back under the chin (Manefield and Tinson, 1996). This should not be confused with paralysis of lower lip as is observed in rabies and a paralysis of unknown etiology, recognized as a disease syndrome in India (Purohit et al., 1988). Trauma to the side of

the head can result in unilateral lip paralysis. Tranquilization will also result in drooping of lower lip. There is frequent loss of hair from the muzzle in camels that have continuous access to mineral licks. Lips of fighting males are frequently injured.

STOMATITIS

Stomatitis is inflammation of the oral mucosa and includes glossitis (inflammation of lingual mucosa), palatitis (lampas) and gingivitis (inflammation of the mucosa of the gums). Clinically it is manifested by partial or complete loss of appetite, smacking of the lips and profuse salivation. It is commonly an accompaniment of systemic disease. Stomatitis may be caused by physical (trauma while dosing, forgien body injury, malocclusion of teeth, sharp awns or spines on plants), chemical (Irritant drugs administered in over strong concentrations, e.g., chloral hydrate, counter irritants applied to skin, left unprotected and licked by the camel, irritant substances administered by mistake including acids, alkalies, phenolic compounds, manifestation of systemic poisoning, e.g., chronic mercury, furazolidone and some fungi cause a combination of focal hemorrhages and necrotic ulcers and erosions), or infectious agents (actinobacillosis, actinomycosis, secondary bacterial infection, camel pox and mycosis), the latter being the largest group of causes. Some times in extreme conditions nutritional deficiencies can also cause stomatitis.

Clinical Findings

The clinical signs of stomatitis are caused by the inflammation or erosion of the mucosa and they vary in severity with the degree of inflammation. There is partial or complete anorexia and slow, painful mastication. Chewing movements and smacking of the lips are accompanied by salivation, either frothy and in small amounts, or profuse and drooling if the camel does not swallow normally. The saliva may contain pus or shreds of necrosed epithelium. A fetid odor is present on breath only if the bacterial invasion has occurred. The latter can also result in enlargement of the local lymph nodes. Swelling of the face can also occur if the deeper tissues are also invaded by microorganisms. An increased desire for water is apparent and the camel resent manipulation and examination of the mouth.

The local lesions vary greatly depending upon the causative agent. Vesicular lesions are usually thin walled, 1-2 cm in diameter, and filled with clear serous fluid. The vesicles rupture readily to leave sharp-edged, shallow ulcers. Usually discrete areas of necrosis, which are not readily seen in the early stages, tend to occur most commonly on the lingual mucosa and at the commissures of the mouth. The necrotic tissues may remain in situ but are usually shed leaving a very shallow discontinuity of the mucosa with a dark-red base which is more readily seen. If recovery occurs, these lesions heal very quickly. Ulcerative lesions penetrate more deeply to the lamina properia.

Catarrhal stomatitis is manifested by a diffuse inflammation of the buccal mucosa and is commonly the result of direct injury by chemical a physical agents. Mycotic stomatitis usually takes the form of heavy, white, velvety deposits with little obvious inflammation or damage of the mucosa. Lesion of the camel pox constitute the formation of pustules on the oral mucosa with the simultaneous presence of pustules on the other parts of the body

The lesions of the stomatitis are produced either by the causative agents being applied directly to the mucosa, or gaining entrance to it by way of minor abrasion, or by localization in the mucosa from a Viremia. In the first two instances, the stomatitis is designated as primary. In the third, it is usually described as secondary because of the common occurrence of similar lesions in other organs or on the other parts of the body, and the presence of systemic disease.

The differential diagnosis of stomatitis is important because of the occurrence of oral lesions in a number of highly infectious viral diseases.

The material collected (swabs or scrapings) from lesions of stomatitis should be sent to the laboratory for the presence of pathogenic viruses, bacteria, and fungi. Complete necropsy examination should be carried out on all fatally affected camels to determine whether the oral lesions are primary or local manifestation of a systemic disease.

Treatment

Affected camels should be isolated and fed and watered from separate utensils, if an infectious agent is suspected. Specific treatments are dealt with each specific disease. Non-specific treatment should consist of frequent application of a mild antiseptic such as 2% solution of copper sulfate, 2% suspension of borax or 1% suspension of sulfonamide in glycerin. Undolent ulcers require more vigorous treatment and respond well to curettage or cauterization with a silver nitrate stick or tincture of iodine. In all cases, soft, appetizing feed should be offered. Intravenous saline glucose should be administered in severe, and prolonged cases.

DISEASES OF TEETH

All of the dental problems seen in ruminants have been seen or are potential problems in camels. The conditions include dental plaque, pigmentation of the teeth, alveolar periostitis, pulpitis, fracture of the incisors and canines, fracture of the mandible, sharp enamel points caused by malocclusion, excessive wear, elongated teeth associated with superior bradygnathism, dental fistula, and other developmental defects causing malocclusion (Kumar et al., 1977; Shabaan et al., 1981; Fowler, 1991).

Clinical Findings

The signs of dental problems include inappetence, weight loss, abnormal chewing, dropping of cud, swelling over the dental roots, mandibular fistulae, and sinusitis (molar roots are contiguous to the maxillary sinus).

Treatment

Plaque will rarely interfere with oral function and will not require removal unless it is causing gingivitis. Pulpitis and alveolar periostitis will necessitate extraction or repulsion of the tooth. If dental fracture opens the pulp cavity, a pulpotomy may be done. If the pulp has become infected, the tooth should be removed. Elongated teeth may be cut off with a circular saw or an obstretic wire.

CHEEK IMPACTION

A bolus of hay may occasionally impact between the cheek and the last back molars resulting in a circumscribed unilateral local swelling. Use of suitable mouth gag and dislodgment attempted through some long instrument is recommended. Sedation may also be required.

EPULIS

A fibrous epulis (a pedunculated growth about 12 mm in diameter), originating from mucous membrane of the dental pad near the corner incisor tooth. This growth of rare occurrence in camels can be removed surgically (Manefield and Tinson, 1996).

PAROTITIS

Parotitis is inflammation of any of the salivary glands.

Etiology

Parotitis may be paranchymatous when the glandular tissue is diffusely inflammed or it may be local suppurative process. There are no specific causes in camels, cases occurring only sporadically and due either to localization of a blood

borne infection, invasion up the salivary ducts associated with stomatitis, or irritation by grass awns in the duct.

Clinical Findings

In the early stages, there is diffuse enlargement of the affected gland accompanied by warmth and pain on palpation. The pain may interfere with mastication and swallowing. There may be marked local edema in severe cases. Diffuse paranchymatous parotitis usually subsides with systemic and local treatment within a few days but suppurative lesions may discharge externally and form permanent salivary fistulae.

Careful palpation is necessary to differentiate the condition from lymphadenitis, abscesses of the throat region, and metastasis to the parotid lymph node of a malignant neoplasm. A needle may be inserted into the fluctuant swelling to establish the presence of an exudate, serum, or blood.

Treatment

Systemic treatment with sulphonamides or antibiotics is required in acute cases especially if there is a systemic reaction. Abscesses may require draining and, if discharge persists, the administration of enzymes either parenterally or locally may be necessary. A salivary fistula is common sequel.

DISEASES OF THE DULLA, PHARYNX, AND ESOPHAGUS

TRAUMATISATION OF DULLA

The Dulla is frequently injured by the camel's own canine teeth, by other camels while fighting, or by thorns and wires etc. The signs may include edema, perforation, and hematoma depending upon the causal agent and degree of trauma (Gahlot and Chouhan, 1992). Infection mostly supervenes, the Dulla may not be drawn back and due to compression of blood vessels necrosis and gangrene may follow (Gahlot et al., 1988; Bhargava, 1973). If the inflammed Dulla is retracted in the mouth, it causes hindrance to normal breathing.

The injured Dulla should be cleaned and painted with some suitable antiseptic e.g. glycerin, as soon as possible after injury. It should be tried to keep it inside the mouth by applying net on the muzzle. Antibiotics and anti inflammatory drugs may be given by injection for 5 days. In the case of necrosis and chronic conditions, amputation of the Dulla is recommended. In racing camels, Dulla is sometimes surgically removed, as it is believed that it causes hindrance to increased demand for respiration and poor racing performance.

IMPACTION OF DULLA

The food sometimes becomes impacted in the Dulla. The size of impaction may sometimes go up to the size of a football. The mass is very hard to touch. If the mass is not very large, it should be moved backwards and then forwards. In the case of larger mass, the resection of the organ is required. The surgery should be done under general anesthesia. Tracheotomy should also be done before resection.

Abscess formation and development of fibroma has also been reported to occur in camels (Barvalia et al, 1998; Gahlot, 1986; Vashishtha et al., 1980).

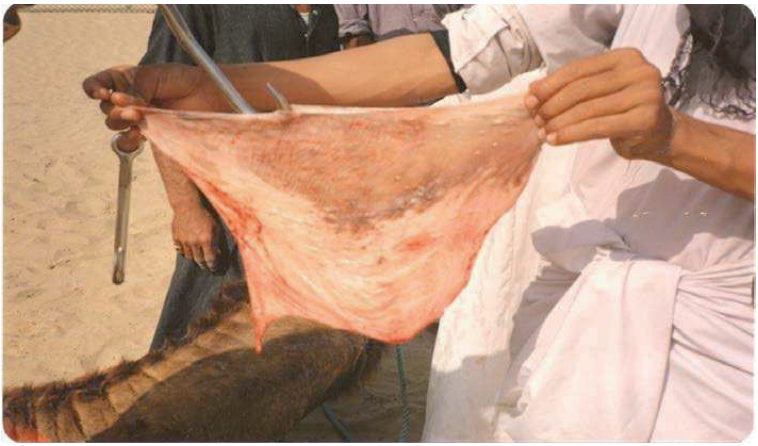
PHARYNGITIS

Pharyngitis is inflammation of the pharynx and is characterized by coughing, painful swallowing, and lack of appetite. Regurgitation through the nostrils and drooling of saliva may occur in severe cases.

Etiology

The distensible part of soft palate of male camels is more prone to injury as compared to other farm animals. Pharyngitis in camels is usually traumatic in origin. Infectious pharyngitis is part of the syndrome with other more obvious signs. The trauma being caused by balling or drenching gun, foreign bodies including grass and cereal awns, wire etc and accidental administration or ingestion of irritant, hot, or cold substances. Infectious pharyngitis may be caused by acute or chronic bacterial infections, especially those of the respiratory system.

Fixing of mouth contraption after sedation of camel perior to amputation of dulla



The dulla after amputation

Clinical Findings

Signs of pharyngitis include anorexia, weight loss, nasal discharge, coughing, fluctuant swelling behind the ramus of the mandible, extended head, pain response to palpation to the throat region, and hyperemia of the mucous membrane when examined with a laryngoscope or endoscope. Pharyngitis may lead to necrosis of 'DULLA' in the camels (Aggarwal and Nanda, 1930).

Treatment

Soft palatable feed should be given to the animal. If the camel is unable to drink water suitable quantities of saline glucose should be given intravenously. Parenteral antibiotics may also be given when indicated.

PHARYNGEAL OBSTRUCTION

Foreign bodies including bones, corncakes and pieces of wire and tissue swellings (lymphadenopathy, abscesses, actinobacillosis, neoplasms) are the usual causes of pharyngeal obstruction (Bhargava, 1973). Impaction of distensible part of the soft palate (Dulla) by feed, in excited males have also been reported to cause obstruction (Ramadan and Abdin Bey, 1987). A syndrome has been observed in camels in which there is mild fever, salivation, repeated swallowing movements and swollen mandibular, and parotid lymph glands. In this condition pharyngitis/tonsillitis has not been confirmed by endoscopy. The condition responds well to antibiotics plus cortisone treatment (Manefield and Tinson, 1996).

Clinical Findings

There is difficulty in swallowing and camels may be hungry enough to eat but, when they attempt to eat and swallow cannot do so and the food is coughed up through the mouth. Drinking is usually managed successfully. There is no dilatation of the esophagus and usually no regurgitation. There is difficulty in breathing. Manual examination of the pharynx may reveal the nature of the lesion but an examination with a fiber optic endoscope is likely to be much more informative.

Treatment

Removal of the foreign body may be accomplished through the mouth. Treatment of actinobacillary lymphadenitis with iodides is usually successful. Parenteral treatment of abscesses with antibiotics may effect a cure. Surgical treatment has been highly successful in cases caused by medial retropharyngeal abscesses.

PHARYNGEAL PARALYSIS

Pharyngeal paralysis is manifested by inability to swallow, absence of signs of pain, and respiratory obstruction.

Etiology

Pharyngeal paralysis occurs sporadically in peripheral nerve injury due to trauma of the throat region and in some encephalitides with central nervous system lesions (rabies, botulism etc.).

Clinical Findings

Inability to swallow and regurgitation are the major manifestation of the disease. The animal is usually hungry but, on prehension of food or water, attempts at swallowing are followed by dropping of the food from the mouth, coughing and the expulsion of food or its regurgitation through the nostrils. Salivation occurs constantly and swallowing cannot be stimulated by external compression of the pharynx. The signs depend upon the nerves involved and the degree of involvement. There is rapid loss of condition and dehydration. Clinical signs of the primary disease may be

evident but, in case of primary pharyngeal paralysis, there is no systemic reaction. There may be an associated laryngeal paralysis accompanied by roaring. Pneumonia may follow aspiration of food material into the lungs and produces loud gurgling sounds on auscultation. Complete pharyngeal paralysis is usually permanent and fatal.

Treatment

Treatment is unlikely to have any effect. Fomentation could be attempted. Intravenous infusions of saline glucose may be tried if disappearance of the paralysis seems probable.

ESOPHAGITIS

It is rare in camels.

Etiology

Primary esophagitis, may be caused by the ingestion of chemical or physical irritants. It is usually, accompanied by stomatitis and pharyngitis. Laceration of the mucosa by a foreign body. Passing of probe of endoscope or probang may cause esophagitis unaccompanied by lesions elsewhere. Inflammation of the esophagus occurs commonly in many specific diseases, particularly those which cause stomatitis, but the other clinical signs of these diseases over shadow those of the esophagitis.

Clinical Findings

In acute stages there is salivation and attempts of swallowing cause severe pain. In some cases, swallowing is impossible and attempts to do so are followed by regurgitation, coughing, retching movements, and vigorous contractions of the cervical and abdominal muscles. In case of the involvement of the cervical region, palpation in the jugular furrow causes pain and the swollen esophagus is palpable. Camels that recover from esophagitis are commonly affected by chronic esophegeal stenosis with distension above the stenosis. If perforation has occurred, there is local pain and swelling and often crepitus. Local cervical cellulitis may cause rupture to the exterior and development of an esophageal fistula, or infiltration along fascial planes with resulting compression obstruction of the esophagus, and toxemia. Perforation of the thoracic esophagus may lead to fatal pleuritis.

Treatment

Food should be withheld for 2-3 days and the camel may need to be fed intravenously during this period. Parenteral antibiotics should also be given. Refeeding of the animal should be started gradually by soft moist feed and the animal be monitored carefully.

ESOPHAGEAL OBSTRUCTION

Obstruction of the esophagus is less common in camels than in equines and bovines because of the ability of camels to regurgitate freely. The camels have more voluntary control of the esophagus than equines. However, choke has been observed in camels. Obstruction may be partial or complete. Any forgein body that becomes lodged in the esophagus will initiate muscular contractions with the purpose of moving the object one way or the other. If neither peristaltic nor antiperistaltic action is ineffective, the end result may be a spasm on either side of the obstruction, leading to partial or complete obstruction.

Etiology

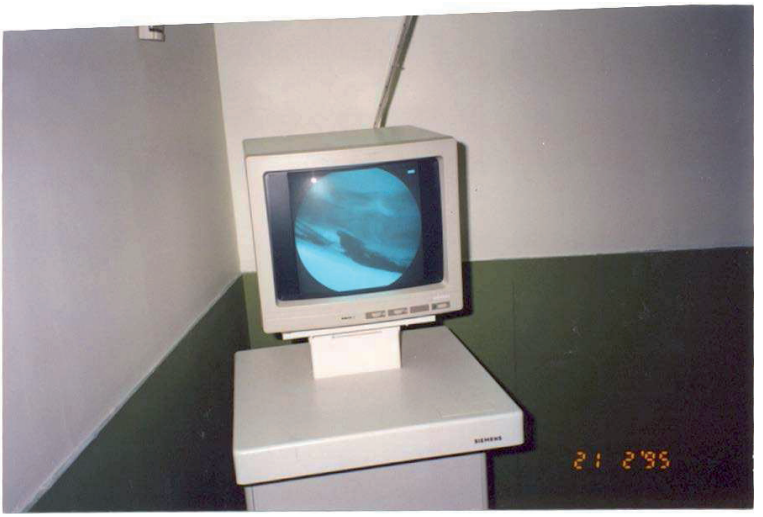
In camel, various feed items such as turnips, carrots, potatoes, rubber ball, cloth, plastic, rough dry feed etc. have become lodged in the esophagus. The problem usually aries when a greedy animal takes a mouthful of feed and attempts to swallow it without chewing. Obstruction can also result because of external pressure on the

esophagus by surrounding organ or tissue such as neoplastic lymph node in mediastinum or at the base of the lung.

Clinical Findings

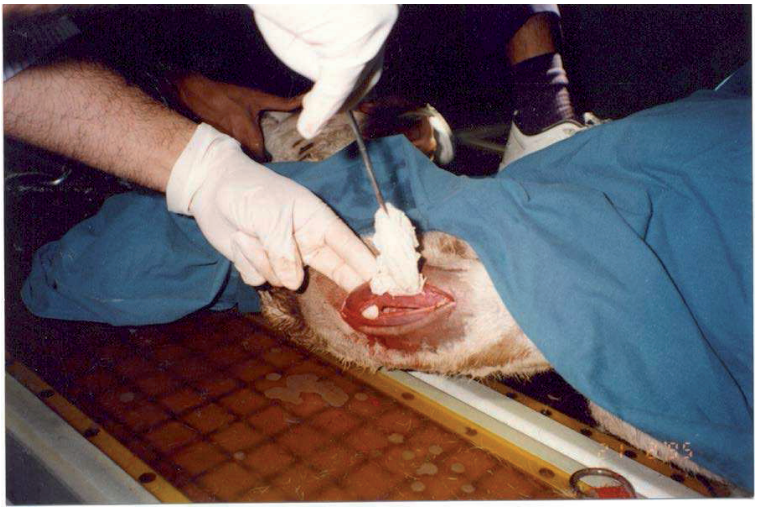
In the choke the behavior of the camel is altered. The animal feels discomfort. The signs could be confused with colic. There is retching, coughing, dysphagia, head shaking, salivation, and nasal discharge. If the animal keeps eating, food may be packed in the pharangyeal area and subsequent retching may drive feed particles into the nasopharynx and out through nose. Aspiration pneumonia may supervene.

The obstruction may lodge any where from the origin of esophagus to cardiac end of rumen. The common area of obstruction is the caudal cervical region where the first pair of ribs and the trachea restricts passage space (Manifield and Tinson, 1996). Obstruction in the cervical region can be seen as a swelling. Radiography or fluoroscopy may do diagnosis of obstruction.



Obstruction of plastic bag in the esophagus of a four month old camel calf confirmed by fluoroscopy

The acute signs, other than, bloat, usually disappear within a few hours. This happens due to relaxation of the initial esophageal spasm and may or may not be accompanied by onward passage of obstruction. Many obstructions pass on spontaneously but others may persist for several days. In these cases, there is inability to swallow, salivation and continued bloat. Persistent obstruction causes pressure necrosis of the mucosa and may cause perforation or subsequent stenosis of esophagus.



Removal of the plastic bag from the esophagus

Treatment

Spasmolytics (atropine sulfate 16-32 mg, hyosin compound) should be given to relax the esophageal musculature and hopefully, allow peristaltic activity to resume. Analgesics may be given. The passage of probang is usually necessary to locate obstruction low down in the esophagus. Gentle attempts may be made to push the obstruction onward by stomach tube but care must be taken to avoid damage to the esophageal mucosa. The tube should be well lubricated with liquid paraffin.

Solid obstruction in the upper esophagus may be reached by a long piece of wire bent into a loop, which may be passed over the object and an attempt made to pull it up into the pharynx.

Accumulation of particulate material in the lower part of the esophagus is more difficult to remove. Small quantities of warm saline with some extract of balladonna should be introduced through a stomach tube passed to the obstruction and then siphoned out. This should be repeated several times. In case of failure surgical removal of the obstructing object should be attempted.

The animal must not be given food and water until obstruction is removed.

ECTASIA

One case of ectasia has been reported in a 6-year old camel (Bolbol, 1992). The animal had a fusiform swelling in the upper third of the neck (at the level of 4--5th cervical vertebrae) seen on radiograph. It was believed that a previous episode of choke had damaged the nerve supply to the dilated paralysed section of the esophagus. The condition was tried to correct surgically, which failed due to excessive tissue damage.

DYSPHAGIA SYNDROME

Difficulty in swallowing can occur in affections of the throat and esophagus. A syndrome has been observed in camels in which there is mild fever, salivation, repeated swallowing movements, and swollen mandibular and parotid lymph nodes. Signs of pharangitis and tonsillitis are not observed. The condition responds well to antibiotics and corticosteroids (Manefield and Tinson, 1996).

DISEASES OF THE FORE STOMACHS

SIMPLE INDIGESTION

Simple indigestion occurs commonly in racing camels and is characterized by inappetence, anorexia, decreased ruminal movements and abnormal feces. The feces may be scanty or voluminous and diarrhetic.

Etiology

The disease is common in racing camels, because of variability in quality and quantity of food consumed. It is not common in grazing camels. The condition is caused by minor dietary abnormalities including dates, milk, succulent roughages like alfa alfa, moldy feeds, and moderate excess of grain and concentrate intake. Cases occur under excellent feeding regimens and are usually ascribed to overfeeding with grain.

Depraved appetite may also contribute to the ingestion of unusual feed material, which can lead to indigestion. It has also been observed that limitation of the available drinking water compel desert camels to come to rural/industrial areas where they can have access to kitchen thresh and unhygienic water. Forced feeding of racing camels can also be considered as a cause of indigestion.

Prolonged and heavy oral dosing with sulphonamides and antibiotics may cause indigestion due to inhibition of the normal ruminal flora and unusual feeding of a special diet to make the camel run fast. Changes in the pH of its contents markedly affects the motility of the rumen and in most of the cases an increase in the acidity is probably of importance. Atony which occurs after feeding on damaged feeds may have the same basis or be due to other unidentified agents in the food. The simple accumulation of indigestible food may physically impede ruminal activity. Putrefaction of protein may also play a part in the production of atony. The toxic amides and amines produced may include histamine which is known to cause ruminal atony.

Clinical Findings

A reduction in the appetite is the first sign and is followed by depression and dullness. Rumination ceases and the ruminal movements are depressed in frequency and amplitude and sometimes are absent. There may be moderate tympany. The feces are usually reduced in quantity and are drier than normal on the first day. However, 24-48 hours later the camel is commonly diarrhetic. The feces are softer than normal, voluminous and commonly smells very bad. Animal may show signs of colic. There may be vomiting as well. When vomiting, a camel will lower its head and neck and by increased intra abdominal pressure, with or without the assistance of ruminal contraction, eject ruminal contents through the esophagus. As the material reaches the mouth the head and neck are shaken and flicked up resulting in the ingesta being scattered in all directions (Manefield and Tinson, 1996).

In the indigestion of short duration there is no systemic reaction. However, if the condition persist the camel exhibits muscle weakness, twitching, incoordination, frequent falling, blindness and inability to rise.

Clinical Pathology

Examination of the urine for ketone bodies is usually necessary to differentiate indigestion from acetonemia.

Two simple laboratory tests have been introduced to assess the activity of ruminal flora. (a) Sediment activity test. It is carried out on aspirated ruminal fluid strained to remove coarse particles. The strained fluid is allowed to stand in a glass vessel at body temperature and the time required for floatation of the particulate

material recorded. The time in normal animals varies between 3 minutes, if the animal has just been fed and 9 minutes if the last feeding has occurred some time previously. Settling of the particulate material indicates gross inactivity, less severe degree being manifested by prolongation of the time required for floatation. (b) Cellulose digestion test depends upon the time required to digest a thread of cotton. A bead is tied to the end of the thread to indicate when separation occurs. Digestion time in excess of 30 hours indicate abnormality.

Simple indigestion must be differentiated from all of the diseases of forestomachs and abomasum in which ruminal atony is common clinical finding, and from diseases of other body systems which cause secondary ruminal atony (septicemia, toxemia, and acetonemia).

Treatment

For the most part treatment is symptomatic, consisting primarily of the use of rumentoric drugs. Parasympathetic stimulants are widely used as rumenatorics, but they have many side effects, their action is transitory, and the large doses depress rumen activity. Carbamylcholine chloride, physostigmine and neostigmine are most commonly used as rumentoric drugs. The latter is the most effective and should be given at a dose rate of 2.5mg/ 45 kg body weight. Epsom salt (0.5-1.0 kg per adult camel) is also helpful. Alkalizers, such as magnesium hydroxide at a rate of 400 gm per adult camel when the rumen contents are acidic is helpful. Patent antacids, given according to the directions of the manufacturer usually give good results. Care must be taken that the animal may not regurgitate the medicine. This can be avoided to hold head up in crouch position after medication.

In the case of prolonged anorexia reconstitution of the flora by the use of cud transfers is highly effective. If the camels show nervous signs injection of thiamine is very effective.

ACUTE CARBOHYDRATE ENGORGEMENT (ACIDOSIS)

An ingestion of large amounts of highly fermentable carbohydrate rich feed cause an acute disease due to the excessive production of lactic acid in the rumen. Clinically, the disease is characterized by severe dehydration, ruminal stasis, weakness, recumbency, and high mortality rate.

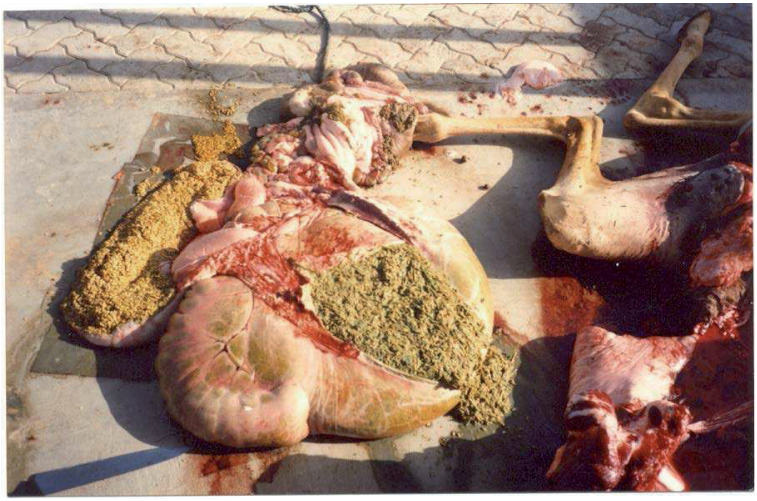
Etiology

The sudden ingestion of large quantities of carbohydrate rich feed, such as grain, dates, jaggery (Gur), bread, dough, sugar beet etc. is the main cause of this condition. The type and level of ration consumed by a ruminant affects the number and species of bacteria and protozoa in the rumen, when a change from one ration to the other occurs, a period of microbial adaptation is required which is variable. Animals which are on low carbohydrate level ration are thus considered to be most susceptible to a rapid change because satisfactory adaptation cannot occur quickly enough, but instead there is the rapid onset of abnormal fermentation.

There is marked increase in the number of Streptococcus bovis, which utilize the carbohydrate to produce large quantities of lactic acid. In the presence of a sufficient amount carbohydrate (a toxic or a lethal dose) the Streptococcus bovis will continue to produce lactic acid which decreases the rumen pH down to 5 or less, at which point the cellulolytic bacteria and protozoa are destroyed. The lactic acid in the rumen has been considered to be intermediate step in the formation of soluble sugars (Balch and Rowland, 1957; Baldwin et al., 1963; Hungate, 1966; Walker, 1968), and starch (Schulam and Valentino, 1976) which get, converted predominantly to propionic acid or acetic acid. Higher rate of lactic acid absorption in the blood leads to acidemia (Heuter et al., 1956). Lactic acid concentration from

0.7 - 11.37 mg/100 ml in the ruminal liquor before feeding is considered normal (Bhatia, 1983).

The concentration of volatile fatty acid (VFA) is also increased initially and contributes to the fall in ruminal pH. The low pH allows the lactobacilli to use the large quantities of carbohydrate in the rumen to produce excessive quantities of lactic acid, which markedly increase ruminal osmolarity and water is drawn in from the systemic circulation causing hemoconcentration and dehydration.

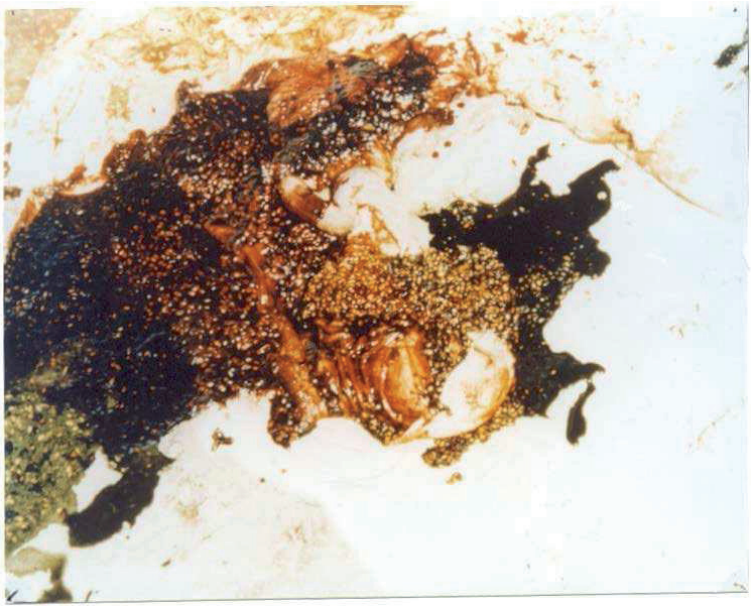


Severe ruminal acidosis after ingestion of large amount of barley

As the ruminal pH is lowered, the amplitude and frequency of ruminal movements are decreased and at about a pH of 5 there is complete ruminal stasis. There is evidence that the increased molar concentration of butyrate causes the ruminal stasis and not the lactic acid. The ruminal stasis could also be due to lactic acid entering the duodenum and exerting a reflex inhibitory action on the rumen (Blood and Radostits, 1989). The diarrhea is considered to be due to the reduction in net absorption of water from the colon.

The high concentration of lactic acid in the rumen is considered to be the cause of the chemical rumenitis, which later on is followed by mycotic rumenitis in those which survive, about 4-6 days later. The low pH of the rumen favors the growth of Mucor, Rhizopus and Absidia spp., which multiply and invade the rumen vessels, causing thrombosis and infarction. Widespread necrosis and gangrene may affect the entire ventral half of the ruminal walls and leads to the development of an acute peritonitis. The damage to the viscera causes complete atony and this, together with toxemia resulting from the gangrene, is usually sufficient to kill the animal.

Severe ruminal acidosis after ingestion of large amount of barley



Abomasum full of blood-mixed barley

In uncomplicated chemical rumenitis the ruminal mucosae will slough and heal with scar tissue and some mucosal degeneration. In this connection, the pathogenesis of hepatic abscesses is considered to be the result of a combination of factors. Rumenitis caused by lactic acidosis allows Fusobacterium necrophorum, Actinobacillus, and Corynebacterium pyogenes to enter directly into ruminal vessels and spread to the liver, which may have under gone injury from the lactic acidosis . Severe diffuse coagulative necrosis and hyperplasia of the bile duct epithelium and degeneration of renal tubules may also be present histologically (Nauriyal et al., 1978).

Several toxic substances other than lactic acid have been proposed as contributory to the disease. Increased concentration of histamine have been found in the rumen of experimentally engorged cattle. Histamine is not absorbed from the rumen except at abnormally high pH values, but is absorbed from intestines. Laminitis occurs in some cases of rumen over load. Other substances, which have been recovered from the rumen in grain over load, include a suspected endotoxin, ethanol and methanol (Blood and Radostits, 1989). Clostridium perfringens and coliform bacteria have also been found in increased numbers but their significance is not known. The electrolyte changes which occur include a mild hypocalcemia due to temporary malabsorption, loss of serum chloride due to sequestration in the rumen and an increase in serum phosphate due to renal failure.

Experimental disease has been produced in camels by feeding jaggery (a byproduct of sugarcane) and the changes similar to the natural disease were observed (Lal, et al., 1994). Changes in the cellular and biochemical composition of the cerebrospinal fluid suggests that blood-brain barrier may be affected.

Clinical Findings

The clinical symptoms observed in camels are almost similar as in other ruminants except evening body temperature is elevated (Lal et al., 1994). The other manifestations observed in camel are dullness, lack of rumination, anorexia and reduced water intake. The passages of loose semi-solid feces dark brown in color may follow by profuse diarrhea and dehydration. Kernels of grain may be seen in feces. Regurgitation (vomiting), profuse salivation and lacrimation, colic, grinding of teeth, and ruminal stasis are signs of acute disease. Extreme depression, and increased respiration (10-12 per minute) and pulse rate (70-90/min) are also observed. Oliguria, incoordination, muscular tremors, ataxia with reduced palpebral reflexes, opisthotonous, recumbency, convulsions, coma and death may ensue. The onset and severity of symptoms depends upon the kind and amount of feed consumed.

The pH of the ruminal fluid obtained by stomach tube passed through mouth can be measured in the field using wide-range pH indicator paper. The test should be performed immediately because the pH will increase upon exposure to air. The microscopic examination of a few drops of ruminal fluid on a glass slide (with a cover slip) at a low power will reveal the absence of ruminal protozoa, which is a reliable indication of an abnormal state of the rumen, usually acidosis. The predominantly gram-negative bacteria of the rumen are replaced by gram-positive ones.

Treatment

The attempt should be made to correct the ruminal acidosis and prevent further production of lactic acid, restore fluid and electrolytes and restore forestomach and intestinal motility. The camel should be prevented to further access to feed responsible for acidosis, and provided no water for 12-24 hours. The animal should be offered good quality hay one half of the daily requirement. The animal should be exercised every hour for 12-24 hours. After 18-24 hours the camels which have continued eating hay may be allowed free access to water.

In severe cases, a rumenotomy is the best course of action. The rumen is emptied, washed out with a siphon, examined for evidence of and the extent of chemical ruminitis and a cud transfer (about 10 liters of rumen juice) is placed in the rumen along with a few handful of hay. The systemic acidosis can be treated with intravenous solution of 5 per cent sodium bicarbonate (at the rate of 5 liters for a 450 kg animal given initially over a period of about 30 minutes). This should be followed by isotonic sodium bicarbonate (1.3%) at 150 ml /kg body weight given over the next 6-12 hours.

In less severe cases, an alternative to rumenotomy is rumen lavage. A long rubber tube having inside diameter of about 20 mm is passed through mouth into the rumen and warm water is pumped in, and then the rumen is allowed to empty by gravity flow. The rumen may be almost completely emptied by 10-15 irrigations.

In moderately affected cases 500 g of magnesium hydroxide or magnesium oxide per 450 kg body in 10 liters of warm water is given by mouth. Simultaneously systemic acidosis should be treated by injecting Sodium bicarbonate solution, intravenously.

Ancilliary treatment includes use of antihistamines, corticosteroids, thiamine, and parasympathomimetics. Calcium borogluconate is also valuable.

OBSTRUCTION OF RUMEN

It is very frequent to see foreign bodies in the rumen of camels in abattoirs and at necropsies (Fahmy et al., 1995). The racing camels are always muzzled to prevent them from eating sand and feces etc., but the scavenger camels eat many odd things like sand, gravel, garbage, rubber, ropes, plastic, plant fiber balls (Phytobezoar), hair balls (Trichobezoars), cloth etc. Depending upon the size and physical nature of the object they affect the ruminal function. These objects can cause intermittent or permanent obstruction of the lumen of the rumen. In camels the ruminal movements are such that almost all foreign bodies remain in the rumen and generally don't migrate to reticulum as is observed in cattle.

Clinical Findings

Clinical findings include persistent poor appetite, pica, and depressed ruminal activity (normally 3-4 sounds of varying intensity are heard per minute). Intermittent bouts of constipation are observed. Colic is also some times observed. The clinician should exclude the possibilities of other chronic illness through laboratory investigation. Many digestive tract disorders e.g., impaction of C-3, ulceration of C-3, impaction of the intestines, tortion of the intestines, and uterine tortion (Ramadan et al., 1986) produce similar signs.

Treatment

Lubricants such as liquid paraffin and purgatives such as magnesium sulfate can help. The surgical removal by rumenotomy is the ideal treatment.

RUMINAL TYMPANY

Ruminal tympany is the over distension of rumen and reticulum with the gases of fermentation, either in the form of persistent foam mixed with the rumen contents (primary tympany or frothy bloat) or in the form of free gas separated from the ingesta (secondary ruminal tympany).

Primary Ruminal Tympany or Frothy Bloat

In the primary ruminal tympany or frothy bloat there is production of stable foam in which the gases of fermentation are entrapped. There is inability of small gas bubbles to coalesce and hence the animal is not able to eructate. The bloat results due to foaming qualities of the soluble leaf proteins in legumes and some

forage crops. Feeding on very succulent rapidly growing legumes is the most common cause of bloat. The disease can also occur by feeding young cereal crop, cabbages, and leguminous vegetable crops including peas, beans and grains having high protein. Feeding of finely ground grain also promotes frothiness of ruminal contents.

Bloat-causing legumes are more rapidly digested by rumen microorganisms than non-bloat-causing forages and the rupture of leaf mesophyll cells leads to the release of chloroplast particles. Rumen microorganisms utilize these particles and gas bubbles are trapped among the particles, which prevent coalescence of bubbles by preventing drainage of rumen fluid from the liquid lamellae between the bubbles. Finely ground grains act as medium for certain bacteria, which produce insoluble slime. The resulting slime entraps the gases of fermentation and bloat ensues. Variation in the susceptibility to bloat has been observed in individual camels.

Secondary Ruminal Tympany

Secondary ruminal tympany or free-gas bloat occurs due to physical obstruction to the eructation of gases of fermentation through esophagus. The obstruction may be due to foreign body, stenosis or pressure exerted from outside on the esophagus by enlarged lymph nodes or some other lesion. Lesion of the vagus nerve may depress the gas eructation centre thus interrupting the reflex responsible for removal of gases from the rumen. Normal tone and motility of the musculature of the rumen and reticulum are also necessary for eructation. In anaphylaxis, bloat occurs due to atony of the muscles of the forestomach, which can be corrected by antihistamines and adrenaline. A sudden marked change in pH (either alkalinity or acidity) can also cause atony.

Cinical Findings

The animal will commonly begin to bloat within one hour after eating bloat-producing feed. The animals may bloat on the first day of feeding but more commonly they bloat on the second and third days of feeding. In bloat, obvious distension of the abdomen occurs suddenly, sometimes as soon as 15 minutes after going on bloat producing forage. The whole abdomen becomes enlarged. The animal feels discomfort frequently lies down and gets up, kicks at the belly or even rolls. Dyspnea, accompanied by mouth breathing, salivation, and protrusion of tongue is also seen. Vomiting and passage of liquid feces is also frequently observed.

Treatment

The source of bloat producing forage should be abandoned. In very severe cases rumenotomy is recommended. In less severe cases it is advised to tie a stick in the mouth of the camel for the production of excessive amount of saliva, which is alkaline and may assist in destabling foam. Drenching of non-toxic oil especially a mineral one, not biodegradable, about 250 ml also helps. Passage of stomach tube through mouth may also help to release free gases in the rumen. Oil containing a detergent such as dioctyle sodium sulfosuccinate is preferred. Synthetic surfactant ‘poloxalene’ is very effective at dose rate of 25.5 g (leguminous bloat). Ethoxylates are also very effective.

ABOMASITIS

Abomasitis is always observed in hemorrhagic disease in camels. It is also seen in enterotoxemia, endotoxemia, and heavy parasitic infestation. Ideal treatment is the use of antibiotics.

ABOMASAL OBSTRUCTION

Feeding large quantities of unseeded dates is the most common cause of impaction in the pyloric area. It can also be caused by ingestion of gravel, pieces of cloths, plastics, etc., and granulomatous lesion. Abomasal obstruction is usually of acute nature. The clinical signs are anorexia, abdominal distension particularly of right flank, discomfort, inappetence, reduced output with reduction in pellet size. Vomiting may occur after drinking water. Leukocytosis with neutrophilia and hypokalemia is also observed. Some times rupture of the abomasum leads to perforation and consequent peritonitis.

Some cases may respond to laxative treatment. Surgical removal of obstruction may be attempted through a right paracostal incision under general anaesthesia (Manefield and Tinson, 1996)

ABOMASAL ULCERS

Any of the stomach compartments may be affected, but they are relatively common in the abomasum.

Etiology

The ulcers are seen in many specific diseases (such as clostridial infections and hemorrhagic disease of camels), and are also observed in many local infections of gastro-intestinal tract. They can also occur as primary ulcers. In this case the etiology of the ulcer is not known in camels as seen in horses. The role of stress in the causation of ulcers is unknown. The condition is observed in camels on good, as well as, on poor diet. Some had been infested with parasites while some were not.

Clinical Findings

Primary gastric ulcers are not usually diagnosed in the living camel. As the condition is seldom diagnosed in the living camel no clinical signs have been recorded, however, abdominal pain and anemia can be anticipated. Perforating ulcers results in peritonits.

Colic – rolling on the ground, common finding in young and old camels

Treatment

No studies have been done about the response of camels to medicines used for the treatment of ulcers. The camel might respond to cimetidine therapy (2.2 mg/kg) given twice daily subcutaneously. The drug should not be given orally as it is degraded in the rumen.

Stretching of the hind leg in colic

DISEASES OF THE INTESTINES

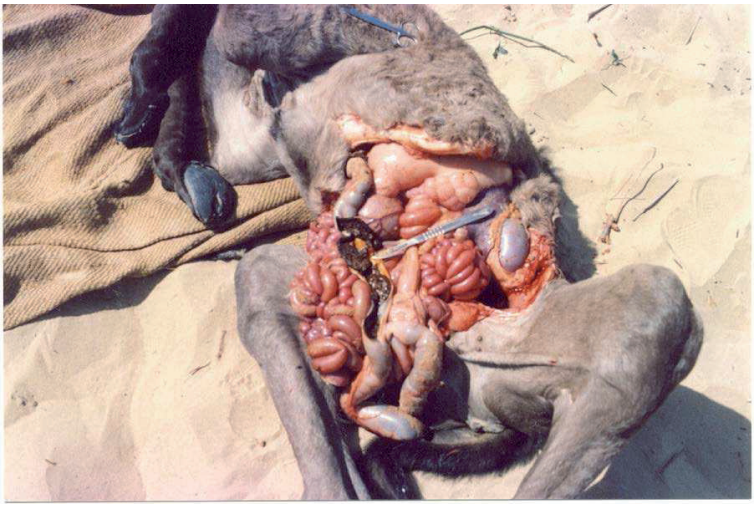
INTESTINAL OBSTRUCTIONS

Either partial or complete obstruction of the intestinal lumen is a frequently seen clinical condition (Manefield and Tinson, 1996). While all the possible causes of obstruction have not been observed in camels, it would be reasonable to assume that any of the types seen in the ruminants may appear in camels. According to Tanwar (1985), Tayal et al. (1985) and Singh et al. (1993) specific causes include impaction (sand, enteroliths, fecoliths), torsion, intussusceptions, compression from the gravid uterus, strangulation from hernias, neoplasia, abscessation, infarction and congenital defects (atresia coli and atresia ani).

Clinical Findings

The signs of blockage vary with the location and completness of obstruction, how rapidly the obstruction developed, the degree of enterotoxemia produced, the extent of vascular occlusion, and production of gas. The major sign is colic, with little or no passage of feces. When an obstruction interferes with circulation, the ensuing events alter the integrity of the bowel. In partial vascular compression, venous drainage will be adversely affected. The continued arterial flow results in edema. If compression is complete, there will be tissue anoxia and increased capillary permeability followed by intramural and mucosal hemorrhage with accompanying edema and breach in the continuity of the epithelium which allows bacterial invasion, which follows ultimately to peritonitis and bacteremia (Jones and Hunt, 1983). Gangrene and shock may ensue.

Hair and sand in the form of semi-solid balls of fecalets. Common cause of death in young camel calves



Blockage of intestines due to sand-mixed fecalets

Treatment

Laxatives and purgatives should be tried to dislodge the impacting body. In case of failure, one has to resort to surgical removal of the obstruction.

ENTERITIS

It is the inflammation of intestines.

Etiology

There are many causes of enteritis in camels and the disease varies considerably depending upon the causative agent. There are many predisposing and exciting causes pertaining to animal and environment which influence the primary causative agent in causing enteritis. The new born camel calves, which are deficient in immunoglobulines are much more susceptible to enteritis and have high mortality rate due to diarrhea, than animals having adequate levels of immunoglobulins. Enteric salmonellosis is commonly precipitated by the stresses of transportation or deprivations of feed, and permit the development of a super infection by organisms, which would normally cause disease.

The causative factors of enteritis in camels includes, bacteria (enterotoxigenic E.coli, Salmonella spp., Clostridium perfringens type B, and C., and Mycobacterium), fungi (Candida spp.), viruses, helminths, protozoa (Eimeria spp., Blantidium coli, Cryptosporidium), chemical agents (Arsenic, fluorine, copper, sodium chloride, mercury, molybdenum, nitrates), poisonous plants, mycotoxicosis, physical agents (Sand or soil; silage or feed containing lactic acid), nutritional deficiencies (copper deficiency), dietary over feeding (simple indigestion), and miscellaneous conditions (congestive heart failure, toxemia).

Hemorrhagic enteritis

Clinical Findings

The major clinical findings in enteritis are malabsorption and diarrhea. Depending upon the causative agent, stage of disease, and age of animal, varying

degree of clinical signs are seen. These include dehydration, abdominal pain, septicemia or toxemia with fever. In acute enteritis, the feces are soft or fluid. There may be blood, fibrin or mucous or foreign material in the feces. The feces may have bad smell. The color of feces vary from pale yellow to dark brown, or blackish. Other signs of enteritis are tenesmus, anorexia, colic, depression, and intestinal borborygmus. Clinical signs should determine diagnosis. An etiological diagnosis will require cultures, examination for cytology and parasitic ova, and hematology and blood chemistry evaluation. Colonic biopsies may be of diagnostic aid. Elecrolyte imbalance is a common sequel of diarrhea.

Treatment

In the management of enteritis, it is important to monitor and correct any fluid and/or electrolyte imbalance. It is particularly important to provide glucose, sodium, and chloride. Acidosis commonly develops in enteritis, so sodium bicarbonate is necessary. Intestinal protectants like, kaolin, pectin, bismuth subsilicate may be used. Broad-spectrum antibiotic therapy should always be given in bacterial and viral enteritis. If the enteritis is parasitic, the therapy should be devised accordingly.

INTESTINAL ULCERATION

Ulceration of intestinal mucosa may follow any severe enteritis and develop anywhere in the gastrointestinal tract. The most common site is the small colon within the spiral colon. In many cases no indication of the cause can be determined, even at necropsy.

Following intestinal spasm around an impaction of dried material, ischemia develops in the intestinal wall. This, in many cases, leads to ulcer formation. Multiple ulcers may develop in such a way. Ulcers may result in perforation of the intestinal wall. It is very difficult to diagnose an ulcer. Elimination of other intestinal disorders should precede the tentative diagnosis.

RECTAL PROLAPSE

It is not uncommon in camels, but has been reported. The most common cause is tenesmus, associated with prolonged severe diarrhea. A mild protrusion, lasting for few minutes, may be observed following defecation. A slight protrusion of rectum along with vaginal /vulvular mucosa has been noted in pregnant female camels, near term, especially while sitting. The protruded mucosae may become traumatized. Edema is likely to develop followed by necrosis.

The primary cause should be corrected. The protruded mass should be cleaned, replaced by gentle pressure. The edema can be reduced by local application of glucose or sucrose. Epidural anaesthesia may be necessary to allow replacement in straining animals. A rectal tube inserted into the rectum and secured by sutures may help. If the protruding segment is necrotic, it may be amputated.

NERVOUS DIARRHEA

Camel feces are voided as dry pellets. Any act that causes the animal stress will result in the passage of soft feces within five minutes. First time loading of a camel on to a truck, putting them in stocks for examination, or treatment, all can cause nervous diarrhea. Many camels when get accustomed to the procedures lose this reaction.

Attempts to take advantage of the reaction are made in camel racing circles. In a procedure known as “Taffiz” camels are taken to the race track and deliberately stressed by noise and threatening aggressive human behavior. This results in diarrhea causing reduction in gut load considerably during the ensuing 30 minutes. It is thought that it reduces the camel’s weight and produces a better power to weight ratio (Manefield and Tinson, 1996).

PERITONITIS

Peritonitis, inflammation of peritoneum, is usually accompanied by pain, tenderness on palpation, rigidity of abdominal wall, inability to pass feces and fever, followed by toxemia. It may be diffuse or focal. Adhesions are a natural sequelae to an inflammed serosa.

Etiology

Peritonitis may occur as primary disease or secondary to some other ailments. As a primary disease it results from rupture of uterus, bladder, gastrointestinal tract, perforation of the abdominal wall from exterior and rupture of abscesses. Peritonitis is seen as a sequel to date seed perfotation of the abomasum, bowel necrosis subsequent to severe impaction and can also be sequel to surgical procedures (Manefield and Tnson, 1996). Septic peritonitis caused by Streptococcus zooepidermicus has been reported in camels by Heller et al. (1998). Traumatic reticuloperitonitis due to piercing by foreign bodies present in the reticulum has been reported by Patel and Suthar (1992).

Clinical Findings

Clinical signs vary depending upon whether the peritonitis is local or diffuse and severity of the condition. In acute diffuse peritonitis signs of toxemia and shock are evident. In chronic peritonitis, formation of adhesions is more important than toxemia and shock. If the healing process is developing satisfactorily and the signs of peritonitis are diminishing, it is a common experience to find that vigorous exercise may cause breakdown of adhesions, spread of peritonitis and return of clinical signs of acute peritonitis.

Accumulation of large quantities of inflammatory exudate in the peritoneal cavity may cause visible abdominal distension and interference with respiration due to pressure on diaphragm. Acute local and chronic peritonitis is usually not fatal.

Treatment

Antibiotics and sulphonamids should be given for bacterial infections (oral, parenteral or intraperitoneal). Analgesics are advised to relieve pain of the animal.

ANAL STENOSIS

It has been occasionally seen as congenital condition. Often repeated injury and cicatrization cause it. The anus becomes frequently injured while correcting prolapse, per rectum examination, perianal abscessation and heavy perianal tick infestation. For treatment one has to go for surgical correction.

HEPATIC INSUFFICIENCY

The liver has a very large reserve of function and approximately three-quarters of its paranchyma must be rendered inactive before clinical signs of hepatic dysfunction appear. Diffuse diseases of liver are more commonly accompanied by signs of insufficiency than are focal diseases, which produce their effect either by the toxins formed in the lesions or by pressure on other organs. Diffuse diseases of the liver can be classified as hepatitis and hepatosis according to the pathological changes, which occur and the classification also corresponds roughly with the type of causative agent. Clinically the difference between the two diseases is not marked.

There are no specific modes of hepatic dysfunction. The liver has many functions and any diffuse disease of the organ interferes with most or all of the functions to the same degree. The major hepatic functions which, when disordered,

are responsible for clinical signs. These include: The maintenance of normal blood sugar levels by providing the source as glycogen; the formation of some of the plasma proteins; the formation and excretion of bile salts; the excretion of bile pigments; the formation of prothrombin; and the detoxification and excretion of many toxic substances including, photodynamic agents.

Clinical Findings

Signs in this condition include anorexia, colic (which aggravates on palpation or percussion), tense abdomen, atony of gastrointestinal tract, weakness, may be fever, diarrhea, painful movements, disinclination to sit followed by recumbency, and deaths in 4-48 hours. The signs observed are:
Jaundice, nervous Signs (Hyperexcitability, convulsions, coma, muscle tremor, and weakness, psychic disturbances including dullness, compulsive walking, head pressing, and failure to respond to signals, and sometimes mania), Edema and emaciation (due to protein deficiency), Photosensitization (Phylloerythrin, the normal breakdown product of chlorophyll in the gut are excreted in the bile. In hepatic insufficiency excretion of these substances is retarded), Hemorrhagic diathesis (deficiency of prothrombin occurs), and abdominal pain (distension of capsule and lesions of the capsule. Alteration in the size of liver). Some times there is displacement of liver or rupture of liver.

Distinctive clinical signs, plus evaluation of clinical pathology parameters like sorbital dehydrogenase, aspartate aminotransferase, alanine aminotransferase, gamma glutamyl transferase, glutamate dehydrogenase help in the diagnosis of liver insufficiency.

Treatment

The prognosis for acute hepatic necrosis is unfavorable. In mild cases, supply of glucose and lipotropic substances such as choline and methionine and broad spectrum antibiotics to check secondary bacterial infection are indicated.

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RESPIRATORY SYSTEM

The camel is an obligatory nose breather like that of equines. It can dilate and constrict the nostrils. The resting camel closes its nostrils during a sand storm, but normally it does not do so. An attempt to inspire through the mouth at exercise is a sign of nasal or nasopharyngeal obstruction. The respiration rate of camel varies from 5-12 / minute (Higgins and Kock, 1984). The nasal turbinates of the camel are more complex than that of the equines, because the camel has to humidify air while inspiration and to conserve water on expiration (Schmidt-Neilsen et al., 1981). The ventral meatus is large enough in an adult camel to permit endoscopy by an instrument having external diameter of 13 mm.

The pharynx of the camel is extraordinarily long and relatively narrow. The camel has a prominent transverse fold or palatine diverticulum (DULLA) on the oral surface of its soft palate. In the male camel this is highly developed as an accessory sex organ. This structure can be inflated from within and is seen projected from the buccal cavity of male camels during breeding season. Even when the bull camel is not in rut, its soft palate is very fleshy and may impede respiration at fast exercise. Surgical resection of the DULLA might enable male camels to compete more effectively. The structure consists of `loose connective tissue and mucous glands covered by mucosa, which is sometimes pigmented. The "DULLA" gets inflated because of the gas eructed from the stomach is forced into the lungs, from where air is forced into the oral pharynx while the soft palate is raised and the entrance of the nasopharynx is closed.

Like oropharynx, the nasopharynx is also divided into cranial and caudal dome shaped compartments, by a transverse fold. The opening of the auditory tube is in the cranial compartment. The nasopharynx lies dorsally to the orthopharynx and stretches from the choanae to the intrapharyngeal opening. The respiratory position of the soft palate in the camel is ventral to the epiglottis (Cook, 1965), permitting the ostium intrapharyngium to create an air tight seal around the larynx (Cook, 1981). During exercise it is important that no air should enter in the orthopharynx. Dorsal displacement of the soft palate above the epiglottis is a cause of airway obstruction. The factors most likely to trigger this displacement are pharyngospasm caused by nasal myiasis, hyperplasia of the soft palate, and regurgitation of rumen contents. Caudoventrally, the nasopharynx communicates with the oro- and laryngopharynx through the intrapharyngeal opening. The Pars laryngea pharyngis is the caudal continuation of the orthopharynx and extends from piriform recess at the base of the epiglottis to the level of the cricoid cartilage. It contains the rostral part of the larynx and the Vestibulum oesophagi, which is marked off from the esophagus proper by a V-shaped fold of mucous membrane, which represents the attachment of the palatopharyngeal arches.

The camel has a large esophageal vestibule and, apparently, a rather slack crico-pharyngeal sphincter. The readiness with which every excited camel regurgitates rumen contents suggests the excitement of racing might precipitate

regurgitation in the camel during exercise. The sudden appearance of food material in the nasopharynx at fast exercise would certainly be a potent cause of pharyngospasm, dorsal displacement of the soft palate and exercise induced dyspnea. Special consideration might be given by trainers to reduce the bulk of rumen contents in the days prior to racing. A reduction in the amount of fluid and solid material in the rumen would reduce body weight, facilitate movement of the diaphragm, lessen the work of inspiration and render respiration more efficient.

The long larynx of the camel is more caudally placed than that of the horse and, although attached to the thyroid cornu of the thyroid apparatus, is much more mobile. The camel appears, like the horse, dog and man, to have a thoraco-laryngeal reflex (Cook and Thalhammer, 1991). The cartilages of the larynx, viz. the epiglottis rostrally, the thyroid cartilage ventrally and laterally, the paired arytenoid cartilages dorsally, and the cricoid cartilage caudally.

The thyroid cartilage resembles that of the horse and possesses well-developed rostral and caudal cornua. The rostral cornu articulates with the free end of the thyroid bone, while the caudal cornu articulates with the cricoid cartilage. There is a thyroid foramen in the cartilage of some specimens, while in others a deep notch is bridged by fibrous connective tissue, for the passage of the cranial laryngeal nerve.

The arytenoid cartilage resembles an anchor. There is a rod-like connection between the apex with its extensive corniculate process, and the base, which contains the muscular and vocal processes. There is a clear demarcation where the yellow elastic cartilage of the rostral part abruptly changes to hyaline cartilage rostrally to the base. The cricoid cartilage is sturdy and relatively long. The epiglottis is approximately 85 mm long in the adult. It has no special features.

The prominent transverse processes of the cervical vertebrae form a gutter which, to some extent, protects the cervical trachea from external trauma, e.g. from the asphyxiating bites of the natural predators. The long neck of the camel means that the cervical trachea and whole of the upper airway is also long. In the cranial mediastinum trachea lies ventrally to the esophagus. In this region the left end of the tracheal rings overlap the right ends. Directly, cranially to the bifurcation of the trachea the tracheobronchus to the cranial lobe of the right lung arises.

The lungs of the camel resemble to those of equines, rather than ruminants. The fissures are not present in the camel lungs. Each lung possesses a cranial and a caudal lobe, and the right lung has an accessory lobe in addition. On closer examination of the surface of the lungs small lobules separated by connective tissue are visible. Abdurahman (1987) has discussed pathology of the camel lungs.

RHINITIS

It is inflammation of nasal mucosa and is of minor importance, as a disease process, except in severe cases when it causes obstruction of the passage of air through the nasal cavities, especially in racing camels. Its major importance is as an indication of the presence of specific disease.

Etiology

Bacteria, viruses, Oestrus ovis larvae, allergy, and fungi cause it. Antibodies to adenoviruses, parainfluenza, bovine syncytical virus and infectious bovine rhinotracheitis have been found in camels (Manefield and Tinson, 1996). Nwasu and Wachy (1998) have reported 97.4 % prevalence of nasal myiasis

(Cephalopina titillator), camel nasal bot fly, in Nigeria. Allergic rhinitis has also been reported in camels (Pal, 1976).

Clinical Findings

There is nasal discharge in rhinitis, which is serous initially then becomes mucous and in bacterial infection it becomes purulent. Redness, erosions, or ulcers may be present on the mucous membrane. Sneezing or snoring may be present. A chronic unilateral purulent discharge from the nostrils which lasts for several weeks is indicative of mycosis.

Treatment

The treatment should be aimed to control the causative agent. The nostrils should be irrigated with saline. A nasal decongestant may be sprayed into the nostrils. The allergic rhinitis should be treated with anti allergic drugs and the causative agent should be identified and eliminated. Antibiotics and sulpha drugs are recommended for bacterial rhinitis.

NARROWING OF NASAL PASSAGES

Lesions within the nasopharynx such as polyps, lymphoid hyperplasia or granulomatous conditions may cause restricted airflow. This can adversely affect the performance of racing camels. Rathore (1986) has reported a condition known, locally, as Paladi, from India. In this condition the septal cartilage of the nose enlarges in size and the camel feels difficulty in breathing. Surgical intervention is usually required to correct the condition (Fowler, 1991).

LARYNGITIS, TRACHEITIS, BRONCHITIS

All the agents responsible for causing pneumonia and rhinitis can cause these three conditions (Vitovec and Vladik, 1983).

Coughing and inspiratory dyspnea are the common clinical signs. In the initial stages the cough is dry and painful and is easily induced by grasping trachea or larynx, or to exposure to cold, dust, smoke etc. Later the cough becomes less painful and productive, thick mucous with blood and fibrin may be coughed up. Fever and toxemia may accompany and the affected animal cannot eat or drink. The dyspnea varies with the degree of obstruction. The inspiration phase is usually prolonged and forceful. Swelling of the larynx may cause deaths from asphyxia.

Treatment

Treatment should be attempted with antibiotics and sulphonamides. Animals showing signs of dyspnea should be operated upon for tracheotomy. Corticosteroides may be used to reduce tracheal edema. Surgical excision of chronic granulamatous lesion or abscesses is indicated followed by long term antibacterial therapy.

PULMONARY CONGESTION AND EDEMA

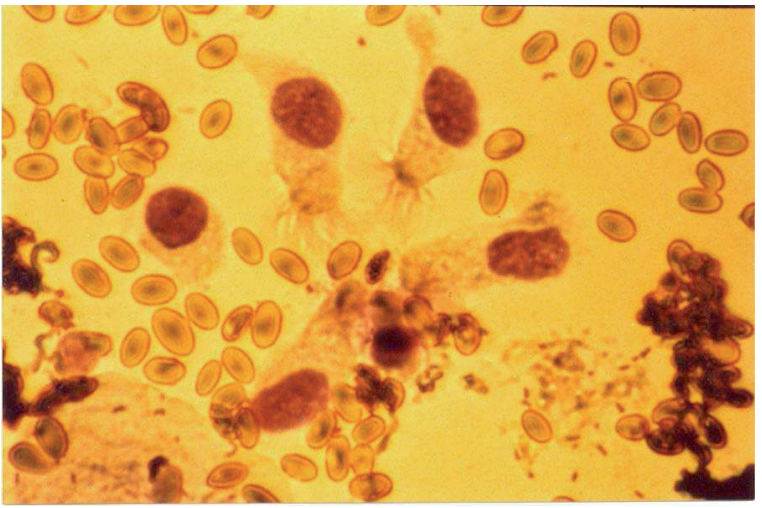
Pulmonary congestion (engorgement of the pulmonary vascular bed), sometimes followed by pulmonary edema, is characterized by respiratory embarrassment. The degree of respiratory embarrassment depends upon the amount of alveolar air space lost.

Etiology

Primary congestion occurs in the early stages of all types of pneumonias by inhalation of smoke and irritant fumes, anaphylactic reaction, and in recumbent animals due to hypostasis.

Secondary congestion is observed in congestive heart failure and after severe exertion in camels, as is found in horses.

Pulmonary edema occurs as a sequel to pulmonary congestion in case of acute anaphylaxis, acute interstitial pneumonia, congestive heart failure, inhalation of smoke and irritant gases, poisoning with organophosphates and viral diseases of lungs.



Ciliated epithelial cells-tracheal wash of one of the EIPH-positive cases

Clinical Findings

There is dyspnea with head extended, nostrils flared and mouth breathing. There are marked abdominal movements seen on respiration. The rate of respiration is increased especially if there is hyperthermia, acute anaphylaxis, and early stages of pneumonia. Pulse rate is also elevated (up to 100/min). The nasal mucosa is either bright red or cyanotic. In acute congestion harsh lung sounds can be heard on auscultation. With the development of edema increased lung sounds and crackles are heard, particularly in the ventral parts of the lungs. In long standing cases emphysema develops at the dorsal parts and the crackles and wheezes are heard at the dorsal parts, especially is cases developed by allergy. On percussion, normal sounds are heard in congestion and dull sounds in edema.

There is coughing, which is soft and moist. Slight to moderate serous nasal discharge appears in congestion, which changes to frothy and voluminous in edema.

The diagnosis of pulmonary edema and congestion is always difficult unless there is history of a precipitating cause such as inhalation of smoke or over

exertion. Pneumonia could be an alternative diagnosis, which is usually accompanied by hyperthermia.

Treatment

Correction of the primary cause should be attempted first. Affected animal should take rest. Adrenaline is recommended in pulmonary edema due to anaphylaxis, which may be followed by corticosteroids. Antihistamines are also good and may be given along with adrenaline. Oxygen therapy may effectively reduce the anoxia. The slow intravenous aminophylline will help dilating bronchioles. Removal of the fluid from the lungs may be assisted by I/V treatment with furosemide or other quick acting diuretic. Reduction of foaming in the airways may be achieved with nebulized 20% ethanol. Morphine is also recommended.

In the case of edema due to organophosphates, atropine should be given promptly (repeated injections are advised).

PULMONARY HEMORRHAGE

The camels are not only kept for milk or meat but also play an important role in the traditional sport of racing. Standard racing distances are 8-10 km. Some camels show some blood coming out of the nostrils after racing or galloping and subsequently those camels do not perform well in races. A study conducted by Akber et al. (1994) revealed that these camels were having hemorrhages of varying sizes in the lungs. The studies were made by fiber optic endoscope (2 m length; 11 mm ext. diameter) passed through nostril. The condition was more common in 8-10 year than 3-4 year old camels. The incidence of the condition is reported to be 30 per cent, which is similar to that reported in racing horses (Pascoe et. al., 1981). The mechanism of hemorrhage is not known. The presence of pre existing lung disease might be a predisposing factor. Usually the hemorrhage is not large enough to cause any apparent clinical signs, except epistaxis in some cases, but these camels do not perform well in the subsequent races. A variety of drugs have been used for the treatment but non has any significant effect. The use of furosemide might help reducing the severity of condition.

PNEUMONIA

Viruses, bacteria, fungi, protozoa, metazoan parasites, and physical and chemical agents may cause pneumonia. Sometimes a combination of two, or more, factors are responsible for the condition. Mostly the pneumonia in camel is bronchogenic, however, it can be hematogenous in some cases.

The opportunistic microorganisms are responsible for pneumonia in majority of the cases when the resistance of the animal is lowered (Farrag et al., 1953). These include E.coli, streptococci, staphylococci, Pasteurella spps, Corynebacterium spps. (Shah and Khan, 1935; Rana et al., 1993), Pseudomonas (Wernery et al, 1997), Kiebsiella pneumoniae (Arora and Kalra, 1973), Mycobacterium (Farrag and Zaki, 1953), and aspergillosis (Bhatia et al., 1983).

Clinical Findings

In the early stages, respiration is rapid and shallow followed by dyspnea in the later stages. Cough is an important sign. There is more painful cough in broncho-pneumonia. Frequently, dry hacking cough is observed in interstitial pneumonia, which is in paroxysms. On auscultation exudate can be detected. Percussion and auscultation can detect consolidation.

Toxemia, anorexia, fever, depression, tachycardia and disinclination to sitting is observed in acute pneumonia.

Treatment

Antimicrobial agents, preferably in long acting base should be injected. Bronchio-dilators such as clenbuterol and nonsteroidal anti-inflammatory drugs should be administered as an adjuvant therapy.

ASPIRATION PNEUMONIA

Aspiration pneumonia or drenching pneumonia is a common disease of farm animals, however, in camels it is not so common. The condition occurs by careless or forced drenching of medicines for the treatment of some ailment. Vomitus some times is also inhaled. Rupture of some pharyngeal abscess and aspiration of pus is also a potential cause of aspiration pneumonia. The animal suffering from paralysis or obstruction of the larynx, pharynx or esophagus may aspire food or water when attempting to swallow. An uncommon but important effect of lodgement of food at the glottis is the production of asphyxia or sudden death due to vagal inhibition and cessation of respiration and circulation.

If large quantities of fluid are aspired death may in this case be instantaneous, but with small quantities the outcome may depend upon the nature of aspired material. The absorption from the lungs is very rapid, thus soluble substances such as magnesium sulfate exert their pharmacological effect very soon, whereas insoluble substances lodge in the lungs resulting in pneumonia, with profound toxemia, which is fatal within 48-72 hours. The severity of pneumonia depends upon the bacteria aspired. The pneumonia usually terminates in gangrene. Occasionally, the animal may survive the acute stage but later the condition usually relapses.

Antibiotics and sulfonamides given soon after aspiration has occurred may prevent development of secondary infection.

PULMONARY ABSCESSES

Pulmonary abscesses may arise on their own as primary disease or arise secondarily due to diseases in other parts of the body (Gautam et al., 1970). Primary abscesses may be due to tuberculosis, actinomycosis, pseudomoniasis (Bergin and Torenbeek, 1991), infections with aspergillosis, histoplasmosis, moniliasis, corynebacteria, and toxoplasmosis.

Secondary pulmonary abscesses may develop as a sequel to aspiration pneumonia or emboli/thrombi carrying infection from endocarditis, metritis, mastitis and omphalophlebitis that may lodge in the lungs resulting in abscesses.

Clinical Findings

In typical cases there is dullness, anorexia, emaciation. The temperature is usually moderately elevated and is fluctuating. There is pronounced coughing accompanied by pain. Respiratory signs vary depending upon size and number of abscesses.

Treatment

Treatment is not usually successful. Antibiotics given daily for several days may help.

PLEURAL EFFUSION (PLEURISY)

It is inflammation of pleura and is almost part of lung inflammation. Primary pleurisy is usually traumatic and caused by penetration of solid objects

into the chest. Secondary pleurisy develops as part of specific infectious diseases including, pasteurellosis, actinomycosis, and tuberculosis. It can also occur as sporadic infection e.g. septicemia due to pseudomonas, streptococci, staphylococci, and E.coli.

In the early stages the respiration is rapid, shallow and abdominal and the animal shows pain. The animal stands with elbows abducted and shows disinclination to move. The animal feels pain by pressure on the thoracic wall and deep digital palpation. Usually there is fever and pulse rate in elevated. Toxemia, anorexia and depression are seen in some cases. On auscultation, in the initial stages, friction sounds are heard, which in the later stages diminish, as the exudate accumulates. Dullness on percussion is evident at this stage. Cough is usually present. The prognosis is usually not favorable. Treatment should be attempted giving antibiotics and sulphonamides. Anaerobic infections may be checked by administration of chloramphenicol and metronidazole. Drainage is also very effective. Analgesics should be given to reduce pain.

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MUSCULOSKELETAL SYSTEM

The diseases involving bones, joints and muscles are being dealt with here together. The reason being that their manifest is common i.e. lameness, failure to support, insufficiency of movement, and deformity.

The clinical and laboratory examination of the musculoskeletal system and feet of the camels are essential to identify lameness and factors which contribute to it. A detailed physical examination of the affected area is necessary to localize the lesion. This includes passive movements of the limbs to identify fractures, dislocations and pain on movement. Muscles can be palpated for evidence of enlargement, pain or atrophy. The use of radiography is a helpful tool to diagnose the diseases of bones, joints and soft tissue affecting limbs. Muscle biopsy may also help in the diagnosis of muscle dystrophies. Microscopic examination of synovial fluid offers great help in the diagnosis of joint lesions. Serum levels of calcium, phosphorus, alkaline phosphatase and muscle enzymes, creatinine phosphokinase (CK) and serum aspartate amino transferase (AST) determination may aid in the diagnosis of muscle cell damage and osteodystrophies (Meyer and Harvey, 1998). Nutritional history should be obtained when the diseases of muscloskeletal system are suspected as most of the conditions are associated with nutritional disorders. This should include analysis of the feed and total amount of feed intake.

The muscular system of camels is adapted for economy of effort than power. Some of the muscles are absent or reduced in size as compared to the other animals. The muscles of the hind limbs are small as compared to the muscles of the fore limbs. The muscles of the neck are also relatively less developed, which makes the neck more mobile.

The predominant muscle types in camel are type I and II A. No relationship has been found to exist between fiber types, composition, and glycolytic or oxidative enzyme activity in the muscle. Enzyme activities and substrate levels are high in muscles of the camels as compared to the other ruminants (Saltin and Rose, 1994).

DISEASES OF MUSCLES

MYOPATHY

It is non-inflammatory degeneration of skeletal muscle, which is characterized by weakness and hyaline degeration of muscle fibers.

Etiology

The most common cause of myopathy is vitamin E and/or selenium deficiency in young calves. The precipitating causes include rapid growth, highly unsaturated fatty acids in the diet and unacustomed to exercise (exertional myopathy). It is aso seen in acidosis.

Some chemical agents in poisonous plants can cause myopathies. These include Cassia accidentalis, Karwinskia humboldtiana, Ixiolena spp, Geigeria spp.,

lupins, Solanum, and Tricetum spp. Myopathies have also been observed in poisoning by coccidostat monensin in camels (Chaudhary et al., 1998).

Ischaemic myonecrosis have been observed in animals recumbent for 48 hours or more. Neurogenic muscular atrophy occurs sporadically due to traumatic injury and subsequent degeneration or complete severance of nerve supply to skeletal muscle.

Clinical Findings

In most animals, including camel, skeletal muscle is composed of mixture of fibers with different metabolic and contraction characteristics. The fibers which have slow contraction time are called type I fibers, and the ones with fast contracting time are called type II fibers. The type II fibers can be subgrouped into type IIA and IIB on the basis of acid preincubation of ATPase (Snow et al, 1981). There exist variations in the percentage and in the composition of muscle fibers within individual camels, between various muscles and within the muscles at different locations. Type I fiber is the most dominant fiber in all muscles studied in the camel, while type II fibers are relatively scarce (Saltin and Rose, 1994). Type I fibers are characterized by strong aerobic capacity, compared with type IIA fibers which are more glycolytic and have strong aerobic and moderate to strong anaerobic capacity. Type IIB fibers are characterized by a relatively low aerobic and relatively high anaerobic capacity and are glycolytic (Kaneko et al., 1997).

In primary nutritional muscular dystrophy associated with a deficiency of vitamin E and/or selenium there is lipoperoxidation of the cellular membranes of muscle fibers resulting in degeneration and necrosis. The lesions only develop in the muscles and are similar, irrespective of the cause. The lesions in most of the cases are of hyaline degeneration to coagulative necrosis of myocardium and skeletal muscles. Because of the necrosis myoglobin is released and excreted in the urine. Myoglobinuric nephrosis also develops.

An important biochemical manifestation of myopathy is increased release of muscle enzyme from the damaged muscle cells. Creatinine phosphokinase (CK) and serum alanine amino transferase (AST) are both elevated in myopathy and are indicative of acute muscle damage. Ischemia also causes focal muscle necrosis and increased release of muscle enzymes. In neurogenic atrophy there is flaccid paralysis, a marked decrease in muscle mass and degeneration of myofibers with failure to regenerate.

The nutritional myopathies associated with deficiency of vitamin E and/or selenium occur most commonly in young growing animals. A sudden onset of weakness and pseudoparalysis of the affected muscles is observed in acute primary myopathy. Usually there is circulatory and respiratory insufficiency. The suffering animal remains alert and seems to be in spasm. The temperature is usually normal. The affected muscle may be swollen, hard and rubbery. Acute case of primary myopathy may die within 24 hours.

Treatment

Vitamin E and selenium are indicated for the treatment of nutritional muscular dystrophy. The treatment of exertional myopathy consists of enforced rest and relief of pain. In recumbent camels, provision of soft bedding and frequent turning is recommended. Fluid therapy to prevent myoglobinuric nephrosis may also be given. In myopathies associated with acidosis sodium bicarbonate is recommended. It should be given 2% of total dry matter intake. Dantrolene sodium at the rate of 4 mg / kg body weight, given orally, immediately upon recognition of clinical signs is efficacious.

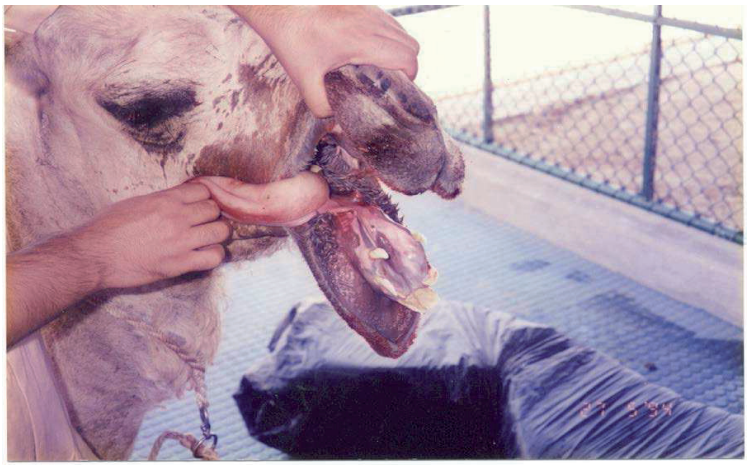
MYOSITIS

Myositis may arise from direct or indirect trauma to muscle and occurs as part of a syndrome in a number of specific diseases, including blackleg, sarcosporidiosis, trichinosis, and toxoplasmosis. Acute myositis of limb muscles is accompanied by severe lameness, swelling, heat and pain on palpation. There may also be accompanying toxemia and fever. In chronic myositis there may be much wasting of the affected muscles and is difficult to differentiate clinically from atrophy due to other causes. Chronic traumatic myositis may result in fibrotic adhesions of the muscles and calcification of fibrous tissue may supervene. Extensive damage to muscles occurs in screwworm and sometimes blowfly infestation. Myositis can also develop in camels by injecting irritant medicines, intramuscularly.

DISEASES OF THE BONES

FRACTURES IN CAMELS

The camel apart from being used as a draught animal is also kept for riding and racing purposes and thus is more prone to injuries and fractures. The incidence of various fractures recorded by Gahlot and Chouhan (1994) was 57% related to the head and neck (mostly mandible) 22% to the forelimbs and 21% to the hind limbs. The highest incidence of mandibular fractures is due to the anatomical weakness of this bone at the horizontal ramus due to presence of alveoli of tusks and also due to presence of dental canal in the region. The stress forces exerted by powerful jaw muscles during "rut" following excitement often lead to mandibular fractures. The sharp edges of the fractured fragments tear the oral mucosa, which usually leads to infection and formation of submandibular abscesses (Kumar et al., 1977).



Fractures of the lower jaw after falling down in the race

OSTEODYSTROPHY

It includes those bone diseases, which occur either due to failure of bone development (rickets), or due to abnormal metabolism of bone, which has already developed (osteomalacia). The clinical manifestations are distortion and enlargement of bones, and increased susceptibility to fracture. This condition is not frequently observed in the camels, however, it has been reported by Caligiuri et al., (1989)

Etiology

These conditions may result from deficiencies of calcium, phosphorus and vitamin D, and imbalance in calcium-phosphorus ratio. Osteodystropies may also be caused due to copper defficiency, inadequate protien in diet, chronic parasitism, hypovitaminosis A, chronic lead poisoning, chronic fluorine poisoning, and grazing on some poisonous plants.

Clinical Findings

In **rickets**, which occurs in the young growing animals, there is a failure of provisional calcification of the osteoid plus a failure of mineralization of the cartilaginous matrix of developing bone. There is also failure of degeneration of growing cartilage, formation of osteoid on persistent cartilage with irregularity of osteochondral junctions, and over gowth of fibrous tissue in the osteochondral zone. Failure of provisional calcification of cartilage results in an increased depth and width of the epiphyseal plates of particularly the long bones (humerus, radius, ulna, and tibia) and the costal cartilage of the ribs. The uncalcified and therefore soft tissues of the metaphyses and epiphyses become distorted under the pressure of weight bearing, which also causes medial or lateral deviation of the shafts of the bones. There is a decreased rate of longitudinal growth of long bones and enlargement of the ends of long bones due to the effects of weight causing flaring of the diaphysis adjacent to the epiphyseal plate.

In **osteomalacia** softening of mature bones occur consequent to resorption of mineralized bone and inability of mineralization of newly formed bone. Enlargement of the epiphyses of the bones does not occur in this condition. Long bones also don't become distorted. However, the bones are fragile and prone to frequent fractures.

Osteodystrophia fibrosa may be superimposed on rickets or osteomalacia and usually occurs in secondary hyperparathyroidism in animals (Blood and Radostits, 1989). Fibrous osteodystrophy of the facial and long bones has been reported in dromedary camels. The cause of the condition is thought to be due to vitamin D deficiency secondary to gastrointestinal malabsorption and inadequate winter exposure to ultraviolet light (Lynch et al., 1999).

The diagnosis can be made, by analyzing serum for calcium, phosphorus, and alkaline phosphatase. Histopathology of the bone and total bone ash determination may also help. The feed should also be analyzed for calcium, phosphorus, magnesium, vitamin D and trace minerals i.e. copper, fluorine and molybdenum.

Treatment

The condition can be treated by administration of calcium, phosphorus and vitamin D.

OSTEOMYELITIS

In camel focal inflammation of the bone can occur following open fractures (Gahlot et al., 1982).

Osteomyelitis is accompanied by severe, persistent pain and usually pus is discharged from the eroded bone. A large number of aerobic and anaerobic bacteria are involved. The treatment comprises of debridgement of necrosed tissue, drainage of pus, and local and systemic use of broad spectrum antibiotics for a long period.

DISEASES OF THE JOINTS

ARTHRITIS

The camels are frequently affected with inflammation of articular surfaces and synovial membranes due to trauma and infection. This condition is relatively more common in racing camels.

Etiology

Localization of infection in the joint is common in new born camel calves having septicemia or bacteremia which arise from naval or intrauterine infection.

It can also occur as a result of traumatic perforation of the joint capsule. The infection may spread from the surrounding tissue. Hematogenous spread from suppurative lesions of the udder, uterus, liver, lymph nodes, infected chest pad or tail, and castration wound may also occur. Al-Afaleq et al. (1992) isolated Chlamydia from joints of dromedary camels in Saudi Arabia. These animals were showing swelling of the carpus-metacarpus and / or tarsus-metatarsus joints. Ramadan et al. (1998) diagnosed carpal hygromas in two she camels associated with Brucella melitensis.

In racing camels sprain during race or training is a very common cause of arthritis.

Clinical Findings

Fetlock and pastern joints are mostly susceptible to injury and inflammation (Higgins, 1986). Rabagliati (1923) has reported lesions of polyarthritis in Somali camels. Arthritis causes pain and lameness in the affected limb. Heat and pain can be detected by palpation. The joint may be swollen and the degree of swelling depends upon the type of infection. In most of the neonatal arthritis some degree of omphalophlebitis and lesions in the other organs (liver, endocardium, meninges) occur, concurrently.

In infectious arthritis of hematogenous origin there is synovitis initially, followed by changes in the articular cartilages and some times bone. In chronic stages there is extensive granulation tissue formation chronic synovitis and degenerative joint disease with osteophyte formation and sometimes ankylosis.

Depending upon the type of organism involved arthritis may be suppurative or serofibrinous. Suppurative arthritis is particularly destructive to cartilage and bone and commonly there is rupture of the joint capsule.

Examination of the joint fluid should be got done by the laboratory. It comprises of culturing for bacteria, TLC, DLC, erythrocytic count and microscopic examination for the presence of cells, crystals etc. Biochemical and immunological tests are also helpful.

Treatment

Early treatment with parenteral and local antimicrobials for several days is recommended. The antibacterial of choice includes synthetic penicillins, tetracyclines, streptomycin, neomycin, gentamycin and kenamycin.

Local application of heat and liniments in the chronic cases are recommended. Analgesics are recommended if the camel experiences great pain.

Failure to respond to parenteral and intra articular medication may require surgical opening of the joint capsule, careful debridement, excision of the synovium and infected cartilage and bone, followed by daily irrigation of the joint cavity with antibiotics. Immobilization of the joint for several weeks, is also recommended. Glycosaminoglycans given orally and injecting in the affected joint may also help. Chronic arthritis may also be treated by subjecting the affected joint to meganetic field.

DORSAL FIXATION OF PATELLA

This condition is frequently observed in the aged and debilitated camels (Rathore and Chouhan, 1971). It is insidious in onset. One or both legs can be involved. There is rigid extension of the stifle and hock joints and flexion of the fetlock. The foot is usually abducted. Some times when the health state of the camel improves, there may be spontaneous remission (Higgins, 1986). Medial patellar ligament desmotomy is reported to be successful in some cases (Dhablania et al., 1971). Injection of counter irritant around the medial patellar ligaments is recommended by Rathore and Chouhan (1971).

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THE SKIN AND APPENDAGES

The skin has many functions. Primarily, the skin serves as a protective barrier between the body and the external environment and is an important organ for homeostasis. The flexibility and elasticity of the skin allow motion and provide shape and form. The skin also plays a vital role in thermoregulation and sensory perception.

The skin of camel is thicker and tougher than that of cattle and horse and affords better protection against penetration of spiny bushes and trees (Manefield and Tinson, 1996). Sweat glands in camel are distributed over the entire body surface (Kamel et al., 1986). However, these glands are absent in the non-hairy skin areas like perennial region, chest pad, and stifle pad (Goswami et al., 1994). Melanin provides the variety of colors found in camels. Precursors for vitamin D_3 synthesis are present in the skin which are acted upon by solar ultraviolet light to produce the vitamin that is later converted to 1, 25 dihydroxycholecalciferol, the active hormone, via metabolism in the liver and kidney. Special appendages of the skin include hair and nails.

The skin of camel is attached rather tightly to the underlying tissue and is relatively immobile. This is a disadvantage to the camel in that, if attacked by the biting and flying insects, particularly in view of its short and ineffectual tail. The animal is thus reduced to stampede, kicking and throwing its head about in the effort to remain comfortable, apparently often without much success. The skin is thicker over the back than elsewhere and particularly over the humps when this is in decline. The epidermis is well developed; the dermis is compact and hard and is rich in elastic fibers, which have long papillae; the sub-epidermal tissue is very hard. Sweat glands occur sparsely all over the body but sweating is restricted to very hot periods and to times when the animal is excessively tired (Wilson, 1984).

The pole glands are more active under conditions of heat and fatigue than at any other time. They are also active when the male is in rut. They may thus be modified sweat glands. They are found in both sexes although they are less active in castrates and females than in entire males and the fluid emitted has a much less noticeable odor. The glands are situated towards the top of the back of the neck and cover an area of about 6x4 cm. They can be distinguished from the surrounding skin, particularly in males, by their slight elevation, their color, and scarcity of hair.

Under the feet, where the skin comes in contact with the ground when the animal is standing or walking, it is modified into pads. The largest pad is generally referred to as the pedestal and is on the chest below the sternum; there are in addition pads on the elbows and stifles and less important ones on the knees and outside the hocks. In young animals the pads are less developed and covered with short hair, which wears away after a few months.

DISEASES OF THE SKIN

The diseases of skin are very common in camels. Included in these are camel pox, contagious ecthyma, papillomatosis, skin abscesses, contagious skin necrosis, staphylococcal dermatitis, dermatophilosis (ringworm), Addison's disease, elephant foot, dermoid cysts, onchocercariasis, sarcoptic mange, psoroptic mange, hypodermatosis, myiasis, saddle sores, whip injuries, crow peck, pedestal injuries, lacerations, branding or firing injuries, allergic dermatitis, neurodermatitis, urine scald, pityriasis, parakeratosis, and hyperkeratosis.

The diseases caused by bacteria, fungi, viruses, and parasites have been discussed elsewhere in this book.

PITYRIASIS

It is characterized by the presence of bran-like scales (dandruff) on the surface of skin.

It can be caused by over production of keretinized epithelial cells as a result of hypovitaminosis A, riboflavin and nicotinic acid deficiencies. It may also be the result of the deficiency of essential unsaturated fatty acids, especially linolenic acid (primary pityriasis).

Ectoparasites (flea, louse, and mange) infestation and ring worm infection are also responsible for pityriasis due to excessive desquamation caused by scratching (secondary pityriasis).

In primary pityriasis there is accumulation of scales without itching. The accumulation of scales occurs commonly where the hair coat is long. The lesions of the primary disease usually accompany secondary pityriasis.

For treatment removal of the primary cause be attempted. Non specific treatment includes thorough washing followed by application of emollient ointment and alcoholic lotion alternately. It is good to incorporate salicylic acid both in ointment and lotion.

PARAKERATOSIS

It is incomplete keratinization of the epithelial cells caused by dietary zinc deficiency, and is often seen in non-specific chronic inflammation of cellular epidermis with resultant faulty keratinization of the horny cells. In nutritional parakeratosis the deficiency must be corrected. The abnormal tissue is first removed by keratolytic ointment (salicylic acid) followed by the application of an astringent preparation. Addition of zinc to the diet will cure zinc deficiency parakeratosis.

HYPERKERATOSIS

Accumulation of excessively keratinized epithelial cells on the skin surface may occur locally at the parts subjected to continuous pressure. Generalized hyperkeratosis is observed in poisoning with highly chlorinated naphthalene and in chronic arsenic poisoning. In this condition the skin becomes thicker than normal and is usually corrugated and hairless. The external surface is dry and scaly. Secondary infection of the area is common. Treatment is comprises of removing the causal factor and use of the keratolytic agents.

URTICARIA

It is an allergic condition in which wheals appear on the surface of skin. Insect stings, contact with stinging plants, ingestion of unusual food, administration of particular drug, and death of parasites or their larvae in the tissues may cause urticaria.

The lesions of urticaria develop suddenly and often in large numbers. They are flat top, elevated and tense and vary largely in size. No exudation occurs. Other manifestations of allergic reaction e.g., diarrhea and fever may accompany the skin eruptions. The lesions disappear soon but may last for 3-4 days. The condition can be treated with antihistaminics. Adrenaline as a subcutaneous injection is also good. Lesions can appear if antigenic encounter is repeated.

ECZEMA

It is an inflammatory reaction of epidermal cells in response to allergens. The allergens may be: endogenous; ingested as food, formed in the gut (amines formed due to over eating or bowel stasis and reflux), or digestion of internal parasites, or exogenous, external parasites, and chemicals etc. The first observable sign is erythema, followed by the appearance of small vesicles, which rupture and cause weeping of the surface. Scab formation follows. The lesions may be isolated patch or be diffuse. Itching and irritation is intense. In chronic cases alopecia and dermatitis may occur. The treatment comprises elimination of causative agent, good hygienic conditions, laxative diet, antihistaminic administration and astringent lotion application.

DERMATITIS

Inflammation of dermis and epidermis in camel is very common and is caused by bacteria (e.g. Staphylococcus aureus), fungi, mange mites, filarial, screwworm, and viruses of pox and vesicular ecthyma. The condition may also be caused by chemical (corrosive agents like strong acids, alkalies etc.) and physical agents (trauma, burns etc.). Sometimes individual alopecic lesions on the hind leg or flank are observed in camels, which are associated with compulsive licking or biting.

Staphylococcus aureus

Affected skin initially becomes red and warm. Later on the lesions progress depending upon the causal agent and degree of exposure. This condition occurs in males and tethered racing camels. It seems to be a behavioral anomaly (Manefield and Tinson, 1996). Examination of the skin scrapings or swabs for the presence of parasites and bacteria or other agents is essential. Culture and sensitivity tests for bacteria are required to prescribe best treatment. Skin biopsy

examination is recommended to know the nature of the disease. Specific treatment depending upon the causative agent is recommended.

Epidemic of Staphylococcus aureus in the herd

ANASARCA

It is the edema of subcutaneous tissue and may be caused due to congestive heart failure, hypoproteinemia (liver damage, kidney damage, intestinal nematodiasis, enteropathy, protein starvation), hypovitaminosis A, allergic reaction (angioneurotic edema, insect bites), inflammatory reaction (clostridial infection, anthrax, pasteurellosis), and compression of lymphatics by tumors or abscesses (local edema). An edema disease in camels due to proliferation of hemolytic E. coli has been reported by Ibrahim et al. (1998).

In affected camels there is visible swelling either local or diffuse. The skin is puffy and pits on pressure. Usually there is no pain except in inflammatory edema. Usually the underside of the trunk is involved.

Treatment should be attempted to remove the primary cause and administration of diuretics and drainage of the fluid. Antihistaminics should be given for allergic edema.

LYMPHADENITIS

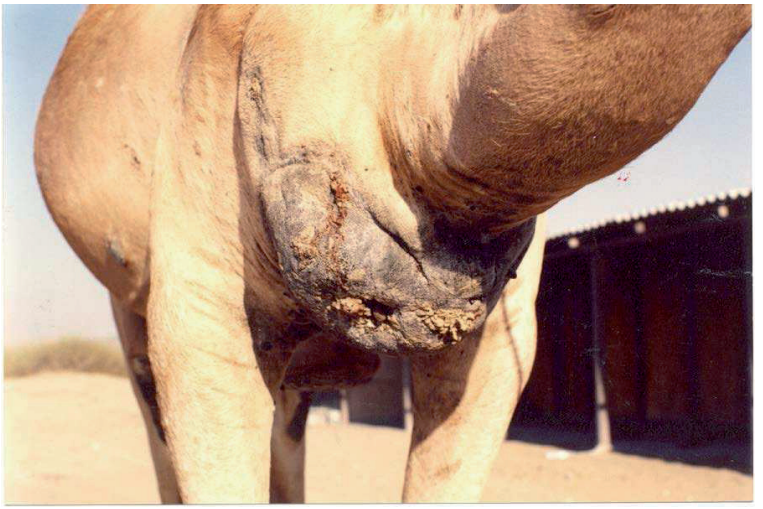
Inflammation of the lymph nodes usually occurs by spread of infection from the skin. The most important causes in the camel include, caseous lymphadenitis due to Corynebacterium pseudotuberculosis, staphylococcal dermatitis, cutaneous tuberculosis, and Histoplasma farciminosum (Spesivtseva and Noskov, 1959).

Abscessation or ulceration usually exists at the original site of infection. The lymph vessels leaving the lesion are enlarged, thickened and tortuous and often have secondary abscesses or ulcers along their course. In chronic cases considerable fibrous tissue may be laid down in the subcutaneous tissue.

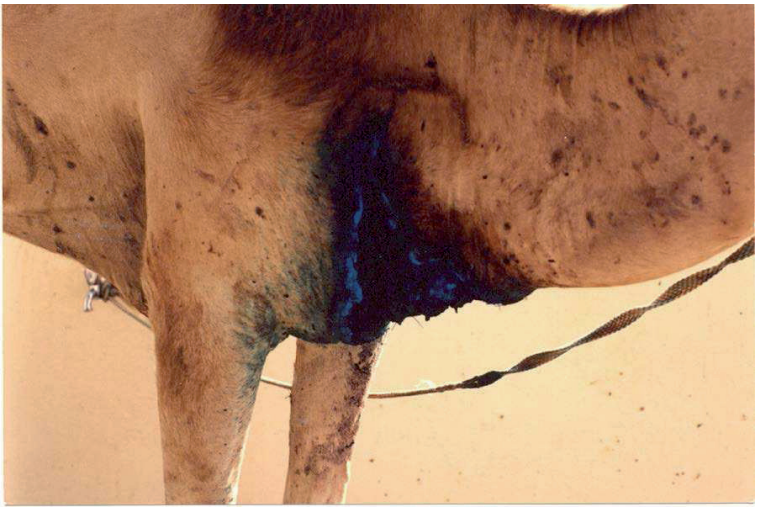
For the treatment it is essential to remove the focus of infection either by medical treatment or surgical excision.

GRANULOMATOUS LESIONS

Granulomatous lesions of the skin occur as nodules, plaques and ulcers. The lesions are hard, cold, and progress slowly. Some of the common causes in camels are infection with Mycobacterium sp., Actinobacillus sp. (Arora et al.,1973; Purohit et al., 1988), Corynebacterium sp., and infestation with



Fibroma, deep-seated abscess



After removal of the abscess

Onchocerca sp. and Hypoderma sp. larvae. Granulomatous lesions can also occur in both the sexes during summer months due to sprinkling of urine on the hind legs (Manefield and Tinson, 1996). Treatment will depend on the causal factor.

CONGENITAL SKIN CONDITIONS

Schwartz and Dioli (1992) have reported idiopathic alopecia, a rare congenital defect.

PEDESTAL INJURIES

The sternal or pedestal pad is frequently injured and is very difficult to treat because it has to come in contact with the ground and bear weight of the animal while sitting. The area becomes bruised, punctured, and infected forming abscesses. The condition is very painful which makes the animal reluctant to sit. A significant amount of fibrous tissue may also form (Purohit et al., 1986; Purohit and Chouhan, 1992).

Occurrence of sternal fibroma and lymphosarcoma is also reported in camels (Bolbol et al., 1992).

Treatment consists of opening the abscess, draining pus, and treating wound with some antibacterial preparation. The fibrous tissue should be removed surgically. A donut shaped pad secured on the area helps in preventing the area being frequently injured.

DISEASES OF FOOT

The camel has ability to traverse through sandy and rough terrain due to special morphology of its foot (Bligh et al., 1976). The soles of the feet in camels are provided with a pad beneath the corium. This pad is composed of much elastic tissue attached to the flexor tendons and the second and third phalanges of each digit. The pad of each limb is fused to enclose the two digits in one pad. The claws or nails do not bear weight, but protect the tip of the toe (Yagil, 1985).

Clusters of branched alveolar glands in the subepithelial connective tissue of the foot pad of camel have been reported (Karkoura, 1986). Evaporation of the gland secretion play an important role in maintaining the foot pad temperature constant when the temperature of the surroundings is high (Saber et al., 1994). Moreover, the highly coiling and branching nature of the glands as well as their deep location from the solar surface of the foot pad seems to match the adaptive feature of the foot pad to the adverse climatic conditions (Saber et al., 1994). It provides profuse amount of secretion, which becomes preserved deeply far from the sole, though, the chance of rapid evaporation will be controlled.

Thick keratinisation at the sole surface makes heat and foreign body particles impermeable, enabling camel to move on hot desert and thorny strech. Despite these anatomical advantages the camels do suffer with occupational hazards i.e., burn injuries on foot while working in kilns, bruised sole in those moving on the roads, fracture of digits in those working in mines, and punctured foot in those being used for draught purposes in the urban areas (Singh and Gahlot, 1997).

Lesions of the foot pad usually result in various degrees of lameness, hot and painful swelling and reluctance to move. Treatment of an injured foot pad presents a serious challenge. Position and function leads to continuous soiling and irritation of the wound. Good nursing, care, and rest have yielded successful results in curing the condition. The treatment should include removal of the foreign body, removing of dead tissue, and application of protective bandage or

boot. In neglected cases surgical amputation of the dead portion of the foot is required.

The following pathological conditions of feet are frequently observed in the camels:

PUNCTURED SOLE

The sole can get punctured by thorns, nails, glass, metallic wire, bones, sharp rock, etc. Lameness ensues suddenly after penetration. Cellulitis can develop in case the wound becomes infected. If the depth of penetration is more, prolapse of the digital cushion can develop. Osteomyelitis can also develop if infection spreads to the bones of the foot. This may lead to necrosis of the bones (Singh and Gahlot, 1997).

For treatment the foreign body should be probed or be located by X rays or ultrasonography and should be removed. Wound should be washed thoroughly and some antiseptic, antibiotic or sulpha drug applied on the wound. The wound should be covered by some bandage. The local treatment should be supported by injection of some suitable antibiotic and antitetanus serum (Manefield and Tinson, 1996).

WORN TOE NAILS

Racing camels suffer from this condition very frequently, especially when they are on rough ground instead of sand. Some camels have more tendency of developing this condition due to their low foot flight in the pacing gait. The hind feet are more prone to this condition. Sometimes the laminae get exposed and bleed due to wear (Manefield and Tinson, 1996).

For treatment the affected animals should be kept on sand and some suitable antiseptic or antibiotic be applied. The foot should be protected by bandage or boots. This should be practised until new nail grows.

CRACKED OR TORN NAILS

If the nails are not trimmed regularly and are left over grown, they tend to crack and may break right off at the nail bed. If the loose nail is not noticed a new nail starts growing beneath it. It takes 6-8 weeks for the regeneration of completely lost nail. If regular trimming of the nails is done, the condition can be prevented (Manefield and Tinson, 1996).

FOOT GANGRENE

It has been observed in the camel. The exact cause of this condition is not known. Proteus vulgaris has been isolated in one case (Ramadan et al., 1984). The condition commences from the dorsal surface of the foot and descends down. The only treatment suggested is surgical amputation of the foot (Soliman et al., 1983).

WORN PADS

This condition is very common in racing camels. It is caused by excessive wearing of the sole when the camels are kept on rough surfaces or due to over training and over racing. The pad becomes very thin and shiny. Ulcers may develop due to excessive wear. Affected camels should be given rest on sand. Protecting foot pads by boots, made of some suitable material, may prove helpful. Biotin preparations promote healthy growth of the hoof. In case of ulceration some good antibacterial preparation be applied and lesions be covered by bandage.

As the pad wears, sometimes a flap of it lifts off. Sand and / or gravel may get impacted under the flap causing discomfort and pain to the animal. It is recommended to cut the flap, clean the pad thoroughly, and if there is any injury or ulceration underneath the flap it should be dealt, accordingly. After acute illness

fissures may also be formed on the foot pad. These may be treated by application of some antibiotic ointment and improving the nutrition of the affected camel.

HERNIATION OF DIGITAL CUSHION

It may occur due to external trauma where the limiting fibrous tissue of the digital cushion develops a breach and hernia of the digital cushion occurs. It may be unilateral or bilateral. The condition is often painless. It appears as a soft reducible, circumscribed in outline, and decreases in size when the leg is flexed. It becomes tense and non reducible, while the animal is standing. Herniorrhaphy with silk sutures may prove helpful in correcting the condition (Singh and Gahlot, 1997).

ELEPHANT FOOT

It is proliferative chronic granulomatous condition of foot, upto fetlock. It can involve all the soft tissues of the foot, except the pad. Usually, only one foot is affected. Due to cellulitis and fibrosis the foot becomes very much swollen, and breaches in the skin and coronary corium develop. The condition is reported to occur due to bites of sand fly (Higgins, 1986). Usually amputation of the digit is required to save life of the animal.

DISEASES OF EYES

Occular diseases are quite common among herded camels. Most of these are traumatic in origin, including amongst these are due to blows, thorns, and other foreign bodies. Commonly only one eye is affected. In camels browsing on shrubs and Acacia trees the chances of injury are more. Excessive rubbing due to irritation of eyelids caused by flies, mites, and ticks can also lead to eye injuries and secondary bacterial infections. Injuries can also be inflicted in camels by the handler's stick. Occasionally, eye infections result in impaired vision or complete blindness due to opacity and scars. Opacity is a common clinical finding amongst older camels. Cases of temporary blindness have also been reported (Schwatz and Dioli, 1992).

CONJUNCTIVITIS

It can occur due to rubbing of eyes due to irritation, usually caused by fly nuisance, parasitic infestation, or sand getting into eyes, injuries occurring while browsing, and eyeworm infestation. It can also occur in pox infection (Rathore, 1986).

Flies and other parasitic infestation can be prevented by the use of pour on insecticidal preparations. Bacterial infection can be treated by application of some antibiotic ointment or drops, preferably, twice daily.

KERATITIS

The causes of this condition are same as for conjunctivitis. The clinical signs are lacrimation, photophobia, and corneal edema. Ulcers can also develop. For treatment application of some ointment or drops containing antibiotic and corticosteroids is recommended. Healing of ulcers may be achieved by securing the third eyelid on the cornea by a suture. This can be done by passing suturing needle carefully behind the T-shape cartilage in the third eyelid, and the eyelid being pulled laterally across the cornea and sutured on the other side. It is better to put some antibiotic ointment behind the membrana nictitans while operating. The ointment should be applied twice daily, until, recovery (Manefield and Tinson, 1996).

KERATOCONJUNCTIVITIS

This condition, commonly known as pink eye, is frequently observed in young camels. It is caused by Moraxella spp. (Manefield and Tinson, 1996). Application of some antibiotic ointment, twice daily, in the eyes is recommended.

PANOPHTHALMITIS

It results from infection being introduced in the eye by some penetrating object or from deep corneal ulcers. The prognosis is not good in this condition. Broad-spectrum antibiotics given parenterally may be tried. In some cases affected eyeball has to be removed.

EYE WORM

Thelazia leesi may infest conjunctival sac of camel. It is transmitted by flies (Dobrynin, 1972).

HYPHEMIA

Presence of blood in the anterior chamber of the eye is called hyphemia. It is usually, caused by a blow of stick. In more strong blows the bleeding can also occur in the posterior chamber of the eye. It takes several weeks until all the blood is reabsorbed. Permanent blindness can ensue due to clot adherence to the lens and other damage in the posterior chamber. Treatment recommended is parenteral administration of some broad spectrum antibiotic and corticosteroid preparation. An ointment or drops containing broad-spectrum antibiotic and corticosteroid should also be applied, locally.

ENTROPION

It can occur in old camels. The condition is more common in the males. Usually one upper eyelid is affected. Often the upper eyelid sags and the lashes turn in. The herds men often try to relieve the condition by cutting the lashes, which adversely affects eye as the cut lashes become bristle like. This may leads to ulceration of the cornea. The condition can be corrected surgically and the eye treated by local application of antibiotic cream.

DISEASES OF EARS

The camel has relatively small and round ears, which are well haired to give protection for entry against sand etc. It has also been observed that relatively less wax is produced in the camel's ears.

OTITIS EXTERNA

This condition is frequently observed in racing camels. It may be due to repeated wetting of the ear, as the latter are given bath repeatedly. Injuries due to parasitic infestation can often become infected leading to this condition. The infection may spread to meninges causing meningitis. Daily cleaning the area and application of some broad-spectrum antibiotic or antiseptic ointments is recommended for treating this condition.

ACRAL GANGRENE

Acral gangrene in a two-month-old camel calf in which 30% of the ears were dried off has been reported by Nothelfer et al. (1994). The condition was attributed to blockage of blood supply due to multiple thrombi. Staphylococcus aureus was isolated from the lesion.

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CARDIO-VASCULAR AND HEMOPOETIC SYSTEM

The morphology of the camel's heart generally follows the mammalian model. The heart is 0.60-0.75 percent of the total body weight of the camel.

Heart rate of the camel can be determined by auscultation with the stethoscope. The head of the stethoscope, being pushed up to the axilla, medial to the elbow. It is difficult to listen to the heart sounds if the animal is making noises. Resting heart rate is normally 30-44 per minute. Camel has an ability to keep it low in very hot environmental conditions by keeping its metabolic rate low. During exercise maximum heart rate of 150-160 per minute has been recorded. Pulse rate can be determined, while the camel is standing, from the median palmer branch of the radial artery as it passes over the medio-palmar aspect of the carpus.

The blood pressure is normally 130 / 100 mm of mercury. Recording of the electrocardiogram using alligator clips to place the limb leads onto clipped elbow and stifles and the exploring electrode (V) over the dorsal spinous process of vertebra T7 have been described in camels. Manefield and Tinson (1996) have described another method. While the camel is crouching or standing, the leads RA and LA are attached with alligator clips to the points of the shoulders. Leads of RF and LF are attached to the skin, respectively, left and right 5 cm lateral to the posterior end of the pedestal. The exploring electrode is attached over the apex of the heart on the left side. There is generally no need to clip any hair so long as electrode gel is liberally applied and the alligator clip can satisfactorily contact the skin. A normal ECG has a slight irregular rhythm; there is a slight degree of sinus arrythemia (Jayasinghe et al., 1963). In lead II the QRS is tallest and is upright. The lowest amplitude is found in lead I. The P-R interval is 0.24 s; Q-T interval is 0.5 s. There is a physiological inversion of the T-waves.

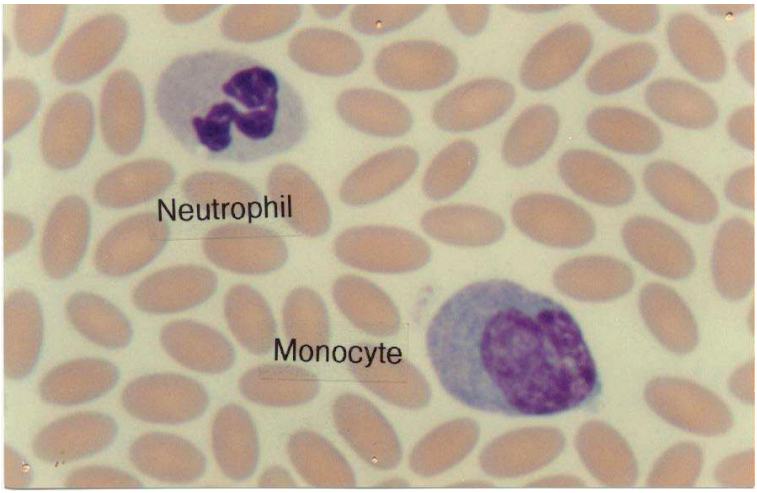
Changes occur in the circulation of camel to get adapted to heat. The camels have comparatively less subcutaneous fat for more successful dissipation of heat, instead the subcutaneous fat is accumulated at one place, the hump (Schmidt-Nielsen, 1964). Skin capillaries of the camel have extremely thick walls and narrow lamina with room for only one erythrocyte to pass at a time. There is no fenestration in the capillary walls. This indicates that the movement of fluid from the lumen to the interstitial space is accomplished by transcellular route. The capillary walls thus prevent the loss of water from the blood vessels during severe dehydration due to non-availability of water. Heat is dissipated from the skin by radiation, conduction, and convection rather than transpiration (Yagil, 1985).

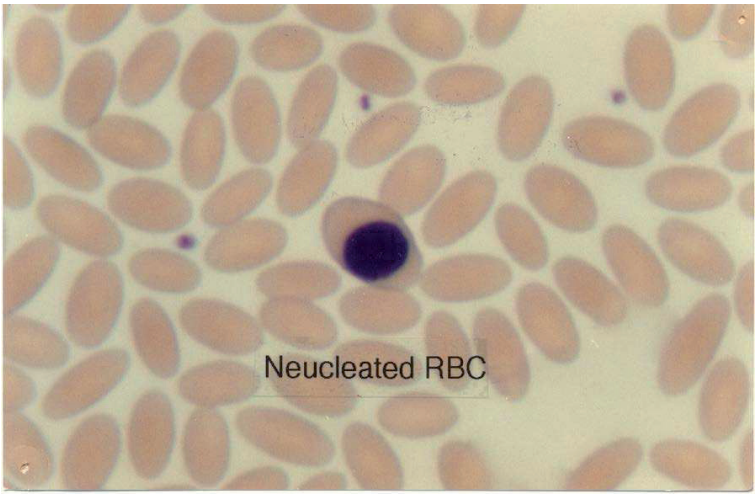
There is special arrangement of cerebral arteries in the camel because of its long neck (Kanan, 1970), which ensures that brain blood pressure will not fluctuate when animal raises its head while browsing trees and then suddenly lowering the head to eat from the ground. This is due to the presence of cusps, which are arranged one above the other along the axis of the veins. Their function is to prevent a back flow of the blood when there is sudden rise in the pressure in main arteries. These valves are also present in the femoral vein (Amonroso et al., 1947).

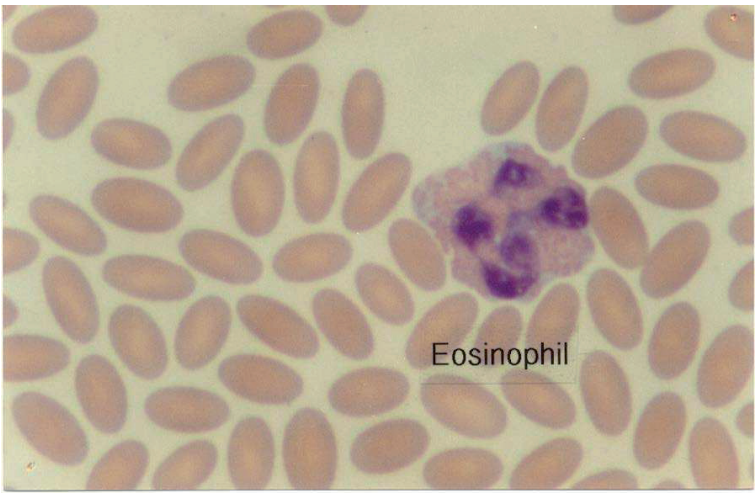
Hematological and serum biochemical values of dromedary racing camels of different ages (Wernery et al., 1999)

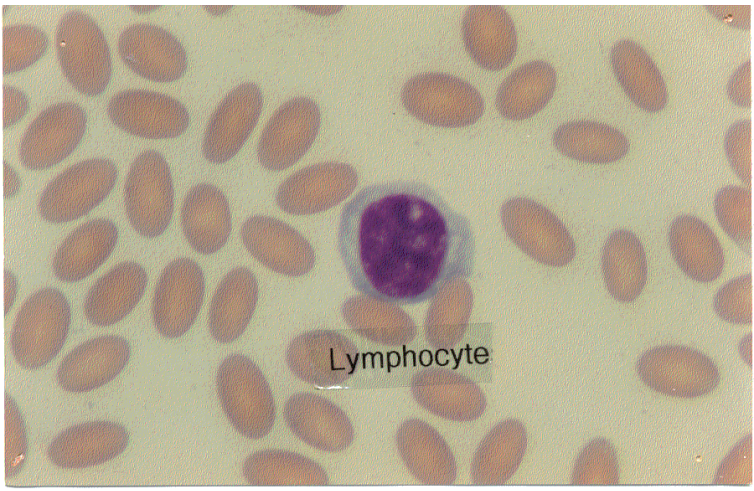
Blood parameters	Age			
	Up to 6 months	6 months to 2 years	2 to 12 years	Unit
ΓHemoglobin (Hb)	10.4-13.9	8.5-12.0	12-15	gm/dl
Packed Cell Volume (PCV)	24.0-31.0	20.0-28.0	26-38	%
Mean Cell Volume (MCV)	29.6-35.1	30.0-34.0	26-34	µm
Red Blood Cell (RBC)	7.4-10.0	6.5-9.0	7.5-12.0	x 10^6/µl
White Blood Cells (WBC)	11.5-18.5	9.5-14.0	6.0-13.5	x 10^3/µl
pH	NA	NA	7.2-7.3	
Neutrophils	53-68	50-60	50-60	%
Lymphocytes	20-44	30-45	30-45	%
Monocytes	0.5-10	0.3-7	2-8	%
Eosinophils	0.3-2.5	1-2.5	0-6	%
Basophils	0.2-2.4	0-1	0-2	%
Platelets	375-820	350-450	200-700	x 10^3/µl
Aspartate aminotransferase (AST)	92-200	100-125	60-120	IU/l
Lactate-dehydrogenase (LDH)	520-730	450-550	400-775	IU/l
Creatine-Kinase (CK)	175-350	85-120	40-120	IU/l
Gamma-glutamyle transferase (GGT)	5-15	4-10	3-15	IU/l
Alkaline-phosphatase (ALP)	350-750	150-240	60-140	IU/l
Total Bilirubin (TBIL)	0.0-1.0	0.0-1.0	0.0-1.0	mg/dl
Alanine aminotransferase (ALT)	3-15	3-15	3-15	IU/l
Blood Urea Nitrogen (BUN)	10.0-20.0	10.0-20.0	3-21	mg/dl
Creatinine (Crea)	1.1-1.5	0.8-1.3	0-2.2	mg/dl
Total Protein (TP)	4.9-6.0	4.0-5.5	5.7-7.5	gm/dl
Albumin (ALB)	2.8-3.5	1.8-3.5	3.0-4.5	gm/dl
Iron (Fe)	37-135	40-120	87-135	µg/dl
Magnesium (Mg)	2.0-2.4	2.5-3.5	1.8-2.4	mg/dl
Calcium (Ca)	10.6-12.0	9.0-11.0	9.5-11.5	mg/dl
Phosphorus (P)	9.6-13.9	6.5-10.0	3.5-6.0	mg/dl
Sodium (Na)	150-160	148-156	150-160	mmol/l
Potassium (K)	6.5-7.7	5.2-7.8	3.5-5.5	mmol/l
Chloride (Cl)	110-120	110-115	90-110	mmol/l
Glucose (Glu)	70-140	75-120	70-110	mg/dl
Fibrinogen	350-450	250-380	250-400	mg/dl

NA = not available


Neutrophil
Monocyte


Neucleated RBC





Under normal conditions water in the plasma constitutes about 16% of the total body water. Plasma albumin, which possesses water-attracting properties, is present in relatively large concentration in camel (Siebert and Mac Farlane, 1975).

As the plasma volume determines the heat transporting capabilities of the blood, the dehydrated camel loses about 5% of its plasma volume compared with other ruminants. Cattle lose 19% of their plasma volume when dehydrated for 2 days

and sheep dehydrated for 3 days lose 8% of their plasma volume (Mac Farlane, 1968). Plasma volume is maintained in the dehydrated camel by a steady absorption of water from its alimentary canal. Other factors aiding in plasma volume are all connected with water-attracting properties are:
Hyperglycemia occurring in dehydrated camel (Yagil and Berlyne, 1977a).
Hypernatremia (Yagil and Berlyne, 1976).
Uremia (Yagil and Berlyne, 1979).

Of essential importance in retaining body water in the dehydrated camel are the recycling of urea, not only as a source of nitrogen for the production of rumen flora protein, but also trapping water. Reabsorbed urea from the kidneys remains only partially in the plasma because it readily enters all of the cells. The passage of urea and water into the stomachs is especially important to camel (Vercoe, 1969). Water that enters the stomachs with the urea is reabsorbed in the omasum and intestines, maintaining plasma water levels.

A healthy camel has an average temperature of 36-36.5^{o}C, rising steadily through out the day to about 39^{o}C in the evening (Higgins, 1986). The dehydrated camel only sweats when its body temperature increases above 42^{o}C (Schmidt-Nielsen, 1964), it sweats very economically (Mac Farlane, 1977). Renal function is greatly depressed during dehydration (Yagil and Berlyne, 1978) and with the general decline in metabolism there is an extremely low water turnover rate (Etzion et al., 1984). The lowest water losses are from the plasma, while highest are from the alimentary tract. The circulatory integrity of the dehydrated camel is thus preserved, including the dissipation of the heat to the periphery.

The white blood cells do not show any functional difference in adaptation to a desert environment. The white cells of camel are responsible for combating inflammatory conditions, like other mammals. Neutrophils are predominating cells amongst the leukocytes. The neutrophils of the camel under transmission electron microscope revealed rounded to spherical outline possessing various sized cytoplasmic processes. The nucleus has 3-4 lobes and has a perinuclear space in some of the cells (Singh et al., 1997). In the ultra structural studies of eosinophils, it is revealed that the nucleus is usually bilobed with chromatin packed densely at the periphery as compared to the central part. At the ultra structural level, the basophils show few cytoplasmic processes. Within the nucleus the densely packed chromatin material is more at the periphery and occupies more space as compared to centrally placed loose chromatin. The nucleus of the lymphocytes has loosely arranged electron dense chromatin as compared to other cell types. It also possesses superficial or deep indentation and in some of the cells, the indentation extends from one pole of the nucleus to the other. The cytoplasm of some of the lymphocytes has very dense granules spread all over. The monocytes at their ultra structural level appear spherical to irregular in shape and have long and wider cytoplasmic processes. Depending upon the plane of section, the nucleus appears spherical or indented, or even lobed. The chromatin material within the nucleus is usually arranged, loosely (Singh et al., 1997).

The size of platelets is smaller than that of human beings. Plasma prothrombin time is slower in camels, 9.1-10.2 seconds compared to 11.5-13 seconds in humans (Yagil, 1985). The scanning electron studies of platelets revealed that the blood platelets of camel appear irregular in out line. They have long, slender and mostly branching cytoplasmic processes (Singh et al., 1997).

The camel red blood cells (RBCs) are ovoid and non-nucleated. They are somewhat flat, and after dehydration, there is no change in the shape of RBCs. However, after rapid rehydration the cells become round (Yagil et al., 1974c). The average dimensions of the camel red cell are: large axis 7.7-10.1 um; short axis 4.2-6.4 um (Yagil et al., 1974b). The average thickness of the cell is only 2.5 um (Adul-

Gadir et al., 1979). The number of RBCs is 7.5-12.0x10^6 /ul in adult camel (Wernery et al., 1999). The mean corpuscular volume (MCV) is 26-34 um, which is relatively low than human beings. Mean corpuscular hemoglobin is relatively low 15.5 pg as compared to human beings, which is 29 pg (Yagil et al., 1974b). Mean corpuscular hemoglobin concentration (MCHC) is much higher in camels (54.4 g /dl) as compared to human beings, which is 34 % (Yagil et al., 1974b).

The scanning electron microscopic studies revealed that camel erythrocytes are thin, flat, elliptical or oval in outline (Singh et al., 1997). Jain and Keeton (1974) reported them to be thin water like, which give these cells a unique capacity to withstand an abrupt decrease in blood osmolarity. Thin erythrocytes are known to be osmotically resistant than the relatively fragile thick erythrocytes. Moreover, it has been shown that camel erythrocytes can double their volume in hypotonic saline solution before lysis occurs (Perk, 1963).

The camel blood cells sediment slowly. Red cell survival time is 90 days in winter and 120 days in summer. Due to water deprivation in summer the survival time increases to 150 days, which is opposite what happens in other species (Manefield and Tinson, 1996).

HEART FAILURE

The function of the cardiovascular system is to maintain the circulation of the blood, so that normal exchange of fluid, electrolytes, oxygen, and other nutrients and excretory substances can be made between the vascular system and tissues. The maintenance of oxygen requirements is very important especially for the nervous system, as it is very much susceptible to oxygen deprivation. Failure of the heart as a pump can result from a defect in filling of the heart, an abnormality in the myocardium or conducting system, an extensive workload, or a combination of any of the three.

Heart failure can be either acute heart failure or chronic / congestive heart failure. Circulatory equilibrium is not maintained when cardiac out put is deficient. If this develops slowly, compensatory mechanisms, plus the failure of heart itself as a pump, result in an increase in the venous pressure and congestive heart failure. If there is an acute reduction in cardiac output, which is caused by sudden cessation of the heartbeat, it deprives tissues of oxygen and acute heart failure ensues.

Congestive heart failure is caused by endocarditis resulting in valvular stenosis or valvular insufficiency, congenital valvular defects, rupture of valves, myocarditis, myocardial degeneration (nutritional or toxic), chemicals affecting cardiac conduction, and pericarditis. In the very early stages when cardiac reserve is reduced and decompensation has not yet occurred there is respiratory distress on light exertion. Congestive heart failure of the left side is manifested by an increase in the rate and depth of respiration at rest, cough, the presence of moist crackles at the base and increased dullness on percussion of the ventral borders of the lungs. In the later stages, there is dyspnea and cyanosis.

In congestive heart failure of the right side, the heart rate is increased and there is anasarca evident on the ventral part of the body, the neck, and the jaw. The urine flow is usually reduced and the urine is concentrated and contains a small amount of albumen. The superficial veins are dilated, particularly, the jugular vein. The prognosis of congestive heart failure in camels is unfavorable.

Symptomatic treatment of the condition may be attempted in congestive heart failure.

ACUTE HEART FAILURE

Acute heart failure can occur where there is severe defect in filling, and heart fails as a pump, either due to severe tachycardia or bradycardia, accompanied by sudden increase in workload. The condition can occur in pericardial temponade (Atrial and ventricular rupture), aortic or pulmonary artery rupture, myocarditis, nutritional deficiency myopathy (copper or selenium deficiency), some plant poisoning, and electrocution. It can be laterogenic due to intravenous injection of calcium preparations, xylazine, concentrated solution of potassium chloride. Rupture of aortic valve due to increased workload and acute anaphylaxis may cause acute heart failure. It has also been reported in monensin poisoning in camels (Chaudhary et al., 1998).

Commonly it occurs during periods of excitement. The animal usually shows dyspnea, staggering and falling, death usually occurs in minutes of the first appearance of signs.

In less severe cases, such as which occur in muscular dystrophy the course of the disease may be as long as 12-24 hours, and clinical syndrome is one of acute left-sided heart failure. Acute heart failure is considered to be the cause of death in a significant proportion of camels that die suddenly during training and racing. In typical acute cases engorgement of visceral veins may be present if the attack has lasted for a few minutes, but there may be no gross lesions characteristic of congestive heart failure.

The only treatment suggested is intracardial injection of adrenaline.

PERIPHERAL CIRCULATORY FAILURE

Failure of venous return towards heart occurs when there is peripheral vasodilatation and pooling of blood in the vessels (vasogenic failure), and when there is a reduction of total blood volume (hematogenic failure). In vasogenic failure, blood collects in dilated splanchnic vessels. In the initial stages the total blood volume is normal but the circulating blood volume is greatly reduced. In later stages there is reduction of total blood volume and irreversible shock develops as the peripheral circulatory failure progresses to the hematogenic type.

Common causes are hypocalcemia, septic shock, endotoxic shock, acute intestinal accidents, severe hemorrhage, and severe dehydration.

Clinical signs are general depression, weakness, fall in body temperature, increase in heart rate with abnormalities of the pulse, and increased shallow respiration. Nervous signs, including depression, listlessness, and coma are also observed. Blood examination usually shows eosinopenia, lymphocytopenia, thrombocytopenia, and hyperkalemia. Metabolic and lactic acidosis is also evident.

Treatment comprises of restoration of circulating blood volume. In hematogenic failure lost fluids should be replaced, the type of replacement depends on how the loss has occurred; in shock, plasma is required; in dehydration, isotonic fluids, and in hemorrhage, whole blood. Large quantities of fluid may be required to restore circulating blood volume. Disturbances in acid-base status are usually corrected by reestablishment of adequate tissue perfusion, however, where there is marked lactic acidosis specific bicarbonate therapy should also be instituted. A large dose of corticosteroids may be beneficial. The cyclooxygenase inhibitor flunixin megalamine, and phenylbutazone may be of value in endotoxic shock.

CARDIOMYOPATHY

A large number of bacterial, viral, parasitic diseases and nutritional deficiencies can cause cardiomyopathies. Calcific cardiomyopathy has been described in camels (Finlayson et al., 1971). It is caused by bacteremia (navel ill), clostridial infections, sarcosporidiosis, chronic copper deficiency, and poisoning by

selenium, arsenic, mercury, phosphorus, lithium, xylazine, monensin, gossipol from cotton seed cake, and some plants. It can also occur due to embolic infarction. In most of these conditions myocardial involvement is part of the total spectrum, although, cardiac manifestation may be clinically predominant.

In early cases, a decreased exercise tolerance is the usual initial presenting sign. This is usually accompanied by an increase in heart rate and heart size. There may be clinically recognizable abnormalities of rate and rhythm, particularly tachyarrthmia associated with multiple ventricular extra systoles. In cases with more severe myocardial damage, there may be sudden death or attacks of cardiac syncope due to acute heart failure, or severe dyspnea and general edema due to congestive heart failure.

Electrocardiography gives a good indication of the status of the myocardium even though the type of lesion cannot be diagnosed. Elevated ALT, AST, and lactic dehydrogenase may be of diagnostic significance in cardiomyopathies.

Treatment consists of dealing with the primary cause.

PERICARDITIS / TRAUMATIC PERICARDITIS

Perforation of pericardial sac by an infected foreign body occurs commonly in cattle, but it has also been reported in camel (Hegazy et al., 1995). Localization of a blood born infection of the pericardium occurs sporadically in many diseases such as pasteurellosis, colibacillosis, Pseudomonas aerogenosa, and streptococcus infection. Direct extension of infection from pleurisy or myocarditis may also occur.

Said (1963) induced traumatic pericarditis experimentally in a camel. The percentage of ingestion of sharp metallic foreign objects is very low in camels (Purohit et al., 1982). Several factors may affect the low prevalence of traumatic pericarditis in camels. It can be grazing nature and anatomy of mouth and lips (Higgins, 1980) and selective prehension by lips (Gahlot and Chouhan, 1992). The relative location of reticulum and heart is also a factor, which prevents the occurrence of traumatic pericarditis in camels (Smuts and Bezuidenhout, 1987).

Hegazy et al. (1995) have reported traumatic pericarditis in a 3-year old male camel at autopsy. They observed that the animal was unable to stand after repeated attempts to make him stand, so the camel was slaughtered. Postmortem examination revealed severe emaciation, hydrothorax, hydroperitoneum, and heart was greatly hypertrophied with thickened pericardium resulting from enormous fibrin deposition. The pericardial sac contained serofibrinous exudate. Ventricular hypertrophy and subendocardial hemorrhages were seen. There were clear adhesions between the reticulum, diaphragm and heart. A cord like fibrinous sinus tract, about 2 cm in length connecting the reticulum with the pericardium near the apex of the heart was observed. Sharp foreign bodies were not detected. However, there were about 7 blunt undigested hard desert fruits and one plastic bag in the reticulum or the rumen contents.

SPONTANEOUS ATHEROSCLEROSIS

Atherosclerosis is focal thickening of the tunica intimidalis of arteries due to deposition of lipids. Ezzi and Zakarian (1979) have reported this condition, in camels from Iran. They found the prevalence of condition to be 1.5% in 200 camels.

KAPALI

It is an Indian term for a condition in which there is specific inflammation of the facial vein (Rathore, 1986). The infection sometimes spreads to the orbital cavity and consequently results in the formation of supraorbital and retrobulbular abscesses. Fever and anorexia accompany the condition. The affected vein becomes

very prominent and there is diffuse swelling of the face, exophthalmia, and the animal keeps its head high to relieve pain. If untreated the infection may spread to the brain, causing death of the animal. Sometimes, the condition has been seen associated with neglected gangrene around the nose peg.

For the treatment, affected eye should be removed and some suitable antibiotic should be given by injection, along with local antiseptic treatment.

HEMORRHAGIC DISEASE

A condition associated with acidosis, mostly observed in racing camels of both sexes, known as hemorrhagic disease (HD) is prevalent in UAE (Wernary et al., 1992). Camels between 2-3 years of age are usually affected. This condition prevails during the hot and humid summer months. Several animals in a herd can be affected.

Etiology

The exact cause of disease is not known. Much aerobic bacterial growth is seen in the tissue samples. The predominent bacteria isolated were: E. coli, Pseudomonas aerogenosa, Proteus spp., Klebisiella pneumoniae,Staphylococcus aureus and other species of Staphylococcus, and Diplococcus.Clostridium perfringens (type A) was olso isolated from various organs and intestinal tract contents in camels which had died from HD. In two cases Aspergillus fumigatus was isolated from macerated lung and ruminal contents (Wernary et al., 1992). El-Khouly et al. (1992) observed Aspergillus fumigatus in the smears from trachea, bronchi, and bronchioles of some necropsied animals. No viruses have so far been isolated from the camels suffering from HD. According to Wernary et al. (1992) Bacillus cerreus and its toxins, which are already present in fresh alfalfa plants when fed to camels may be responsible for HD.

It is quite difficult to differentiate between Clostridium perfringens and HD. Both diseases usually start with acidosis. Stress of early training, heat, and severe training could be an exciting cause of the disease.

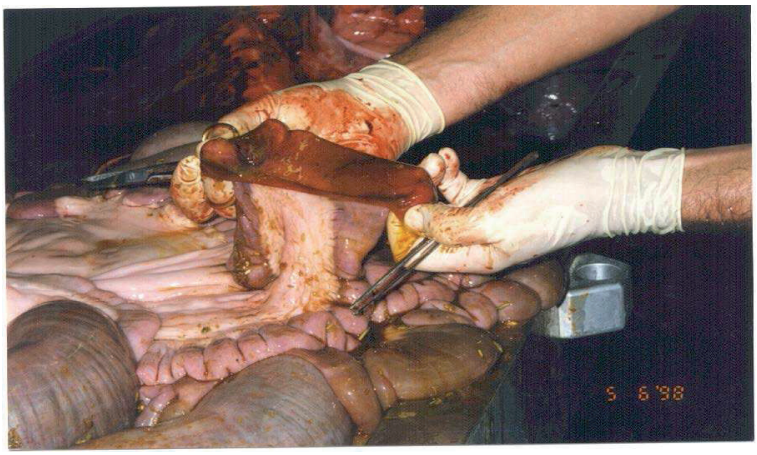
Clinical Findings

Affected animals are off feed, depressed and have fever upto 41^{o}C. During early stages some camels have cough, and a marked uni or bilateral enlargement of the submandibular lymph nodes. There is complete ruminal stasis with gurgitation and abdominal pain. By the third day, blood coated whole fecal droppings could be removed from the rectum. No diarrhea is apparent although in some animals, unclotted blood is seen to be dripping from the anus. The animals become recumbent 2-3 days before death at which time most animals develop lacrimation and salivation. Some animals may develop nervous signs, like muscle fasciculation, head shaking, and opisthotonus, before death. There is marked leukopenia and massive increase in AST, ALT, GGT, ALP, CK, and LDH. The pH of the ruminal fluid of the necropsied animals ranges from 4-5.

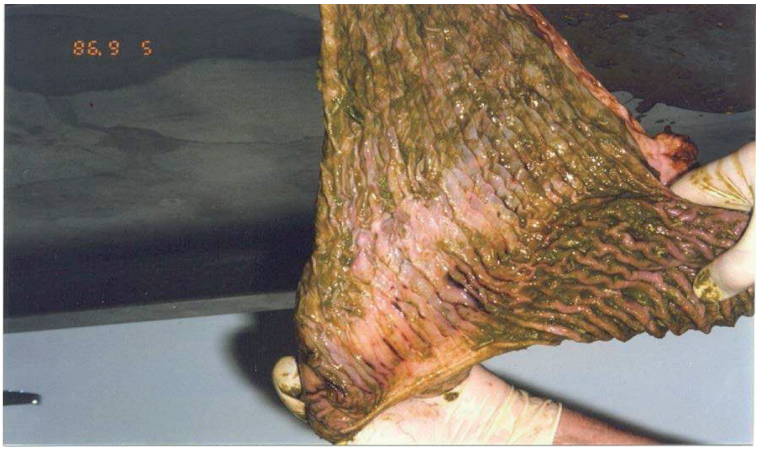
At necropsy hemorrhages of varying sizes and severity are seen in the mucous membrane of pharynx, trachea, abomasum (some times there is ulceration), intestines (mainly ascending colon), and renal pelvis. Hemorrhages are also seen on the epicardium and under the endocardium. Lymph nodes are enlarged and contain hemorrhages of varying sizes. Corresponding to gross changes histopathological changes observed in the lymphoid tissue include necrosis of follicular centres and hemorrhages of varying sizes. There is loss of lymphocytes. Hemorrhages of varying sizes are seen in many tissues of the body.

Treatment

If the disease is diagnosed in early stages on the basis of clinical signs and clinical pathology the prognosis is favorable. Treatment should be focussed upon neutralization of ruminal acidity through administration of antacids. Sodium bicarbonate (500 gm given twice daily per os) should be commenced immediately. Antibiotics are also considered to be effective by some veterinarians. Administration of isotonic intravenous electrolytes with vitamins and glucose are also helpful. It is recommended to store alfalfa in a way that heat within it should not reach at a point for maximal growth of bacteria and fungi. As soon as the bundles are recieved on the farm it should be spread out on racks (Manefield and Tinson, 1996).

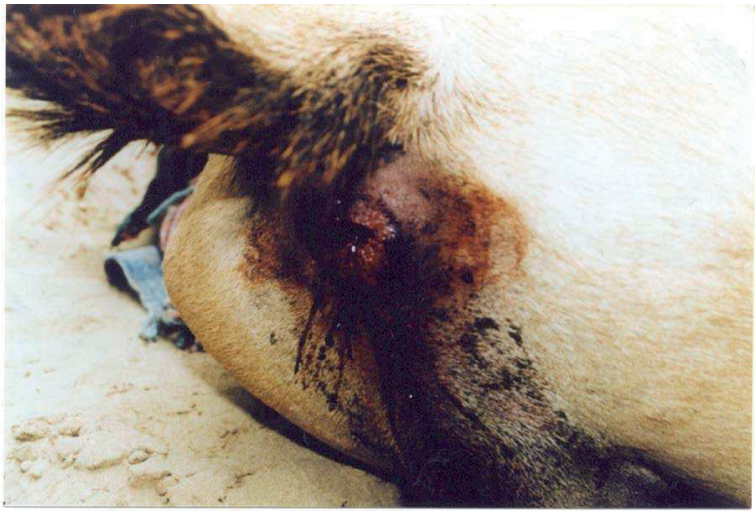


Intestine full of blood-mixed feces

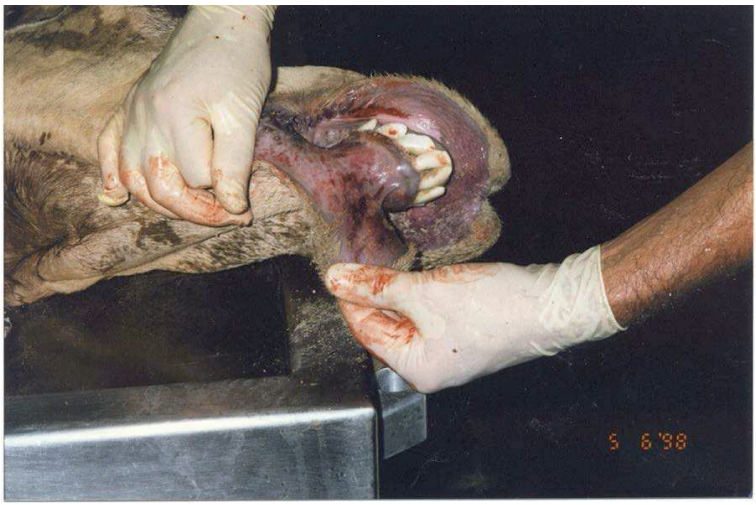


Streaks of blood mixed with ingesta in abomasum

Hemorrhagic lesions in the heart



Tarry feces



Ulcerative lesions under the lips

Unchecked bleeding after removal of canula. Blood does not clot.

EDEMA

Edema, excessive accumulation of fluid in the tissues, can affect camels like other animals. It can be local or general.

Etiology

It can result from increased hydrostatic pressure in the capillaries (congestive heart failure, portal hypertension due to hepatic fibrosis causing ascites, compression of mammary veins by the large fetus causing mammary or ventral edema in late pregnancy). It can occur due to decreased plasma osmotic pressure (hypoproteinemia) due to continued blood loss due to parasitic infestations, renal disease causing continued protein loss, liver damage causing failure of synthesis of plasma proteins, and malnutrition. It can also occur due to obstruction of lymphatic flow (due to inflammation and pressure of tumors etc.) and vascular damage to small vessels (allergic edema, toxic damage in anthrax, gas gangrene, colibacillosis (Ibrahim et al., 1998), certain plant poisons, and trypanosomiasis).

Clinical Findings

Accumulation of edema transudate in the subcutaneous tissues (anasarca) is usually confined to the ventral wall of the abdomen and thorax. Edematous swellings are soft, painless and pit on pressure. In ascites there is distension of the abdomen and the fluid can be detected by a fluid thrill on tactile percussion. In the fluid accumulation in the pleural cavity (hydrothorax) pericardial sac (hydropericardium) the clinical signs produced include restriction to cardiac movements, embarrassment of respiration, and collapse of the ventral part of lungs.

Localized edemas cause localized signs; pulmonary edema is accompanied by respiratory distress; cerebral edema is manifested by severe nervous signs.

Treatment

The treatment should be focused at correcting the primary cause. In protein deficiency edema the animal should be given protein rich food. Administration of intravenous amino acids may also help. Diuretics help in relieving the symptoms. Aspiration of fluid is sometimes indicated to relieve excessive pressure, which should be done very carefully to prevent shock.

ANEMIA

Anemia is defined as deficiency of erythrocytes and / or hemoglobin. It can occur due to hemorrhage and intravascular hemolysis. It can also be caused due to decrease in the production of the erythrocytes in the hemopoetic tissue.

Hemorrhages can occur due to spontaneous rupture or traumatic injury to large blood vessels. Important causes of hemorrhagic diseases in camels include: Thrombocytopenia induced by drugs that cause bone marrow suppression or premature destruction of circulating platelets due to probably, mycotoxins; massive haemonchus infestation; gastric ulcers in racing camels; heavy tick and sucking lice infestation; pulmonary hemorrhage in racing camels; bleeding from the kidneys, ureters, and urethra due to infection or calculi; coccidiosis; hemorrhagic diathesis, which is believed to be caused by toxins in connection with acidosis (Wernery et al., 1992).

Hemolytic anemia is caused by trypanosomiasis, poisoning by kitchen waste (onions, tomatoes etc.), treatment with long acting oxytetracycline, and due to biting by some snakes.

Anemia due to decreased production of erythrocytes or hemoglobin is observed in nutritional deficiencies, chronic diseases, or depression of hemopoitic tissue by drugs (chloramphenicol) and exposure to radiation. Nutritional deficiencies include cobalt, copper, iron, pyridoxine, and folic acid deficiency. Chronic diseases causing anemia include suppurative processes.

Paleness of the mucous membranes is the out standing clinical sign, but appreciable degree of anemia can occur before this change is noticed. The anemia affects the performance of racing camels. In clinical cases of anemia there is muscular weakness, depression, anorexia, increased heart rate, the pulse has a large aptitude, and the absolute intensity of the heart sounds is markedly increased. Clinical signs do not appear until the hemoglobin level of the blood falls below about 50% of normal. The erythrocytic count and hematocrit are usually decreased. In hemorrhagic and hemolytic anemias there is an increased number of immature red cells in the blood. In hemorrhagic anemia there is fall in total serum protein. In hemolytic anemia plasma is discolored by hemoglobin. The anemia in iron deficiency is microcytic and hypochromic. In vitamin B deficiency anemias the number of immature erythrocytes increase in the circulation. In copper deficiency anemia number of nucleated erythrocytes increase in the blood (Wernery et al., 1999).

Treatment of the primary cause of the anemia is essential. In acute hemorrhagic anemia blood transfusion is recommended.

LYMPHOBLASTIC LEUKEMIA

Afzal and Hussein (1995) have reported lymphoblastic leukemia in camels. Some cases have been observed in Central Veterinary Laboratory, Dubai. In our laboratory (Veterinary laboratory, Abu Dhabi Municipality), ten cases of lymphocytic leukemia have been diagnosed in a period of seven years. Chouhan et al. (1986) were not able to find antibodies to enzootic bovine lekcosis (EBL), from sera of camels, from India. Wernery and Wernery (1990) also found sera of 986 camels negative for EBL. Wernery and Kaaden (1995) reported that sera from all the cases of camel lymphoblastic leukemia to be negative to EBL. The etiology of the disease is not known. The condition is usually fatal. At autopsy enlargement of the lymph nodes and cachexia was consistent in all the cases died of lymphoblastic leukemia.

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NERVOUS SYSTEM

The structure of nervous system of camel is almost same as that of other ruminants. Smuts and Bezuidenhout (1987) have given good description of the structure of nervous system of dromedary.

Discrete lesions of the central nervous system resulting in well-defined neurological signs are not common in camels. Most of the nervous diseases encountered are characterized by diffuse lesions caused by viruses, bacteria, toxins, nutritional disorders and embryological defects. The camel practitioners should look for whether the animal has meningoencephalitis, cerebral edema, increased intracranial fluid as occurs in vitamin A deficiency, or whether the lesion is at the neuromuscular junction as occurs in tetany due to magnesium deficiency. Examination of cerebrospinal fluid is a great tool in large animals for the diagnosis of the nature of nervous system diseases.

Because of limitations in the neurological examination of camels, there must be more emphasis on the history and epidemiological findings. Many of the diseases have epidemiological characteristics which give the clinician a clue to the possible causes.

The functions of the nervous system are directed at the maintenance of the body's spatial relation with its environment. Several divisions of the nervous system perform those functions including: (a) the sensory motor system responsible for the maintenance of normal posture and gait; (b) autonomic nervous system controlling the activity of smooth muscles and endocrine glands, and thereby the internal environment of the body; sensory system of special senses; (c) and the psychic system which controls the animal's mental state. The nervous system itself is not independent of other organs and its functional capacity is regulated to a large extent by the function of the other systems, particularly the cardiovascular system. Hypoxia (anoxia), due to cardiovascular disease, quite commonly leads to altered cerebral function because of the dependence of the brain on an adequate oxygen supply.

Nervous tissue is limited in the ways in which it can respond to noxious influences because of its essentially coordinating function. The transmission of impulses along nerve fibers can be enhanced or depressed in varying degrees, the extreme degree being complete failure of transmission. Increased activity of the reactor organ occurs because of an increase in the nerve impulses received either because of excitation of neurons or by facilitation of the passage of stimuli. The excitability of nerve cells can be increased by many factors including stimulant drugs, inflammation, and mild degree of those influences which in a more severe form may cause depression or excitability. Thus early or mild anoxia may result in increased excitability while continued or severe anoxia will cause depression of function or even death of nerve cells. Hypoglycemia also may cause increased excitability, as manifested by hypoglycemic convulsions in insulin therapy, but if sufficiently severe or prolonged causes a fatal hypoglycemic encephalopathy. Irritation phenomenon may result from many causes including inflammation of nervous tissue caused by bacteria or viruses, certain nerve poisons, and anoxia. In

those diseases which cause an increase in pressure within the cranial cavity, irritation phenomenon result from interference with circulation and development of local anemic anoxia. The major manifestation of nerve excitability is in the form of convulsions, muscle tremors, hyperesthesia, and paresthesia. The area of increased excitability may be local or generalized. A local lesion in the brain may cause signs of excitatory nervous dysfunction in one limb and a more extensive lesion may cause complete convulsions. Exaggeration of normal nervous system activity occurs when lower nervous centres are released from the inhibitory effect of higher centres. Cerebellar ataxia is a classical example. In the absence of cerebellar control combined limb movements are exaggerated in all modes of action including rate, range, force and direction. In general, release phenomenon are present constantly while the causative lesion operates, whereas excitatory phenomena fluctuate with building up and exhaustion of energy in nerve cells. Excitation states include mania and frenzy. In mania the animal acts in a bizarre way and appears to be unaware of its surroundings. The animals lick, chew or even eat the foreign material, bite themselves and other animals persons or objects. There is abnormal voice, constant bellowing, apparent blindness, walking in circles, drunken gait and aggressiveness. Diseases characterized by mania include: encephalitis e.g. furious rabies; degenerative disease of brain, e.g., early polioencephalomalacia, poisoning by Astragalus sp; toxic and metabolic diseases of brain, e.g. nervous acetonemia, acute lead poisoning, poisoning with carbon tetrachloride, severe hepatic insufficiency. Frenzy is characterized by violent activity with little regard for surroundings. The movements of the animal in frenzy are uncontrolled and dangerous (Blood and Radostitis, 1989).

Depressive mental states include somnolence, lassitude, nacrolepsy/catalepsy, syncope and coma. These conditions are caused by those influences which depress nervous system function generally.

Coma may result from: encephalomyelitis and encephalomalacia, toxic and metabolic diseases such as uremia, hypoglycemia, hepatic insufficiency, toxemia, septicemia and most toxins which damage tissues generally. Hypoxia of brain due to peripheral circulatory failure, milk fever, and heat stroke, specific poisons which cause somnolence e.g. bromides.

Syncope (sudden fainting) may occur due to acute heart failure leading to acute cerebral anoxia, spontaneous hemorrhage, traumatic concussion or contusion, lightening stroke and electrocution.

Narcolepsy (catalepsy) is a condition in which animal experiences episodes of uncontrollable sleep. It has not been reported in camels.

INVOLUNTARY MOVEMENTS

Convulsions

Convulsions are violent muscular contractions affecting part or all of the body and occur for relatively short period. They may be clonic, in which repeated muscle spasms alternate with periods of relaxation. Tetanic or tonic convulsions are less common and are manifested by prolonged muscular spasms. The latter are found in strychnine poisoning and in tetanus. Convulsions can result from disturbances anywhere in the proencephalon, including cerebrum, thalamus or even hypothalamus. However, the initiating cause may be in the nervous system out side the cranium or in some other systems, so that the convulsions can be intracranial or extracranial.

Intracranial convulsions can occur in meningitis, encephalomyelitis, encephalomalacia, acute brain edema, brain ischemia, local lesions caused by trauma

(concussion, contusion), abscesses, tumors, parasitic injury, and hemorrhage in the brain.

Extracranial convulsions are caused by brain hypoxia in acute cardiac failure, toxic and metabolic diseases (hepatic encephalopathy, hypomagnesemia, inorganic poisons, poisonous plants and fungi, and bacterial toxins).

Tremors

It is continuous, visible, palpable repetitive twitching of skeletal muscles and is indicative of cerebral involvement. True tremor is often severe enough to cause incoordination. The tremor may become intensified when animal starts some action. This condition is caused in toxic and metabolic diseases due to a number of poisons, especially poisonous plants and fungi, probably by some bacterial toxins, and occasionally in metabolic defects, such as hyperkalemia.

Posture and gait

One of the major clinical syndromes encountered in camel is ataxia or incoordination of gait. This condition is encountered in toxemia caused by bacterial, fungal and plant toxins, and in acidosis.

Paralysis

The motor nervous system comprises pyramidal tracts, which originate in the motor cortex, the extrapyramidal system originating in the corpus striatum, red nucleus, the vestibular nucleus and the roof of the mid brain.

DIFFUSE DISEASES OF THE BRAIN

CEREBRAL ANOXIA

An acute or chronic syndrome caused by oxygen deprivation to the brain is exhibited initially by excitement, followed by depression and loss of function.

Etiology

This condition may be caused by poisoning by hydrocyanic acid or nitrite, terminal stages of pneumonia, congestive heart failure in calves born after prolonged parturition and increased intracranial pressure, and edema due to intracranial lesions.

Clinical Findings

The central nervous system is extremely sensitive to anoxia and even after few minutes deprivation of oxygen degeneration of brain cells ensues. Acute cerebral anoxia is manifested by a sudden onset of signs referable to paralysis of all brain functions; including flaccid paralysis and unconsciousness. There are muscle tremors, which start from the head and spread to the trunk and limbs, followed by recumbency, clonic convulsions and death or recovery. In chronic anoxia there is lethargy, dullness, ataxia, weakness, blindness and in some cases muscle tremor or convulsions. In both acute and chronic anoxia the signs of the primary disease are also evident.

Treatment

The supply of oxygen is badly needed and could only be provided by removing the cause. The respiratory stimulants may help in acute cases and artificial respiration may keep the animal alive.

HYDROCEPHALUS

It causes increase in intracranial pressure resulting in irritation. Signs such as mania, head pressing, muscle tremor, and convulsions are seen when the onset is rapid. Signs of paralysis including dullness, blindness, and muscular weakness are noticed when the condition develops gradually.

Etiology

The condition may be congenital or acquired. In congenital hydrocephalus the causes may be viruses, vitamin A deficiency and unknown factors. Acquired hydrocephalus may occur due to hypovitaminosis A in young calves, tumors and chronic inflammatory lesions.

Clinical Findings

Due to increased intracranial pressure, in most of the cases, there is gradual onset of general paralysis. In the beginning of the disease the animal is depressed, shows disinclination to move, an expressionless stare blindness, and incoordination of movements. It follows the stage of somnolence and the animal has slow reflexes.

Congenitally affected animals are usually alive at birth but are unable to stand and most of them die within 48 hours. The cranium is sometimes domed, the eyes protrude and nystagmus is often evident. Congenital hydrocephalus may be mistaken for vitamin A deficiency, toxoplasmosis, and hydroencephaly if there is no distortion of the cranium. Recently, a case of hydrocephalus has been reported in camel calf by Abubakar et al. (1998)

Acquired hydrocephalus needs to be differentiated from other diffuse diseases of the brain, including encephalitis and encephalomalacia and from hepatic dystrophies.

DIFFUSE EDEMA OF THE BRAIN

It occurs rarely as a primary disease. Mostly, it accompanies other diseases and usually its occurrence is transient. It may terminate fatally, complete recovery or recovery with residual signs.

Etiology

It accompanies brain abscesses, hemorrhage, lead induced encephalopathy, purulent meningitis, hypoxia, polioencephalomalacia, and hydrocephalus.

Clinical Findings

The development of clinical signs takes place over a period of 12-24 hours and cerebral shock does not occur. There is blindness, and periodic attacks of opisthotonus, nystagmus, muscle tremor and convulsions. In the intervening periods the animal is dull, depressed, and blind. Papilledema can be observed on ophthalmoscopic examination. Diffuse brain edema causes a syndrome not unlike that of encephalitis although there are fewer irritation phenomenon.

Treatment

Decompression of the brain is desirable in acute edema. The treatment will depend in part on the cause. The edema associated with polio-encephalomalacia will respond to early treatment with thiamine. In general terms, edema of the brain responds to parenteral treatment with hypertonic solutions and corticosteroids. Mannitol at 2 g/kg body weight and dexamethasone at 1 mg/kg body weight, both, I/V, are advised. The mannitol is given as a 20% solution I/V followed 3 hours later by dexamethasone. Diuretics may help in the chronic cases.

ENCEPHALITIS

It is inflammation of brain and is characterized by initial irritation signs followed by signs of depression due to loss of nervous functions.

Etiology

Encephalitis is encountered in rabies (Bildfell, 1996), toxoplasmosis, and sarcocystosis. It may also occur due to some bacteria e.g., Streptococcus zooepidermicus.

Clinical Signs

The clinical signs of encephalitis are usually referable to the general stimulatory or lethal affect on neurons in the brain. This may be in part due to the general effect of inflammatory edema and partly due to the direct effect of the agent on nerve cells. There is sometimes fever accompanied by anorexia depression and increased heart rate. There may be initial periodic episodes of hyperesthesia including blind charging, bellowing and pawing. Mental depression, including head pressing, may occur between episodes. Signs of irritation are very variable in their occurrence and may not appear at all. The loss of function varies in degree from paresis with knuckling at the lower limb joint, to spasticity of the limbs with resultant ataxia, to complete paralysis. More restricted paresis do occur and may be manifested by deviation of the head, walking in circles, abnormalities of posture, ataxia and incoordination but these are more commonly residual signs after recovery from the acute stages. A case of female camel suffering from infection due to Streptococcus zooepidermicus, showing signs of congestion of the brain have been reported by Ibrahim et al. (1998). Bildfell (1996) has reported a case of camel showing nervous signs prior to death. The microscopic lesions found were nonsuppurative meningo-encephalitis with vasculitis and extensive necrosis and edema. Intranuclear inclusion bodies typical of Herpes virus were observed in neurones and glial cells.

Treatment

Specific treatment is being dealt with under each disease condition. Supportive treatment by intravenous glucose should be given in acute phase of the disease. Sedation during the excitement stage and nervous system stimulants during the period of depression may maintain life through the critical phase.

ENCEPHALOMALCIA

It is degeneration or softening of brain. Leukoencephalomalacia and polioencephalomalacia refer to softening of white matter and gray matter, respectively. Polioencephalomalacia is very commonly observed in racing camels, fed high concentrate feed (Manefield and Tinson, 1996).

Etiology

Encephalomalacia occurs as a consequence of liver damage, which results in high blood ammonia. It may occur in poisoning by mercury, lead, selenium, and plant poisons. Clostridium perfringens type D toxins can cause this condition. Thiamine deficiency also causes polioencephalomalacia.

Clinical Findings

Paralysis of varying degree accompanied by dullness or somnolence, blindness, ataxia, head pressing, circling, and terminal coma. In the early stages,

particularly in polioencephalomalacia, there are irritation signs including muscle tremor, opisthotonus, nystagmus, and convulsions.

Treatment

In grain fed racing camels thiamine injected in the early stages may be helpful. Encephalomalacia is usually irreversible and no treatment is recommended.

TRAUMATIC BRAIN INJURY

It results either due to direct trauma applied from exterior or flexing or stretching of the head or neck or by migrating parasitic larvae, internally. The effect of trauma varies with site and extent of injury. There are chances of nervous shock in the beginning, which could follow either by death or recovery, or the persistence of residual signs.

Clinical Findings

With severe injury there is cerebral shock in which the animal falls unconscious with or without transient clonic convulsions. Consciousness may never be regained, but in animals that recover it returns, within a few minutes to several hours. During the period of unconsciousness, clinical examination reveals dilatation of the pupils, absence of the eye perception and pupillary light reflexes, and a slow irregular respiration. Bleeding from the nose or ears may be present. The site of injury may be revealed by palpation of the cranium. Residual signs vary a great deal. If the optic cortex is damaged blindness occurs. In the mild brain lesions hemiplagia may occur. Traumatic epilepsy may occur with lesions in the motor cortex. The other signs of severe trauma to the brain include opisthotonus with blindness and nystagmus, and if the brainstem has been damaged, quadriplegia. There may also be localized signs including head rotation, circling and falling backwards.

Treatment

If the consciousness is regained within few hours the prognosis is favorable and in this case treatment may not be necessary, however, when coma lasts for more than 6 hours the prognosis is not favorable and the slaughter of the affected animal is recommended. In the case of valuable animals, treatment outlined in the treatment of brain edema is advised. The animals with prolonged coma (more than 12 hours following treatment) are unlikely to improve.

FOCAL DISEASES OF THE BRAIN

BRAIN ABSCESSES

Abscesses are encountered more in young camels as compared to old ones. The clinical signs vary depending upon the location and size of the abscess.

Etiology

The common way of spread of infection to the brain is through the circulation. However, direct spread from injury to the skull or nasopharynx may occur. The organisms causing brain abscesses may be Mycobacterium tuberculosis, Actinobacilli, Streptococci, Fusobacterium necrophorum, Pseudomonas, Staphylococci, Actinomyces, and Listeria.

Clinical Findings

The animal is depressed, presses its head and, may show blindness (from one or both eyes). There are transient attacks of motor irritation, during which the animal is very much excited and is unmanageable and shows signs of convulsions.

There may be rise in temperature. Nystagmus is common when the abscess is near vestibular nucleus. There may be cerebral ataxia, deviation of the head with circling and falling, hemiplegia or paralysis of individual or groups of cranial nerves often in a unilateral pattern. In the later stages there may develop edema of brain or hemorrhage in the brain.

Treatment

Parenteral administration of broad-spectrum antibiotics is suggested, but the results are usually not satisfactory.

Encephalomalcia caused by Clostridium perfringes Type-D toxins

COENUROSIS (GID)

It results from the presence of cysts (intermediate stage) of Taenia multiceps in the brain or spinal cord. The clinical signs manifested are those of space-occupying localized lesions of central nervous system.

Etiology

The disease is caused by the invasion of nervous tissue by Coenurus cerebralis. It is intermediate stage of Taenia multiceps, which inhabits the intestine of dogs and wild Canindae. Consumption of feed contaminated by the feces of infested dogs, hatch in the intestine, liberating embryoes, which pass into the blood stream. Only those embryos, which lodge in the brain or spinal cord, survive and continue to grow to the coenurid stage. Coenurus cerebralis can mature in the brain and spinal cords of sheep, goats, cattle, horse, camel and wild ruminants and occasionally, in man.

Clinical Findings

The early stages of larval migration (in light infestation) through nervous tissue usually pass unnoticed, but in heavy infestation encephalitis may be produced. It takes 6-8 months for mature Coenurus to develop to a size of about 5 cm, after which usually the signs of the disease develop. The symptoms include, in the beginning, wild expression, salivation, frenzy and convulsions. Deviation of eyes and head may also occur. Some animals may die at this stage but others develop partial or complete blindness of one or both eyes, dullness, clumsiness, head pressing, ataxia, incomplete mastication and periodic epileptic convulsions. Deviation of head and circling towards the blind eye may be present.

Treatment and Control

Surgical drainage or removal of the cysts may be attempted in valuable animals. The control of the disease is possible by treating the dogs for tape worms, and eradication of stray dogs.

MENINGITIS

In most of the cases, it is caused by bacterial infections, and is exhibited by fever, hyperaesthesia, and muscular stiffness. Singh et al. (1995), and Saini and Sreemannarayan (1980) have reported its occurrence in camels.

Etiology

The organisms responsible are Listeria, Pasteurella, and Staphylococcal sp. Toxoplasma gonadii may cause lesions on the meninges. In the young animals some species of streptococci and E. coli may also cause the disease.

Clinical Findings

Acute meningitis usually develops suddenly and is accompanied by fever toxemia and nervous signs. There is trismus, opisthotonus and rigidity of neck and back. Motor irritation signs (tonic spasms of the muscles of the neck, muscle tremors and paddling movements) and varying degree of hyperesthesia may also be evident. There is evidence of irritability (mania) in the early stages, followed by drowsiness and coma. Blindness may occur in some cases.

In camels the meningitis is usually diffuse, however, it may be localized in some cases. In this case localizing signs including involvement of the cranial or spinal nerves, localized muscle tremors, hyperesthsia or rigidity may result.

Treatment

Parenteral antibiotics for several days are used as treatment. Chloramphenicol is the drug of choice in meningitis. If the animal does not respond to the treatment within 3 days, the prognosis is unfavorable. The bacterial inhibitory therapeutic levels may not be attained in young camels. The drug of choice in the latter cases is cephalosporins, which resist hydrolysis by beta-lactamase, and easily penetrates into CSF and is bactericidal at very low concentrations. Moxalactam and cefotaxime are widely used in human beings for the treatment of Gram negative bacillary meningitis.

TOXIC AND METABOLIC DISEASES OF THE NERVOUS SYSTEM

A large number of plant poisons, farm chemicals and some metabolic defects cause abnormalities of the function of nervous system.

The factors which affect the consciousness and behavior of the animal are: hypoglycemia and ketonemia/acetonemia, hypomagnesemia, high blood levels of

ammonia in hepatic insufficiency, uremia, exogenous toxins including carbon tetrachloride, hexachlorethane, trichlorethane, cyanide or nitrite poisoning.

Convulsions may result due to: hypoglycemia; hypomagneseimia, deficiencies of vitamin A, pyridoxine, inorganic poisons including lead, mercury, farm chemical such as arsenicals. organophasphates, chlorinated hydrocarbons, strychnine, urea, metaldehyde, bacterial toxins, including Clostridium tetani, Clostridium perfringens type D, fungal toxins e.g., Penicillium cyclopriumm; some green weeds and trees.

Tremors and ataxia are caused due to some weeds, bacterial toxins, and fungi. Many toxic substances and metabolic defects listed above cause ataxia when their influence is mild and paralysis when it is severe

TRAUMATIC INJURIES TO THE SPINAL CORD

There occurs a brief complete flaccid paralysis caudal to the injury in sudden severe trauma to the spinal cord due to spinal shock. This soon follows to flaccid paralysis in the area supplied by the injured segment and apastic paralysis caudal to it.

Etiology

The spinal cord can be injured when the camels fall off the vehicles. It is common in osteoporotic or oesteodystrophic animals, especially aged breeding camels while jumping or leaning on fences. Spondylosis and fracture in old bull camels while matting and some migrating parasitic larvae are also potential cause of spinal cord injury.

Clinical Findings

The lesions caused by traumatic injuries to the spinal cord consist of disruption of nervous tissue or its compression by displaced bone or hematoma. Minor degrees of damage may result in local edema or hyperemia, or in the absence of microscopic lesions, transitory injury to the nerve cells classified as concussions. The spinal cord injury reflects itself by flaccid paralysis and there is concurrent fall in local blood pressure due to vasodilation and there may be local sweating. Stretch and flexor reflexes and cutaneous sensitivity disappear but reappear within a few hours. The animal is unable to rise in most of the cases due to lose of control over the extremities. The muscles controlling respiration may also be affected causing difficulty in respiration. The area supplied by the segment will eventually show flaccid paralysis and muscle atrophy.

Treatment

Caring of affected animal on thick bedding with frequent turning, and message of bony prominences may help the animals, having a minor lesion, to recover. In most of the cases prognosis is not favorable.

PERONEAL NERVE PARALYSIS

Nassef (1996) has reported a case of seven-year-old camel with knuckling of fetlock of right hind limb. The paralysis occurred due to injury to peroneal nerve due to injection given by large bore needle by an inexperienced person. In this camel the hock was dropped. Loss of sensation was observed on the dorsum of the digit.

FACIAL PARALYSIS

This condition is frequently observed in camels, especially male camels. It is because of their long neck. The face gets injured when it is violently turned to one

side and it strikes to some hard or sharp object. It is always one sided. There are several reports on the paralysis of face. Lower lip paralysis has been reported by Leese (1927), and Purohit et al. (1988); tongue paralysis by Gahlot et al. (1989); one sided facial paralysis by Schwartz and Dioli (1992), Stanic et al. (1972), and Singh et al. (1995). The cause of paralysis is reported by the latter workers to be due to injury followed by infection. The symptoms reported are loss of reflexes and flaccid paralysis of the muscles of the affected area. Dropping of the ears, eyelid and cheek of the affected side may be observed.

OTHER CONDITIONS RESULTING IN NERVOUS SIGNS

Due to shortage of food the camels are compelled to eat **poisonous plants**. A plant from Africa "*Capparis tomentosa*" when consumed by animals results in neurological disorders such as twisting of the neck, progressive loss of condition, paraplegia, and convulsions (Schwartz and Dioli, 1992).

In Australia ironwood ingestion has been associated with staggering, star gazing, and apparent blindness (Manefield and Tinson, 1996).

Various nerve dearrangements and paraplagia has been reported to occur in camels due to **botulism** (Provost et al., 1975).

The **snake** having venom containing neurotoxins, when bites animals, including camel, causes initial excitement followed by depression and paralysis (Tagra and Yadev, 1998). **Scorpion** bitten camels hold their head and neck high (Manefield and Tinson, 1996).

The camels also can get affected by **wryneck syndrome**. The characteristic clinical finding is a S-shaped deformation. The onset of the disease is acute and spontaneous. The recovery can occur without any treatment. The sick animals respond to vitamin B complex. The etiology is not known (Schwartz and Dioli, 1992).

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URINARY SYSTEM

The kidneys in dromedary camel are bean shaped and smooth externally. The right kidney is more elongated than the left. They are located against the dorsal body wall and are retroperitoneal. The right kidney lies below the transverse processes of the second to the fourth lumber vertebrae. Its cranial pole is round and it fits into the renal impression of the caudate lobe of the liver. The caudal pole is not as rounded and is slightly flattened dorsoventrally. The left kidney is regular in shape and lies below the left transverse process of the last three lumber vertebrae. The hilus of each kidney faces medially and is well defined. The renal artery bifurcates before entering the hilus. The renal vein lies ventrally to the arteries. The ureter is directed caudally (Smuts and Bezuidenhout, 1987).

On section made through the hilus and the poles, the cortex and medulla are easily distinguished. There is well-developed crista renalis. Renal cortex comprises about 50% of the volume of the kidney and the thickness of medulla is four times than that of cortex. This is designed for the production of the hypertonic urine and preservation of water in the camel.

The ureter takes the usual retroperitoneal course. It enters the pelvic aperture medially to the round ligament of the bladder, and in the male dorsally and laterally to the deferent duct to pass along the genital fold or broad ligament. It pierces the wall of the bladder near the neck (Smuts and Bezuidenhout, 1987).

The alimentary canal of camel provides the fluid for the body, while the kidneys are responsible for retaining as much water as possible. The renal function of the camel is greatly depressed when the animal is dehydrated (Yagil and Berlyne, 1978). It has been calculated that the daily urine volume excreted by the dehydrated camel is 1/1000 of the animals weight (Macfarlane, 1977). In comparison, dehydrated sheep excretes 1/100-1/200 of their body weight. Urine of dehydrated camels is extremely concentrated, but it has tremendous capacity to return to normal within half an hour after drinking water.

The ability of camels to concentrate their urine is partly due to the structure of kidney itself. Camel kidneys have long loops of Henle, which enable the camel to reabsorb greater amount of water by concentration multiplier system. Daily volume of urine voided by the camel ranges from 1.1 to 2.2 liters (Siebert and Macfarlane, 1971). About 450 ml urine is excreted at each micturition (Schmidt- Neilsen et al., 1956). The water content of the feed affects drinking rate, amount of water intake, and volume of urine. More urine is excreted when camels eat fresh green fodder than when they eat dry food (Gautheir-Pilters and Dagg, 1981).

Salt is well handled by the camel kidneys. Charnot (1960) noted that the camel can excrete urine with a salt concentration almost twice that of sea water. This is valuable mechanism as the camel is exposed to fodder and water with high salt concentration. A peculiar variation occurs in the ratios of the salts excreted in the camel's urine. In most urine samples potassium is the dominant

ion excreted, but when the camel feeds on certain types of fodder, sodium can become dominant (Schmidt - Nielson et al., 1957). As urine flow is decreased there may be a change in the balance of ions excreted. Products of nitrogen metabolism are normally excreted in the form of urea by terrestrial animals. The urine concentration of this compound can reach high levels. As the volume of urine output decreases urea content increases.

In a study carried out by Yagil and Berlyne (1978) it was shown that in spring only marginal changes in the kidney functions occur in the dehydrated camel. The camel could urinate on the water available in the food. In the heat of summer significant changes in the renal function occur after water deprivation for 10 days. The glomerular filtration rate declines by 75% (from 0.81 ml/min./kg to 0.23 ml/min./kg). The renal plasma flow declined by 72% (from 5.5 ml/min./Kg to 1.5 ml/min./kg). Urine flow rate dropped by over 50% (from 3.3 ml/min. to 0.7 ml/min.).

Cattle have a higher basic glomerular filtration rate than camels, while sheep have glomerular filtration rate between those of cattle and camels. Cattle cannot concentrate their urine more than 200 osm/kg, compared with sheep which excrete urine with 3.5-3.8 osm/kg (Macfarlane, 1968) or camels which excrete urine with 2.8 osm/kg (Yagil and Berlyne, 1978). There appears to be discrepancy with the fact that on the one hand sheep lose more water via their urine than camels and on the other hand they excrete urine with a higher osmolality. It must be remembered that plasma volume of sheep is affected to a large degree than that of the camel and therefore sheep need to concentrate their urine to a greater degree. The camel is able to concentrate its urine because its kidneys respond a hundred times more readily to ADH than kidneys of cattle (Macfarlane, 1977).

After taking excess water, regulatory attenuation of vasopression secretion results in a water diuresis, which eliminates the danger of over hydration. The delayed water diuresis following rehydration (Zine Filali, 1987) or water-loading (Benlamlih et al., 1992) observed in camels might be caused by higher initial plasma arginine vasopressin (AVP) levels or a slower metabolic clearance rate of AVP. Plasma AVP levels in the camel (Yagil and Etzion, 1979) are similar to those in sheep and goats, both in hydrated and in dehydrated states (Olsson et al., 1982; Blair-West et al., 1985). In addition, the plasma half-life (11 $\pm$ 2 min) and the body clearance of exogenous AVP (13 $\pm$ 2 ml/min/kg) in camels are in the range as those reported in other mammals (Laurson, 1967).

According to Siebert and Macfarlane (1971) the prolonged antidiuretic activity before the onset of water diuresis is that camels are more sensitive to the antidiuretic action of vasopressin than ruminants.

After rehydration, plasma aldosterone concentration increases within 24 hours following given water to camels (Dahlborn et al. 1989), and renal sodium excretion falls (Zine Filali, 1987). This would attenuate the decrease in plasma osmolality and therefore the magnitude of water diuresis occurring after rehydration. Accordingly, water loading in normohydrated camels induces a rise in aldosterone concentration. On the other hand, saline loading lowered plasma renin activity and plasma aldosterone concentration (Benlamlih et al., 1992). However, despite the inhibition of the renin-angiotensin-aldosterone system, the saline loaded camels are not able to increase renal sodium excretion as efficiently as goats, probably because of their compartively low glomerular filteration rate.

The comparatively low glomerular filtration in camels, not only induces a slow excretion of fluids, but may also influence excretion of drugs eliminated through kidney. The pharmocokinetics of benzylpencillin, a drug excreted mainly by the kidney, showed that body clearance was 4.9 ml/kg/min in camel

and 9.2 ml/kg/min in sheep and the mean residence times were 27 and 15 minutes, respectively (Oukessou et al. 1990). Consequently, direct extrapolation to camels of doses established in ruminants may lead to a higher plasma concentration of drugs eliminated by the kidneys and, therefore, to risk of toxicity.

The high retention capacity of fluid in camels can be used efficiently in racing camels. Long race distances, in a hot environment, involve potential disturbance of fluid and acid base balance. The use of the physiological fluid retention capacity to prevent such disturbances may improve the racing performance of camels.

In conclusion it can be stated that despite the fact that levels of vasopressin and renin-angiotensin-aldosterone are similar to ruminants, camels are able to keep surplus fluid within the body for days instead of hours because of low capacity of the kidney to excrete water and sodium. This allows the camel to delay the effect of water deprivation when water is available intermittently.

The camel's urinary bladder is relatively small and urination is relatively frequent. The posture adopted for urination is a simple straddling of the hind legs. Camels have an instinct to urinate on their hind legs during hot weather, which might be associated with desire for evaporative cooling. This practice sometimes results in scalding and granuloma formation on the hind legs. Bull camels flick urine on their backs with their tails during rutting season.

The urine of camel is normally pale yellow and clear. The pH ranges from 6-7. The pH tends to increase to 8-9 after exercise. The specific gravity varies markedly from 1.027-1.070, depending upon season and water intake. The crystalline substances seen in the centrifuged samples include calcium oxalate, calcium hydrogen phosphate, ammonium urate, and triple phosphate. All types of castes are observed, but mainly observed are granular or hyaline (Manefield and Tinson, 1996).

Urinary tract diseases are diagnosed by a combination of clinical signs, urinalysis, serum biochemistry, hemogram, and special procedures as renal angiography, radiogrphy, ultrasonography and cytoscopy. An elevated, blood urea nitrogen (BUN) level may simply be reflecting anorexia seen as an ancillary finding in many sick camels. However, an elevated creatinine, accompanied by high BUN is suggestive of kidney disease.

ABNORMAL CONSTITUENTS IN URINE

PROTEINURIA

It is mostly called albuminuria as the greater proportion of urinary protein is albumin. It occurs in congestive heart failure, glomerulonephritis, renal infarction, nephorosis and amyloidosis. The degree of proteinuria varies, the greatest concentration, being found in amyloidosis. Small amounts may be present when mild glomerular damage occurs in fever and toxemia. When the urinary sediment contains formed elements, like casts, the significance of proteinuria increases.

CASTS AND CELLS

Casts, which appear as organized tubular structures and vary in appearance according to their composition, are indicative of nephritis and degeneration of kidneys. RBCs, WBCs and epithelial cells may originate in any part of the urinary tract.

HEMATURIA

The causes of hematuria include trauma to the kidney, septicemias and purpura hemorrhagica accompanied by vascular damage. Renal causes are glomerulo-nephritis, renal infarction, embolism of renal artery, tubular damage as caused by sulphonamide intoxication, and pyelonephritis. Postrenal hematuria occurs particularly in uroliths and cystitis. In severe cases the blood may be voided in the form of clots but more commonly causes a deep red to brown coloration in the urine. The blood, which originates from the kidney is usually intimately mixed with the urine and is present in equal concentration in all samples. When it originates from a urinary bladder lesion it is usually most concentrated in the final sample. Blood from the urethral lesion is most evident in the first part of the flow.

Occurrence of hematuria of unknown etiology has been described in camels. Bloody urine syndrome is very common in racing camels of both sexes in UAE. The condition is not commonly observed in the breeding stock. The condition is not associated with any infection. There is no concomitant increase in blood urea and creatinine values in camels suffering from hematuria indicating normal kidney function. No changes in the urinary sediment have been observed other than increased number of erythrocytes. The condition seems to be associated with increased concentrate and carbohydrate intake in racing camels.

HEMOGLOBINURIA

True hemoglobinuria gives deep red coloration to urine. A positive reaction to chemical test for hemoglobin and protein is given and there is absence of cellular debris. There are many causes of intravascular hemolysis, which is the source of hemoglobinuria. The specific causes are listed under hemolytic anemia.

MYOGLOBINURIA

The presence of myoglobin in the urine is an evidence of severe muscle dystrophy. It gives brown discoloration to urine.

PYURIA

Pus in the urine indicates inflammation somewhere in the urinary tract, usually in the renal pelvis or bladder. It is detectable mostly by microscopic examination.

CRYSTALURIA

Crystals of calcium, carbonate and triple phosphate are commonly present in the urine, however, if present in large numbers they suggest that the concentration of urine is above normal and the possible future development of uroliths.

GLYCOSURIA

Glycosuria, is not commonly observed in the camels, but is associated with enterotoxemia due to Clostridium perfringens type D and occurs after parenteral treatment with dextrose solution, adrenocorticotropic hormone or cortisone analogs. It also occurs in nephorosis due to failure of tubular reabsorption. Ketonuria is a more common finding and occurs in starvation and acetonemia.

INDICANURIA

The presence of indican (potassium indoxyl sulfonate) in excessive amount indicates increased absorption of this detoxification product of indol from the large intestines and occurs when there is increased alimentary sojourn.

CREATINURIA

Excessive endogenous breakdown of muscle causes an increased concentration of creatinine in the urine. It is associated with muscle dystrophy.

UREMIA

The causes of uremia are anuria or oliguria, the latter being more common unless there is complete obstruction of the urinary tract. Chronic renal disease may be manifested by polyuria but this is essentially a compensatory phenomenon and oliguria always appears in the terminal stages when clinical uremia develops. The animal is depressed, shows muscular weakness and muscular tremor and the respiration is usually deep and labored. If the disease has been in progress for some time body condition is poor, due probably to continued loss of protein in the urine.

DISEASES OF KIDNEYS

URINARY ISCHEMIA

General circulatory failure (shock) usually causes reduction of blood flow through the kidneys. Chronic circulatory insufficiency such as congestive heart failure results in chronic renal ischemia.

Acute ischemia of kidneys occur when compensatory vasoconstriction affects the renal blood vessels in response to a sudden reduction in circulating blood volume. There is an immediate reduction in glomerular filtration and an elevation of blood urea nitrogen. There is a concomitant reduction in urine flow. If the ischemia is severe enough and persists for long enough, the reduction in glomerular filtration which is usually reversible by a return of normal blood flow, becomes irreversible because of anoxic degenerative lesions in the renal parenchyma. This is most likely to occur in acute circulatory disturbances and is an unlikely event in chronic congestive heart failure. As a consequence of renal ischemia there results necrosis of glomeruli and tubules.

Renal ischemia is not a disease itself, because it is masked by the clinical signs of primary disease. The oliguria and azotemia will go unnoticed if the circulatory defect is corrected in early stages. However, failure to respond completely to treatment results in renal insufficiency and the clinical picture would be that of uremia.

The treatment should be directed at correction of the circulatory disturbance. If renal damage has occurred, supportive treatment , including the parenteral administration of fluid and sodium and possibly calcium salts, may enable the animal to survive an acute renal insufficiency until an infectious process is brought under control.

GLOMERULONEPHRITIS

A relatively high incidence of proliferative glomerulonephritis has been recorded in camels and especially in racing camels. The earliest and most consistent sign of chronic renal failure is weight loss; other signs are anorexia, polyuria, polydipsia and ventral edema. The consistent clinical pathological findings are high blood urea nitrogen and creatinine. Urine analysis findings vary with the nature and stage of the disease. The cause of the condition is obscure. It

might be due to feeding concentrated diet, lack of water or deposition of antigen-antibody complexes.

PYELONEPHRITIS

It usually develops by ascending infection from lower urinary tract. It usually accompanies ureteritis and cystitis.

The bacteria usually isolated in this condition are Pseudomonas aerogenosa, Corynebacterium spps, E. coli, streptococci, and staphylococci. The condition is usually unilateral. Toxemia and fever are also present and if the condition is bilateral and the lesions are extensive uremia may also be present. Pyelonephritis is always accompanied by pyuria because of the inflammatory lesions of the ureters and bladder.

The treatment should base on the results of culture and sensitivity to sulphonamides and antibiotics. Antibiotic treatment is required for a lengthy period. It should be supplemented by parenteral administration of enzymes (chymotrypsin etc.).

NEPHROSIS

It includes degenerative and inflammatory lesions of the renal tubules and / or glomeruli.

Etiology

Most cases of nephrosis are caused by toxins, i.e. mercury, arsenic, selenium, cadmium, copper, vitamin K, oxalate, thiabendazole, benzimidazole, neomycin, gentamycin, plant poisons, poisonous mushrooms, aldrin, anti inflammatory drugs including phenylbutazone and flunixin meglumine, highly chlorinated naphthalenes and sulpha drugs. It can also result from non-specific endogenous or exogenous toxemias. Other important causes include hemodynamic factors such as dehydration leading to concentration of toxic substances in the tubules, renal ischemia, and factors causing hemoglobinuria.

Clinical Findings

Other signs of primary disease often mask clinical signs. In acute nephorosis there is oliguria and protein urea and clinical signs of uremia in the terminal stages. The signs of uremia are anorexia, hypothermia, depression, slow heart rate, small weak pulse, inappetance, and diarrhea. The diarrhea may be intense to cause dehydration. Polyuria is characteristic of chronic cases but oliguria usually follows when secondary glomerular damage prevents glomerular filtration.

In many illnesses there is sufficient toxemia to cause temporary nephrosis. If the degree of epithelium lost is not enough to cause renal failure and, degree of renal damage is small, complete function can be restored.

For the treatment of the condition primary cause has to be removed. Supportive treatment suggested for urinary ischemia may be attempted.

EMBOLIC NEPHRITIS

A small embolic lesion in the kidney usually causes no clinical signs, unless it is very extensive. In this case toxemia may follow uremia. Proteinuria and pyeuria may occur some where in the course of disease.

The septic emboli carried from suppurative lesions in the other tissues may lodge in the kidneys, resulting in abscess formation.

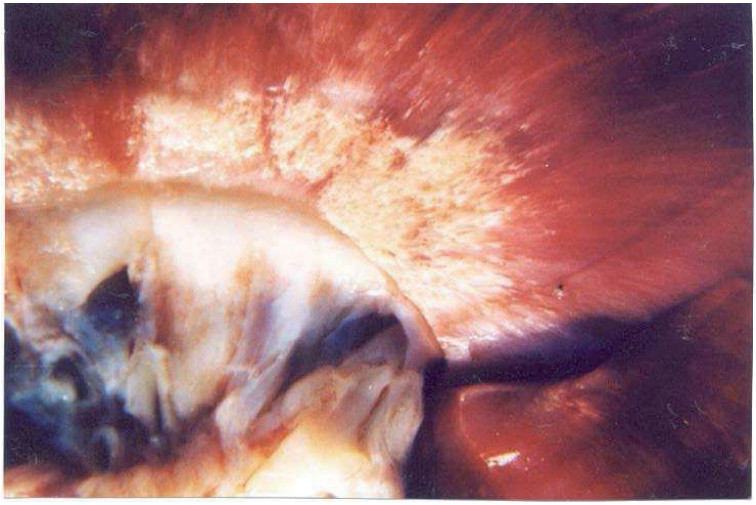
The treatment consists of standard antibiotics and sulphonamides, for a considerably longer time and can be supplemented by the administration of parenteral enzymes.

CYSTITIS

It is of sporadic occurrence due to the introduction of infection into the bladder or when there is stagnation of the urine. It is usually observed in association with vesicular calculi, difficult purturition, contaminated catheters and late pregnancy. The infection may spread from the kidney or reach through urethra from exterior. The organisms commonly involved are Pseudomonas, Corynebacterium, E. coli, staphylococci and streptococci.

There is frequent and painful urination, sometimes accompanied by grunt. The animal remains in the posture of urination for a long time. The amount of urine passed each time is small. Sometimes in very severe cases abdominal pain is evident.

Treatment should be attempted by the use of antibiotics, after determination of sensitivity. The treatment should be given for a longer period. Prognosis is usually not good.



Calcification of renal papilla

UROLITHS

The urinary calculi or uroliths result when urinary solutes (mostly inorganic, but sometimes organic) get precipitated. The precipitates may be present in the form of crystals or in the form of amorphous deposits. This usually happens around a nidus, and after a considerable time a calculus is formed. Factors which affect the urinary concentration of specific solutes, the ease with which the solutes are precipitated out of solution, the provision of nidus, the tendency of concretion, all affect the rate of occurrence of uroliths. Uroliths have been associated with a diet high in concentrated feeds (Kock and Chapman, 1986). The occurrence of uroliths in camels is reported (Gahlot, 1992; Nigam, 1992). The uroliths in the urinary bladder and urethra are encountered

commonly. In the male camels the calculi usually lodge in the urethra before or in the region of sigmoid flexture. The formation of uroliths in the males are associated with habitual masterbation, resulting in soiling of the preputial orifice with sand leading to trauma and inflammation. Urinary calculi are formed in males and females equally, but the diameter of the female urethra generally allows free passage of a calculus that may enter the urethra. Thus obstructive urolithiasis is rare in females (Fowler, 1996).

In urethral obstruction caused by uroliths there is anuria and abdominal distension. In cases with prolonged retention of urine the bladder can rupture (Kock, 1985).

The treatment indicated is the surgical removal of stones under epidural anaesthesia.

URINARY RETENTION

Retention of urine is commonly observed in male camels. It can be fatal if urine flow is not restored (Gahlot et al., 1995). It is usually caused by urolithiasis. The dynamics of obstructive urolithiasis is not very well known. Clinical cases of retention of urine develop hypochloremia, hyperkalemia, and azotemia. The calculi can be removed by posterior urethrostomy under epidural anaesthesia and flow of urine facilitated by passage of 12-15 cm long polythene catheter in the caudal part of the urethra through urethrostomy site (Choudhary et al., 1995). Urinary retention can also result from cystitis and uretritis (Manefield and Tnson, 1996).

In certain breeding herds of U.A.E., recurrent urinary retention is frequently seen in 2-4 week old dromedaries. The calves affected no longer suckle, exhibit fever up to 41ºC and die within 2-4 days. Some of the affected animals develop torticolis. At autopsy, urinary retention without urethral obstruction is observed. Urine filled cysts of varying sizes are found in the kidneys caused by urine reflux. There is also brain lesions observed in these animals. Organism Pseudomonas putida was regularly isolated from the lesions of these animals (Wernery and Kaadanen, 1995). These authors have suggested that this infection might be secondary to vitamin B deficiency.

Urine retetion due to cystic and urethral fibroma has also been reported by Gahlot et al. (1995).

URETHRITIS

This is more commonly observed in female camels and mostly is a result of ascending infection due to E. coli. It causes urethral obstruction and results in frequent passage of small volumes of urine, straining without passage of urine, and adoption of urinating posture (Manefield and Tinson, 1996). Treatment consists of administration of antibiotics, cortison, and spasmolytics.

URETHRAL RUPTURE

Urethral rupture in male camels is usually caused by trauma due to an over tight girth strap, impaction of urethral calculi or ascending infection subsequent to damage from sand masturbation (Gahlot, 1992). In the female camels it has been observed in ischial fracture.

In the male camels it is characterized by edematous swelling of the prepuce, subcutaneous tissue of the ventral abdomen, scrotum, and perineal region. The urine is not voided from the normal urethral /preputal orifice and leaks out into the subcutaneous space and can lead to extensive necrosis and possibly fatal uremia, if no intervention is attempted.

The treatment is surgical and aims at reestablishing free urine passage. It is usually done by multiple incisions in the swollen area, removal of necrotic tissue, urethrostomy or penile amputation, as indicated.

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REPRODUCTIVE SYSTEM

The discussion in the current chapter will mainly be confined to the dromedary. The literature reveals that regarding reproduction in the Bactrian camels the anatomy and physiology are common to both varieties, although some differences in the seasonal pattern of reproductive events are observed (Arthur et al., 1986). This is an often quoted comment that 'the camel is an animal designed by a committee' was never more true than when applied to the reproductive processes of camels. Aspects of the reproductive mechanisms appear to be borrowed from many different genera as viewed by reproductive physiologists. But it is a most successful mixture considering that, with the minimum of modern veterinary treatment and management, and in the harshest of climatic conditions, nutrition and environments, there are still so many camels in the world.

PUBERTY

The female camel reaches its puberty at about 3 years of age, but she is not mated until she reaches 4 years (Bodenheimer, 1954; Leonard, 1894; Williamson and Payne, 1987; Yasin and Wahid, 1957; Ywema, 1960). In, India puberty in the female camel is reported to begin at the age of 5 years (Rathore, 1986). Late maturity in the camel is one of the factors responsible for lowered reproductive performance. The prepuberal gonades and genital tract have been shown to be responsive to the exogenous gonadotropic hormones (Yagil and Etzion, 1984). This property has been utilized in reducing the age of puberty and thus improving the reproductive performance in dromedary camels (Rai et al., 1990).

Breeding activity in male dromedary camels in nomadic herds starts at five to six years of age and continues until 14-15 years, with some minor differences according to breed and geographical location (El-Wishy, 1990).

According to a report from Pakistan the male camels are used for breeding, from 6 years of age and continued to breed up to 20 years (Yasin and Wahid, 1957). In Somalia, the male camel usually arrives the age of puberty when he is 5 years old (Mares, 1954).

MALE DROMEDARY REPRODUCTIVE TRACT

The testes of camels are ovoid in shape and are found in the scrotum in a perineal position, a few inches below the ischial arch. The length of the long axis and weight of the camel testes vary in animals older than 3 years, from 2.8 to 4 inches and from 80 to 110 g, respectively. Each testis is located in its pouch, lying obliquely to the vertical axis. The anterior edge is nearly straight and is linked with the epididymis. The posterior edge is free and convex, and the inferior and superior extremities are rounded (Tayeb, 1951-52).

The epididymis is formed on the anterior edge of the testis, extending from the inferior extremity to just above the upper edge. The head of the epididymis is joined to the testis. The spermatic cord is 18 to 20 inches long, and is enlarged at its point of issue, at the inferior extremity in association with the venous

pampiniform plexus. The ductus deference extends from the tail of the epididymis to the pelvic part of the urethra. It passes forwards between the thighs to the inguinal region where it passes upwards through the inguinal canal to enter the pelvic cavity and opens into the urethra under the prostate (Tayeb, 1951-52).

The non-erect penis is directed backwards; otherwise it has close resemblance to that of the bull, the shape of the glans is like a crochet-needle in the camel and like a sharp needle in the bull. Moreover, in the camel penis, the sigmoid flexure is prescrotal whereas, in the bull, it is post scrotal. The elongation of the penis at coitus is caused by straightening of its sigmoid flexture (Tayeb, 1951-52).

The prepuce is pendulous and is formed by two layers, parietal and visceral. Between these two layers, three group of muscles are found: the anterior muscles, or protractors of Lesbre; the posterior muscles, or retractors of Lesbre; and the lateral muscles. The fibers of these muscles join to form conical mass around the preputial orifice. Due to the action of these muscles, the prepuce could be moved onwards or backwards during erection or urination, respectively. These muscles also control dilatation and constriction of the preputial orifice. The prostate gland in the camel is a discoidal mass, dark yellow in color and located on the superior edge of the first portion of the pelvic urethra, at the level of the neck of the bladder. It measures 3.7 to 5 cm in its longitudinal and transverse axis, respectively. It has several ducts on either side, which perforate the urethra. The bulbo-urethral glands are formed by two lobules situated on either side of the terminal portion of the pelvic urethra. They are whitish in color, almond shaped, and measure 2.5 X 1.2 cm. The seminal vesicle in the camel is not present (Tayeb, 1951-52).

Spermatogenesis is found to occur throughout the year. The spermatogenic activity and the diameter of the seminiferous tubules are highest in the breeding season. During the non-breeding season a shift of the frequency of the stages of the seminiferous epithelial cells is observed. This shift indicates that fewer tubules enter into final maturation and meiotic phases of primary spermatocytes and the elongation of the spermatids require a longer time. The spermatogenesis is impaired further by an increase in the abnormal division of spermatogonia, degeneration of leftotene, zygotene and pachytene stages of spermatocytes (Gupta, 1992).

Mature male dromedary camels show a seasonal sexual activity known as the rut. In the northern hemisphere the rutting season is roughly in the later half of the cold weather (Arthur et al., 1986). The rut occurs in spring in Egypt (Abdul-Rouf and El-Naggar, 1964), and from November to February in India (Khan, 1971). In Pakistan it is reported to occur from the beginning of December to the end of March (Yasin and Wahid, 1957) and from mid January to the end of May in the Turkoman dromedary (Abdunazarov, 1970). The rutting period in male has many physiological and behavioral peculiarities. There is significant increase in FSH, LH, testosterone and cortisol during the rutting season (Abdul-Rouf et al., 1975; Agarwal et al., 1987; Al-Mougy et al., 1984; Azouz et al., 1992; Dixit et al., 1987; Osman et al., 1979; Yagil and Etzion, 1980). The poll glands become active and secrete dark brown material with a pungent odor that attracts females (Tingari et al., 1984; Yagil and Etzion, 1980). The male camel loses a considerable amount of weight during the rutting season because its sexual activity prevents normal feeding. In the Bikaneri breed in India, 16 to 25 per cent loss in body weight by the breeding male dromedary has been reported (Khanna et al., 1990) during a rutting season.

Gonadotrophin-releasing hormone (GnRH) is a deca-peptide hormone secreted from the hypothalamus in a pulsite manner. Its synthesis and release is affected by season, photoperiod and endocrine status. It acts on the pituitary gland to stimulate release of LH and FSH, in a ratio influenced by feedback effects of steroid hormones. Sexual activity in normal male camels outside the breeding season could be stimulated by GnRH treatment (Moslah et al., 1992). It is also likely that the phermonal "male effect" may induce female camels to cycle earlier in the breeding season. There also seems to be an alteration in the semen consistency with GnRH administration (Willmen et al., 1992).

In the non-breeding season hyperprolactinemia, accompanied by decreased serum levels of FSH, LH, testosterone and cortisol have been observed in dromedary camels. It is proposed that hyperprolactinemia is a causative factor of low fertility and libido in the male camel during the non-breeding season (Azouz et al., 1992). This is probably due to its action in reducing the synthesis and secretion of FSH and LH. There is also possibility that prolactin has an anti-gonadotropic action at the gonadal level (Besser et al., 1974; McNeilly et al., 1978).

In the male camel, as in other species, instances of lack of sexual desire and inability to copulate during the rutting season have been observed. Because of the length of copulation and the fact that several ejaculations may occur during mating, wide variation in ejaculate volume and in sperm concentration and motility have been reported. Considering the relative short breeding season together with the fact that sometimes excessive number of females have to be mated by one male, poor semen quality could cause herd infertility, under range conditions (Arthur and El Tigani, 1990).

COPULATION AND MATING BEHAVIOR

The male camel behaves in an aggressive manner during the mating season, and grinding of the teeth and protrusion of the “Dulla” is observed. Although this latter organ is present in both sexes, it is never extruded in the female. It has been reported that the soft palate is increased in length during the rutting season and is capable of evagination (Charnot, 1963). In the breeding season the males have also been seen standing frequently with their hind legs spread, wagging the tail vigorously when urinating so as to spray urine over the hind quarters and hind legs. The female usually crouches and voids some urine when the male approaches. The male smells the urine on the ground, or the vulva, and exhibits the olfactory reaction known as "flehmen", holding the head in the vertical position and averting the upper dental pad and lower incisors (Abdel-Rahim and El-Nazier 1992).

Before copulation, the male tries to make the female sit down by biting at her shoulders and by applying pressure on her neck with his own. During copulation, both sexes adopt a squatting posture and the male grips the female with his forelegs. Copulation lasts for 7 - 22 minutes and is accompanied by oral frothing, vocal gurgling, intermittent protrusions of the soft palate by the male, and bleating by the female (Leese, 1927, Rai et al., 1988; Singh and Prakash, 1964). Interspersed with these activities are several bouts of male pelvic thrusting (Agarwal and Khanna, 1990), with corresponding louder vocal responses from the female.

Erection of the penis does not occur during courtship in the standing position, which indicates that foreplay and a set period of time for full arousal are not necessary for successful breeding in camel (Abdel-Rahim and El Nazier, 1992).

Females that have mated successfully two or three weeks previously stand still with their tails raised in a characteristic erect position when approached by the male and continue to show the stance throughout pregnancy when approached by the male (Abdel-Rahim and El Nazier, 1992).

FEMALE REPRODUCTIVE TRACT

The Ovaries

The size and shape of camel ovaries vary with age and with their content of follicles and corpora lutea (CL). In anoestrus nulliparous female, the ovaries are oval or circular and laterally flattened. The surface is irregular with many small follicles of 3-5 mm diameter. Each ovary weighs 3-4 gms and measures 2-3 cm in length and 2-2.5 cm in width and 5-9 mm in breadth. In multiparous camels the presence of corpora albicantia of different sizes and in different stages of regression, causes marked distortion of the ovarian contour (El-Wishy, 1992).

The Oviducts

The oviduct in the camel is 25-28 cm long. The isthmus is less coiled than the ampulla and the ovarian part of the fallopian tube. The fimbriae lie in the bursa at a short distance from the ovary. The lumen of the ampulla is 4 to 5 cm in diameter at its ovarian opening in the depths of the fimbriae. The lumen of the isthmus is 1 to 2 mm in diameter. Each oviduct opens into the uterine horns by a narrow orifice at the summit of a papilla 3 to 4 mm in height (Tayeb, 1953).

The Uterus

The camel uterus is T-shaped rather than 'Y' shape. The right cornu is shorter than the left one and body of the uterus is relatively short (Smuts and Bezuidenhout, 1987), even in the nulliparous females and virtually all coceptuses implant in the left horn, regardless of the side of ovulation. The division between the uterine horns is pronounced and it proceeds well back into the uterine body so that the bifurcation is frequently only 2 to 4 cm cranial to the internal os of the cervix. The stage of ovarian cycle has profound effect on the shape and tone of the uterus in the dromedary camel. The follicular phase is accompanied by pronounced myometrial tone and minimal curl to the uterine horns. In the luteal phase, on the other hand, the curl is more pronounced and tone is less during oestrus and it increases progressively in diestrus and especially after day 18 of pregnancy (Ginther, 1979). The mucous membrane of the uterine body and both cornua are smooth and do not have cotyledons (Shalash, 1965).

The Cervix

The cervical canal protrudes about one centimeter posteriorly into the vagina forming two blind cavities, one situated dorsally and the other ventrically. The average length and diameter of the cervical canal during follicular activity has been recorded as 5.32 ± 0.98 and 5.96 ± 0.92 cm respectively, while during ovarian inactivity the length and the diameter were found to be 4.96 ± 1.25 and 5.79 ± 0.99 cm. The cervical mucosa is thrown into several longitudinal mucosal folds. Also, two to six complete and incomplete annular mucosal folds are observed. The folded cervical mucosa is lined by simple columnar to stratified columnar epithelium that reacts positively with both Period Acid Schiff's (PAS) and Alcian Blue (AB) stains, especially at its apical half. The cervical submucosa propria is highly vascular and shows branched secretory crypts (Ali et al., 1992).

The Vagina

The mucosa of the cranial part of the vagina bears longitudinal mucosal folds as well as two or eight circular musculo-mucosal folds. The muscle fibres of the circular folds run parallel with the long axis of the folds. The vaginal epithelium is thin stratified squamous non-keratinised and shows PAS positive granules in the cells of the upper layers. At the most cranial part of the vagina the squamous cell layers disappear and the epithelium becomes transitional to stratified columnar. Lymphoid tissue and crypts that enclose PAS and AB positive material are noticed in the vaginal stroma (Ali et al., 1992).

The Placenta

The placenta in dromedary is diffuse in nature, as in the mare, and noncotyledonous as in ruminants. The chorionic membrane is thick being covered with short, shrub-like tendrils, or villi, in which arterio-venous connections sometimes occur (Shalash, 1965).

OVARIAN FOLLICULAR WAVE PATTERN IN THE FEMALE CAMEL

Most investigators have concluded that waves of follicular growth, maturation and atresia occur throughout the breeding season. The time-course of such events can vary with latitude, stage of season, age and nutritional status of the camel (El-Wishy, 1987). It is difficult to define a clear-cut period of increased sexual receptivity in relation to any stage in the follicular growth cycle in dromedary camel. Waves of growing (2-14 days; mean = 6 days), mature (5-19 days; mean = 8 days) and regressing (7-10 days; mean = 8 days) follicles have been described (Musa and Abusinenia, 1978b). When regression is complete another wave of follicular development starts and follow the same pattern. Two or more follicles of different sizes are often found in the ovaries of unmated camels (El-Wishy, 1992). During the follicular phase in camels, basal concentration of LH is 2.7 ± 1.2 ng /ml. By 4 hour after insemination, peak values of 6.9 ± 1.0 ng /ml, occur. In addition, a smaller LH peak (5.4 ± 2.5 ng/ml) appears 1 day before the regression of the follicle begins, in unmated camels. During the follicular phase peripheral plasma progesterone values are low (0.36 ± 0.28 ng /ml), but values increase to reach 1.73 ± 0.74 ng /ml at 3 days and 2.4 ± 0.86 ng /ml at 7 days after ovulation. The plasma estradiol-17-beta concentrations are 26.8± 9.0 pg /ml during follicular phase and 30.8± 5.1 pg /ml when the follicle is of maximum size. Values fall after ovulation but rise to 29.8 ± 6.5 pg /ml 3 days later (Xu et al., 1985).

Histologically, the granulosa cells of large follicles are arranged in columns that radiate from the basement membrane. A rich bed of capillaries is found among the epithelial cells of the theca interna and the spindle shaped theca externa cells. An area of loose connective tissue separates the thica externa and the dense ovarian stroma (El-Wishy, 1992).

Developing and Regressing Corpora lutea

Ovulation in the camel is induced by mating (Chen et al., 1985; Elias et al., 1984) and the CL that forms after a sterile mating has a short life span (Marie and Anouassi, 1987; Musa and Abusinenia, 1978b), developing and regressing Corpora lutea are rarely seen in non-pregnant camels.

Soon after ovulation a dark haemorrhagic area of 10-15 mm diameter appears on the surface of the ovary. On sectioning this area, a thickened, luteinised wall surrounding a central haematoma is seen, similar to the developing CL in the mare (Ginther, 1979). A few days latter the young, mature CL becomes reddish

brown in color and projects from the surface of the ovary. It has a diameter of 12-15 mm and weighs 1.5-2 g. The regressing CL is light brown to fleshy in color and has a firm consistency. Microscopically, large polygonal granulosa cells containing fine granules and a lightly stained cytoplasm form the main mass of the CL. They have large, spherical, vesiculated and centrally located nuclei, with prominent nucleoli. Small lutein cells of the thecal origin, with dense, acidophilic cytoplasm and darkly stained round nuclei, form a minor part of the CL (El-Wishy, 1992).

Ovarian Changes During Pregnancy

Growing follicles (5-15 mm diameter) are observed frequently during the first 90 days of pregnancy but only occasionally thereafter. In contrast to the rapid regression of the corpus luteum formed after a sterile mating, a definite increase in corpus luteum size occurs in the early stages of gestation. Hence, large and well-developed CLs are found in pregnant animals (El-Wishy et al., 1981; Tayeb, 1948). Measurements of progesterone concentration in milk (Abdel-Rahim and El Nazier, 1987) or in serum provides a useful indication of pregnancy before palpable changes in the in the uterus can be recognized (El-Wishy, 1988). Like follicles, the CL of pregnancy protrudes prominently, above the ovarian surface. In cross section, a central plug of connective tissue is a consistent feature. The presence of two (occasionally three) luteal bodies in one or both ovaries occur frequently in pregnant camels (El-Wishy, 1992). The left ovary is more active than the right in this respect. Twin fetuses are found in the very early stages of pregnancy but one of them dies at around day 40 to 45 when its body length is about 30 mm. In most cases the twin CL are fairly equal in size.

Sometimes, however, one of the luteal bodies is markedly smaller (5-10 mm diameter) and may lack the central plug of connective tissue that indicates ovulation. Hence, it seems possible that follicullar luteinisation can occur during pregnancy in the camel. The regression of these bodies during pregnancy is probably the source of the excess numbers of corpora albicans found in the ovaries of older camels, than can be accounted for by the number of possible pregnancies for that age (Arthur et al., 1986). The right and left ovaries tend to alternate in their ovulatory activity.

Regression of the CL of pregnancy occurs over an extended period after parturition in the camel. This contrasts with the situation in cattle where the CL of pregnancy regresses rapidly and is usually not palpable per rectum by day 14 postpartum (Marrow et al., 1968). Seven days after parturition in the dromedary camel, the CL is still present and remains spherical in outline. On section, the outermost capsule is 1 mm thick and the lutein tissue remains light brown in color. With time, the outer capsule becomes thicker and the concentric fibrous core becomes relatively wider. Decrease in size is, however, slow and hard, protuberant, spherical, laterally compressed (10 to 15 mm) or button shaped (5 to 8 mm) corpora albicans are frequently found in completely involuted genital organs in multiparous camels. In contrast, follicular growth is encountered rarely in the ovaries of post partum camels in which the uterus is not yet involuted. Histologically, the corpora albicans in the early post partum period are characterized by cells with shrunken and vacuolated cytoplasm and pyknotic nuclei, together with thick-walled arterioles. When regression is more advanced, the parenchymal cells show hyalinization and the thick-walled arterioles are sometimes obliterated (El-Wishy, 1992).

PREGNANCY

The period of gestation is from 365 to 410 days (average 370 days). When there is a male fetus it will be 3 to 4 days less. In a first pregnancy the period is longer by 15 to 20 days. About 6 months after conception, the she camel may show evidence of pregnancy such as enlargement of belly and general lethargy. As the time of parturition approaches, she may sometimes show edema on the belly and legs, increase in the size of udder, some swelling around the vulva, and depression on each side of the root of the tail (Rathore, 1986).

Although the left and right ovaries function equally, about 99 per cent of the pregnancies are in the left uterine horn and uterine body. This is because of the embryonic migration from the right horn to the left and seems to occur when the right ovary ovulates and the left ovary does not. When both ovaries ovulate at the same estrus, embryos develop initially in both horns but the one in the right horn dies when it reaches a size of two or three centimeters. This selective death of the right horn embryo occurs despite coalescence of the chorions of the two conceptuses. However, vascular anastmosis does not take place as it does in the bovine. There is no record of a freemartinism in the small number of twins born. The incidence of twin ovulation is 14 percent and the twins are born in only 0.4 per cent of all births (Musa, 1969).

The results, obtained by video-endoscopic hysteroscopy have revealed (Skidmore et al., 1992b) that in dromedary the coceptus begins to elongate prior to day 20. Then it extends to occupy the whole of the left uterine horn between days 20 and 25, with further extension resulting in occupation of the whole of the right uterine horn between days 25 and 35. This is much faster than the rate of allantochorionic extension in the mare which like the camel, produces a diffuse non-invasive micro cotyledonary placenta, that does not become attached to the endometrium over the entire internal surface of the uterus until day 85 to 90 of gestation (Allen, 1982). This rapid rate of placental elongation and attachment in the camel is similar to that in sheep and cattle (Moor, 1968). In these ruminant species, however, intimate contact and placental exchange is reserved to the discrete endometrial protuberances known as cotyledons, which are absent in camels (Arthur et al., 1989). Two other features of conceptus development were observed with the videoendoscope (Skidmore et al., 1992b). First, considerable elongation of the allantochorion of the conceptus occurs before sufficient fluids have accumulated to distend the chorion and force it against the endometrial epithelium, thereby facilitating microvillous attachment and true, permanent placentation. It may be speculated that this early extension of the allantochorion may be necessary to provide sufficient direct contact between trophoblast and endometrial surface to enable direct transmission of the maternal recognition of pregnancy signal (Short, 1969) to the endometrium, so preventing it from releasing prostaglandin F2 (Allen, 1982).

The second finding of interest was the presence of structures which, appear to be some sort of homologue of the equine hippomanes. These have not been reported in the full term placenta of the camel (Arthur et al., 1986), so their origin and fate during the early stages of pregnancy has to be explored.

The amount of allantoic fluid increases rapidly from about 1.5 liters at 0-10 cm fetal body length to approximately 5 to 6 liters at fetal body length 11- 20 cm. This volume is maintained fairly constantly until fetal body length reaches 90-100 cm when it rises to 6 liters, and finally at fetal body length 101-107 cm, the allantoic volume is about 8.5 liters. The allantoic fluid resembles pale urine and sometimes contains yellow-brown hippomanes. The volume of amniotic fluid rises from 13 ml at fetal body length 0 to 10 cm to a final volume of nearly one liter, its amount being always small relative to the allantoic fluid. The amniotic

fluid is usually watery but sometimes cloudy, with brown bits of meconium and hippomanes (Arthur et al., 1989).

From gestation stage fetal body length 41.0 cm (Musa, 1969), noticed two amniotic membranes: amnion proper, which contained the amniotic fluid, and an inner membrane which very closely invested the fetus, except at its orifice, which opened directly into the true amniotic cavity.

PREGNANCY DIAGNOSIS

A practical consideration of any indirect method of pregnancy diagnosis in the camel is that the camel will almost certainly have to be restrained in a sitting posture in order to obtain a blood or urine sample and to make a rectal exploration, for pregnancy diagnosis.

The only method investigated sufficiently, to be recommended is the clinical method of rectal palpation (Arthur et al., 1986; Barmintsev, 1951). In the dromedary the technique of palpation of the genital organs is the same as for the cow. It is important to remember the following features which are peculiar to the camels: (a) large corpora lutea are only present during pregnancy; (b) 99% of pregnancies are in the left uterine horn; (c) the empty (or early pregnant) right horn is congenitally shorter than the left; and (d) the amount of fetal fluid at all stages is less than in the cow (Musa and Abusineina, 1976; Musa and Abusineina, 1978a).

From the foregoing it follows that the presence of a CL in one or both ovaries is a very strong indication of pregnancy. Presumably, however, a CL could develop after a sterile mating and would be expected also, initially, in cases of recent embryonic death; but in both these instances the CL would be unlikely to persist. The palpable enlargement of the pregnant (left) uterine horn in the dromedary can be detected not until the eighth week, when the whole of the left horn is enlarged. At this time, both ovaries (one or both with CL), together with the uterus, are within the pelvis. It should be noted that because the camel placenta is non-cotyledonary, it is not possible to slip the fetal membrane between the palpating thumb and fingers as in the cow. By the eighth week vaginal inspection, or palpation, reveals a plug of adhesive mucus in the os uteri externum.

At the end of the third month the pregnant left horn is clearly larger, softer and in front of the non-pregnant right horn; it is at the pelvic brim and its corresponding ovary is in the abdomen. At the fourth month the uterus is just in front of the pelvic brim but most of it can be felt. A month later, the limits of the uterus cannot be defined, although its dorsal surface is still palpable. During the sixth month and for the remainder of pregnancy, the fetus can be felt and the ovary on the non-pregnant (right) side can be palpated until the tenth or eleventh month. From the seventh month individual parts of the fetus, namely its head and legs, can be identified

External observation of the she-camel's right flank reveals spontaneous fetal movements from the ninth month and the fetus can be observed externally from the tenth month. In the eleventh month the vulva is slightly swollen and growth of the udder is first noticeable. In the following month there is obvious abdominal enlargement and the camel is lethargic. The sacro-sciatic ligaments begin to relax and rectal palpation reveals the uterus projecting backwards and occupying the anterior two-third of the pelvis. Finally, in the thirteenth month relaxation of the sacro-sciatic ligaments is pronounced, tumefaction of the vulva is marked and there is much greater enlargement of the udder. The fetus can be balloted from both flanks.

The presence of follicle-stimulating activity has been identified in the blood of camels pregnant with fetuses of fetal body length 11 to 85 cm (Musa and Abusineina, 1976). The latter authors have not found any spate of follicular development comparable to that which occurs in the mare. At which time it shows high levels of pregnant mare serum gonadotrophin between 40 and 120 days; neither have they seen anything resembling the endometrial cups in the camel uterus which, are the source of the equine follicle-stimulating hormone of pregnancy. This matter needs further investigation to see whether it has any, even limited, application to pregnancy diagnosis in camels.

It has been found that the Cuboni test for the demonstration of estrogens in urine can be successfully applied in the camel, as in the mare. It should be noted, however, that in the mare this test could only be used from mid-pregnancy onwards. In the mare the source of the pregnancy estrogen is believed to be the markedly enlarged fetal gonads which reach a huge size in the horse.

Since well developed corpora lutea are known to be present only during pregnancy and because progesterone level of >1.0 ng /ml is reached only after fertile mating (Elias et al., 1984), blood or milk progesterone assays can be a valuable tool for diagnosis of pregnancy in camel.

Early pregnancy diagnosis is very important for the successful application of modern breeding technologies such as artificial insemination and embryo transfer. Significant structural changes in the reproductive tract of female animals even during pregnancy can be visualized by ultrasonic image detection (Schels and Mostafawi, 1978; Tinson and McKinnon, 1992), and videoendoscopic monitoring (Skidmore et al., 1992b).

For ultrasonography the linear array transducer is passed into the evacuated rectum and rotated slowly from left to right so that the 'curtain' of high frequency of sound passes over the body and two horns of the uterus, and both ovaries. During pregnancy in dromedary camels, the conceptus is first identified at day 17 or 18 after ovulation as a typical 'black hole' in the uterine lumen. By day 20-21, the conceptus appears as a discrete and easily recognized accumulation of conceptus fluid, the diameter and out line of which varies appreciably in different parts of the uterine horn. The embryo itself is first recognizable at this stage as a small echogenic 'blob' in the central region of the conceptus, apparently attached closely to the endometrium in the ventral region of the uterus. The embryogenic heart beat is first discerned around day 22 or 23, as a rapid rhythmical fluttering movement in the centre of the echogenic embryo (Skidmore et al., 1992b).

Videoendoscopic hysteroscopy during early pregnancy in the camel is an exciting tool for descriptive and investigative research purposes. Videoendoscopic hysteroscopy is a new method for observing the interior of the vagina, cervix and uterus in non-pregnant large animal species and for monitoring fetal development and recovering samples of fetal fluids and tissues for research investigations during pregnancy. The flexible videoendoscope utilizes a powerful xenon light source, the rays of which pass through a rotating strobe filter before they pass down the endoscope via a collated bundle of glass fibers. The rays reflected from the object being viewed pass back through a lens onto a charged couple device (CCD) microchip at the anterior tip of the endoscope. This converts the light rays into electronic signals for transmission back up the endoscope. The signals then pass through a videoprocessor, which reconverts them to form an integrated picture on the video monitor. The videoendoscope gives greatly improved brightness, depth of focus and color reproduction compared to the conventional fiberoptic endoscope and it has the great advantage that several

people can watch simultaneously and discuss the examination (Skidmore et al., 1992b). The process of endoscopy usually terminates pregnancy. Hence it is not a recommended procedure for routine pregnancy diagnosis. Ultrasonography, on the other hand is safe and accurate method for the diagnosis of important reproductive events including pregnancy diagnosis.

PARTURITION

Camels in advanced gestation, with distended abdomens and enlarged udders are known to be near parturition when there is milk in the teats and the vulva is swollen. First-stage labor lasts 24 to 48 hours and is characterized by intermittent restlessness. The period of expulsion averages half-an-hour and during it the camel adopts a sitting posture. Almost 100% of presentations are anterior. There are bouts of straining at intervals of 1/2 to 1 minute and the allanto-chorion ruptures before it reaches the vulva. The fetal nose, covered by amnion, rather than the fetal feet (as in other ruminants and the horse) appears first. With further expulsive efforts, one front foot and then the other emerge alongside the fetal head, and more straining then leads to complete emergence of the head and almost simultaneously the rest of the body is expelled. Birth appears to be achieved more easily than in the mare and cow, the fetal body being well lubricated and beautifully stream-lined while the mother is not over-taxed by her expulsive efforts (Arthur et al., 1986).

The umbilical cord ruptures as the offspring wriggles away from its mother, or when the mother gets up. She noses and nibbles at the calf but does not lick it like ruminants do. The third-stage of labor usually lasts less than half-an-hour and during it the mother shows intermittent restlessness and may get up and down several times. The after-birth progressively emerges and includes large retention sacs containing a gallon, or more, of fetal fluid which probably exert a gravitational pull on that part of the after-birth still attached. Sometimes the fetal membranes may be completely expelled very soon after the fetus. The membranes are usually not eaten by the mother. The camel calf can stand, after many unsuccessful attempts, within half-an-hour of birth (Arthur et al., 1986).

IMPROVING CAMEL REPRODUCTION

It is not possible for camels to produce a calf each year, because of long gestation period and relatively short breeding season. However, reproductive performance in camels can be improved by keeping the animals in good body condition by providing the animals satisfactory nutrition. Adequate number of males should be kept in the herd to cover the females during the breeding season and fertility of the stud males should be assessed as far as possible. It is also reasonable to consider modern breeding technologies as discussed below. Neonatal mortality is major constrain on herd productivity in pastoral system of camel husbandry. It is very important to reduce neonatal losses to the minimum.

Induction of Estrus and Management of Reproduction

During estrus, which lasts for four to five days, plasma estrogen levels are high while progesterone concentrations are always low (El-Wishy, 1987). In non-mated females the mature follicle becomes atretic and a new follicular wave soon initiates a new period of estrous and sexual receptivity. It has been observed (Marie and Anouassi, 1986) that ovulation can occur in females exposed to the sight, sound and smell of a male after a prolonged absence. However, plasma progesterone measurements in such animals indicate short life-span of the resulting CL, as is the case in females that ovulate after a sterile mating (Elias,

1990). A fully developed and functional CL is normally associated with pregnancy.

In many species, progesterone priming of the follicle is an important pre-requisite for normal development of the CL. Consequently, the first estrus after puberty or at the start of the new breeding season, in sheep and goats, and the first estrus post partum in cows, frequently gives rise to a short-lived CL so that mating is followed by early abortion due to premature luteolysis.

In the dromedary camel a single fetus can often develop in the uterus with more than one CL present in the ovaries (Shalash, 1965). There is a distinction between pregnant and non-pregnant CL (Arthur and Tagani, 1990) and, early in the breeding season, many fertile matings occur 12-13 days after a sterile mating (Marie and Anouassi, 1987). All these observations support, indirectly, the concept that the absence of any progesterone priming of follicles induces a short-lived CL which, in turn, is responsible for the failure of pregnancy at the first estrus of the breeding season.

Single injections of equine Chorionic Gonadotrophin (eCG) in doses ranging from 1,000 to 8,000 i.u. can induce estrus in both the non-breeding and breeding seasons, but the number of pregnancies achieved by this therapy is very low (Elias et al., 1985; Rai et al., 1990; Yagil and Etzion, 1984). Camels that conceived to matings during the first estrus after eCG treatment showed a high rate of pregnancy loss (75%) subsequently, whereas those mated in the next estrus sustained a much higher rate of pregnancy (Rai et al., 1990).

It is possible to advance the limited breeding season of the dromedary camel by a single treatment with 2,000 iu eCG, alone or associated with progesterone. It has also been shown that follicular growth can be stimulated by hormonal treatment in camels in all reproductive states, including maiden, aborted and lactating animals. There is a higher pregnancy rate in the camels treated prior to the start of the breeding season than those treated similarly during the season itself. The repeated matings observed in the post partum females treated only with eCG during the season may be related to a reduced life span of first CL induced by the treatment. The results obtained by injecting eCG in association with progesterone are slightly better than those obtained by eCG alone in all the categories of camels (Minoia et al., 1992).

Artificial Insemination in Camels

Like in other species, artificial insemination (AI) is a useful tool for the rapid genetic improvement in the camel. Camels offer more advantages than any other livestock for the use of AI techniques for improving genetic traits, such as milk, meat, hair production, and racing ability. Since, females demonstrate continuous estrus, they are ready for insemination at any time, provided ovulation is properly induced (Williamson and Payne, 1987).

Some pioneer studies on the use of artificial insemination in camel have been reported (Chen et al., 1990; Sieme et al., 1990). Remarkable fertility rate has been reported. A major difficulty with camel artificial insemination is to ensure that the inseminated animals ovulate. More work is required to determine the minimum required amount of natural semen in the inseminating dose, whether service by a vasectomised male can regularly induce ovulation and how reliable hormone preparations, like human Chorionic Gonadotropin (hCG) and GnRH, are for stimulating ovulation. Synchronization of estrus makes artificial insemination more convenient (Arthur, 1992).

Estrus Detection

Estrus in camel could easily be recognized by restlessness, swelling and mucous discharge from the vulva and bleating of the female (Khan and Kholi, 1973; Novoa, 1970; Yasin and Wahid, 1957). Apparently when mature follicles are available in the ovary, the female becomes willing to accept the male. The estrus female seeks male, stands besides him, becomes restless, wages her tail, and is very ready to be mounted. Females that are not in heat do not mount others in heat, but they will run after them in a playful manner and attempt to bite their vulva. Males seek out females by smell, usually sniffing along neck and not at vulva (Williamson and Payane, 1987). On approach of a male or hearing the gurgling voice of rutting male, the female moves her tail up and down in rapid succession (Arthur et al., 1986).

Estrus detection can be facilitated by introducing a male or vasectomised male to the corralled females and observing their characteristic behavior (Anouassi et al., 1992). Whether the time of onset and cessation of estrus in camels is controlled by diurnal changes is not reported. There is good evidence that more signs of estrus are observed during the hours of night in cattle, perhaps when the animals are less disturbed (Arthur et al., 1986).

The estrus cycle in camel is follicular and the increasing values of estrogens during follicular development are probably the stimuli for behavioral estrus (Homeida et al., 1988). During follicular cycle, the concentration of the serum estradiol-17-beta varies between 9 and 110 pg /ml. In early estrus, the peak level of estradiol (74.7±6.61 pg /ml) was maintained for three days (Elias et al., 1984). Blood estradiol concentration averages 26.8±9.0 pg /ml when follicles are fully matured. This level decreases immediately after ovulation (Gao et al., 1987).

Synchronization of Estrus

Because effective estrus detection requires much time, labor, skill and expense, it has often been sited as a major factor limiting the wide spread use of AI in cattle. Therefore, the elimination of estrus detection from artificial programs was the principal stimulus that led to the research in development of prostaglandin and progestational compounds that have the ability to control estrous without affecting fertility of the animal (Wenkoff, 1986). Extensive research and development has been achieved in estrus synchronization and controlled breeding in cattle but not in camel. Camel is an induced ovulator and offers great prospects of natural synchronization of estrus (Yagil and Etzion, 1984; Yagil and vonCreveld, 1990). By synchronization of estrus problem of estrus detection could be solved and makes AI more convenient (Helmy, 1991; Minoia et al., 1992). Synchronization of the ovulation can be achieved successfully by the use of progesterone releasing intravaginal device (PRID; Sanofi Animal Health, U.K.), which is a flexible stainless steel coil coated with an inert elastomer that is impregnated with 1.55 g of progesterone. A capsule containing 10 mg of estradiol benzoate is attached to the device. When placed in the vagina, PRIDs provide a convenient method for delivering progesterone over a long period (Coopers et al., 1992; Skidmore et al., 1992a). Injection of 100 mg progesterone-in-oil (progestin; Intervet (Aust) Pty Ltd, Sydney, Australia) for 10 to 15 days have also been used to synchronize estrus in dromedary camels (McKinnon and Tinson, 1992). This treatment limits further follicular development and provides an environment suitable for recrudescence of follicular activity which is later on stimulated by gonadotropin treatment on the last day of progesterone therapy.

Induction of Ovulation

Ovulation failure is one of the major contributing factors towards low fertility rates in camels (El-Wishy, 1987). Camel is an induced ovulator, the ovulation being induced mainly by coitus (Cristofori et al., 1989; Homeida et al., 1988; Musa and Abusinenia, 1978b). However, mechanical stimulation, which triggers ovulation in species such as the cat and the rabbit, does not induce ovulation in the camel (Elias et al., 1985; Musa and Abusinenia, 1978b; Musa et al., 1990). It has also been shown that ovulation in both dromedary and bactrian camels can be induced by depositing semen in the uterus (Chen et al., 1983; Musa et al., 1990).

An ovulation-inducing factor has been demonstrated in the seminal plasma of the bactrian camel and that this factor is probably a protein (Zhao et al., 1990). Ovulation in the camel can be induced by seminal plasma, given either as intramuscular injection or vaginal insemination (Pan et al., 1992). Four hours after administration of seminal plasma, preovulatory LH and FSH peaks appear in the peripheral blood and ovulation occurs 30-48 hours later (Zhao et al., 1990). However, there is an opinion that, at least in dromedary camel, mechanical stimulation of the cervix or some other physical aspect of coitus is also important for induction of ovulation. It is suggested that acceptable conception rates following insemination could only be achieved if ovulation is induced reliably (Anouassi et al., 1992).

Variation exists in the individuals in the response of individual females in the ability of particular males to induce ovulation. Semen of all camels do not induce ovulation (Chen et al., 1985; Marie and Anouassi, 1986). However, LH, hCG, LHRH, GnRH, PMSG induce 100 per cent ovulations (Chen et al., 1985). Hormone induced ovulation occurs a little earlier than natural mating or insemination; 36 hours in camels (Chen et al., 1980). Ovulation time is not much different in AI or after natural mating (36-48 hours), however, the incidence of ovulation is 87 per cent and 100 per cent, respectively (Chen et al., 1985).

Semen Collection, Evaluation, and Preservation

Methods of semen collection, evaluation and preservation in camels have been discussed (Anouassi et al., 1992; Musa et al., 1992; Tingari et al., 1987). Semen can be collected either by electro-ejeculation (Ali et al., 1992) or by the use of bull artificial vagina (Anouassi et al., 1992; Billah and Skidmore, 1992). The volume of semen recovered by electroejaculation was usually less (3.9 ml) than that collected by artificial vagina (7.5 ml) but the other sperm parameters (sperm concentration, sperm motility, percentage of dead spermatozoa, morphological abnormalities and percentage of acrosomal abnormalities were similar (Merkt et al., 1990; Taha Ismail, 1988). As most of the rubber liners used in artificial vagina have a deleterious effects on camel spermatozoa, it was recommended to construct artificial vagina in such a way that either the semen is ejaculated directly into the graduated collection vessel or a plastic jacket is inserted inside the inner rubber liner (Musa et al., 1992).

Considerable variation in the semen characteristics of dromedary camel has been reported (Billah and Skidmore, 1992). The semen was greyish-white in appearance. Its total volume range from 2-8 ml. Sperm concentrations range from 256-440 X 10^6 /ml. Sperm motility in raw semen, examined 15 minutes after collection, range from 30-50 per cent and motility is preserved best at room temperature by diluting the semen 1:2 in skim milk/glucose diluent containing antibiotics.

Good quality semen to be used for artificial insemination in camels should have at least sperm concentration of 331-325 X 10^6 /ml, and sperm motility of 49.7-50.5 per cent. The proportion of dead spermatozoa should not be more than 18-19 per cent, total sperms showing morphological abnormalities 27.4-27.7 per cent and sperms showing acrosome abnormalities 8.1-8.5 per cent (Merkt et al., 1990; Musa et al., 1992; Taha Ismail, 1988; Tingari et al., 1987).

The sperm concentration is usually measured with haemocytometer. The sperm motility is evaluated using phase contrast microscope at a magnification of X128 slide being put on warmed stage (38° C). Standard commercial stains such as Krass (Krass, 1952) for examining morphology and Eosin color test of Bartmann (Bartmann, 1959), for recording percentage of dead spermatozoa are used (Anouassi et al., 1992; Musa et al., 1992).

Different extenders have been tried with varying success for short-term preservation of liquid semen. Extenders containing egg yolk and lactose were best suited to liquid preservation (to be used within 36 hours) of camel semen. Anouassi et al. (1992) and Musa et al. (1992) also extended semen with 11 per cent w:v lactose and 20 per cent v:v egg yolk to a final concentration of 50 X 10^6 /ml.

Different methods to deep freeze camel semen by testing the motility and morphology of the spermatozoa and their life-span in 1 per cent sodium chloride solution at 38°C, after thawing was evaluated (Musa et al., 1992). The best method found was modification of the technique recommended by Westendrof et al. (1975). According to this method raw semen at 20-30° C is diluted (1:1) with cooling extender (containing egg yolk 20 parts and 11 per cent lactose 80 parts) and cooled to +15° C over 2.5 hours. Cooled diluted semen is further diluted with freezing extender (cooling extender 95.5 ml, glycerol 6 ml, and OEP-Equex emulsifier 1.5 ml) to give final sperm concentration of 150 X 10^6 /ml. This semen is placed in straws and frozen for 20 minutes in liquid nitrogen vapors (+5 °C to –120 °C) and plunged into liquid nitrogen (-120 °C to –196 °C). The semen was diluted, cooled (4 °C over 90 minutes) and loaded into 0.25 ml straws and frozen in liquid nitrogen (Billah and Skidmore, 1992). The latter authors observed vigorous motility in high proportion of spermatozoa, after one day to 13 months, extended with commercial medium 'Laicipho' as compared to two other commercial media used by them. Better post-thawing semen quality was observed in small straws (0.25 ml), however, bigger straws will hold sufficient quantity of seminal plasma to induce ovulation (Musa et al., 1992).

Optimum Time of Insemination

Even the best procedures for depositing the right volume of semen containing the right number of live sperms in the right location are virtually useless if the time of insemination is wrong. Life of the sperm and ovum in the female reproductive tract is an important factor for consideration in determining time of insemination. Spermatozoa usually have longer life than ova. In-vitro studies show that camel sperm has short life of 1-6 hours (Kasymov et al., 1982) at 0-4 °C. This may be due to some inherent metabolic entity or to cold sensitivity of the camel semen, which have high lipid content (El-Manna et al., 1986). However, in nature, ovulation occurs 36-48 hours after natural mating or insemination (Chen et al., 1985), which conceivably corresponds to and indicates camel sperm life in the female reproductive tract. One factor, which may contribute to prolonged sperm life in the female reproductive tract of camel is the presence of seminal mucopolysacchride gel of bulbourethral glands (Perk, 1962).

The spermatozoa are entangled in the seminal mucopolysacchride gel and are slowly released by its progressive liquefaction (Merlian et al., 1979). Liquefaction time of the camel semen has been reported to vary from 4.50 - 9.61 minute (Abdul-Rouf and El-Naggar, 1976). Liquefaction time in-vivo and in-vitro needs to be studied. Spermatozoa have to go capacitation before getting fertilizing ability. Literature lacks information on capacitation and acrosomal reaction of camel spermatozoa.

Proper time, not only involves, the time during an estrus period best suited for the successful union of the ovum and the sperm cell, but also involves the optimum cyclic conditions of the female reproductive tract that affect the likelihood that the female will conceive. Gupta et al (1978) and Musa et al., (1992) have recommended that the optimum time for insemination with fresh semen is the first day on which the camel shows signs of estrus; ovulation usually occurs 24-36 hours later. However, when inseminating with frozen semen, it is good to inseminate twice, 24 hours apart, to be sure of supplying enough ovulation-inducing factor. Best results have been obtained when semen is inseminated 24 hours after mating with a vasectomized male (Anouassi et al., 1992).

Site of Insemination

Artificial insemination in camel is either vaginal or uterine. The uterus is bicornuate. The cervix is dilatable and two fingers can easily be inserted into it at the time of follicular activity. This suggests an easy bypass of cervix by AI gun for insemination or for embryo transfer (Musa and Abusieina, 1978a). The incidence of ovulation after deep vaginal and uterine inseminations were reported 87 per cent and 100 per cent, respectively, reflecting the possibility of higher conception rate with uterine insemination in camel (Chen et al., 1985).

Amount of Semen and Sperm Number to Inseminate

Artificial insemination is a tool of rapid genetic improvement using superior genome of male on population basis. Thus in exploiting available superior genome to a maximum number of females, naturally interest arises to number of spermatozoa required per insemination for optimum conception rates. Camel semen is peculiar in that around 70-80 per cent of the ejaculate is composed of secondary sex gland secretions. This makes the semen very thick and gelatinous immediately after collection. The volume of the ejaculate ranges from 2 to 10 ml (average 3 ml) and sperm concentration is around 200-300 x 10^6/ml in dromedary camels (Anouassi et al., 1992).

Much work has been done on dairy cattle and the use of $10x10^6$ / ml., motile spermatozoa per insemination, for optimal conception rate, is almost universal. However, the information available on camel is limited and primarily related to ovulation studies. In the bactrian camels, insemination with 1.2-4.0 ml of camel semen, having 5-8 x 10^6 /ml sperm cells (at maturation of Graffian follicles) deep into the vagina, all the females ovulated by 30-48 hours after insemination (Xu et al., 1985). These authors suggested that the least amount of semen required to elicit ovulation is 1.0 ml. Good results have also been obtained, in the dromedary camels, by the use of diluted semen with an extender containing 11 per cent w:v lactose and 20 per cent v:v egg yolk to a final sperm concentration of $50x10^6$/ ml (Anouassi et al., 1992).

Insemination Technique and Equipment

The technique of rectal palpation and gynaecological examination of the camel have been described (Arthur et al., 1986; Mobarak and El-Wishy, 1971). Use of long insemination tubes in the first successful AI has been reported in camel (Elliot, 1961). A disposable plastic pipette such as used for bovine insemination or a rubber insemination tube of the type used for horse AI (Chen et al., 1985) inserted gently and as deeply as possible into the vagina or uterus can be used for camel insemination. Liquid semen could be injected through the tube by means of a syringe. For semen frozen in straws, the same type of equipment has to be used as with bovines.

The rubber insemination tube or insemination gun should be more than 37 cm long in relation to the length of vulva to uterine body of the camel (Djang et al., 1988) or simply 42 cm long x 0.9 cm out side diameter.

Unlike in cattle, buffalo and other farm animals, camel is inseminated restrained preferably in sitting position. However, some authors (Anouassi et al., 1992) inseminated 32 female camels in standing position using raw or diluted semen, depositing semen into the uterus using a mare inseminating catheter guided through the cervix by manipulation per rectum. Their results suggested that two of the six females ovulated following insemination with the whole ejaculate of undiluted fresh semen. Two of the five camels ovulated following insemination with fresh diluted semen and six camels ovulated out of ten that were inseminated with 2 ml fresh diluted semen after mating with vasectomised male.

Embryo Transfer Technology

Embryo transfer technology offers many advantages to commercial animal breeder and is the routine practice in several species, including the cow, sheep, goat and horse. The method offers the chance to increase the overall rate of progress in genetic improvement, to increase the productivity of a particular female and to shorten the gestation interval. In the camel breeding, embryo transfer would be of particular value to increase the number of progeny from desirable male and female genetic combinations, whether this is for racing or production of meat or milk (Yagil and vonCreveld, 1990). Furthermore, due to camel's long gestation period (13 months) and its restricted breeding season, judicious use of embryo transfer could increase reproductive efficiency in this species.

The results of the preliminary studies have highlighted some important facts about embryo transfer in camels. On the positive side, they show that the female dromedary can superovulate in response to readily available gonadotropin preparations and will yield multiple embryoes if mated optimally in relation to follicular growth. These embryoes can be transferred non-surgically to synchronized luteal-phase recipient camels, or to unovulated progesterone treated camels, to achieve some pregnancies. On the negative side, however, the superovulatory response to exogenous gonadotripin therapy varies tremendously between individual camels. The difficulty in inducing and synchronizing ovulation in groups of unmated camels to create recipients have also been observed, which shows disappointingly low pregnancy rates following non-surgical transfer of embryos to the recipients. Finally, the difficulty of inducing female camels to enter the stocks, and the great resentment of camels to rectal and/or vaginal penetration, make the processes of embryo recovery and transfer manipulatively difficult (Skidmore et al., 1992a).

One further potential problem for embryo transfer in the camel concerns the lifespan of the corpus luteum, the nature of the maternal recognition

pregnancy signal that ensures its maintenance of function during pregnancy (Short, 1969), and the time after ovulation when the developing embryo transmits this vitally important message to the maternal organism. Measurement of peripheral plasma progesterone profiles in camels, in which ovulation has been induced hormonally or by a sterile mating, shows a precipitous drop in levels to baseline around days 9 and 10 after ovulation (Agarwal and Khanna, 1990; Marie and Anouassi, 1987). This suggests that, in the pregnant animal, a viable conceptus must be present in the uterus and releasing its maternal recognition of pregnancy signal by no later than day 9, if it is to prevent luteolysis and maintain pregnancy (Skidmore et al., 1992a). This is appreciably earlier in gestation than the equivalent obligatory presence of the conceptus in the uterus to prevent cyclical prostaglandin F_2 alpha release around day 14 to 16 in the sheep and cow (Moor, 1968) and in the horse (Hershman and Doughlas, 1979). Because the camel embryo does not begin to elongate until day 10 (unpublished observations), it is probable, that as in the horse between days 6 and 16 after ovulation (Ginther, 1985), the camel embryo remains mobile in the lumen of at least the left uterine horn between days 4 and 10 after ovulation to release its anti-luteolytic maternal recognition of pregnancy signal onto the surface of the endometrium. There is good evidence in cattle, sheep and other species (Adam et al., 1992) that the shock and trauma of the recovery and transfer processes will induce a temporary slowdown in development of most embryos. If this occurs in the camel, recovery of day 7 expanding blastocysts, and transfer to synchronous on day 8 recipients, may not allow the necessary interval for the embryo to recover and be sufficiently advanced developmentally to release enough maternal recognition of pregnancy message to suppress release of uterine luteolysin that is poised to commence by as early as day 9. Therefore, it may prove advantageous to flush the donor animals on days 5 or 6 after mating and to transfer embryos to recipients that are one, or possible two, days negatively asynchronous with the donors. This works well in the horse (Allen, 1982) and it may improve the results in the dromedary camel (Skidmore et al., 1992a).

More fundamental research on the embryology and reproductive physiology of dromedary is required to achieve maximum response and success rates in embryo transfer.

PATHOLOGY OF MALE AND FEMALE REPRODUCTIVE SYSTEM

INFERTILITY

The fertility of the female camels is maintained throughout their lives and breeding in alternate years is the usual practice. The fertility of female camels is apparently high and the herd owners claim that 80 - 90 per cent of those mated in one season produce calves (Arthur et al., 1986). A similar, fertility rate has been reported in herds managed intensively (Abdel-Rahim and El Nazier, 1990; Yagil, 1985). The abattoir studies confirmed that the structural defects of the genitalia, including cystic ovaries and ovarobursal adhesions, are relatively rare (Arthur et al., 1989; El-Wishy, 1990), and endometritis, associated with a partially involuted uterus and a regressing corpus luteum is sometimes seen (Arthur et al., 1989). According to some reports the reproductive performance of camels is poor. It has been found that the fertility in the pastoral herds of dromedary camels was unlikely to be higher than 50 per cent, or up to 65 per cent under improved management conditions (Mukasa-Mugerwa, 1981). It has also been seen (Wilson, 1989) in field studies involving a large number of Asian and African countries that delayed puberty (First pregnancy at five years of age), long

interval between births (>24 months) and early culling of breeding females limit the average production of calves to less than three per female. Anestrus, due to malnutrition or debilitating disease, is probably a major cause of infertility. Embryonic death is known to occur, especially in twin gestations. Abortions are reported occasionally but the causative role of Brucella abortus in the dromedary camel is not yet clear. Poor nutrition and trypanosomiasis are probably responsible in part for the embryonic death (Arthur, 1992).

Reports of bacterial infertility in female dromedary camels are scarce and most investigations have been in slaughtered camels with no previous breeding histories (Merkt et al., 1990). Corynebacteria, Anthracoids, Micrococci, Sarcina, Gram-positive and Gram-negative bacilli were isolated from the normal genital tract of pregnant and non-pregnant slaughtered camels (Zaki and Mousa, 1965). The latter workers suggested that the bacteria isolated from the amniotic fluids in some pregnant camels either ascended through the cervix or came via the maternal bloodstream from a focus in the dam. Almost, the same spectrum of bacterial species were found in an other study (Eidarous et al., 1983) in which Staphylococcus epidermidis and Escherichia coli were also identified. In 94 dromedary camels (4.5 %) out of 2,075 slaughtered at the Cairo abattoir, lesions such as uterine abscesses, cattarhal endometritis, haemorrhagic endometritis, pyometra with macerated fetuses were recorded (Nawito, 1973). Micrococcus pyogenes var. aureus played the predominant role in the endometritis cases but B-hemolytic streptococci, E. coli, and Pseudomonas aerugenosa were also recovered from the uteri. No bacteria were isolated from some endometritis cases (Nawito, 1973). The author suggested that other microrganisms such as protozoa or virus could be involved in such cases.

Also in Egypt, a wide range of organisms was recovered from 24 uteri, which showed clinical signs of endometritis (Hegazy et al., 1979). They included Proteus sp., Serratia sp., Enterobacter sp., Klebsiella sp., E. coli, enterococci, Bacillus sp., Corynebacterium renale, C. pyogenes, Staphylococcus aureus, Micrococcus sp. and Streptococcus pyogenes. These authors observed that the most severe purulent cases of endometritis were associated with presence of C. pyogenes. Campylobacter fetus and Trichomonas fetus have also been isolated from the uteri of breeding camels in two different herds (Wernery and Ali Amjad, 1989; Wernery, 1991; Wernery and Wernery, 1992). Despite being mated to different bulls, the infected camels had remained barren for the previous one to four years. Endometritis was also diagnosed in the camels from which opportunist pathogens were isolated. Campylobacteriosis and Trichomoniasis are notifiable diseases in cattle, characterized by profuse mucoflocculent discharge from the uterus, early embryonic death and prolonged infertility. The studies of the latter authors show that both these venerial diseases exhibit a similar pathogenesis in camels and cattle. Streptococcus zooepidermicus and Taylorella equigenitalis a common cause of endometritis in mares (Fontijne et al., 1989), have not been reported in camels.

In light of the above the detection, prevention and control of venereal diseases should be an important component of camel stud management to improve reproductive performance in this species.

OVARIAN ABNORMALITIES

CYSTIC OVARIES

Large, thick walled, fluid filled ovarian cysts, 50 to 80 mm in diameter and 83 to 250 g in weight are occasionally, observed in the ovaries of camels. They may occur either singly or in pairs and they often replace most, if not all,

the ovarian stroma. The fluid within the cyst varies from yellow and watery to brown and viscous. Hemorrhagic cysts containing reddish-brown fluid are also observed (El-Wishy, 1992).

Histologically, these cysts vary according to their type. The granulosa layer is sometimes reduced to one or two flattened cells or it may have disappeared completely to be replaced by a dense layer of fibrous tissue adjacent to a broad layer of luteinised theca cells. In cattle, variation in the width of the granulosa layer of cysts might be related to their age, with older cysts being devoid of granulosa cells altogether (Al-Dahash and David, 1977). Moreover, it has been proposed by the latter authors that variation in the structure of the granulosa and theca layers may have a bearing on the hormonal activity of the cysts and, therefore, the behavioural patterns of the affected animals. In this respect, very high levels of progesterone have been measured in the fluid of camel ovarian cysts (El-Wishy, 1988). However, the possible influence of ovarian cysts as a cause of infertility in camels is not known.

Unilateral or bilateral, single or multiple ovarian teratomas, 10 to 30 mm in diameter, may occur in the ovaries of camels. They have been reported to occur quite frequently in the ovaries of alpacas (Sumar, 1983) and camels (El-Wishy, 1988) but are observed rarely in the ovaries of other domesticated animals (Jubb et al., 1985). They appear as smooth, thick walled, grey-colored structures, which on sectioning exude sebaccious material and hair. While they remain under 30 mm in diameter, these ovarian teratomas do not seem to influence ovarian activity. Follicles and corpora albicans can be observed along side a teratoma in the same ovary (El-Wishy, 1992).

The cysts may be present within the ovarian tissue or outside the tissue. The cysts outside the ovaries are called parovarian cysts. Two types of ovarian cysts are encountered i.e. follicular cysts and luteal cysts. The cysts are very frequent in camels if they are not bred, as the camels are induced ovulators. The effect of cysts on fertility is not well studied in dromedary. The cysts can be detected by rectal palpation or by ultrasonography. Parovarian cysts are located in the broad ligament near the ovary or uterine tube. They can be single or multiple, unilateral, bilateral, round, or oval, measuring 0.5-5 cm in diameter.

The variation of the effect of cysts on follicular activity may be dependent on the nature of the cyst and its endocrine activity (Bravo et al., 1996).

Treatment

Treatment for most of the cysts is often not required. The reason being, that these are normally encountered in unmated females. Practice of manually breaking the follicles should be avoided. Leutinized anovulatory follicles may be treated with prostaglandin F2-alpha. The ideal approach suggested by Tibary and Anouassi (1997) is to first induce leutinization with 5000-10,000 i.u. of human chorionic gonadotropin, followed 5-6 days by an injection of PGF-alpha. The latter may be repeated at 24 hours.

OVARIAN HYPOPLASIA

This condition is very common in females coming back from race and in under nourished females. The condition can be diagnosed by either rectal palpation or ultrasonography. The size of hypoplastic ovary would be ½ or 1/3 of the normal size (Bravo et al., 1996).

Treatment

Dietary regime of the animal should be improved. Chronic illness should be dealt with accordingly. Administration of follicular stimulating hormone and

equine chorionic gonadotropin is recommended for the treatment of this condition.

OVARIO-BURSITIS WITH ENCAPSULATION

The condition is characterized by encapsulation of ovary and accumulation of fluid of varying quantities in the capsule. The non-affected side continues functioning properly. It can be unilateral or bilateral.

An incidence of about 9.3% has been noted in dromedary camel (Tibary and Anouassi, 1996). The incidence is even higher in animals with history of reproductive failure. It has also been reported to be associated with echinococcus infestation (Shalash and Nawito, 1963). Diagnosis can be made by rectal examination and by ultrasonography. The condition is suspected when there is difficulty in retracting uterus or while reaching for grasping the ovaries. Tibary and Anouassi, (1997) have suggested three causal factors.
1). Hereditary predisposition (incidence more in racing varieties); 2). possible relationship between recurrent anovulatory haemorrhagic follicles and bursal adhesions; and 3). Infection spreading from else where.

Treatment

The only recommended treatment for valuable camels is the surgical removal of ovary and bursa.

OVARIAN TUMORS

Ovarian neoplasms do not cause a major reproductive problem in camels. The incidence reported is about 0.06-2.0%. Mostly teratomas have been reported (El-Khouly et al., 1991; El-Wishy, 1990). Teratomas usually affect one ovary. They are benign tumors and grow very slowly. The tumors are detected through rectal palpation and through ultrasonography. Dysgerminoma is another type of very rare tumor of ovary (El-Khouly et. al., 1990).

OOPHORITIS

Inflammation of ovaries may be caused by ascending infection from the uterus or may come from the peritoneum. The follicular activity is lost because of the adhesions formed between the ovaries and surrounding tissue. Diagnosis is based on rectal palpation and ultrasonography. The condition can be confirmed through laproscope. A number of organisms, including Mycobacterium, Brucella and Campylobacterium, may be involved. The lesions depend upon, the causative agent. Abscesses and adhesions are commonly formed.

DISEASES OF THE OVIDUCTS

The most common condition in camels is inflammation and adhesions in oviduct with occlusion and accumulation of clear fluid or pus in the tubes (Nur, 1984). The accumulation of serous fluid may also be caused due to congenital segmental aplasia. Diagnosis can be made by rectal palpation or by ultrasonography. The condition usually is not manifested clinically, unless it is bilateral and causing infertility. The infection may have spread from ovary or uterus. A large number of organisms have been reported to cause the infection including Campylobacter fetus, Escherichia coli, Actinomyces pyogenes, Brucella, Mycobacterium and Mycoplasma. The fertility prognosis in the case of salpingitis is not good. Surgical intervention in unilaterally affected animals may be considered; if it is certain that, the other side is potent. The condition may be prevented by limiting, the risks of infection.

DISEASES OF THE UTERUS

CONGENITAL ABNORMALITIES

A case of segmental aplasia of uterus in a newborn female dromedary with atresia ani and direct communication between the rectum and the vaginal / uterine cavity with accumulation of fecal material in the uterus has also been reported (Tibary and Anouassi, 1997).

METRITIS

Uterine infections are the most common cause of infertility in camels (Nur, 1984; Wernery, 1991; Wernery and Amjad Ali, 1989).

The uterus is prone to more risk of infection during breeding and after parturition in camels. Infection is usually from outside (posterior). In the initial stages infection is attempted to be over come by the defenses of the body. If the defense mechanisms fail the infection is established. The major defense mechanisms include local immunoglobulin activity, phagocytosis, and mechanical elimination by mucous entrapment and myometrial contractions. These mechanisms are said (Tibary and Bakkoury, 1994) to be more effective during high blood estrogen levels (follicular phase). A number of organisms can cause metritis which include: Corynebacterium pyogenes, E. coli, beta-hemolytic streptococci, Staphylococcus sp., Klebsiella pneumonie, Aspergillus sp., and Mucor sp. and Compylobacter faetus (Wernery and Amjad Ali , 1989).

The organisms responsible for purulent metritis (pyometra) are Actinomyces pyogenes, Pseudomonas aeroginosa, E. coli, Staphylococcus epidermidis, Streptococcus pyogenes, and Proteus sp. (Nawito, 1973).

Clinical Findings

Metritis should be suspected with the history of repeat breeding or early embryonic death after normal pregnancy. Examination of the prenium, base of the tail and vulva may reveal presence of mucous or pus. On rectal palpation and ultrasonography the wall of the uterus is thickened. The uterine cavity contains mucous or pus. The condition may be confirmed by vaginoscope. The cause, may be determined by culture, and extent and type of inflammation may be determined by biopsy and histological examination. A particular form of endometrial granulomatous nodular lesion, caused by Campylobacter fetalis, has been observed in dromedary camels (Tibary and Anouassi, 1997).

Treatment

For treatment uterus should be washed and infused with some antibiotic. Some antibiotic should also be given parenterally. Penicillin gives good results. Trimethoprime plus sulphadiazine is also effective.

OTHER UTERINE DISEASES

Other diseases of uterus reported in camels are uterine cysts, abscesses, peri uterine adhesions, and polyps (Ribadu et al., 1991). The lesions can be detected by ultrasonography and by fibroscope. The cysts result from enlargement of endometrial glands or lymphatics and can badly affect the fertility of the animal. The abscesses result of mal handling while passing pipette or follow caesarian section or retention of placenta.

DISEASES OF THE CERVIX

Remnants of the mesonephric ducts can lead to the formation of cervical cysts. These cysts have been found in 0.06% of the camels out of 1701 camels studied in abattoir (Shalash, 1965).

El-Wishy (1989) have described segmental aplasia to occur in dromedary camels. Incomplete development of the cervix has been reported in 0.12% dromedary camels out of 850 cases studied in slaughterhouse. These anomalies usually result in the accumulation of mucous in the uterus (El-Wishy, 1990). A case of double cervix with a complete vaginal septum and communicating uterine horns have been described (Tibary and Anouassi, 1997).

The most common acquired disease of cervix is its inflammation, mainly due to injuries encountered during parturition or gynaecological manipulations. It usually co occurs with metritis and / or vaginitis. The most common type of inflammation is mucopurulent, however, one may also find adhesions.

DISEASES OF VAGINA AND VULVA

Occurrence of **segmental aplasia, persistent hymen, vaginal constriction,** and **presence of vaginal septum** have been reported (El-Khouly et al., 1990; Shalash, 1965). These anomalies impose hindrance to penile intromission. These conditions may be corrected by surgery. Two cases of **atresia vulvi** in newborn camel calves have been reported. The urine was retained in the vagina thus creating a soft flucuating swelling ventral to the anus. A vulvular opening was made in each animal, successfully (Ramadan, 1997).

VAGINITIS

This is a very common condition observed in camels. It occurs due to mal handling during breeding examination of genital tract and parturition. It gets started as traumatic. The mucous membrane is lacerated, congested and sometimes there is discharge depending up on the type of infection. It may be mucous or purulent.

EMBRYONIC DEATH

Early embryonic death during the first 50 days of pregnancy has been reported in 35% of all bred females (Tibary and Anouassi, 1997). This phenomena is well known to herdsmen and is recognized by them if the dromedary female fails to show tail curling, a typical pregnancy behavior in the presence of a male or a person, after the camel had been declared pregnant. The condition can be diagnosed by ultrasonography or by hormonal assay (low progestron). The tone of the uterus is increased when examined per rectum due to decrease in progesterone level. Corpous luteum either disappears or decrease in size.

There are many causes of early embryonic death. It may be due to abnormalities of the conceptus, failure of conceptus to develop because of bad uterine environment and abnormalities of corpus luteum function. According to Tibary and Anouassi, (1997) most common factors causing early embryonic death are inflammation of the uterus and failure of corpus luteum to be maintained. Some authors believe that most of the early pregnancy is due to failure of conceptus to move to the left horn, where almost all conceptuses develop in camels. Luteolysis is caused by the release of PGF2-alpha, metritis, or

manipulation of genital tract is probably the most common cause of embryonic death. An abortion due to Bacillus cereus toxicosis has been reported by Wernery et al. (1996).

Treatment

The cause of early embryonic death should be established. This consists of thorough clinical and hormonal profile examination. The object of this practice is to establish whether ovulation has occurred and corpus luteum has formed after breeding. Corpus luteum can be detected by ultrasound and by progesterone monitoring.

If the problem is with the uterus, it should be washed and disinfected. Deficiency of progesterone may be treated by exogenous progesterone administration, 50-100 mg daily by injection (Tibary and Anouassi, 1997).

ABORTIONS

The expulsion of dead or nonviable fetus, before term, is called abortion. Abortion rates in dromedaries range from 2-18% in various studies (Agarwal and Khanna, 1990; Agarwal et al., 1987; Bhargava et al., 1963; Enany et al., 1990; Saley, 1990). Suspected causes of abortion include brucellosis, uterine infections, toxoplasmosis, trypanosomiasis, twining, stress and trauma (Al-Khalaf and El-Khaladi, 1989; Enany et al., 1990; Hagemoster et al., 1990; Egbe Nwiyi and Chaudhary, 1994; Musa and Abusineina, 1976; Tibary and Anouassi, 1997).

PROLAPSE OF VAGINA

The prolapse of vagina can occur at any time of pregnancy, however, the condition is more common during last 3 months of pregnancy (Arthur and Al-Rahim, 1982). The cause of the condition may be due to loosening of the tissue due to increased esterogen levels.

According to Arthur and Al-Rahim (1982) and Gitao and Akabwai (1989) the condition is more common in older females and females with good body condition (overfed and lack of exercise). The prolapse can be very little or can be as big as up to cervix. If the prolapsed portion remains out for sometime inflammation increases and even necrosis of the tissue can occur. In advanced cases risk of abortion and prolapse of rectum increases. This can lead to other complications and even death of the animal.

The condition should be treated at the earliest to save life of fetus and dam. The prolapsed portion should be cleaned with mild antiseptic solution, some antibiotic cream or sulphonamide should be applied and prolapsed portion be replaced manually (one may use epidural anaesthesia and spray the prolapsed vagina with local anaesthesia) and secured by applying sutures around the vulva. The sutures can be removed if signs of parturition are observed. Application of truss may also be helpful in securing the prolapse.

POST PARTUM DISEASES

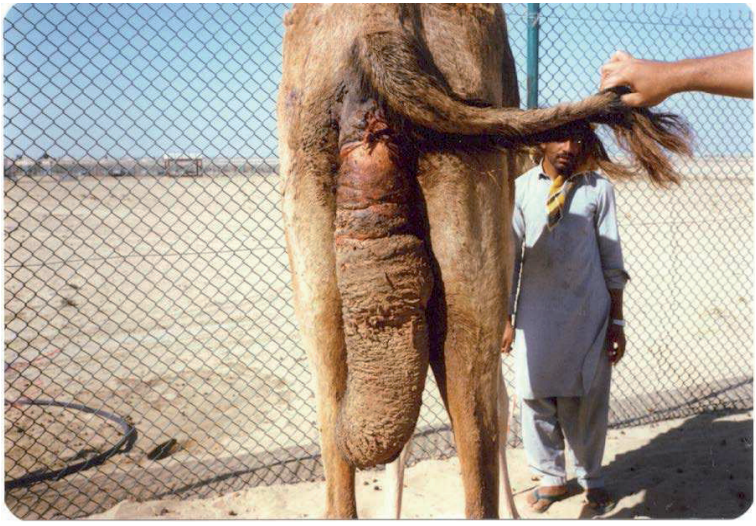
RETENTION OF PLACENTA

It is failure of placenta to be expelled within normal time (12 hours after birth in dromedary). The prevalence of retained placenta is low in camels. The chances of retained placenta increase following dystokia and cesarean section. Retained placenta may require manual removal, observing all aseptic precautions. Local and parenteral antibiotic treatment is indicated after removing the placenta to prevent complications. Oxytocin may also be given to expel any exudate collected in the uterus.

PROLAPSE OF UTERUS

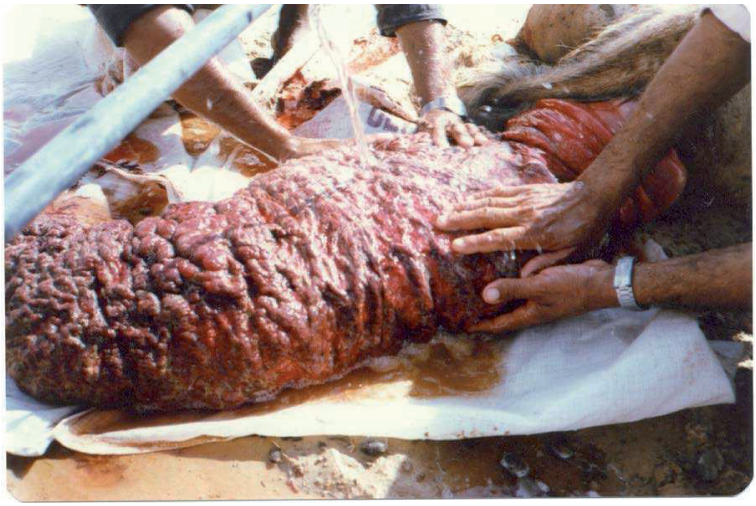
Uterine prolapse usually follows dystokia, retained placenta, or excessive obstetrical manipulation. Old age is considered to be predisposing cause, because of the relaxation of ligaments. It is also said to be common after abortion (Tibary and Anouassi, 1997). In most cases, there is complete prolapse of uterus. The prognosis of the prolapse depends upon the size of uterine prolapse, the time for which it has occurred, extent of infection, and the extent of tissue damage (hemorrhage etc.). Increase in all of these factors can lead to shock.

Recent uterine prolapse is easy to replace. But in case of complete and complicated prolapse it is advisable to inject epidural anaesthesia. The uterus should be replaced in sitting position on a slope with hind part elevated. It is also good to calm the animal with xylazine (this should be avoided if the animal has already gone in shock). The uterus is put on prolapse tray or plastic and washed thoroughly, with mild antiseptic. Placenta is removed and if there is laceration or rupture, it should be sutured, and ruptured blood vessels should be ligated. Wrapping the prolapsed uterus in the towel and squeezing it can reduce the edema. The uterus is then replaced commencing at the portion near the vulva and



Post-partum uterine prolapse

forcing it in by holding the organ slightly above the vulva. Once the uterus is partly replaced, the rest of the portion will be replaced easily. When the entire uterus has been replaced, antibiotic pesseries should be put in the uterus. Oxytocin should be injected. The vulva should be sutured. Parenteral antibiotics should also be given.



Post-partum uterine prolapse. Washing and cleaning before replacing back

DYSTOKIA

Difficult birth is known as dystokia. The incidence of dystokia in camels is very low (Arthur et al., 1986). Incidence of 4.6% has been reportd by Tibary and Anouassi (1997).

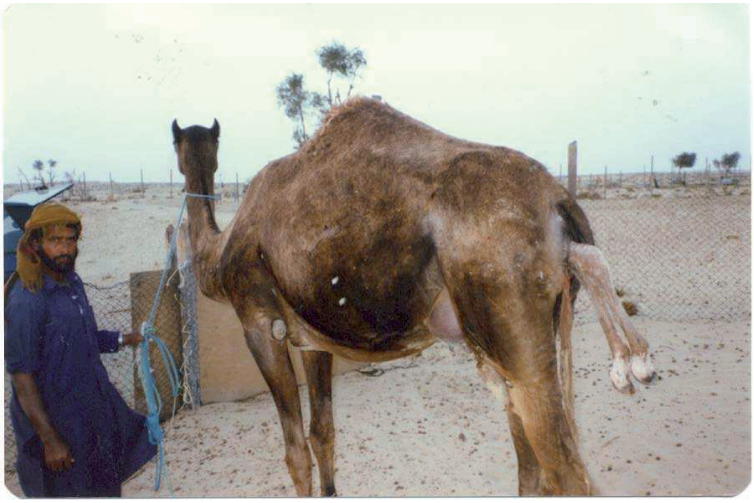
At any time or stage of parturition dystokia can occur. The most important sign of dystokia is increased duration of stages of labor. Dystokia should be suspected if the first or second stages of labor exceed, respectively, 6 and 2 hours. The animal also shows signs of distress with frequent alteration between standing and sitting positions, frequent rolling from side to side, and excessive straining. Many dromedary females will show profuse diarrhea and frequent vocalization in case of dystokia.

The examination should be accomplished on the standing animal or in sitting position, restraining the animal adequately. The tail should be secured by tying it to one side. Rectal palpation facilitates to see the occurrence of torsion. After thoroughly cleaning and disinfecting the exterior of the animal and lubricating the hands with antiseptic gel examination of the birth canal should be done. One should examine for the presence of torsion and if the cervix is open and the fetus is alive. This can be done, by pinching the fetus at its various parts and observing the reaction.

The dystokia can be classified in two types, i.e. maternal dystokia and fetal dystokia.

Maternal dystokia: Maternal dystokia develops due to displacement of uterus, uterine inertia or due to disparity between fetal size and pelvic canal. Uterine torsion is probably most common cause of maternal dystokia. The best solution is to perform caesarian section to deliver the calf. Uterine inertia is observed in very

weak and old animals or when the pregnancy is delayed. Injection of calcium borogluconate and oxytocin may be of value in helping this type of dystokia.



Fetal dystokia

Fetal dystokia: Fetal dystokia is due to abnormal position or posture of the fetus and monstories. Fetal monstories are rare in camels but have been reported (Chandolia et al., 1991) and also seen by the author in dromedaries.

Dystokia due to the small size of the dam is not frequent if breeding management is adequate. However, some cases of feto-maternal disproportion have been described in dromedaries (Sharma and Pareek, 1970). The dystokia may be helped by stretching the vulva or by performing episiotomy. In case of failure caesarian section should be attempted.

Abnormal presentation, posture, or position of the fetus, are the most common causes of fetal dystokia. Correction of the position is difficult in camels because of the long legs and neck and relatively narrow birth canal. Position needs to be corrected by various techniques used in other large animals to deliver the young one. If not successful, one has to resort to fetotomy or caesarian section.

TORTION OF THE UTERUS

It is rotation of the uterus on its long axis during pregnancy. The uterus may rotate clockwise or anti clockwise. It may be complete or partial. Uterine torsion is one of the most important causes of maternal dystokia (Arthur and Al-Rahim, 1982; Nigam et al., 1977).

The female is referred to the doctor after a long unsuccessful first stage of labor. The animal shows sign of discomfort, is off feed and gets isolated, frequently sitting and rising up. Diagnosis can be made by rectal and vaginal palpation. Rectal palpation shows a tense dorsally displaced broad ligament. Right broad ligament pulled strongly downwards under the twisted uterine body towards the right side suggests right uterine torsion. At the same time the left

broad ligament is pulled tightly across over the top of the cervix, the body of the uterus and the vagina towards the right side. The situation is the reverse in left uterine torsion. The fetus is often not possible to be reached in torsion more than 180 º.

Vaginal palpation reveals the presence of spirals on the wall of the vagina, directed to the right in the case of right side torsion and towards the left in left sided torsion. The birth canal is felt very tight and cervix and fetus are not palpable in the case of torsion more than 180º. The torsion compresses the blood vessels and decreases the blood supply to the uterus, which may cause death of the fetus, uterine wall necrosis, and rupture. Prolapse of uterus may also follow.

If treated early by caesarian section, the prognosis is good. Rolling of the female while maintaining the uterus in place by pressure against the abdominal wall don't work well in camels because of their long legs and presence of hump.

The other conditions that can be encountered during pregnancy are hemorrhages of the uterus, ventral edema (lack of exercise), fetal maceration or / and mummification.

AGALACTIA

Agalactia is the absence of lactation due either to a failure in milk let down or a failure of development of the mammary glands. It may be observed in camels (Kohli et al., 1988). It is likely to be caused by inadequate milk let down due to nervousness of the female or due to udder edema and increased pain. Failure of milk let down can be resolved by ensuring that the female is calm and by I/M administration of oxytocin (40 IU). The injection should be repeated every 2 hours. Massage of the udder and stripping of the teat may aid in reducing edema and udder congestion. Agalactia may also be associated with rejection of the new born in many primiparous females. These cases require close monitoring of the dam and her young until they bond. Colostrum feeding should be done in adequate volume within the first 12 hours of life. Colostrum from a recently calved dam should be bottle-fed to the newborn. It is important to keep a stock of frozen colostrum on hand for such cases.

DISEASES OF MALE REPRODUCTIVE ORGANS

The reproductive failure in the male camel is atributed mostly to traumatic orchitis, balanoposthitis, phimosis, and paraphimosis (Hemeida et al., 1998).

INFLAMMATION OF THE PREPUCE

Inflammation of the prepuce can occur due to various types of irritants, i.e. chemical, physical, and infectious. Tick infestation can also cause inflammation of the prepuce. Swelling of prepuce may also be seen in ventral edema, which may be due to trypanosomiasis. Thorough cleaning and application of suitable antiseptic or antibiotic lotion or cream could treat the condition.

PARAPHYMOSIS

Inability of penis to be retracted in the prepuce is rare in camels, however, it can be seen during sexual activity and in camels having tendency for masturbation. If not treated promptly paraphymosis becomes complicated by accumulation of sand in the prepuce and can lead to balanoposthitis and sometimes necrosis of the tip of the penis. Treatment comprises replacement of

the prolapsed tip. This may require sedation of the animal, debridgement, urethrostomy or amputation of the penis if there are bad adhesions or gangrene of the tip of penis. After retraction, it is recommended to apply anti-inflammatory and antibiotic cream twice daily for several days.

PHYMOSIS

It is the inability of the penis to protrude from its preputial sheath. Mainly it is due to congenitally small preputial opening or due to abscesses or other nodular lesion preventing the penis to come out for copulation. Surgical correction of the hindrance is recommended.

HYDROCELE AND PYOCELE

Hydrocele is the abnormal collection of various quantities of fluid between the visceral and parietal layers of the tunica vaginalis. The condition may be inflammatory or it may be due to accumulation of edema fluid. The latter condition is painless, cold testes are usually free in the scrotum. Exploratory puncture yields transudate. If the condition is inflammatory, it is painful and exudate is recovered instead of transudate. The condition develops following obstruction of the normal flow in the cord. Long standing hydrocele affects the thermoregulation of the testicles and decreases the semen quality and quantity.

Nigam (1992) has reportd occurrence of pyocele (pus in the tunica vaginalis) in dromedary camel. The condition is very painful. Treatment comprises drainage of pus and application of antiseptic or antibiotic solutions plus parenteral administration of antibiotics.

HEMATOMA OF THE TESTES

It can result from severe trauma. In the absence of infection, the hematoma resolves within few days.

TUMORS OF THE TESTES

The occurrence of tumors in camels is rare, however, Homeida et al. (1985) have reported one seminoma out of 310 testes examined by them.

FUNICULITIS

Acute or chronic (Schirrous cord) infection of the testicular cord is usually a complication of castration. It can also occur due to scrotal bite (Chouhan et al., 1981). Treatment for schirrous cord is to remove it surgically under general anaesthesia.

ORCHITIS

The condition is rarely observed in camels. A prevalence of 7.7% due to Dipetalonema evansi (filaria) infestation has been reported (Homeida et al., 1985). Infectious orchitis may be treated with parenteral administration of antibiotics. Castration is advisable in unilateral orchitis.

CRYPTORCHISM

Failure of testes to be descended into scrotum is termed cryptorchism. This condition occurs very rarely in camels (Homeida et al., 1985).

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NUTRITIONAL DEFICIENCIES

Camels thrive in areas where there is scanty and unreliable rainfall resulting in seasonal availability of fodder mainly of poor quality. The desert living camel is renowned for finding its sustenance from fodder that is left untouched by other animals or having too low nutritional value for them. Camels have physiological digestive capabilities that enable them to extract adequate nutrition from almost any plant material. The fodder that is normally available consists mainly of grasses, leaves and twigs of shrubs and trees. The long legs allow the camel to browse from upper-story and lower-storey vegetation, covering long distances each day in search of food and water. The camel is a browser by nature and therefore does not destroy its food sources as do the intensive grazers (Newman, 1975). The camel takes a bite from here and a bite from there, continually moving, often covering about 50 km a day, even if good feed is available. This method of grazing is beneficial for the vegetation, acting as a natural pruning. Plants that are naturally available and preferred by the camel over others, belong to the species Acacia, Salsola, and Atriplex (Westrn, 1989). These plants have high salt, as well as water content. They are mainly bitter and thorny as well, and are not liked by other animals grazing in the same area.

The desert plants fall into two general biological types: (a) the perennials, which are adapted to the scarcity of water and heat and (b) the annuals, which show virtually no adaptation to the harsh climatic conditions. The adaptation of the perennials to the harsh desert environment includes thickening of the plant covering, reduction of the leaf size, and curling of the leaves, especially the grasses. There is often even a complete disappearance of leaves. All these features aid in reducing transpiration losses by the plants. These changes also make the plants virtually inedible for animals other than the camels. This is especially true for those plants whose leaves have evolved into thorns. The thorns not only reduce transpiration losses of the plants but also protect the plants from grazing animals. The desert perennials are further characterized by their extended root system, which is necessary for gaining adequate moisture for the plant. Therefore, drier the climate, the farther apart plants grow from each other. The camel does not dig for roots, as do goats, therefore the plants are unaffected by grazing. The browsing habits of the camels lend themselves to this plant growth system. The annuals undergo morphological and 'behavioral' adaptations in the desert environment. Morphologically they are small plants, thereby diminishing the transpiration surface. 'Behaviorally' they have a very short life span. They are able to respond to a single rainfall by sprouting and maturing in a matter of days. These plants have small roots and large tender leaves. In drought years, the seeds of these plants lie dormant. Only after the autumn rains do the annuals flourish and provide the fodder for grazing animals. Therefore, in drought periods there is a continued dormancy of seeds. The plants remain green for about 4 months and are still edible for a further 4 months when dried (Johnson, 1969).

The idea of changing the farming of camels from free ranging to corralling is slowly becoming acceptable. The benefits range from a better

production of the camels to better social, health, education and economic status of the herder. Not a lot is known about the feeding requirements, especially concerning the food needed for growth, fertility, pregnancy, and lactation. The hand-fed camels eat 25-40 kg of good fodder per day. This amount seems to be far more than the optimum. It is concluded that the anatomy and physiology of the camel's digestive system are adapted to the type of fodder available, which gives the animal its advantage over the true ruminants in arid zones.

Energy deficiency is the most common nutrient deficiency, which can badly affect the performance of animals. The deficiency may be quantitative or qualitative. Quantitative deficiency may be due to inclement weather or the farmer may not be able to feed his animals.

In case of scarcity of feed the camel can decrease its feed intake, as well as it can reduce its metabolic rate. In these circumstances, production is adjusted to energy intake, which in part explains the supposed poor production of the camel. The worst effects are encountered when high demanding physiological states coincide with the dry season. To improve overall camel productivity, it is necessary to establish the minimum nutritional requirements to keep the animal’s body in a stable energetic state (Guerouali and Filali, 1992).

Gihad and El-Bodevy (1992) suggested that maintenance requirements of protein are lower as compared to the cattle, sheep, and goats. Urea seems to be most important end product of nitrogen metabolism in camels, as is the case with other ruminants. In most animals endogenous urea is excreted via kidneys, however, in herbivorous animals not all the urea is excreted in the urine. Some amount returns to the digestive tract via saliva, rumen epithelium, and through the walls of the lower digestive tract. Urea has an important function due to the presence of microorganisms in the rumen of the ruminants. The urea nitrogen is partially incorporated into the microbial proteins, which after proteolysis in the small intestines will be available to the host animal in the form of amino acids thus making a substantial contribution to the nitrogen economy of the host animal. It has been observed (Mousa et al., 1994) that in camel recycling was consistently high (94%) and significantly higher than that of sheep and goats, which was 75 and 78%, respectively. It appears that a physiological limit will be reached by feeding 9.6% crude protein. Increasing the level of protein intake does not enhance the utilization. Increasing the amount of feed protein and elevated ammonia concentration in rumen may depress urea entry from blood to the rumen, which points to the regulating control of urea degradation by the intermediates of the rumen fermentation. Urea metabolism is also closely related to the energy value of the diet. Water deprivation for short periods is beneficial to the camel and other ruminants with respect to urea metabolism and energy utilization (Mousa et al., 1983).

The effect of deprivation will depend upon the age of animal, pregnancy, concurrent deficiency of other nutrients, and environmental conditions. In young animals the growth will be retarded. In mature animals the production will be lessened, they will lose weight, and fertility will be affected. In the pregnant animals prolonged energy deficiency can affect fetal growth or even fetal death and abortion may occur. A sudden dietary deficiency of energy in the camels, particularly pregnant and lactating camels can result in starvation ketosis.

Protein-energy malnutrition occurs in neonatal calves, which are not allowed to suckle by the dam and are given poor quality milk indigestible by the new born calf. Diarrheic calves, which receive only electrolytes and remain deprived of milk for few days, can lose a significant amount of body fat.

The effects of the protein deficiency are usually not so severe as are of energy, at least in the early stages. Deficiency in protein intake or malabsorption in the young animals, result in reduced appetite, lowered feed intake, inferior growth

rate, lack of muscle development, and a prolonged time to reach maturity. In mature animals there is loss of weight and decreased milk production, reduction in total serum protein and albumin. In the male camels, reduction in sperm count, and libido have been observed (Tibary and Anousi, 1997).

The diagnosis is based on estimating the energy and protein contents of feed and requirements of these ingredients in camels, according to the age and physiological status (milk production, gestation, exercise, etc).

For the treatment of acute energy deficiency (ketosis) oral fluid, glucose, and electrolytes may be given as indicated. The provision of high quality feeds appropriate to the species is recommended.

DISEASES CAUSED BY MINERAL DEFICIENCIES

At present, 22 mineral elements are believed to be essential for the higher forms of animal life. These are comprised of seven major or macro-nutrient minerals (calcium, phosphorus, potassium, sodium, chlorine, magnesium and sulfur), and 15 trace elements or micro-nutrient mineral elements (iron, iodine, zinc, copper, manganese, cobalt, molybdenum, selenium, chromium, tin, vanadium, fluorine, silicon, nickel and arsenic).

The mineral elements exist in the cells and tissue of the animal body in a variety of functional chemical combinations. The concentration of minerals varies in the different tissues. The concentrations must be maintained within quite narrow limits, or normal ranges, if the functional and structural integrity of the tissues is to be safeguarded and the growth, health and productivity of the animal are to remain unimpaired. Continued ingestion of diets that are deficient, imbalanced or excessively high in a mineral induces changes, in the form or concentration of that mineral in the body tissues and fluids, so that it falls below or rises above the permissible limits or normal range. In such circumstances biochemical lesions can develop, physiological functions can be affected adversely and structural disorders may arise in ways which vary with the element, the degree and duration of the dietary deficiency or toxicity, and the age, sex and species of animal involved. Homeostatic mechanisms in the body can be brought into play, which delay or minimize the onset of such diet-induced changes. Ultimate prevention of the changes requires that the animal be supplied with a diet that is palatable and non toxic and which contains the required minerals, as well as other nutrients, in adequate amounts, proper proportions and available forms.

Inadequate or excessive intakes of a single mineral element are uncommon in most natural environments. They are often ameliorated or exacerbated, i.e. “conditioned” by the extent to which other dietary components, with which the mineral interacts metabolically, are present or absent from the whole diet or the environment.

With copper the question of mineral balance or dietary ratios is of crucial importance because of the potent influence of molybdenum and sulfur on copper retention but it should be recognized that metabolic interaction significantly affecting minimum requirements and maximum tolerances are widespread and pervasive among the mineral elements.

The incidence and severity of mineral mal nutrition in livestock can be further influenced, both directly and indirectly, by climate factors, such as sunlight and rainfall incidence. Sunlight promotes vitamin D formation in the animal, which facilitates calcium and phosphorus absorption. The phosphorus concentration in herbage plants fall with increasing maturity of the plant and shedding of seeds. In any area the relative length of the dry, mature period of the plants (low herbage phosphorus) and of the green, growing period (high herbage phosphorus) are

determined largely by incidence of rainfall. Heavy rainfall resulting in water logging also increases the availability of some soil minerals to plants, notably cobalt and copper, so affecting the concentration of those elements in the grazed herbage.

Three broad types of functions for minerals exist, although they are not exclusive to particular elements and may all be discharged by the same element at the same time. The three types of functions are:

i). Structural components of the body organs and tissues, such as calcium, phosphorus, magnesium, fluorine and silicon in bones, and phosphorus and sulfur in proteins.

ii). Constituents of the body fluids and tissues such as electrolytes concerned with the maintenance of osmotic pressure, acid base balance, membrane permeability and tissue irritability: sodium, potassium, chloride, calcium and magnesium in blood, cerebrospinal fluid and gastric juice provide examples of such functions.

iii). Catalysts in enzyme and hormone system, as integral and specific components of the structure of metallo enzymes, or as less specific derivatives within those systems.

In the metallo enzymes the metal is firmly attached to the protein moiety with a fixed number of metal atoms per mole of protein. The metal cannot be removed without loss to enzyme activity and usually cannot be replaced by any other metal, though the native zinc atoms in several zinc enzymes can be substituted by cobalt and cadmium without complete loss of activity, and individual metallo enzymes are not confined to single metals. Superoxide dimutase, which catalyses the dismutation of the super oxide free radical, may contain copper and zinc, or manganese, depending on its source. The concentration and activities of many of the mineral element enzyme associations, in particular cells and tissues, have now been related to the manifestations of deficiency and toxicity of these elements in the animal body. In some cases serious clinical and pathological disorders arise as a consequence of dietary mineral abnormalities.

Evidence is usually available, from the experimental work, to indicate clinical signs and necropsy findings one can expect to be produced, by each deficiency. Several modifying factors may confuse the issue. Mineral deficiencies have been reviewed by Faye and Bengoumi (1994) and Abu Damir (1998), in camels.

COBALT DEFICIENCY

Cobalt is peculiar as an essential trace element in ruminant nutrition. It is stored in the body in limited amounts only and not in all the tissues. In the adult ruminant it functions only in the rumen, so it must be present all the times in the feed. Cobalt is required for the synthesis of vitamin B12 (cyancobalamin) and as compared to other species the requirements for vitamin B12 is very much higher in ruminants. The essential defect in cobalt deficiency in ruminants is an inability to metabolize propionic acid, which is accompanied by a failure of appetite and death from inanition.

Clinical Findings

No specific signs are characteristic of cobalt deficiency. A gradual decrease in appetite is the only obvious clinical sign, accompanied by loss of body weight and emaciation. The affected animals are anemic (normochromic, normocytic anemia). A marked pallor of the mucous membranes and affected animals are easily fatigued. Growth and lactation may get retarded. There may be loss of hair. Wasting may be precipitated by the stress of parturition or abortion.

Estimation of the cobalt or vitamin B12 content of the liver is the most valuable diagnostic test available. The radioassay of serum and liver vitamin B12 may be used as a diagnostic tool, if interpreted correctly.

Presence of methylmalonic acid (MMA) and formimino glutamic acid (FIGLU) in plasma and urine is a diagnostic and prognostic indicator. The concentration of FIGLU is considered to be a reliable indicator of the cobalt status of lambs. Levels of 0.08 – 20 umol /ml in the urine of affected lambs return to zero after treatment (Rucker and Morris, 1997). Methylmalonic acid is ordinarily metabolized in ruminants by a vitamin B12 enzyme system. In a cobalt deficient animal the methylmalonic content of urine is abnormally high and this has some merit as a test for the presence of the deficiency. Neither MMA nor FIGLU is a normal constituent of urine and their presence in urine is a positive indication of cobalt deficiency.

This can be treated by oral administration of cobalt.

COPPER DEFICIENCY

When the copper is deficient in diets, it is called primary copper deficiency. When the copper taken in the feed is not utilized in the tissues, it is called secondary copper deficiency. There are several disease conditions in ruminants in which administration of copper is curative, although the amount of copper in the diet is adequate. Dietary excess of molybdenum is one of the conditioning factors. Zinc, iron, lead and calcium carbonate are also thought to be conditioning factors. Dietary inorganic sulfate in combination with molybdenum has a profound effect on the uptake of copper by ruminants. Increases of sulfate content in diet can potentate the molybdenum content (as low as 2 mg/kg) to reduce copper intake below normal levels. Additional sulfate in the diet also has a depressing effect on the absorption of selenium (Blood and Radostits, 1989).

Many diseases are attributed to copper deficiency throughout the world, young ones are more susceptible to copper deficiency than adult animals. Copper is required for hemoglobin synthesis, erythrocyte production, for collagen synthesis and maturation, and melanine synthesis. It is a component of cytochrome oxidase, lysyl oxidase and super oxide dismutase, which are necessary for the oxidative processes in the body (Fraser et al. 1991).

Physiological levels of Cu in serum of the camel has been reported by many authors and these levels are comparable to those of sheep, goat and cattle. The normal plasma concentration for the camel is considered to be 70-120 ug/dl and values below 60 ug /dl indicate deficiency. Plasma concentration of Cu decreases during pregnancy (El-Tohamy et al., 1986). This could be attributed, as is the case in bovines, that the developing fetus obtains its copper by placental transfer and at birth the liver concentration of copper is high and declines postnatal to adult levels with in the first few months. The value of copper also increases in dehydration but not by hemolysis (Tartour, 1975).

Ceruloplasmin (copper oxidase) is an alpha glycoprotein, synthesized predominantly in the liver and contains >95% copper in the plasma.

The activity of ceruloplasmin enzyme in the camel is reported from 31.8-41.4 UO. This is similar to that in cattle, sheep, and goats. The activity gets affected by age and sex. Direct correlation exists between ceruloplasmin activity and copper concentration in camel serum, as is in other ruminants (Abdul Rahim, 1983).

Camel can store large quantities of copper in the liver. Mean liver copper concentration is reported to be 155 mg to 275 mg/kg DM (Khalifa et al. 1983). In the spleen the copper concentration is reported to be 6.4-43 mg/kg DM (Tartour, 1975), cartilage 5.9 mg/kg DM, bone 1.15 mg/kg DM (Khalifa et al., 1973), kidneys 2.4 – 12.2 mg/kg wet weight (Abu Damir et al. 1983).

Clinical Findings

Copper deficiency or borderline deficiency is wide spread in other ruminants, in countries with large camel populations. In a survey, 46% of camels had serum copper levels below the critical value (Faye, 1989). Copper deficiency symptoms are being observed in young camels in UAE (personal observation), and copper supplementation is routinely practiced. In a survey carried out in Dubai (Wensvoort, 1992), very low levels of copper have been reported in breeding (43.3 ± 35.4 mg / kg DM), racing (41 ± 40.3 mg/kg DM), calves (29.7 ± 25.1 mg/kg DM), and mature camels (43 ± 38.3 mg/kg DM). The border line copper deficiency is thought to be conditioned and is mainly due to the high content of sulfate and molybdenum in the diet and drinking water coupled with low copper content in feed (Wensvoort, 1992). Low serum copper levels in racing camels, occasionally having low PCV and Hb concentration, have also been reported by Abdulla et al (1988). Hastings and Gascoyne (1992) have reported anemia and weight loss in lama and guanaco. A sway disease, accompanied with pica, emaciation, dyskinesia, low blood copper concentration and cerulosplasmin, microcytic hypochromatic anemia, liability to fall and fractures, have been observed in camels. The condition is attributed to secondary copper deficiency resulting from high levels of molybdenum in soil and forage. The concentration of molybdenum in soils and forage is reported to be 4.8 ± 0.02 and 4.8 ± 0.25 mug / g (dry matter), respectively. The concentration of copper in blood and hair of camel is 0.28 ± 0.17 mug / ml and 3.50 ± 1.0 mug / g, respectively. A large number of nucleated erythrocytes have been observed in camel calves, which respond well to copper administration (Wernery et al., 1999).

The diagnosis of copper deficiency can be established by observing low serum copper or ceruloplasmin estimation along with anemia and /or high sulfate and molybdenum in diet.

Treatment

Inclusion of copper in diets of both milking and racing camels is recommended at the rate of 10 mg/kg feed on dry matter basis, as is recommended for dairy cattle (ARC, 1980, Mc Dowell, 1985, Thompson and Fowler, 1990). Margin of safety is high in the camels (Abu Damir et al., 1993). Most of the prepared animal feeds are supplemented with copper sulfate. Using feeds from the areas where soils are rich in sulfate content should be avoided.

IRON DEFICIENCY

About 70% of the total body iron is stored in the hemoglobin, 25% in the liver, spleen and bone marrow and the rest is contained in myoglobin and cytochromes (Varley et al., 1980, Whitby et al., 1988, Schreiber, 1989). About 0.1% of total body iron is contained in the plasma. Most of the iron in plasma is attached to a protein called transferrin, however, a small amount of iron is free. In the liver, spleen and bone marrow iron is stored as ferritin, which is water-soluble. When iron concentration increases it is stored pathologically in the liver, spleen, and bone marrow as water insoluble hemosiderin.

Iron deficiency states are reported to be not very common in farm animals except in very young who are confined to milk (Blood and Radostitis, 1989). Normal values in camels are comparable to those of other ruminants and horses (Abu Damir et al., 1993). However, Faye et al., (1986) observed lower iron level in camels as compared to other ruminants. Camel milk iron values are reported to be higher than in cow, sheep and goats. Total iron binding capacity (TIBC) is the amount of iron which can combine with transferrin to full saturation. Normally, only

33% of the transferrin is saturated with iron in vertebrates (Dacie and Lewis, 1991). Serum iron x 100 / TIBC is considered as a sensitive indicator of early iron deficiency. The difference between total serum iron and total iron binding capacity is unbound iron. Sex or age does not affect total serum iron values. However, male camels have significantly higher TIBC and saturation concentration (SC) values as compared to female camels (Tartour and Idris, 1970). The total serum iron values decrease in pregnancy. Iron levels also decrease during nephrosis, renal insufficiency, acute and chronic blood loss, acute hepatitis, and other acute and chronic infections and malignancies (Varley et al., 1980). Iron deficiency anemia is not reported and is unlikely to occur in unweaned, naturally reared camels. Splenic iron content of camels is lower than of other ruminants, and is reported to be higher than its liver content. Fetal liver iron content is higher than in the adult (Abu Damir, 1998).

Etiology

The occurrence of primary iron deficiency is seldom observed in grazing ruminants. However, it can happen in intensive farming, secondary to intervascular hemolysis, defective iron absorption and metabolism, chronic infections and chronic hemorrhages (due to ulcers, parasitic infestation, and tumors). The common parasites involved are Haemonchus longistipes, Trichuris species and Trichostrongylus species (Faye et al., 1986). Trypanosomiasis, a very common disease of camels, causes hemolytic anemia and consequent iron deficiency. Mange, caused by Sarcoptes scabie (cameli) is a common disease in camels (Higgins, 1986). The mites feed of camel's lymph. The demand of the parasite for iron seems to be very high. Low iron values (41ug/100 ml) have been reported in camels affected with mange. Heavy tick infestation has a similar effect.

Clinical Findings

Iron deficiency results in anemia, which is usually hypochromic and microcytic. Anemia accompanied by hypoferremia is a common finding in racing camels in UAE despite the continuous efforts to supplement iron. It is possible that the anemia is due to copper deficiency secondary to elevated levels of sulfate and molybdenum, however, some camels with poor racing performance respond well to treatment with iron. Low serum iron is observed in association with inflammatory conditions in camels. This might be due to an increase in macrophage activity which has been stimulated in response of illness (Manefield and Tinson, 1996).

Diagnosis

Diagnosis of iron deficiency in the camel is based on low serum iron concentration (<50 ug/dl), microcytic hypochromic anemia, low hemoglobin, and decreased serum ferritin with low saturation concentration.

Treatment

Treatment may be given orally or where ever necessary, parenterally. Organic preparations of iron are preferred i.e. iron dextran, iron-sorbitol-citric acid complex, iron saccharate or gluconate. The drugs should be used as per directions of the manufacturer.

IODINE DEFICIENCY

Iodine is essential for synthesis of thyroid hormones i.e. thyroxine (T4) and tri iodothyronine (T3). These hormones are required for oxidation process in the cells, reproduction, growth and differentiation, neuromuscular functions and for

integument in mammals. Iodine deficiency, in addition to disturbing the above mentioned functions causes enlargement of thyroids (goiter).

Etiology

Causes of goiter are primary iodine deficiency, goiterogenic substances, and metabolic defects in thyroid homeogenesis. High levels of rubidium, arsenic and fluorides can also cause goiter. The degree of disease depends on the nature and duration of exposure to causative agent (Underwood, 1977). The relative thyroid weight of all the domestic animals, including camel is about 0.1 gm / kg body weight.

A mean weight of 52.7 gm of fresh thyroid glands of healthy camels has been reported by Abdel Wahab and Hamza (1970). Thyroid stimulating hormone triggers the trapping of iodine in the thyroid glands and subsequent thyroid hormones secretion. Abdel Wahab and Osama (1971) reported lesser radioactive iodine accumulation in camels as compared to sheep and goats. Thyroid scanning by radioactive iodine is extensively used in humans for diagnosing goiter, extra thyroid iodine concentrating tissues and for detecting nodules of increased or decreased iodine uptake within the gland (Guiles, 1991). This method can also be used in animals, including camels. Venkatesh and Deshpande (1996) have reported values of iodine content of camel thyroid and thyroglobin.

Clinical Findings

Clinical and sub clinical goiter has been reported in adult camels from Darfur and Kodafan regions of the Sudan. The affected thyroid glands bulge from the side of neck. It may be unilateral or bilateral. Normocytic and normochromic anemia is also seen in camels affected with goiter (Abu Damir et al., 1990).

At postmortem examination, the thyroids appear to be pale or brown, heavier, symmetrically or asymmetrically enlarged. The cut surface may be smooth, rough or cystic. On cutting gelatinous or watery yellowish fluid oozes out. The histopathological changes of thyroid glands generally correlate, with those seen in colloidal goiter in other species. The follicles are usually enlarged to variable extent and are seen either filled with colloid or are empty. Cellular hyperplasia, rarely metaplasia and often fibrosis of inter follicular connective tissue is observed, resulting in distortion of follicular morphology. Low serum levels of T3 and T4 are observed in affected camels.

Iodine deficiency can cause reproductive disorders in camels. The newborn calves of iodine deficient dams are weak and are usually, goiterous. There is high rate of neonatal mortality. Loss of libido in male camels has also been observed.

Diagnosis

Prompt diagnosis of iodine deficiency in the camel, as in other animals, is based on the presence of non-malignant and non-inflammatory enlargement of the thyroid gland, together with decreased serum levels of the thyroid hormones (total T3 and T4 and free T3 and T4) and decreased iodine concentration in milk (<10 ug /100 ml). Exceptionally high cholesterol concentrations are detected in milk of affected camels without clinical goiter.

Treatment

The dosage for the treatment should be calculated very carefully as over dosage can cause intoxication. Iodine salt licks should be provided for prevention. The use of iodine in fertilizer also helps. Potassium iodide is preferred to treat and prevent the disease.

SELENIUM DEFICIENCY

Selenium is a component of the enzyme glutathione peroxidase (GSHPX) in erythrocytes. Both GSHPX and vitamin E interrupt the process of oxidation, which kills the cells and tissues. The disease, which results as a consequence of selenium or vitamin E deficiency, is called white muscle disease or ill thrift. The deficiency of selenium and vitamin E also causes retained placenta in cattle, and exudative diathesis, encephalomalacia, and pancreatic fibrosis in chicks and reproductive disorders in all species (Blood and Radostits, 1989).

Glutathion peroxidase catalyses the removal of hydrogen peroxide in the presence of reduced glutathione (GSH) which in turn changes to the unreduced form (GSSG) and then GSH is regenerated by the enzyme glutathione reductase.

The concentration of 82-175 ng /ml of selenium were reported in the serum of healthy adult camels. (Hamliri et al., 1990). Dromedary erythrocytes have a lower concentration of GSH (77.5 ± 4.5 mg %) and intermediate glutathione reductase activity (4.1 IU/g Hb), as compared with other animals and man. However, Agar and Suzuki (1982) found that GSH in erythrocytes of camels is not different from that of buffaloes (74 ± 2.83 mg, camel versus 80.9 ± 4.3 mg, buffalo), but the GSH regeneration rate is significantly higher in the camel (0.162 ± 0.009 mmol /min /g Hb versus 0.082 ± 0.011 mmol /min /g Hb). It is not known whether the low concentration of GSHPX in the camel erythrocytes has any effect on the rate of removal of peroxides and hence on the etiology of the white muscle disease.

Mean vitamin E levels of 1.44 $\pm$ 0.47 mg/ml for 6 groups of camels kept under different conditions, are reported: The breeding females, which received no vitamin supplements, showed the lowest concentration of vitamin E (0.9 $\pm$ 0.23 mg/ml). Vitamin E concentration in camels given a vitamin supplement containing 5 gm of vitamin E for 9 days increased from 1.2 – 2.15 mg/ml. Generally, serum vitamin E level in the dromedary (0.4-1.5 mg/ml) is similar to that of bactrian camel (Higgins and Kock, 1986). Concentration of selenium in various organs of camels are as (mg/kg DM basis): kidneys 6.7; lungs 3.89; adrenal glands 2.39; lymph nodes 1.77; large intestines 1.23, abomasum 0.81; brain 0.8; and heart 0.73 (Awad and Berschneider, 1977).

Deterioration of vitamin E in dry pasture by ultraviolet rays is of special importance in the etiology of the disease especially in areas where selenium concentration in pasture is critical or marginally deficient. High levels of polyunsaturated fatty acids in diet, is another contributing factor. Clinical myopathy is seen in cattle fed on pastures having high level of linolenic and low in linoleic acids (Rice et al. 1983).

Clinical Findings

Selenium and/or vitamin E deficiency cause acute or chronic white muscle disease in ruminants, including camel (Manefield and Tinson, 1996). It is especially important in young animals (Hamliri et al., 1990). The disease can cause high mortality up to 30% in camel calves. Poor racing performance, heart and respiratory disturbances and reduced fertility, in males and females, are observed in adult camels. In young animals, lethargy, anorexia, increased heart and respiratory rates, locomotor disturbances, and exhaustion at exercise are observed (Manefield and Tinson, 1996). Death may also occur after stress, following heart fibrillation. Sudden deaths have also been observed in peracute cases (Zhang et al, 1986).

Lesions of vitamin E /selenium deficiency include degeneration and necrosis of myocardium, diaphragm, intercostal muscles, and gastrocnemius muscle. Varying degree of myocardial mineralization, hydropericarditis, ventricular hyperemia and congestion and / or edema of lungs have also been reported. Section

of diaphragm and gastrocnemius muscle revealed degeneration and necrosis with loss of striations of muscle fibers and myocardium showed hyaline degeneration with nuclear pyknosis or karyolysis,. Some degree of calcification, fibrosis and cellular proliferation of myocardium was also reported (Finlayson et al., 1971; Decker and Mc Dermid, 1977; and Manefield and Tinson, 1996).

Musa and Tageldin (1994) described "swelling disease of the dromedary camels" and attributed it to a relative vitamin E and selenium deficiency conditioned by stress. Moderate to severe edema of limbs, ventral abdomen, neck, and testicles is characteristic of the disease. Microscopic lesions comprise focal myocardial degeneration and loss of myofibrils, capillary dilatation, swelling of endothelial cells, and thickening of tunica media of large arteries.

Diagnosis in camels is based on lesions and low serum concentrations of selenium and/or vitamin E, and low activity of glutathione peroxidase in the blood. CPK is useful at early stages of the disease. The high activity of GOT, LDH and SD and increased concentration of linolenic acid in serum (highly poly unsaturated fatty acids in the feed) may aid in the diagnosis.

Treatment

A combined mixture of selenium and alpha-tocopherol may be used for treatment. Provision of <0.5 mg /kg DM of selenium (Hamliri et al., 1990) and <100 mg of vitamin E in feeds is adequate.

ZINC DEFICIENCY

Deficiency of zinc causes parakeratosis, alopecia, wool eating, abnormal hoof growth, lameness and unthriftiness in ruminants. Little work has been done regarding zinc in camels and clinical deficiency is not known.

Mean serum Zn concentration of 93.4 $\pm$ 4.2ug/100 ml has been reported by El-Tohamy et al (1986). Somewhat higher values have been found (104.8 $\pm$ 9.5 ug/100 ml) by Abu Damir et al. (1993). The range in values is similar as reported in other animals (Underwood, 1977).

Plasma zinc levels have been reported higher in adult camels as compared to young ones. In hemolytic blood zinc levels increase and also the value increases in contaminated collection material. Values of 143$\pm$ 4.8, 187.0$\pm$ 23.95 and 112.00 $\pm$ 12.5 mg/kg, respectively, have been found in liver, heart, and spleen (Awad and Breschneider, 1977). Zinc deficiency is observed comparatively rare in field conditions.

Clinical Findings

Symptoms observed in various animals are parakeratosis; anorexia, and stunted growth, cell mediated immunodeficiency, and impaired reproductive function (Underwood, 1977). There is low serum zinc (<60-80 ug/100 ml). Although no clinical cases have been reported in camels, there are indications that deficiency or borderline deficiency prevails. Very low levels of serum zinc values (41 ug /100 ml) have been reported in camels by Abdulla et al. (1988). Wensvoort (1992) have reported that the pastures of UAE are also deficient in zinc (1.2-21.88 mg/ kg dry weight), which is far below the ARC recommendations for the cattle (30 mg/kg DM). This is also thought to be a cause of low fertility in camels.

Treatment

Zinc deficient camels promptly respond to injection of zinc preparation. Manefield and Tinson (1996) have claimed that 18-20 mg of zinc sulfate is required to keep the camels in good health. 30-50 ppm zinc is recommended by ARC (1980) to be added in the feed of domesticated animals for optimal growth and production.

CALCIUM DEFICIENCY

About 99% calcium in the body is present in the bones. The calcium is mobilized to the blood when required. The calcium metabolism is regulated by parathyroid hormone, calcitonin, and vitamin D. Total plasma calcium reflects calcium status in animals. However, ionized calcium is biologically more important. Critical plasma level of calcium is 8 mg /dl, which is same as in other ruminants (Mc Dowell, 1985). Both vitamin D and calcitonin are elevated in dehydrated camels (Yagil, 1985). Calcium concentration also elevates significantly after exercise (Snow et al., 1988). The calcium levels decrease by long serum separation time and during active trypanosomal parasitemia (Boid et al., 1986). Simple calcium deficiency is rare in ruminants. It only occurs when pasture contains less than 0.2% calcium. A number of disease conditions can occur due to calcium deficiency or due to disturbed calcium, phosphorus ratio.

Hypocalcemia

Hypocalcemia commonly known as parturient paresis or milk fever in cattle is a metabolic disease. It occurs most frequently in adult females after parturition and after milking. It is not very common but has been reported in camels (Straten, 1998).

A depression of the ionized calcium in the tissue fluids is the basic biochemical defect in parturient paresis. The calcium ions are known for maintaining muscle tone. Serum calcium levels fall in all adult cows at calving due to the onset of lactation. Hypocalcemic syndromes are also observed at times other than related to parturition. It can be part of an early or mild over eating of fermentable carbohydrates (Blood and Radostitis, 1989).

The symptoms observed in camels are same as reported in cattle: First stage of disease is brief excitement and tetany. The second stage is of recumbancy, depressed consciousness, and atony of rumen. Body temperature is normal or decreased. Normal levels of serum calcium in camels are 9.5 – 11.5 mg /dl (Wernery and Kaaden, 1995). In hypocalcemia the levels can drop to 2.41-3.94 mg /dl (Straten, 1998).

The condition can be treated by I/V administration of calcium borogluconate 500 ml.

Wilson (1984) has reported this condition in camel calves from Kenya. This condition is also observed in camel calves in UAE and Sultanate of Oman. The disease is characterized by bone taxity (mostly fore legs), with lameness, abduction of legs, bending of radio-ulnar bone, and malformation of joints.

The disease results from feeding low quality roughage poor in calcium and /or phosphorus. This may not be true in suckling calves, in which a number of factors might be incriminated. Copper deficiency may have a role. More research is needed to explore the etiology.

PHOSPHORUS DEFICIENCY

Critical plasma phosphorus level in camels is 4-4.5 mg /dl, which is same as reported in other ruminants. Phosphorus concentration is high when young and increases by cereal feeding, hemolysis, and defective kidney filtration. Phosphorus deficiency is global (Reid and Horvath, 1980).

Phosphorus deficiency is characterized by pica (depraved appetite). Parasitic infestation causes secondary hypophosphatemia (Bansal et al., 1971; Kapur and Sharma, 1972; Sharma and Satija, 1974). In this condition animals eat large amount of mud, sand, pieces of stones, and bricks instead of normal feed. The animals are emaciated with rough hair, reduced hump, sunken eyes, and pale

mucous membranes with diarrhea or constipation. Serum calcium, phosphorus, and total protein concentrations are low. Macrocytic, hypochromic, anemia also accompanies. Heavy infestation with parasites accompanied by reduced feed intake may deplete camel of calcium, phosphorus, and magnesium.

A condition known as *Kraft* has been observed in camels (Durand and Kchouk, 1958; Kchouk and Durand, 1958) in areas of calcareous soils, poor in phosphorus with a wide calcium /phosphorus ratio in soil (25:1) and pasture (11:1 or more). The disease is characterized by spontaneous fractures of the limb bones, intense joint pain, lameness, reluctance to move, and death due to starvation. The histological lesions in long bones, ribs and vertebrae, and the blood chemistry are suggestive of rickets.

According to our personal observation cases of idiopathic hematuria in racing camels respond well to I /V phosphorus administration.

MAGNESIUM

Magnesium is abundant in most common feedstuffs, and constitutes about 0.05% of total bodyweight of animals. About 60-70% of it is found in the skeleton. Normal serum content of magnesium in camel is reported to be 1.8-2.4 mg/100 ml.

In ruminants the major Mg absorption site is the reticulo-rumen portion of the digestive tract. Magnesium has many diverse physiological functions. The Mg in the skeleton is important for the integrity of bones and teeth. It is present mainly as the Mg ion as $Mg(OH)_2$ held within the hydrated shell of the apatite crystal surface. Magnesium is the second most important cation, after K, of the intracellular fluids. Although only about 1% of the total magnesium is in the extracellular fluid (blood plasma and interstitial fluid), this magnesium baths the body cells and is of great importance. When this ion in the extracellular fluid declines substantially below normal, the consequences are quite serious (e.g., tetany).

Magnesium is an active component of several enzyme systems in which thiamin pyrophosphate (TPP) is a cofactor. Oxidative phosphorylation is greatly reduced in the absence of Mg. It is also an essential activator for the phosphate transferring enzymes myokinase, diphosphopyridine-nucleotide kinase, and creatine kinase. It also activates pyruvic acid carboxylase, pyruvic acid oxidase, and the condensing enzyme for the reactions in the Kreb's cycle. Magnesium also appears to facilitate the transketolase reaction in the pentose monophosphate shunt.

Intracellularly, Mg is predominantly associated with the mitochondria. Its main role in this respect is an activator of enzymes. Magnesium is vitally involved in the metabolism of carbohydrates and lipids as a catalyst of a wide array of enzymes. It is essential for cellular respiration, and in certain tissues, it is complexed with adenosine diphosphate (ADP), and adenosine monophosphate (AMP). It is clear that Mg is important in almost all functions of the body, since ATP is required in numerous different functions such as muscle contractions; in oxidation phosphorylation; and many more.

Magnesium is involved in protein synthesis through its action on ribosomal aggregation, its role in binding messenger RNA to 70S ribosomes, and in the synthesis and degradation of DNA. It is also essential for the formation of cyclic AMP and other second messengers. Magnesium plays an important role in neuromuscular transmission, acting at some points synergistically with Ca, while at others, as an antagonist.

The functions of Mg can be demonstrated by the diverse physiological properties modified during a deficiency, which include growth, immunity and allergy, muscle contraction, red blood cell survival, occurrence of neoplasms, metabolism of collagen rich tissues, and Na and K metabolism. Its role in reproduction has also been suggested.

Its daily requirements in camels is not known. A concentration upto 0.20% in the diet of dairy cows is recommended (NRC, 1989).

Clinical Findings

Grass tetany (hypomagnesemia) is complex ruminant metabolic disorder affected by mineral composition of forage species, soil properties fertilizer practices, season of the year, temperature, animal species, breed, and age. Occurrence of this condition has not been reported in camels. Grass tetany is quite rare in livestock consuming predominantly legumes, as these are generally higher in Mg than grasses. The disease is characterized by convulsions rather than tetany. El-Magawry (1998) diagnosed hypomagnesemia in six camels from Egypt suffering from severe nervous manifestations. This was confirmed by biochemical analysis of serum.

Treatment

Females that develop tetany should receive medical teatment immediately by I/V injections or enemas. Treatment can include subcutaneous injection of a single dose of 400 ml of a 25% solution of $MgSO_4$ or I/v injection of a similar dose of Magnesium lactate.

SULFUR

Shortage of sulfur (S) containing amino acids is a world wide problem in animal nutrition. Sulfur is part of the amino acids methionine, cystine, and cysteine, and occurs in animal tissues in many sulfate forms. One of these compounds is chondroitin sulfate, an important component of cartilage, bone, tendons, and walls of blood vessels. The blood anticoagulant heparin is sulfuric acid ester of a polysacchride. Sulfur enters into metabolic pathways at sulfate or sulfide oxidation levels. Forms of S must either be oxidized to sulfate or reduced to sulfide before they are utilized by ruminants. Sulfate reductions and sulfide oxidation constitute a process called S. cycle. It is largely a microbial process, but both animals and plants make some application of the cycle.

Sulfur-containing compounds have vital metabolic functions in all living cells. Methionine, cystine, and cysteine are S-containing amino acids. Liver enzymes are able to produce cystine and cysteine from methionine; but methionine is required by all living animals. Methionine may be demethylated to form homocysteine, then combined with serine to form cystathionine, which upon cleavage produces cysteine and homoserine. Thus cysteine and cystine are non essential amino acids, but a large part of the S amino acid requirement of animals can be met by cystine and cysteine.

The S amino acids are incorporated into polypeptide chains. Free sulfhydryl groups participate in hydrogen binding, and covalent disulfide bonds between cysteine molecules aid in maintaining the spatial configuration of the tertiary structure of the protein molecule. Disulfide bonds contribute to the biological activity of enzymes and many proteins. Sulfhydryl groups provide sites for hydrogen bonding, and sites for the attachment of prosthetic groups of enzymes to substrate and for the binding of substrates to active sites of enzymes.

Hemoglobin, cytochromes, coenzymes A, coenzyme M, lipoic acid (coenzyme involved in the decarboxylation of pyruvic acid and other keto acids), S-adenosyl-methionine (methylating agent in the synthesis of methyl-containing substances such as N-methyl nicotinamide, creatine, choline, epinephrine, anserine, and glycocyamine), glutathione, and heparin all contain S.

The microbes of the rumen can incorporate inorganic S into organic compounds. Forages contain significant amounts of inorganic sulfate. The

incorporation of inorganic sulfate into S-containing amino acids by rumen microbes is an important part of their function.

Dietary Molebdenum (Mo) decreases liver Cu storage. Inorganic sulfate in feed potentiates the Cu-Mo antagonism. It has also been shown that sulfate influence Mo excretion in the urine and level in the blood. Cu utilization is restricted by depressed Cu solubility in the digestive tract from participation of insoluble Cu sulfide. It is suggested that when thiomolbidate, which is not complexed with Cu in the rumen, is absorbed into the blood, reactes with plasma Cu causing tissue Cu to be mobilized. It has also been suggested that an antagonistic relationship exists between dietary Se and S. The field studies have shown considerable muscular dystrophy in lambs born of ewes fed alfalfa and grass hay high in both Se and S.

Sulfur Toxicity in ruminants is most likely to occur when animals are fed diets supplemented with high S-containing substances such as ammonium sulfate to provide NPN, or calcium sulfate to provide calcium. It is indicated that hydrogen sulfide formed from sulfate by intestinal flora is the cause of poisoning. Sulfide reduces rumen activity and causes nervous and respiratory distress. It appears that 0.4% S is a maximum tolerable level of S as sodium sulfate for sheep. Data for camels is not available.

SODIUM AND CHLORINE (COMMON SALT) DEFICIENCY

Sodium and chlorine (Cl), in addition to potassium, maintain osmotic pressure and regulate acid-base equilibrium. These electrolytes in body fluids are involved at the cellular level in water metabolism, nutrient uptake, and transmission of nerve impulses. Acid base status is determined by the differences between total cation and anion intake and their excretion. Sodium makes up over 90% of the total cations, and Cl, two thirds of the acid ions. Sodium and Cl are crucial to the maintenance of normal fluid volume and osmotic pressure relationship. Sodium has a major role in the transmission of nerve impulses, and in maintaining proper muscle and heart contractions. In respiration and regulation of blood pH, Cl is transferred between plasma and erythrocyte through a process known as the chlorine shift. Sodium and Cl help control the passage of nutrients into the cells and waste products out. Sodium ions must be present in the lumen of the small intestine for absorption of sugars and aminoacids. Insufficient sodium lowers the utilization of protein and energy. Dietary electrolytes affect calcium absorption and possible mobilization in parturient paresis. Acidic diets result in greater Ca absorption, whereas diets containing excess cations, including those containing dietary buffers, reduce Ca availability. Absorption of the water soluble vitamins (riboflavine, thiamine, and ascorbic acid) may be sodium coupled. Water absorption in the intestines may also be closely linked to Na ion transport (McDowell, 1992).

Chlorine is the major anion of extracellular fluid and is also found in gastric secretions, where hydrochloric acid is important in protein digestion. It is also found in fairly large concentrations in bile, pancreatic juice, and secretions from the intestines. Chlorine is essential for activation of intestinal amylase. It is known that camel requires about 140 gm of salt per day to keep in top condition. If the camel is not grazing on salt containing bushes, free salt must be given to the camel (Perk, 1939).

Etiology

An excess of potassium will aggravate a deficiency of sodium just as too much sodium heightens effects of potassium deficiency. Body depletion of salt occurs from diarrhea and vomiting, with less of these nutrients absorbed. Animals with kidney or adrenal gland damage have increased depletion of these elements. A dietary deficiency of sodium is most likely to occur in rapidly growing

young animals fed cereal based diets or forages inherently low in sodium, during lactation as a consequence of sodium (also Cl) losses in milk, and during hot weather, especially in camels getting hard training (the sodium and Cl being lost in sweat).

Clinical Findings

The first sign of sodium deficiency is a pica or craving for salt manifested by licking of wood, soil, or sweat of other animals. A prolonged deficiency causes loss of appetite, decreased growth, unthrifty appearance, reduced milk production and loss of weight. More pronounced signs of sodium deficiency include shivering, incoordination, weakness and cardiac arrythemia, which can lead to death. The effects of deficiency of Na and Cl in camels are not much documented. According to Perk (1938) the camels are frequently salt starved, and that this starvation is likely to be related to the causation of necrotic skin lesions and obscure lameness.

POTASSIUM

Potassium (K) carries out the same function inside the cell that Na performs in the plasma and interstitial fluid, i.e. maintenance of acid-base relationship and proper osmotic balance. Sodium, K, and Cl are the three major electrolytes in the body and function to maintain cation-anion balance. Sodium is the major extracellular cation, providing greater than 90% of the total cations in the plasma and interstial fluid, whereas K, the major intracellular cation, provides approximately 75% of the total cation within the cell. Within each cell compartment, the net charge equals zero, and electroneutrality is maintained. Normal K content in serum of camels is reported to be 3.5-6.0 mmol/l.

Active transport, in which energy is required, mechanism regulates the concentration of specific electrolytes in the extracellular and intracellular compartments. The intracellular-extracellular separation of Na and K is handdled by a Na/K pump. Maintenance of these concentration gradients is important for transport of substrates into and out of the cell as well as regulation of osmotic pressure.

Potassium contributes 50% of the osmolality of intracellular fluid, whereas Na and Cl contribute 80% of extracellular osmolality. Diffusion of water maintains equilibrium on either side of the membrane. If the concentration of the molecules outside the cell is greater than the intracellular concentration, the cell loses water and dehydrates, while the extracellular fluid volume increases and edema develops.

Principal base in the body tissues is potassium and plays an important role in the regulation of acid-base balance. Extracellular pH is maintained within narrow range (7.4±0.05). Maintenance of this range is a complex process involving respiration, blood buffering, renal excretion and reabsorption. Potassium is importat in the transport of oxygen and carbondioxide carrying capacity of the blood. Potassium is also important in the transmission of nerve impulses to muscle fibers and in the contractility of the muscle itself. An ionic balance exists between K^+ , Na^+, Ca^+, and Mg^+. These ions affect capillary and cell function and the excitability of nerve and muscle. Potassium acts as a brake in regulating heart beat and suppresses heart flutter. It also helps prevent tetany, convulsions and an unsteady gait.

Potassium functions as a cofactor in several enzyme systems. These include energy transfer and utilization, protein synthesis, and carbodydrate metabolism. Some of the enzyme systems that are activated by K include adenosine triphosphate, hexokinase, carbonic anhydrase, salivary amylase, pyruvic kinase, and fructokinase.

Potassium also affects the uptake of amino acids into cells, and thus influences the growth.

According to Mc Dowell (1992) ruminant species require K between 0.5 and 1.0% expressed as per unit animal feed as fed (approximately 90% dry matter). Lactating animals require more K. Stress tends to increase urinary loss of K, and diseases with fever or diarrhea further increase K loss.

Potassium concentration is highest in plant leafs and less in the seeds. The average concentration of K in the sweat of camels is 4.0 mM/litre and sodium concentration is 9.5 mM/litre. This is due to large potassium content of plants (Mc Farlane, 1964). The potassium blood serum levels are reported to be 3.5-5.5 mM/litre (Wernery et al., 1999).

Clinical Findings

For all species studied, reduced appetite is one of the first signs of K deficiency. With K depletion in the body, there is depressed growth, muscular weakness, stiffness, and paralysis.

Ruminants are much more likely to develop K deficiencies than do monogastric, as they have higher dietary requirement for K than than do other species. Milking animals have greatest need for K. Ruminants consuming high-concentrate diets are likely to develop K deficiency more commonly. Various types of stress for ruminants have resulted in higher K requirements.

Continued K deficiency results in intracellular acidosis, degeneration of vital organs, and nervous disorders. Potassium loss accompanies persistent diarrhea. Young animals with diarrhea develop acidosis and a K deficit more rapidly than do mature animals.

Potassium toxicosis is not likely to occur under natural conditions. Maximum dietary tolerance level of 3% have been reported. Clinical findings of K toxicosis include cardiac insufficiency, edema, muscle weakness, and death (NRC, 1980).

HYPERGLYCEMIA

The normal level of serum glucose in the camels is 5-8 mM/liter. Camel is able to decrease renal glucose threshold level during water deprivation. This phenomenon enables the animal to draw water in the blood to maintain plasma volume. The glucose level can go up to 72 mM /liter and insulin level to 40 IU (normal 14 IU). Hyperglycemia together with low serum iron is indicative of systemic illness. Hyperglycemia also occurs after exercise and dehydration.

VITAMIN DEFICIENCIES

Vitamins are known to have a vital role in numerous functions of the body. Ruminant's vitamin requirements may increase to meet the various immune and metabolic stresses imposed during hard exercise. There is a scarcity of information on the vitamin requirements of camel. Snow et al. (1992) have reported the plasma levels of different vitamins.

VITAMIN A

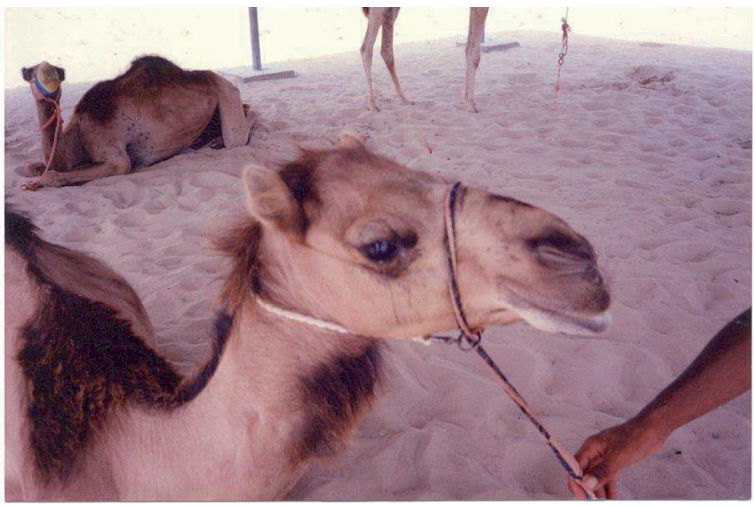
In nature, vitamin A is largely present as lipid esters in animal tissues and as provitamin forms vitamin A in plants. Beta-carotene concentrations of most of the animals are below the levels of detection (5 ug /liter). It is postulated that ingested carotenoids undergo marked conversion to vitamin A within the intestinal wall, and any amount that may pass intestinal conversion is stored in the liver. In plants and prokaryotes, carotenoids serve as mediators of photo-energy-related process by

capturing energy from light. Carotenoids also quench singlet oxygen and may act as both antioxidants and pro-oxidants.

The overall nutritional status and the integrity of the intestinal microvillus affect the bioavailability and digestion of vitamin A and carotenoids. Inside the target cells, vitamin A, as retinol, interacts with cellular binding proteins that function to control its subsequent metabolism, for example, oxidation to retinal and to retinoic acid. Some of these cellular binding proteins are also a part of the super family of glucocorticoid-retinoid-thyroxine transcriptional factors. It is the role of such proteins in transcription and regulation that makes vitamin A important to so many facets of cellular regulation.

The major roles of vitamin A are in cellular differentiation, tissue growth, and vision. In vision, vitamin A, as a component of rhodopsin, facilitates the efficient transfer of energy from photons of light to electrochemical signals. In dietary vitamin A deficiency sensitivity of the visual apparatus is decreased so that light of low intensity is not perceived, leading to the clinical condition of nyctalopia or night blindness.

In response to very low doses of retinoids, epithelial cells undergo a “terminal differentiation”. Vitamin A derivatives control the expression of various proteins important to mucus formation and cytoskeletal integrity, such as keratin and transglutaminase, and the rate of cell cycling. In response to an abnormally low level of retinoids, cells lose their normal columnar shape, become flattened or squamous, and increase their cytosolic content of keratin. In dermis, this process results in a protective outer layer, scales, and other specialized surfaces. However, where the primary function of epithelial cell is provision of a moist surface or absorption, squamous overkeratinization leads to loss of functional integrity. Lack of protective mucous secretion facilitates the establishment of infection of the lungs and other tissues that depend on a mucus barrier. In the intestine, keratinization induces premature sloughing of enterocytes and malabsorption.



Opacity of cornea due to vitamin A deficiency. The animal responded to vitamin A & D supplementation

For optimal maintenance, the allowance for many animals ranges from 100-200 I.U. per kilogram of body weight per day (one I.U. is equal to 0.3 ug of retinol). Vitamin A plasma concentration in camels is 422 ± 102 ug / l, as reported by Snow et al. (1992).

VITAMIN E (Alpha-tocopherol)

The principal dietary sources of tocopherols are plant oils. Tocopherols are unique among the vitamins as they act primarily at a chemical level as antioxidants, that is, they do not serve as cofactors or appear to be involved directly as a specific factor in cellular regulation. Primarily, vitamin E protects unsaturated fatty acids in the phospholipids of cell membranes. The quinone moiety of tocopherol is capable of quenching free radicals, such as free radicals of hydrogen, superoxide, hydroxl radicals, and other lipid-derived radical species.

Cell membranes contain vitamin E at a concentration of approximately 1 mg per 5-10 g lipid membrane, a concentration sufficient to retard membrane-lipid oxidation. Membrane lipids are constantly engaged in the process of turnover and repair. By prolonging the initiation time before a free-radical chain reaction occurs, vitamin E gives time to replace damaged membrane lipids through the process of normal cell turnover.

The nutritional status of vitamin E is difficult to assess. A number of factors can influence the concentration of tocopherols in cells. Vitamin E acts as the last line of defense for lipid oxidation, primarily residing in lipid membranes. Consequently, enzymes such as superoxide dismutase, catalase, glutathione peroxidase, and related systems for oxidant defence can moderate the absolute need for vitamin E. Further, high intake of polyunsaturated dietary fats increase the vitamin E requirement because of their eventual deposition in cell membranes and higher susceptibility to oxidation.

Plasma vitamin E concentrations in camels were 1.4 ± 0.47 ug /l. which are relatively low than reported in other animals. The levels are potentially hazardous to the ruminants; hence they should be supplemented with vitamin E.

BIOTIN

Biotin functions in enzymatic carboxylation reactions as a cofactor for the CO_2 –fixing enzymes: acetyl-CoA carboxylase, which is essential for fatty acid synthesis; propionyl-CoA carboxylase, which participates in odd-chain fatty acid metabolism; and pyruvate carboxylase, which is involved in the formation of oxaloacetate, an important obligatory step in reverse glycolysis and glyconeogenesis. Biotin is also involved as the coenzyme in the carboxylation of beta-methylcrotonyl-CoA (a product of leucine metabolism). In food, biotin is found in highest concentrations in cereals, including soybean, rice, barley, oats, corn, and wheat.

Plasma biotin concentrations in camels were 328 ± 91 ug /l, which are similar as reported in horse but are lower than in cattle. It is believed that biotin synthesis occurs in the rumen. Oral administration of biotin avoids ruminal degradation.

ASCORBIC ACID

Ascorbic acid functions primarily as a cofactor for microsomal mono-oxygenases (hydroxylases) and oxidases. In most animals, ascorbic acid is synthesized from glucose in the liver or kidney. Ascorbic acid is the most powerful reducing agent available to cells and is of general importance as an antioxidant, because of its high reducing potential. However, under some conditions ascorbic acid can also act as a pro-oxidant. As a cellular reducing agent, ascorbic acid plays a number of very important roles. It serves as cofactor for mixed-function oxidations that result in the incorporation of molecular oxygen into various substrates. Example includes the hydroxylation of proline in collagen, elastin, Clq complement, and acetylcholine esterase. Hydroxylases (monooxygenases) and some P450-dependent hydroxylases that carry out the hydroxylation of steroids, drugs, and other xenobiotics also utilize ascorbic acid as a reductant. Moreover, the hydroxylation steps in the biosynthesis of carnitine and the hydroxylation of tyrosine in the formation of catecholamines represent other important catalytic functions of ascorbic acid. Most of the enzymes involved in these processes are metal requiring enzymes, in which the role of ascorbic acid is to maintain the metal (usually Cu or Fe) in a reduced state. Ascorbic acid also influences histamine metabolism in some animals, particularly humans. There is an inverse correlation between ascorbic acid and serum histamine levels.

To maintain the functions requiring ascorbate, most animals generate 10-60 mg of ascorbic acid per Kcal utilized in the course of normal metabolism. Camel's plasma ascorbic acid concentration was 4.5 ± 1.3 ug /l, which is similar to that in cattle. It is likely that the camel's ascorbic acid requirements are met by hepatic synthesis. There is no increase in plasma ascorbic acid after oral administration. Ascorbic acid is destroyed rapidly in the rumen. However, under circumstances, some absorption may occur through the ruminal wall and from the intestines.

FOLIC ACID AND VITAMIN B_{12}

They are required for the synthesis of thymidylate, which is essential to cell division and proliferation. The requirement for folic acid ranges from 1-10 mg per kg diet for most animals. There are some conditions, in which the folic acid requirements are conditionally high, such as when either natural or pharmacological folic acid agonists are present in the diet. The best example is methotrexate, which ultimately inhibits the proliferation and regeneration of rapidly replicating cells. Cell division is blocked in the S phase because of impaired DNA synthesis. As a consequence, drugs such as methotrexate are widely used in cancer chemotherapy. Deficiencies of both vitamin B_{12} and folic acid produce clinical signs of macrocytic anemia and desynchronize in growth and development owing to the importance of folic acid to purine and DNA synthesis. Chronic deficiencies of either folic acid or vitamin B_{12} can also promote fatty liver disease and indirectly influence extra cellular matrix maturation stability by causing abnormal elevations in homocysteine

Plasma folic acid concentrations were 7.6 ± 3.3 ug /l, which are similar to that reported in the horse. There is no increase in the plasma folic acid after oral administration. There is extensive destruction / microbial uptake of folic acid in the rumen.

THIAMINE

Thiamine in cells occurs, either as the pyrophosphate (TPP) or the triphosphate (TPPP). There are two general types of reactions wherein pyrophosphate functions as magnesium-coordinated coenzyme for active aldehyde transfer reactions. One example is the decarboxylation of alpha-keto acids, designated as transketolase reaction. Decarboxylation of alpha-keto acids occurs

twice in the tricarboxylic acid cycle (TCA): in the conversion of pyruvic acid to acetyl-CoA and in the conversion of alpha-ketoglutarate to succinyl-CoA. The other reaction of pyrophosphate is the facilitation of the transformation of "ketols" (ketose phosphates) in the pentose pathway. In this pathway, NADP is reduced to NADPH, an essential reducing agent for synthetic reactions. A deficiency of thiamine impairs metabolism of carbohydrates because of defective TCA regulation. Further perturbations in the pentose phosphate related carbohydrate pathway lead to decreased production of NADPH, which affects synthetic processes such as fatty acid biosynthesis. TPPP predominates in neural tissue and in brain. In the brain, TPPP is proposed to be involved in sodium-gated processes, that is, the flux of sodium ions across neuronal cell membranes. This aspect of thiamine metabolism may be related to the neuromuscular control. Predominant characteristic of thiamine deficiency is polioencephalomalacia. Primary lesions of the brain are hemorrhages of the periventricular gray matter and pathology of the inferior colliculli, medial vestibular, and lateral geniculate nuclei. A relationship between excessive production of hydrogen sulfide in the rumen of sheep and cattle and polioencephalomalacia has been demonstrated. High levels of tannins reduce the bioavailability of thiamine in foodstuffs.

Plasma thiamine concentrations in camels were 39 ± 11.5 ug /l. Blood thiamine concentration of racing camels was reported to be lower than the breeding camels. This may be due to increased stress of exercise /training in racing camels or decreased synthesis in the rumen due to increased grain content in the diet. Thiamine deficiency can occur in ruminants (Rammell and Hill, 1986). If thiamine is given in high doses orally, absorption is likely to occur.

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INFECTIOUS DISEASES

Infectious diseases play a prominent role in adversly affecting animal production. Prevention, diagnosis, and treatment are vital to the management of these animals. Camels are susceptible to many infectious diseases (Higgins, 1983), some of which have been amply investigated because they affect all species of farm animals, such as anthrax, rabies, tuberculosis, brucellosis, pasteurellosis, necrobacteriosis, pox, and ringworm. They are also susceptible to some of the diseases which affect ruminants, such as foot and mouth disease, contagious bovine pleuropneumonia and malignant edema, and some of those which affect equines, such as glanders, strangles, and equine infectious encephalomyelitis (Buchnev et al., 1987). There has not been much research into the specific infectious diseases of camels, and some have not been investigated at all. The diseases which, are commonly encountered in the camels are being discussed in the pages to follow.

VIRAL DISEASES OF CAMELS

The camel is resistant to many deadly viral diseases, such as rinderpest and African horse sickness (Wernery and Kaaden, 1995). Only few viral diseases of the camels have been reported. These include: rabies, camel pox, contagious ecthyma, papillomatosis, influenza, and rota virus diarrhea (Hedger et al., 1980). A large number of viral antibodies have been detected in camel's serum (Abu Elzehin, 1985; Bildfell et al., 1996; Mousa et al., 1987 and Olaleye et al., 1989). These results do not confirm whether these antibidies are produced as a result of disease or otherwise.

RABIES

Rabies is a highly fatal disease of warm-blooded animals, including camels. The disease is caused by lyssavirus which, belongs to the Rhabdoviridae family.

Rabies is prevalent in most of the countries of the world, except in some island countries, where the disease has been eliminated through strict quarantine measures. The source of infection is always an infected animal and method of spread is almost always by the bite of an infected animal, although contamination of skin wounds by fresh saliva may result in infection. Rarely infection by inhalation and ingestion of virus can also lead to infection (Afshar, 1979), Traditionally, the dog, and to a minor extent the cat, have been considered to be the main source animals. However, native fauna, including foxes, wolves, coyotes, vampire bats etc may act as main source of infection. Kumar and Jindal (1997) have reported occurrence of rabies, in dromedary camels from India and Afzal et al., (1993) in UAE. The epidemiological pattern of the rabies in these camels was intriguing. The history of bites could not be traced and no wounds were seen. However, all these animals were kept and

grazed in one locality which, harbors wild life including foxes, jackals and mongooses.

Following the bite of rabid animal, the virus multiplies in the striated muscle fibers. After this, neuromuscular junctions are affected, through which the virus enters nervous system. The virus after having entered the nerve ending invades the brain by passive movement within axons, through spinal cord.

The lesions of rabies are produced in the central nervous system and the virus moves from the site of infection through peripheral nerves. This pattern of spread is responsible for such a variable incubation period. Nearer the site of bite to head shorter is the incubation period as compared to bites on the extremities .There is not much published work on rabies in camel, however, rabies has been reported. At necropsy inclusions have been seen in the neurones (Richard, 1979; Bah et al., 1981; Ata et al., 1993; Wernery and Kumar, 1993; Afzal et al., 1993).

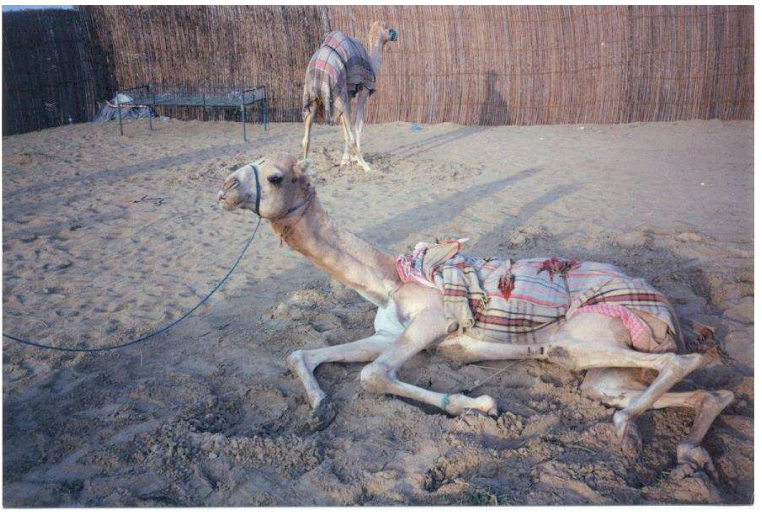
Rabies – facial paralysis. Known history of being bitten by a fox

Clinical Findings

The most common symptoms in camel are restlessness, hyperexcitability, aggressive biting, scratching, self biting, drooling of saliva, muscular tremor, colic followed by paralysis (usually hind legs), yawning, and subsequent death (Higgins, 1986; Gahlot, 1994; Ahuja et al., 1994), within 3-7 days. It is probable that when the virus invades the brain, irritation of higher centres produces mania, excitement, and convulsions. Paralysis may be because of the destruction of spinal neurones. Death is usually due to respiratory paralysis. The involvement of autonomous nervous system is thought to be the cause of the clinical signs of salivation, indigestion, pica, paralysis of anus and bladder, and increased libido. Presence of the virus has also been demonstrated virtually in every neuron, in the cornea, salivary gland

and many other organs, which could have reached at these locations centrifugally along the peripheral nerves. Passage of the virus along the olfactory nerve to taste buds and other sensory end organs in the oropharynx is considered to be the cause of highly infective nature of saliva, rather than the salivary glands.

Diagnosis is based on careful examination of brain tissue in the laboratory. Fluorescent antibody test of impression smears of brain, histological search for Negri bodies in the neurons and intracerebral inoculation of the brain emulsion of rabid animal into weaned mice are the reliable tests suggested for the diagnosis of rabies.



Camel affected with rabies - paralysis of the hind leg

Treatment

The bite wound should be immediately washed with soap and water. Post exposure vaccination may be of value. Treatment of rabid animal should not be attempted. For the purpose of control stray dogs should be eliminated and pet dogs should be vaccinated regularly. Camels, which are exposed to wild fauna, should also be vaccinated regularly.

CAMEL POX

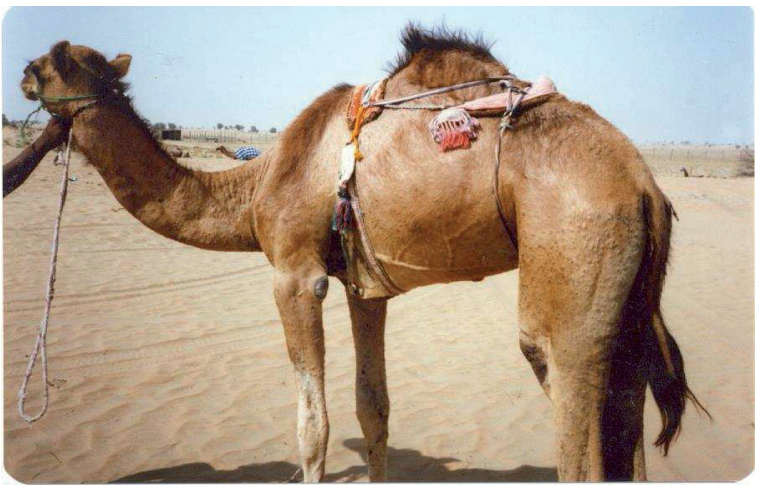
Camel pox is one of the most important viral diseases (Al Ani and Al Saliby, 1988; Chauhan and Kaushik, 1987). It has been reported from all parts of the world where camels are kept (Krupeuko, 1972, Moallin, 1986, Pfeffer et al. 1996; Tulepbaev, 1971; Wernery et al., 1997). Causative agent is an orthopox virus "Orthopox Cameli". There seems to be several distinct antigenic strains of camel pox virus. Sheep pox and vaccinia viruses are non-pathogenic for camels. Zoonotic importance of the disease remains un established (Jezek et al. 1983). The disease is highly contagious. The transmission is mainly horizontal, however, scabs, contaminated tools, utensils, cloths, grazing areas and human beings also can transmit the disease. The disease mostly affects young animals and they bear life long immunity once they are recovered. The

incubation period is about 3 –15 days. Dromedaries are also susceptible to infection with contagious ecthyma. Differentiation between camel pox and contagious ecthyma requires serological and virological examination (Azwai et al, 1996).

Clinical Findings

The camel poxvirus causes skin and mucous membrane lesions. The

Lesions of pox on face of camel



Pox lesions on the back of the thigh

lesions progress, from papules, to vesicles and pustules and eventually scabs are formed. During the vesicular stage pruritis is observed (Kriz, 1982). In this disease, lesions develop on the nostrils, lips, eye lids, and mouth. In acute cases there may be pyrexia, anorexia, diarrhea, and lesions may be present on the entire body, including trachea and lungs (Wernery et al., 1997). Edema of the head, neck and legs may also occur in severe cases (Manefield and Tinson, 1996). Mortality is not usually very high. In acute cases it may reach up to 28% (Jezek et al., 1983).

Several vaccines have been produced for which varying degrees of success have been reported (Hafez et al., 1992; Kaaden et al., 1992).

CONTAGIOUS ECTHYMA

Camel contagious ecthyma is a wide spread disease of camels (Gitao, 1994) and is caused by epitheliotropic parapox virus of the family Poxviridae (Munz et al. 1986). The disease mainly affects young camels and produces localized lesions. Occurrence of generalized lesions is infrequent. Unlike camel pox, the lesions in contagious ecthyma are mild and self-limiting and heal spontaneously (Dashtseren et al. 1984). Contagious camel ecthyma is difficult to differentiate clinically from camel pox. The lesions are mainly confined to the skin around lips and nostrils. Severely affected animals are commonly emaciated and have pendulous lower lips, sometimes the entire head becomes swollen. Skin lesions in other areas consist of nodular masses of variable size and are scattered at different locations, but more pronounced at the medial aspects of the front and hind legs (Osheik et al., 1990). The mouth lesions get frequently injured while grazing thorny bushes and trees, and frequently there is secondary bacterial infection, causing pain. Animal becomes off feed and emaciated. Lameness may also be observed due to the presence of lesions on the feet. The morbidity rate can reach up to 100% in young camels as compared to 10 – 20% in adult camels. Mortality rate is considered to be zero, although some deaths from mixed infection can occur (Wernery and Kaaden, 1995). No reliable vaccine seems to exist. However, Azwai et al. (1995) have studied immune responses of camel to contagious ecthyma.

The lesions should be treated with some antiseptic ointment.

PAPILLOMA

Warts (pamiolloma viral infection) occur frequently in humans and animals throughout the world. Amongst the animals cattle and horses are mostly affected. The warts are caused by papova viridae, family. The virus seems to affect camels also (Khalafalla et al., 1998). Munz et al. (1990) and Wernery (1992) have reported the occurrence of papillomatosis in different parts of Africa and Asia. Morbidity rate is quite high. Development of life long immunity after recovering from the disease is not established. The zoonotic importance, and its mode of transmission is not clear (Dioli and Stimmel – Mayr, 1992).

The lesions in camels are not wide spread. The characteristic nodules are mainly confined to head, neck, shoulders, or udder. The lesions are usually long lasting and painless. The lesions may be surgically removed. The use of autogenous vaccines may also help.

BOVINE VIRAL DIARRHEA

An outbreak of reproductive disease was observed in the camel research herd at the Faculty of Veterinary Medicine, Cairo University, Egypt.

The clinical syndromes were abortion, stilbirths, weak calf syndrome, early neonatal respiratory distress and hemorrhagic diarrhea. Histologically, lymphoid depletion and vasculitis were the outstanding findings in most of the tissues. Optic neuritis, underpopulated cerebral molecular layer and ectopia of Purkinje cells were the histological characteristic of the oculocerebellar syndrome. Cytopathic bovine virus diarrhea virus was isolated from the still born camel calves and from mesenteric and caudal superficial cervical lymph nodes of the camels which died of hemorrhagic diarrhea at day 20 postnatally. The virus was identified by indirect immunofluorescence using fluorescin isothicyanate labeled antiserum, and virus neutralization test. Electron microscopy revealed BVDV-like particles in the cytoplasm of the Maden Darby bovine kidney cells inoculated with homogenates of tissues from the calves. This was the first report of BVD outbreak in dromedary camels (Hegazy et al., 1998).

BACTERIAL DISEASES

ANTHRAX

It is peracute septicemic disease encountered in almost all the mammalian species, including man. It is characterized by sudden death, absence of rigor mortis, exudation of tarry unclotted blood from the natural orifices of the dead body and splenomegaly. The disease is prevalent in all of the regions inhabited by camels.

Etiology

Bacillus anthracis is the causative agent, which is large, Gram positive non motile spore forming rod. Bacillus anthracis endospores can survive in soil for up to 50 years in endemic areas. Bacillus anthracis has a plasmid endcoded tripartite protein toxin with protective lethal and edema factors. The toxin is leukocidal, increases vascular permeability and produces capillary thrombin causing shock. The polypeptide capsule is antiphagocytic. To be fully virulant, B. anthracis must produce both the tripartite toxin and the capsule. The main routs of entry of endospores are by ingestion from soil when grazing or in contaminated food and by infection of wounds. Transmission by biting insects may be important especially during an outbreak of anthrax.

Clinical Findings

In peracute cases the camels may die without showing any symptoms. In acute cases there is high fever, diarrhea, colic, bloat, and respiratory distress. In the dead body there is discharge of dark tarry blood from the natural orifices. The rigor mortis does not develop and the blood does not clot. There is enlargement of spleen along with other lesion of septicemia (Barakat, et al., 1976). The decomposition of the carcass sets in very soon. The necropsy of animals suspected dying of anthrax is forbidden. The carcass should be burnt or burried deep under soil. For confirmation of diagnosis, the discharge from the natural orifices may be cultured.

Treatment

Treatment can be attempted by injecting streptopenicillin or oxytetracycline before the on set of shock in less acute cases. Simultaneous injection of antisera may also be given. The disease can be prevented by vaccination. In case of out break the spread of the disease can be prevented by strict quarantine.

CORYNEBACTERIUM INFECTIONS

Corynebacterium spp. are ubiquitous organisms and are responsible for many animal diseases. They are either the primary cause or present as secondary invaders. They can cause metritis, arthritis, bursitis, orchitis, mastitis, pyelonephritis, cystitis, balanoposthitis, caseous lymphadenitis, lymphangitis, pastular dermatitis, pneumonia etc. in different species of animals including, camels (Hassanein et al., 1984, Barbour et al., 1985).

Corynebacterium pseudotuberculosis causes caseous lymphadinitis or pseudotuberculosis in sheep, goats, and camels. The prevalence of the disease is quite high and has been reported from many countries of the world (Esterabadi et al., 1975 ;Domenech et al. 1977; Hoste et al., 1985; Bergin, 1988, Radwan et al. 1989; Purohit, 1985; Sadykov and Dadabaev, 1976; Afzal et al., 1996). Other microorganisms have also been reported to cause similar type of lesions as those of pseudotuberculosis (Domenech et al., 1977; Radwan et al, 1989). This infection is spread via ingestion, inhalation or directly through wounds.

Clinical Findings

It is a chronic disease and is characterized by abscess formation, usually lymph nodes are involved (both external and internal). However, internal lesions occur rarely in camels (Radwan et al., 1989). Generalized cutaneous form is also rare (Eldisougi, 1984). Usually camels more than 5 years of age are involved and most frequently the cervical, subscapular, and inguinal lymphnodes are affected. The size of the lesion varies, usually they are quite large (orange size), painless, and cold. The pus is creamy, thick, and yellowish. A generalized lymphadenopathy without abscess formation has also been reported (Radwan et al., 1989).

Treatment

The condition can be treated by parenteral administration of antibiotics, preferably erythromycin and by surgical intervention and dressing of the lesions. No trials have been made regarding use of vaccine in camels .

CONTAGIOUS SKIN NECROSIS

Cross (1917), and Leese (1927) considered contagious skin necrosis as one of the most important diseases of camels, which is characterized by the formation of hot, painful swellings, which suppurate and sloughs, leaving a raw circumscribed skin lesion. Peck (1939) considered that the etiology of contagious skin necrosis was related to faulty diet and that deficiency of common salt was probably concerned.

Cross (1917) found streptococci in pus from the lesions. Curasson (1936) isolated Streptothrix. Edelsten and Pegram (1974) found that Streptococcus agalactiae (group B) was the main bacteria isolated from the lesions. They also isolated coagulase positive, streptococci from the lesions. Domenach et al. (1977) found streptococci (group B) alone or in association with Staphylococcus sp., Corynebacterium sp. and Lactobacillus sp. from the

lesions. Wernery and Kaaden (1995) named the disease pyoderma. Yaqoub and Mohamed (1996) isolated Staphylococcus sp., Streptococcus sp., Corynebacterium sp., Nocardia cameli and Erysipelothrix sp. from the lesions. Streptococcus sp. were however, most common.

Clinical Findings

According to Yaqoub and Mohamed (1996) the lesions begin as small nodules, which later increase in size. The lesions are painful in the beginning. The centre of the lesions then becomes necrosed and gets separated from the surrounding healthy tissue and sloughs off leaving an ulcer (5-7 cm) which remains for long time. The animal experience itching at this stage. The lesions are mostly seen on the root of the neck, head, flank, gluteal region, root of the tail and medial aspect of thighs. Body temperature, heart and respiratory rates usually remain normal. There is a controversy regarding the involvement of lymph nodes. Cross (1917) and Leese (1927) reported that the lymph nodes around the affected parts become swollen, hot, and painful. However, according to Yaquob and Mohamed (1996) the lymphatic system is not commonly affected.

MASTITIS

Inflammation of mammary gland (mastitis) is one of the most important diseases of domestic animals, especially cattle, buffaloes, sheep and goats. According to Leese (1927) mastitis is rare in camels. However, Hafez et al. (1987) have suggested that the condition in camels is probably not uncommon. Moreover, Quandil and Quder (1984) reported in United Arab Emirates that most bacteria isolated from few cases of mastitis in female camel were those, which cause cow mastitis. Barbour et al. (1985) have shown that in many cases of infection with a variety of bacteria the organisms were present less than 3 x 10^3/ml of milk and only a very small number of cases exceeded this level. They suggested that there is a limit to bacterial multiplication in the camel's udder probably due to its complex immune system. It is also possible that the twin duct anatomy of the camel teat in some way protects it against infection (Manefield and Tinson, 1996). The practice of hand milking instead of machine milking might also be a reason of camels being not much susceptible to mammary gland infection.

A large number of bacterial species have been reported to cause mastitis in camels. They include Escherichia coli, Klebsiella pneumoniae, Staphylococcus aureus, Streptococcus sp., Pseudomonas sp., Pasteurella hemolytica, Corynebacterium sp., and Clostridium sp. (Quandil and Quder, 1984; Hassanein et al., 1984; Mostafa et al., 1987; El-Jakee, 1998). Abdurahman (1996) and Abdurahman et al. (1995) have suggested methods for the diagnosis of mastitis in camels.

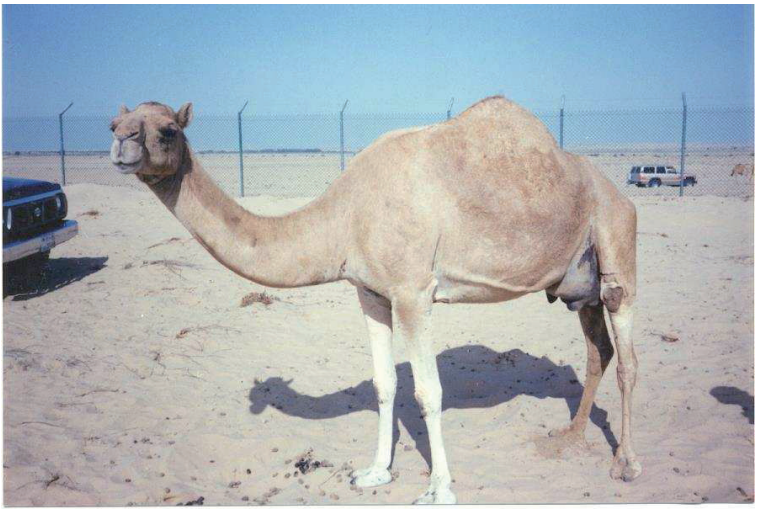
Clinical Findings

The symptoms vary according to the severity of the condition, which depends on the causative agent (Ramdan et al., 1987). Subclinical mastitis seems to be more common (Barbour et al., 1985; Mostafa et al., 1987). In less severe cases varying degree of inflammatory changes have been observed. One or more teats may be affected. The changes in the milk include discoloration, flocculation, change in viscosity, or pus may be present in the milk, depending

on the causative agent (Hassanain et al. 1984). Peracute, gangrenous mastitis may also be observed, which usually results in sloughing of the affected quarter (Kapur et al., 1982).

Treatment

Depends upon the type of signs. Generally, the disease can be treated by intramammary preparations, containing one or more antibiotics, suggested by antibacterial sensitivity test. Cortisone, oxytocin and enzymes (chymotrypsin) may also help, depending upon the nature of the case. This therapy may be supplemented by, parenteral administration of antibiotics, massage of mild counter irritants, and fomentation (hot or cold as needed). In gangrenous mastitis amputation of udder is recommended. In case of abscess formation drainage of pus either through teat canal or from exterior is required. In chronic mastitis sometimes fibrous tissue has to be removed to facilitate flow of milk and inflammatory exudate.



Mastitis in a ten year old female camel after calving

PASTEURELLOSIS

Organisms of Pasteurella spp. are responsible for causing different diseases in various animal species, mostly causing septicemia and affecting respiratory system.

The pasteurellae are small (0.2 um by upto 2.0 um) Gram negative rods or cocco-bacilli. They are non-motile, non-spore forming, facultatively anaerobic. Types or serogroups of P. multocida have been identified based on differences in capsular substances (polysaccharides). They have been designated A, B, D, E and F. Pasteurella multocida type A causes shipping fever complex, enzootic pneumonia complex (in calves), pleuropneumonia and mastitis in cattle and sheep. Pasteurella multocida type B is responsible for epizootic hemorrhagic septicemia in almost all ruminants. Type E causes epizootic hemorrhagic septicemia in cattle and buffalo in Africa. Pasteurella

hemolytica type A is responsible for shipping fever, pneumonia, septicemia and mastitis in cattle and sheep. Pasteurella hemolytica type F causes pneumonia in sheep. P. caballi causes pneumonia in horses. Pasteurella granulomatis causes fibrinous granulomatous disease in Barazilian cattle. Viruses share the pasturellae in causing shipping fever.

Camels are also susceptible to infection with Pasteurella spp. Pasteurellosis have been reported in camels in different parts of the world (Fazil and Hoffmann, 1981; Mustafa, 1987; Schwartz and Dioli, 1992; Momin et al., 1987; Hassan and Mustafa, 1985; and Richard, 1979). Awad et al. (1976) suggested that camels are less susceptible to P. multocida infection as compared to other ruminants. Hassan and Mustafa (1985) have reported an outbreak of paseurellosis in Sudan, where more than 50 camels died. Stress is thought to be of high importance in the initiation of pasteurellosis.

Clinical Findings

There is quite a variation in the clinical signs observed in camels as observed by different workers. The symptoms observed in camels are rise in temperature, lacrimation, excessive salivation, cessation of rumination, inappetance, rapid pulse and respiration (Awad et al., 1976). Momin et al. (1987) while reporting an outbreak of pasteurellosis in camels observed swelling at the throat regions with difficulty in breathing and death. Higgins (1986) has suggested three types of disease i.e. acute, peracute and abdominal form. Schwartz and Dioli (1992) reported the signs of pasteurellosis in camels as pyrexia, tachycardia and tachypnea, anorexia, painful swelling of the neck, enlarged cervical lymph nodes, tar like feces and chocolate colored urine. Morbidity in camels is reported to be low, but mortality can reach upto 80% (Schwartz and Dioli, 1992). Camels are also susceptible to P. pestis infection (Sotnikov, 1973) and it occurs in Mongolia, China, India, Iran, Africa, and USSR. Fleas are thought to be the cause of spread of disease (Higgins, 1986).

Treatment

The treatment is based on the clinical experience. Most of the affected animals recover within 24 hours if treated with almost any of the common antibacterials (penicillin, oxytetracycline, trimethoprim-sulphonamides, and chloramphenicol).

For prevention of disease one should minimize the effects of predisposing causes (stress) and vaccinate the animals.

DISEASES CAUSED BY CLOSTRIDIUM SPECIES

Pathogenic clostridia are commonly present in soil and water. They are also found in the intestinal contents of normal animals and cause disease only under special circumstances. The clostridia produce potent exotoxins that are responsible for the development of the disease. Each exotoxin has its specific effect on the animal system. Following are the most common diseases caused by different clostridial species.

TETANUS

This is a highly fatal disease of animals and human beings and is characterized by hyperesthesia, tetany and stiffness of the muscles. Camel

seems to be relatively resistant to tetanus. However, the occurrence of the disease has been reported (Rabagliati, 1920; Morcos, 1965). It is caused by Clostridium tetani, which is a spore forming organism and is capable to survive in the soil for many years. Clostridium tetani organisms are commonly present in the feces of animals especially horses and in the soil contaminated by these feces. The port of entry is usually through deep puncture wounds but the spores may lie dormant in the tissues for sometime and produce clinical illness only when tissue conditions favor their proliferation. For this reason the portal of entry is often difficult to determine. The endospores enter traumatized tissue or surgical wounds, especially after castration or docking, via the umblicus or into the uterus following dystokia. Presence of facultative anaerobes and necrotic tissue create anaerobic conditions and the C. tetani spores germinate. The vegetative cells multiply at the entry site and produce the patent tetanospasmin. This travels via peripheral nerves or blood stream to ganglioside receptors of the motor nerve terminals and eventually to cells of the ventral horn of the spinal cord, thus affecting many groups of muscles at various levels. The toxin acts presynaptically on motor neurones blocking synaptic inhibition and causing a spastic paralysis and the characteristic tetanic spasms. Tetanospasmin binds specifically to gangleosides in nerve tissue and once bound cannot be neutralized by antitoxin. Death is usually the result of asphyxia due to spasm of the muscles of throat.

Clinical Findings

Signs reported for camels are similar as reported for other animals i.e. hyperaesthesia (over reaction to external stimuli), protrusion of the third eyelid, muscle tremors and stiffness of the muscles (saw horse appearance). In camels the hind limbs are spread apart on standing, with tucked up abdomen. Walking, backing and turning is difficult, however, the camel can lie down easily (Marcos, 1965).

Treatment

The response to treatment is generally poor. Attempts should be made to eliminate the organism, which can be accomplish by giving penicillin or some other suitable antibiotic in large doses. Neutralizing the toxin by giving antiserum (given parenterally and around the wound). Relaxation of muscles can be achieved by administration of chlorpromazine or some other muscle relaxant. The animal should be kept at a dark quiet place.

Control could be attempted by application of antiseptics on the injuries (accidental, castration, docking) and injecting antiserum subcutaneously to achieve passive immunity.

BOTULISM

It is a highly fatal intoxication caused by ingestion of the toxins of Clostridium botulinum, which proliferates in decomposing organic matter and remains viable for many years. The condition can occassionally be caused by infection of the wounds by the organism. There are five (A, B, C, D and E) antigenically distinct types of C. botulinum. Under favorable climatic conditions warmth and moisture the organism proliferates and secretes highly lethal toxins which when consumed by the animals cause disease. The incidence of the disease increases in areas where phorsphorus is deficient in the soil and the animals eat organic substances rich in phosphorus. The organic materials may harbor toxins of C. botulinum. The absorbed toxins through the blood stream, bind to the susceptible cells and suppress the release of

acetylcholine at the myoneural junctions. This results in flaccid paralysis, causing death of animal due to circulatory and respiratory failure.

The incidence of the disease in camels is rare. Provost et al. (1975) have reported occurrence of the disease in camels.

Clinical Findings

The symptoms include difficulty in standing, paralysis of hindquarters and death in a few hours. Signs usually appear 3-17 days after the animals gain access to the toxic material, variation depends upon the amount of toxin absorbed. Peracute cases die without showing signs. In such cases toxin can be detected in the blood by mouse inoculation tests (Blood and Radostits, 1989).

There are no lesions detectable at necropsy, however, any abnormal material present in the stomach could be suspected suggestive of the condition. Clinically and at necropsy the disease resembles parturient paresis / hypocalcemia. Many other diseases of nervous system should be considered while making diagnosis.

Treatment

Specific or polyvalent sera may be of some help in the early stages of the disease. Purgation could be of some help. Central nervous system stimulants are sometimes administered. Other animals in the group should be vaccinated. Hygienic disposal of carcasses is advisable to prevent contamination.

GAS GANGRENE CLOSTRIDIA

The disease syndrome can vary from simple wound infection, anaerobic cellulitis to severe and fatal gas gangrene. Gas gangrene clostridia include C. chauvoei, C. septicum, C. novyi type A, C. sodellii, and C. perfringens type A. The infections can either be endogenous or exogenous. Endogenous infections often occur with C. chauvoei in black leg. Endospores are ingested and pass normally harmlessly through the intestinal tract but occasionally the spores pass from the intestine via the lymphatics and blood stream to muscle masses, usually muscles of the hindquarters but sometimes in cardiac muscles. Trauma to the area where spores are lodged causes tissue necrosis and hence anaerobic conditions favoring germination of the spores and a supply of amino acids and other nutrients for vegetative cells. Toxin is produced followed by localized damage and finally a terminal toxemia and bacteremia. In exogenous infections spores are introduced into wounds where they may germinate in the anaerobic necrotic material and toxin is produced by the vegetative cells.

According to the old literature the causative agents of gas gangrene were seldom isolated from camels. Clostridium chauvoei infections in dromedaries have been reported (Gatt Rutter and Mack, 1963).

HISTOTOXIC CLOSTRIDIA AFFECTING THE LIVER

Clostridium novyi type B is common in soil and in the normal intestinal tract of herbivores. It produces black disease “necrotic hepatitis” in sheep. Clostridium novyi type D (C. hemolyticum) found in the ruminants digestive tract, liver , and in the soil is the cause of bacillary hemoglobinurea in cattle. The alpha toxin produced by the C. novyi type B is lethal, necrotising and phage- mediated. The beta toxin produced by C. novyi type B and D is a phospholipase and is responsible for hemolytic crisis and death in bacillary hemoglobinurea. There is no report of the disease occurring in camels.

ENTEROTOXEMIA COMPLEX

Clostridial enterotoxemias are acute highly fatal intoxication that affect almost all the domestic animals, particularly the young ones. The diseases are caused by the major exotoxins (enterotoxins) of C. perfringens type B, C and D and occasionally type A and E. Clostridium nordelli can also cause enterotoxemia complex.

Acute and subacute entero toxemias as well as hemorrhagic enteritis due to C. perfringens, type A, C, and D have been described in camels (Moebuu et al. 1966; Ipatenko, 1974; Chauhan et al., 1985; Gameel et al., 1986) and in South American Camilids (Fowler, 1989).

Peracute and acute outbreaks of enterotoxemias, myocardial degeneration, and pulpy kidney disease in breeding and racing camels have been described in UAE (Wernery et al., 1991; Seifert et al., 1992; Wernery et al., 1992). According to Wernery and Kaaden, (1995) trypanosomiasis may be a predisposing factor for the outbreaks of enterotoxemias. An additional aspect in the development of clostridiosis in dromedary camels is the amount of serum immunoglobins in the young camels. The camelidae have an epitheliochorial placenta so the calf gets passive protection against disease through the intestinal absorption from colostrum. Although the newborn calve is immunocompetent at birth, the endogenous antibody production is not sufficient to produce a protective immunoglobulins level within the first month of life. Even after the ingestion of colostrum, the globulin levels decline after the seventh day and reach the lowest level between the 20^{th} and 30^{th} day post partum. The highest losses, due to C. perfringens enterotoxemia occur during this time (Unger-Waron et al. 1987; Hannant et al., 1992). Vitamin E / Selenium deficiency is also thought to be predisposing factor in causation of clostridial infections (Wernery, and Kaaden 1995).

Clinical Findings

The affected camels exhibit perspiration, muscular tremor, ataxia, hyperesthesia, seizures and death (Wernery and Kaaden 1995). According to Ipatenko (1974) symptoms of enterotoxemia in Bactrian camels include excitement, staggering, convulsions and death. Sanousi and Gameel (1993) observed yellowish, pasty, diarrhea and death, in suckling camels, in Saudi Arabia.

High protein or carbohydrate diets, abrupt change of weather and handling of animals (e.g., transport, weighing) are thought to be predisposing factors (Chauhan et al., 1985).

Clostridium perfringens types C and D have been incriminated in the causation of camel enterotoxemia in Russia (Ipatenko, 1974). Chauhan et al., (1985) described an outbreak of enterotoxemia in camels in India caused by C. perfringens type A in association with C. novyi. Gameel et al. (1986) isolated C. perfringens type A together with C. sordellii and Aeromonas hydrophila from camels with hemorrhagic gastroentritis in Sudan. An outbreak of enterotoxemia was observed in S. Arabia (Sanousi and Gameel, 1993).

Encephalomalacia caused by Clostridium perfringens Type-D toxins

The lesions in enterotoxemia as found by Sanousi and Gameel (1993) were hydrothorax, pulmonary congestion, and edema. Serofibrinous exudate in the pericardial sac was also observed. Epicardium was streaked yellowish brown, endocardium was whitish, and myocardium was soft and had chalky granular appearance. Peritoneal cavity contained considerable amount of serofibrinous fluid. Congestion and petechiation were seen in the abomasum. Intestines, particularly jejunum and ileum were congested, slightly edematous and had patchy mucosal hemorrhages and in some cases ulceration. Marked congestion was seen in mucosal lymph nodes, liver, kidneys and brain. Wernery et al. (1992) have reported severe myocardial degeneration and pulpy kidney in 4-6 week old dromedary calves.

Treatment

Hyper immune antisera administration is the only treatment likely to be of value. Doses of 25 ml of Clostridium perfringens type C antiserum have been used successfully in calves. Oral administration of penicillin may also help. Chelating agents may also help. Vaccination, preferably with type-specific toxoid or bacterin, is the only preventive measure available (Blood and Radostits, 1989). In the case of out breaks the dams should be vaccinated to protect their progeny. This should be done at least 2 weeks before parturition.

LEPTOSPIROSIS

The order spirochaetes includes the families Spirochaetaceae and Leptospiraceae. The genera of significance in animals and humans are Serpulina, Treponema, Borrelia (Spirochaetaceae), and Leptospira (Leptospiraceae). The genus Leptospira is at present divided into two species, L. interrogans (parasitic) and L. biflexa (saprophtic). Leptospires are present in the tubules of mammalian kidneys and are excreted in the urine, often for several months. Characteristically, a reservoir host shows minimal or no clinical signs. All domesticated animals including camels (Shigidi, 1974), wild

game, rodents, as well as man are susceptible to infection. Occurrence of leptospirosis seems to be insignificant in camels. There has been no report on the clinical presentation of leptospirosis in camels.

Krepkogorskaya (1956) has isolated Leptospira in the camel's organs. Presence of agglutinating antibodies for various serovars of leptospira in camels have been reported from various countries (Werney and Kaaden, 1995; Sebek, 1974 and Moch et al, 1975).

SALMONELLOSIS

Salmonellae inhabit the intestines of man and animals throughout the world. Majority of the infected animals become subclinical excretors. Salmonellae can survive for 9 months or more in the environment in sites such as moist soil, water, feces and animal feed. Infection of camels, as well as other animals, with various species of Salmonellae can result in serious clinical disease and always constitute a vast reservoir for infection of humans. The genus Salmonella comprises a single species that has been divided into over 2000 serotypes based on the somatic (O), flagellar (H), and occassionally capsular (Vi) antigen. The genus has been divided into 7 subgroups. Subgroup 1 contains most of the salmonellae that are significant animal pathogens and most have been given names e.g. dublin, typhimurium, etc.

Transmission is usually through oral route, however, this can happen through mucous membranes of conjunctiva and upper respiratory tract.

There are several reports on the occurrence of salmonellosis in camels (Curasson, 1918; Olitzki and Ellanbogen, 1943; Donatien, and Boue, 1944 ; Bruner and Moran, 1949; Kwaga, 1985; Cheyne et al., 1977; Pegram and Tareke, 1981; Refai et al., 1984; Yassien, 1985; Wernery, 1992). Salmonellae colonize the distal small intestine or colon before initiating enteric disease. Volatile organic acids produced by normal gut flora usually block access to attachment sites required by the Salmonella species. Disruption of the normal intestinal flora by factors such as antibiotic therapy, diet and water deprivation increases the host's susceptibility to infection. Reduced peristalsis, stress due to transportation and overcrowding also predisposes to colonization of the intestine by the salmonellae. The attachment of salmonellae is usually by fimbriae. Some strains producing enteritis and diarrhea appear to be capable of forming enterotoxins and cytotoxins.

The invasive strains that produce septicemia are able to escape destruction by the host and to multiply within the macrophages of the liver and spleen as well as intravascular. Any of the salmonellae, in nonimmune animals that are phagocytosed tend to survive within the phagocytes. Multiplication of the organisms in the body leads to a severe endotoxemia. The predisposing factors are transportation, nutrition, parturition, overcrowding, surgery, and medication (Blood and Radostits, 1989).

Clinical Findings

In camels salmonellosis may be enteric or septicemic. Chronic salmonellosis is characterized by diarrhea, weight loss and death within a few weeks (Fazil and Hafmann, 1981). Cheyne et al. (1977) reported unthriftyness and intermittent attacks of diarrhea in two severely ill animals shortly before death in salmonellosis. One of the camels, which had been sick for only 24 hours was recumbent with rectal temperature of 102.5^{o}F, pulse rate of 50 per minute, and rapid respiration. The prescapular and submaxillary lymph nodes were enlarged and easily palpable, the muscles of the head and neck twitched continuously, and the feces were predominantly fluid but with some hard

pellets. The second camel had been ill for 5-6 days and was in very poor condition. The rectal temperature was below 98° F, the pulse was thready, and the respiration labored. The superficial lymph nodes were grossly enlarged, and the visible mucous membranes were congested. The feces were black, liquid, and foul smelling. The animal died next day. At postmortem examination the mediastinal, mesenteric, and the peripheral lymph nodes were deeply and irregularly congested. Both the camels had haemorrhagic gastroenteritis.

Treatment

Early treatment with broad-spectrum antibiotics and sulphonamides and fluid electrolyte therapy is highly efficient. The choice of drugs to be used depends on a test of drug sensitivity in each case of outbreak but failing this the following generalization can be applied. The antibiotics of choice are gentamycin, ampicillin, chloramphenicol, and enrofloxacin. Combining antibiotics with sulphonamides may also help.

Preventing the introduction, into the herd, of carrier animals and contaminated food can control the disease. The disease can also be controlled by culling of carrier animals, prophylactic use of antibiotics, disinfecting farms, utensils, and by immunization.

COLIBACILLOSIS

Colibacillosis caused by Escherichia coli (E. coli) occurs in all species of newborn animals. Escherichia coli is a natural inhabitant of the large intestine and lower small intestine of all mammals. Escherichia coli is excreted in feces and can survive in fecal particles, dust and water for weeks and months.

The predisposing causes are of great importance and to a large extent determine whether or not the clinical signs of illness will occur. Neonates obtaining insufficient passive immunity from colostrum are predisposed to infection. This might be due to either a quantitative or qualitative deficiency of immunoglobulins. Poor hygiene often allows a built up of pathogenic strains in the young animal's environment. A large dose of pathogenic E. coli may overcome colostral immunity. Neonates, under one week of age, are particularly susceptible, the reason being that the normal flora of the intestines is not fully established, they have a naive immune system, and receptors for the adhesion of E. coli are present for the first week of life in bovine calves (for camel calves not known).

Escherichia coli strains, normally regarded as non pathogenic, can cause opportunistic infections in sites of the body such as mammary glands (mastitis) and uterus (metritis). Escherichia coli strains that cause enteritis have been classified as enterotoxigenic E. coli (ETEC) which has the fimbrial adhesions K88, K99 or others. The production of these colonization factors correlates with enterotoxin production. These strains cause the majority of cases of neonatal colibacillosis. Enteroinvasive (EIEC) strains adhere to cells of the distal small intestine.

Escherichia coli infection in dromedary calves have been described by various authors. Schwartz and Dioli (1992) reported a morbidity of 30% in neonatal dromedary calves in East Africa. Without immediate veterinary intervention, mortality can reach 100 per cent.

Clinical Findings

The camel calves suffering from colibacillosis exhibit anorexia, dysentary, abdominal pain, and dehydration. Death occurs within a few days (Chauhan et al., 1986; Rombol, 1942). A fever, of between 40° C and 41° C followed by death in 2-3 days was observed by Wernery and Kaaden (1995). On autopsy, extreme pallor of the whole carcass and inflammation of the intestinal mucosa and presence of sand in compartment 1 is also reported by the latter authors. In severe cases of E. coli dysentary, a fibrinous exudate covers the abdominal organs. In the adult camels E. coli has been recovered from sporadic cases and is involved in infection of uterus, urethra, bladder, kidneys, gastrointestinal tract, and mammary glands. Ibrahim et al. (1998) recorded edema disease in camels, caused by E. coli, in Bahrain. The disease appeared suddenly in a group of camels. The incidence of the disease was 50% or more and the mortality rate approached 90% of the affected animals. No rise in body temperature was noticed throughout the course of the disease. Affected animals had reluctant and painful gait. Some animals showed obvious nervous signs. Edema swelling started at the lower part of the body, i.e. abdomen, udder and brisket area. The distension of the bowel with fluid, in the late stages of the disease was the characteristic clinical sign. Usually affected animals died as a result of heart failure due to abnormal pressure exerted on the heart as a result of the sustained volume overload on the heart due to the presence of a huge amount (100-150 liters) of edema fluid. Lesions indicated heart failure.

Treatment

It is advisable to provide readily absorbable substances such as glucose electrolyte mixtures. Parenteral fluid therapy should also be given. The antibiotics of choice are chloramphenicol, neomycin sulfate, sulfonamides, trimethoprim-sulphonamide mixtures, nitrofurazone, ampicillin, and enrofloxines. Any of the above mentioned antimicrobials may be used but should be discontinued after 3 successive days of treatment to avoid elimination of too many species of drug sensitive normal intestinal flora.

As regards control of the condition, an attempt should be made to reduce exposure of newborn calves by observing all hygienic measures. Providing adequate colostrum and increasing the specific resistance of the newborn by vaccination. Maternal vaccination has also proved useful in the control of colibacillosis in camel calves (Strauss, 1991).

TUBERCULOSIS

It is a chronic infectious granulomatous disease of most species of animals including camels. Littlewood (1888) first reported it in camels.

Tuberculosis can be caused by Mycobacterium bovis, M. tuberculosis and M. avium and results in progressive development of tubercles in various organs of the body (Bush et al., 1986). The disease is of great zoonotic importance (Seifert, 1992). Inhalation is the most common port of entry, however, entry of the organisms may be effected by ingestion. Virulence of the organism appears to reside in the lipids of the cell wall. Mycosides, phospholipids, and sulpholipids are thought to protect the tubercle bacilli against phogocytosis. Glycolipids cause a granulomatous response and enhance the survival of phagocytosed mycobacteria. In previously unexposed animals, local multiplication of the mycobacteria occurs and the resistance to phagocytic killing allows continued intracelluler and extracelluar multiplication. Infected host cells and mycobacteria can reach local lymph nodes and from there may pass to the thoracic duct with general dissemination. After the first week of introduction of bacteria, cell mediated immune response starts modifying the

host's response and activated macrophages are able to kill some mycobacteria. The aggregation of macrophages contributes to the formation of a tubercle. A fibrous layer may encapsulate the lesion. Caseous necrosis occurs at the centre of the lesion and this may proceed to calcification or liquefaction.

Tuberculosis is not very common in camels, which are normally under nomadic conditions. The disease is normally observed in those camels kept in confinement, especially when they are kept along with cattle (El-Afifi et al., 1953; El Mossalami et al., 1971; Damodaran et al., 1969, Schillinger, 1987, Wernery and Kaaden, 1995).

Clinical Findings

The symptoms in camels, are progressive debility and coughing. Caseous nodules are found in lungs, liver, spleen and lymph nodes. Granulomatous masses with caseation were seen in the mediastinal lymph nodes and pleural cavity (Pellegrini, 1942). A change in the serum protein pattern in camels has also been described (Donchenko and Donchenko, 1978). Hematological changes occurring in camels are same (Fedchenko, 1971) as in other animals (Blood and Radostits, 1989). Herd diagnosis can be achieved by intradermal tuberculine test in camels (Kennedy and Bush, 1983).

Use of isoniazid, combination of streptomycin and para-aminosalicylic acid can be attempted for the treatment of valuable animals.

PARATUBERCULOSIS

It is characterized by chronic granulomatous enteritis in ruminants, including camels (Chauhan et al., 1986; Amand, 1974; Radwan et al. 1991; Esterabadi et al., 1975). It is caused by three strains of Mycobacterium paratuberculosis. The disease is not very common in dromedaries.

Cell mediated immune phenomenon appear to be involved in the pathogenesis of this disease. The organisms are found within macrophages that are unable to kill them, in the submucosa of the ileocecal area and adjacent lymph nodes. The mucous membrane becomes thickened and permanantly corrugated as a result of cellular infiltration. There is profuse diarrhea, and protein loss.

Diagnosis can be made by allergic tests (Khon, 1983).

Streptomycin is the most effective drug (50 mg / kg body weight) for treatment.

BRUCELLOSIS

Brucellosis is a disease of zoontic importance and is characterized by abortions in late pregnancy and infertility in female as well as males. Brucella species are facultative intracellular bacteria, which can survive and multiply within the cells of the macrophages system. It is fairly common in camels. It has been reported from all parts of the world where ever the camels are kept (Hamada et al., 1963; Abu Damir et al., 1989; Waghela et al., 1978; Andreani et al., 1982; Domenech, 1977; Okoh, 1979; Radwan et al., 1995; Gameel et al., 1993; Afzal and Sakhir, 1994; Chukwu, 1985; Kagunya and Waiyaki, 1978; Kudi et al., 1997; Zowghi and Ebadi, 1988). The disease is caused by infection with Brucella sps. (B. abortis, B. melitensis, B. suis, B. ovis, B. canis), particularly B. abortis. Transmission is by direct or indirect contact with infective excretors. The route of infection is often by ingestion but venereal transmission may occur. Less commonly, infection may occur via conjunctiva or by inhalation. The brucellae possess an endotoxin that contributes to the pathogensis, as does a surface cell wall carbohydrate that is responsible for

binding to B-lymphocytes. Soon after entry into the host, the brucellae are engulfed by phagocytic cells. In the latter cells the organisms survive, multiply and are transported to the regional lymph nodes. The organisms pass to the thoracic duct and then via the blood stream to parenchymatous organs and other tissues such as joints. Granulomatous foci can develop in tissues with occasional suppuration and caseation. Brucellosis is essentially a disease of sexually mature animals, the predilection sites being the reproductive tract of males and females, especially the pregnant uterus. Allantoic factors stimulate the growth of most brucellae. These factors include erythritol. Erythritol, possibly steroid hormone, and other substances are present in the placenta and male genital tract of animals. A pyogranulomatous reaction occurs in affected placentae and abortion occurs from mid-gestation onward. Apparently normal, but infected neonates can be born but the infection is of limited duration in these animals. Females usually abort only once, after which a degree of immunity develops, and the animals remain infected and large numbers of brucellae can be excreted in fetal fluid at subsequent parturitions.

The majority of reports on brucellosis in camels utilize serological methods of identification (Chichibabin, 1971). It has been observed (Wernery and Wernery, 1990) that incidence in racing dromedaries in UAE is higher than the breeding camels and is attributed to feeding of raw cow's milk to the racing camels. The lower incidence of infection in breeding camels as compared to racing camels is supposed to be due to spontaneous recovery among non-reproductive dromedaries (Fazil and Hofmann, 1981; Gatt Rutter and Mack, 1963; Ostrovidov, 1954a, and b). An incorrect diagnosis of brucellosis may occur when based on serology alone, which is due to cross reactivity with other bacteria (Osman and Adlan, 1987; Vanstraten et al., 1997). Although isolation of the organism is difficult, however, it has been isolated from the milk and lymph nodes.

The treatment of brucellosis in the cows has generally been unsuccessful because of the intracelluler sequestration of the organism in lymph nodes, the mammary gland and reproductive organs. Treatment failures are considered not due to the development of anitimicrobial resistance but rather the inability of the drug to penetrate the cell membrane barrier. The use of long acting oxytetracycline at 20 mg /kg body weight intramuscularly at 3-4 day intervals for five treatments in combination with streptomycin daily for 7 days consecutively is reported to be partially successful in the treatment of infected cows. (Blood and Radostits, 1989).

CAMEL PLAGUE

The causal organism is Yersinia pestis. The virulence factors of Yersinia species include antiphagocytic outer membrane proteins, a plasmid-encoded exotoxin, a bacteriocin, a coagulase and a fibrinolytic factor, all of which correlate with virulence. The virulent strains of Y. pestis resist phagocytosis and are able to grow within macrophages. Endotoxin is also thought to contribute to tissue damage and clinical signs in plague. The symptoms in camels vary. There may be vague illness, enlargement of lymph nodes, generalized cutaneous abscesses, septicemia or pneumonia (Manefield nd Tinson, 1986).

There is an extensive literature blaming camel to spread plague to humans (Fedorov, 1960; Wu et al., 1936; Pollitzer, 1954). Outbreaks of plague

have been reported from India, Iran, Iraq, Africa and Russia (Sotnikov, 1973). There are conflicting reports on susceptibility of camel to plague. Fedorov (1960) suggested that although camels did vary slightly in susceptibility to plague, the mechanism of their infection was the same as in plague susceptible rodents and humans. Ticks and fleas that have recently fed on septicemic plague affected rodents can infect camels. Christie et al (1980) reported that 19 human beings became infected with plague from contact with killing of diseased camel and goats.

The organism of plague is susceptible to most of the commonly used antibiotics. The organism is sensitive to tetracyclines, aminoglycosides and sulphonamides. The treatment should be attempted with antibiotics and supportive therapy.

FUNGAL DISEASES

RINGWORM INFESTATION (Dermatophytosis)

The dermatophytes are group of closely related fungi that utilize keratin for growth. They tend to be confined to the superficial integument including the outer stratum corneum of the skin, nails, claws and hair of animals and man. Traditionally the dermatophytes are placed in the fungi Imperfecti but the perfect state has been described. Over 38 species of dermatophytes are known. Those affecting animals are placed in one of two genera Microsporum or Trichophyton. The dermatophyte species affecting animals are known as ectothrix as the septate hyphae invading the skin and hairs fragment into arthrospores and these form a sheath around the infected structures (Gupta et al., 1970).

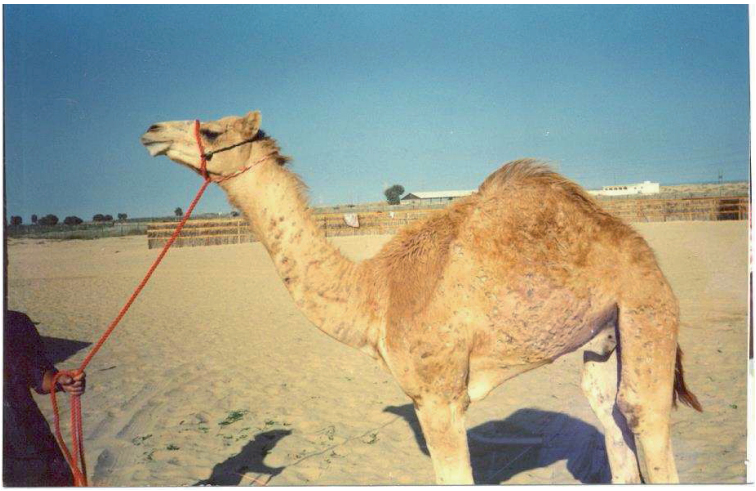
The ability of the dermatophytes to hydrolyse keratin may cause some damage to the epidermis and hair follicles. The mechanism by which they cause lesions is through a hypersensitivity reaction in the host to the fungal metabolic products. The host mounts an inflammatory response that is harmful to the fungus, so the dermatophytes move away peripherally towards normal skin. The result is the commonly seen circular lesions of alopecia with healing at the centre and inflammation at the edges. (Polyakov and Ivanov, 1985, Sarkisov et al., 1989; Khamiev, 1981, Kamel et al., 1977).

Lesions of ringworm infestation

Ringworm (dermatophytosis) is a very common disease of young camels. Peak incidence being in the 1st year of age. Mostly the camels get infected by Trichophyton species (T. mentagrophytes and T. verrucosum), but infection by Microsporum congolensis, Aleurisma lugdunese, Penicillum vinaceum, Pseudeurotium spp., Pseudoarachniotus spp., Allescheria spp., and Mycelia sterial have also been reported (Gitao, 1992; Chatterjee et al., 1978; El Timawy et al., 1988 and Khamiev, 1982). Outbreaks are associated with winter rains, and in late summer when there is high humidity. The disease has zoonotic importance and the camel-handlers get frequently, affected by the disease.

Clinical Findings

The early skin lesions are in the form of circular, crusty areas of alopecia, 1-2 cm in diameter. The lesions usually coalesce. The young camels are more severely affected and almost all of the animal's body gets involved. In the old animals lesions are localized. The disease is self-limiting in well-fed camel calves. The morbidity rate is very high, reaching to 100% in the young camels. Infection with D. congelensis causes an exudative inflammatory reaction, resulting in a bulging of the slow growing epidermis away from the corium, thereby allowing growth of a new layer of epidermal cells (Seifert, 1992). Drying of the serous exudate forms a crust which, is a distinguishing characteristic of this disease. When the crusts are removed a wet reddish area is revealed. Losos (1986) and Gitao et al. (1990) have described two forms of dermatophitosis: An early or acute form and a chronic form. Long hairs in the vicinity of the exudate become matted together yielding the characteristic "paint – brush" effect. The human beings can contact disease by handling animals, wool, and hides of infected animals.



Ringworm in a 3-year old male camel

Most of the fungi causing ringworm disease produce certain metabolites when growing on hairs and skin that will fluoresce a vivid apple green under ultraviolet light (366 nm) of a Wood's lamp. The hair should be plucked and material should be obtained from the edge of the lesion for making diagnosis. The KOH wet preparation method examined under low power objective (with condenser lowered slightly) may reveal presence of spores. The scrapings or hair can also be grown on special media (Emjon's sabouraud dextrose agar) for identification and sensitivity test.

Treatment

A number of fungicidal drugs, for topical use, are available. They include: copper sulfate 10% ointment, iodine preparations, propionic acid, natamycinum (Mycophyte, Myofarm), enilcozolum (Imaverol, Jenssen), mercury preparations (Sporyl, Richter Pharma), and Halamid (Intervet). Sodium iodide 500 mg / ml (Sodide, Parnell) given as injection gives good results. All the medicines should be used as per manufacturer's directions.

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ECTOPARASITES

Infestation with ectoparasites is one of the most important setback in livestock health in tropics and subtropics. They not only inflict direct injuries but also transmit a wide range of diseases among various animals, including camels. Flies transmit trypanosomiasis caused by Trypanosoma evansi, one of the most important diseases of camels. Mange caused by Sarcoptes scabei is often regarded as second in importance to trypanosomiasis. Ticks play a direct major role in morbidity and mortality as is the case in helminthiasis.

Tick–borne diseases in camels are not so important. However, unconfirmed anaplasmosis associated with Hyalomma species have been recorded (Wilson, 1984). Ticks are also believed to transmit viral diseases e.g., posterior paralysis (UNESCO – IPAL, 1985). Amblyomma lepidum is suggested to transmit heart water in camels (Karrar et al, 1963). The ticks can inflict considerable mechanical injury and subject the camels to secondary bacterial infections. The most affected sites to injury are around the eyes and feet. Several other conditions of camels, causing paralysis and ataxic / nervous symptoms, which can be attributed to viruses or some other toxic agents transmitted by ticks. Further studies on this aspect are suggested.

Of the hematoparasites, Trypanosoma evansi is the most important and is transmitted by numerous species of biting flies. Flies may also be involved in epidemiology of several bacterial diseases including anthrax and salmonellosis. Globidiosis, a skin disease of the camels caused by a protozoan parasite, is thought to be spread by biting flies. They can also transmit Thelazia species in various species of animals, including camel. Mosquitoes of Aedes species transmit Dipetalonema evansi. Although the filarial worms are mostly found in the heart, but sometimes sheathed microfilariae can also occur in pulmonary circulation, in lymph nodes, and in spermatic cord.

Culticoides species may transmit Onchocerca fasciata, which is a specific camel parasite. Although the prevalence of camel onchocerciasis can be high, the disease generally causes few problems other than unsightly subcutaneous nodules, mostly in the neck and head region.

The ectoparasites of camels consist of two zoological classes, the Arachnida (mites and ticks) and the Insecta (insects), within phylum Arthropoda.

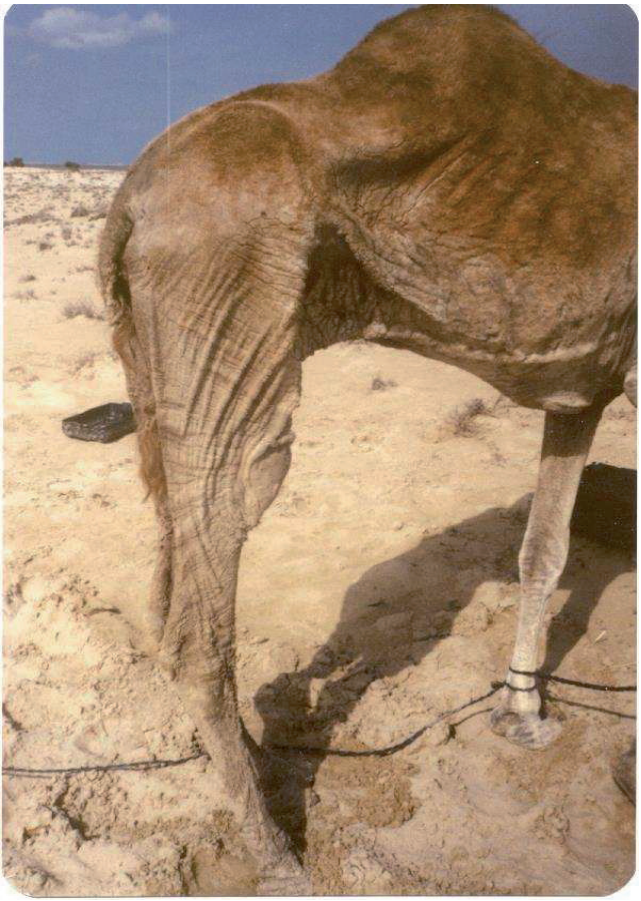
ORDER ACARINA

Severe mange can reduce productivity in camels of any age. Ticks can cause losses resulting from calf mortality. This is because young camels are very susceptible to ticks (Pegram and Higgins, 1992). In northern Kenya about 20-70% of camel calf mortality is attributed to Rhipicephalus pulchellus and Hyalomma truncatum. Under favorable circumstances Hyalomma dromedarii and R. pulchellus multiply very fast. Steward (1950) has reported death in camels infested with up to 100 nymphs and adults of Hyalomma species / 25 cm^2. High infestation causes reduction in milk yield and depression in growth rate. Although ticks may be of

direct economic importance in camel production, their role as vectors of tick borne diseases is very minor as compared to cattle, sheep and goats.

FAMILY IXODIDAE (TICKS)

The most important genus of ticks, parasitising camels is Hyaloma with four host specific species. H. asiaticum, H. dromedarii, H. franchini and H. scupense. The other genera infesting camels, which can be identified easily, are



Demodectic mange (invasion of hair follicles leads to chronic Inflammation; staphylococcal pustules on the lower leg)

Amblyomma and Dermacentor, as these are ornate. Only three species of soft ticks (Argasidiae) occur on camels, the most important of which is the desert adapted sand tampan, Ornithodoros savignyi. Frequently, it invades camel pens and yards. Ornithodores tholozani and O. laborensis have also been recorded in camels (Abu Elela et al., 1981; Al Asgah, 1985; Dolan et al., 1983; Drerup-Eilker, 1980).

About half of the Hyalomma ticks which infest camels exhibit a typical three-host (triphasic) life cycle. Notable exceptions are the one host H. scupense and the four species of H. marginatum, which are two host ticks. The latter sub species are also associated with birds, which migrate towards north and south, and assist their distribution over wide areas. The type of life cycle may vary, however, according to environmental conditions and availability of hosts. Ticks of this family possess a hard, chitinous shield or scutum which extend over the whole dorsal surface of the male and covers only a small portion behind the head in the larva, nymph and female. The mouth parts are anterior and well visible from the dorsal aspect. Eyes when present consist of one pair situated on the lateral margin of the scutum. The imago has one pair of spiracles situated posteriolaterally to the fourth coxae.

The basis capituli or capitulum, which is inserted into the body anteriorly and carries the mouth parts and palps, shows two dorsal porose areas in the female. The scutum has bilateral cervical and lateral grooves, varying in depth and length in different species. The body of the female may have a pair of lateral marginal grooves behind the scutum, while posteriolateral and median grooves are usually present on the dorsum in both sexes. The posterior border of the body may be notched, forming the 'festoons', which are generally 11 in number. The genital opening is a ventral transverse slit in front of the middle, the anus being posterior. The male may have ventral plates. Ornate ticks have colored, enamel-like areas on the body, inornate ticks have not (Soulsby, 1978).

The Ixodidae lay their eggs in sheltered spots: under stones and clods of soil or in crevices of walls and cracks of wood near the ground. The eggs are small, spherical, yellowish-brown to dark brown in color and are laid in large masses. The female lays all her eggs in one batch, up to 18000 in some species, and then dies. The whole process of subsequent development to the adult stage is greatly influenced by the prevailing temperature, cold weather causing marked prolongation of the different stages, especially hatching of the eggs and the pre-oviposition period of the engorged female.

The newly hatched larvae or 'seed ticks' climb on to grass and shrubs and wait there till a suitable host passes, to which they attach themselves with their claws.

After having engorged, the larva moults and becomes a nymph. The integument of the latter requires a few days to harden and then the nymph engorges and moults to become an imago. After hardening of the integument, and often also after copulation, which may take place on the ground or, more usually, on the host, the female engorges, drops off and seeks a sheltered spot to lay her eggs. The males remain much longer on the host than the females, in some cases four months or even longer, and consequently they accumulate on the host. Although it is not known definitely whether the males of all species feed on the host, many of them certainly do so for a few days and then go in search of females. If no males are present on the host, the females may remain attached for much longer periods than normal.

According to the number of hosts they require during their life cycle, ticks can be classified into three groups:

One-host ticks

All three instars engorge on the same animal, the two ecdyses also taking place on the host. Examples: Boophilus decoloratus and B. annulatus. B. microplus infesting camels have been reported by Kennedy and Green (1993).

Two-host ticks

The larva engorges and moults on the host and the nymph drops off after also having engorged; it moults on the ground and the imago seeks a new host. Examples: Rhipicephalus evertsi and R. bursa.

Three-host ticks

These require a different host for every instar; they drop off each time after having engorged and moult on the ground. Examples: Ixodes ricinus and Rhipicephalus appendiculatus.

Each species of tick is adapted to certain ranges of temperature and moisture, some occurring only in warm regions with a fair degree of humidity, while others are winter ticks most active in a dry climate. They suck blood and sometimes lymph and are in general not very specific with regard to hosts, although some species, or certain instars of a species, show a particular preference for certain host species, or there may be a definite adaptation to certain hosts. When a tick attaches itself to feed, it buries its mouth parts deeply into the tissues of the host and remains attached until it is engorged. The feeding mechanism of ticks has been reviewed by Tatchell (1969). This is assisted by the host response to the tick and infiltration of neutrophils, which leads to destruction of collagen resulting in a cavity beneath the mouth parts.

Various species of Ixodidae are vectors and reservoirs of important viral, rickettsial and protozoan parasites of man and other animals (Hoogstraal, 1981).

Most of the 32 species belonging to genus Hyalomma are found frequently on camels. This genus, has been notoriously difficult taxonomically. Individual species show much variation, which cause large numbers of descriptions of new species, later to be synonymised.

Hyalomma dromedarie is a highly desert adapted tick which occassionally uses three hosts. Peak density of these ticks occur usually in January and June-July. However, in arid regions they occur through out the year. Hyalomma schulzea is a large tick and occurs in Iran and neighbouring countries (Hoogstraal, 1981).

Hyalomma anatolicum anatolicum is a small two host tick, which infests wide range of animals including camel. This tick seems to be active through out the year. Cracks in walls or buildings and stony premesis often harbor numerous nymphs and adults.

Hyalomma anatolicum excavatum is large tick widely distributed. It has two to three host life cycle, virtually always found in rodent burrows.

Ornithodoros savignyi is called sand tampan. It commonly attacks camels in hot, arid, desert areas, and can also parasitize man and other domestic animals, especially goats. It hides just below the soil surface and large numbers can be seen scurrying towards a potential host in animal farms. These ticks are rarely found in buildings and are one of the most highly desert-adapted of all arthropodes. It takes less than 30 minutes to complete a feed and its bite is very painful.

Rhipicephalus pulchellus is one of the species infecting camels. It is the easiest member of the genus to identify definitively. Nearly half of the rhipicephalus ticks infesting camels are members of notorious R. sangumueus group which like some Hyalomma ticks, has been confused due to proliferation of synonyms and its use as a collective term for unidentified ticks.

Most observations on camel ticks are a part of general surveys rather than specific studies on the camel (Liebisch and Zukar, 1974; Mc Cartan et al., 1987; Osman, 1978; Osman et al. 1982; Pegram, 1976; Pegram et al., 1982; Rutagwenda and Munjua, 1984).

Death of camels has been reported following infestation with up to 100 nymphs and adult Hyalomma species ticks per 2.5 cm^2 of skin (Steward, 1950). There is a loss of 1-3 ml blood for every tick completing its life cycle on an animal. Distraction due to irritation by ticks can result in reduction in the production. Ticks also damage hides. The damaged sites caused by ticks on the skin attract flies, leading to bacterial / protozoal infection and myiasis. The attachment of Amblyomma lepidium results in the development of large sores. The ticks can affect the camels throughout the year, however, the survival rating of eggs and immature stages are reduced in hot desert conditions (Higgins, 1985).

Ticks can attach anywhere on the body of camels in the long hairs, but most common sites are perineal, inguinal, axillary regions, around the eyes, nostrils, lips and between the toes.

Some camel ticks have been reported to be able to transmit diseases of veterinary importance. Amblyomma lepidum has been proposed as an important vector of heart water (Cowdria ruminantum) in camels (Karrar, 1963). H. dromedarii is a possible vector of theileriosis of camels (Hoogstraal, 1981). There has been controversy whether ticks can transmit some viral diseases in camels, however, camel ticks can be vectors of some viruses affecting man e.g. H. anatolicum is a major vector of Crimean Congo haemorrhagic fever virus in USSR, Pakistan, Nigeria, Iran, and UAE (Suleiman et al., 1980).

CONTROL

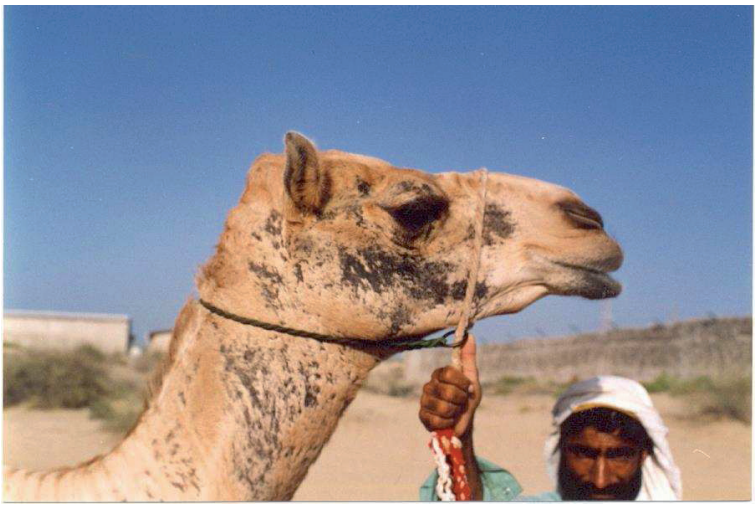
Complete eradication is not possible because of the persistence of ticks on wild fauna and their ability to live for very long periods apart from the camel. An attempt should be made to reduce the tick population by periodic spray of insecticides on farms and camels.

Development of resistance against insecticides should also be considered. The use of pour on applications, which allow a longer period between treatments e.g., ivermectin may be tried. Ornithodorus species ticks are difficult to control because the nymphs and adults attach to feed for brief periods only. Flumethrin pour on gives good results (El Azazy, 1996).

FAMILY SARCOPTOIDAE (MITES)

Mange has been reported from Egypt (Radwan et al., 1987), India (Lodha, 1966; Rathore and Lodha, 1973; Raisinghani and Kumar, 1990), Saudi Arabia (Higgins et al., 1984), Sudan (Abu Samara et al., 1981), France (Richard, 1987), Germany (Grigoryan, 1987), and Nigeria (Basu et al., 1995). Stress, age, malnutrition, overcrowding, poor skin condition, long hair coat, and worm burden have all been suggested as important predisposing factors for camel mange. Young or aged camels are more prone to infection, probably reflecting lowered body defences. Nomadic camels on poor nutrition and higher worm burden are more susceptible to mange infection. Debilitating diseases such as tuberculosis and trypanosomiasis also precipitate this condition. Bernstein et al. (1997) has detected antibodies against mange scabies in camels. Kumar et al. (1992) and Higgins et al. (1984) has reported high incidence of scabies in camels. Sarcoptic mange, caused by S. scabei var. cameli, is often ranked second in importance to trypanosomiasis in dromedary camels. Mange is usually a chronic debilitating condition with high morbidity and low mortality. It is difficult to confirm mange microscopically because of the burrowing effect of the parasite, and even more difficult to treat it

except in recent acute cases. The parasite is circular and minute. The size of female mite being 330-600 x 250 – 400 um and of male 200-240 x 200 um. (Soulsby, 1978). Hot weather in Arabia predisposes to acute outbreaks of camel mange, however, in other parts of the world the incidence seems to be more during winter months (Higgins et al., 1984; Raisinghani and Kumar, 1990).



Breeding camel suffering with sarcoptic mange

Mange is usually diagnosed frequently on clinical appearance. Nayel and Abu Samra (1986a; 1986) experimentally infected camels with Sarcoptes scabei and reported clinico-pathological findings. Mange lesions first appear as small nodules, often unnoticed but associated with initial burrowing of the mites, intense irritation leads to extensive rubbing and biting which results in hair loss, crusting and exudative dermatitis. Infestation starts in the inguinal region, neck and flanks. If untreated lesions spread quickly throughout the body. The female digs tunnels in the keratinous layers of the skin. These tunnels are used for depositing eggs, which are laid one or two at a time and three to five each day. Up to 50 eggs are laid by the female ticks, which hatch into larvae within four to five days. There after they pass rapidly through two nymphal instars to become reproductively mature within three weeks of age. All stages are capable of infesting another host. Quiescent phases usually coincide with winter. The mites multiply so rapidly that a single ovigerous female could theoretically give rise to over a million descendents within three months. If untreated, the camels will rapidly lose condition and within 2-3 weeks, the acute disease may give way to a chronic state. Excessive keratinization and proliferation of connective tissue will lead to the skin becoming thickened and thrown up into corrugations or folds often spread over with a fine “chalk – like” covering of dandruf. Ram et al. (1987) found a camel developing orchitis due to infestation with Sarcoptes cameli.

For treatment the camel may require three or four applications of acricide at 7-10 days interval with vigorous scrubbing over the body, unless powered spraying equipment is available. Saddles and other clothing must also be treated (Opferman, 1985). Sulphur and coal tar in the ointment form can be helpful to some extent in

treating mange. Chlorinated hydrocarbons, organophosphate insecticides, hexachlorocyclohexane, lindane, and gamma isomer of benzene hexachloride are also being used as acaricides (Draz, 1947; Lodha, 1966; Ismail and Amer, 1978; Ram et al., 1980; Higgins, 1984).

Ivermectin and other similar drugs seem to be most effective and convenient for the treatment of mange (Lumsden, 1992). A single subcutaneous injection of ivermectin (200 ug / kg body weight) usually resolves clinical signs of mange. A second injection a day later may be required for complete control (Hashim and Wasfi, 1986). It is better to treat all the animals in a group (Chellapa et al., 1989). Pathak et al. (1995) found that charmil gel gives good results against sarcoptic mange in camels. Amitraz applied locally, also gives good results (Singh et al., 1996).

Chorioptic mange has only been reported in Bactrian camels. The man can contact mange from camels (Basu et al., 1996).

THE INSECTA

MYIASIS PRODUCING FLIES

Myiasis results from invasion of living animal's tissue by the larvae of diptera (flies). There are specific myiasis producing species such as Chrysomyia bezziana, facultative species such as Lucillia cuprina, and accidental species such as Musca domestica. Calliphoridae and Sarcophidae are large families in which the adults have functional mouth parts. Only a few species are obligate agents of myiasis. The Oestridae is one of the four highly specialized families in which mouth parts of adults are nonfunctional, but the larvae are obligatory, often host-specific, parasites of mammals.

The calliphoridae are divided into several subfamilies; two are of biomedical importance. The Chrysomyinae contains the two most important screw worm species, both of which are obligatory agents of myiasis. Cochliomyia hominivorax, the new world screw worm, has in the past few years, become of global importance since it invaded north Africa (Husni and Elowni, 1992). Chrysomyia bezziana, the old world screw worm is distributed widely in Asia, Arabia and Africa and is attracted to tick bites and other wounds. Several females may be attached to the same wound, due to phermone production, leading to production of up to 3000 larvae. The other subfamily, the Calliphorinae includes the genus Lucillus. Lucillus cuprina is associated with sheep but can infect camels also.

The Sorcophidae contains the genera Sorcophaga and Wohlfabrtia. Wohlfabrtia magnifica is thought to be the most important of the obligate myiasis-producing flies affecting the camels. Adult flies deposit larvae in any wound or tick-bite lesion including those in the nasal cavity. In Bactrian camels W. magnifica may be involved in vaginal and preputial myiasis. Genital myiasis has also been reported by Valentin et al. (1997) and Ribbeck and Beulig (1977), in camels. Wohlfabrtia nuba, a similar parasite, occurs in the wounds of camels and man. Sarcophaga misera is a facultative parasite, which is also associated with camels. Cochliomyia hominivorx is reported to be the cause of wound myiasis in camels (El Azazy, 1989)

NASAL MYIASIS

These are obligate parasites and develop in the nasopharyngeal cavities. Cephalopina titillator is seasonal, host specific nasal bot fly of camel (Abul Hab and Al S'Adi, 1973; Abul Hab and Affas, 1977; Elias et al., 1984; Musa et al., 1989; Hadani et al, 1989; Nwosu and Wachy, 1998). This fly deposits its larvae on the nostrils of camels. After moulting twice, the larvae ingest a large amount of blood.

The life cycle may take several months. Normally two generations occur every year. The adult flies are short-lived (Fatani and Hilali, 1994; Charyev, 1981).

The infested animals are sneeze and shake their heads. In severe infestations the bots may perforate the sinuses leading to nervous symptoms (Hussein et al, 1982), which can be confused with several other diseases. Antibodies have also been detected against bot flies in camels (Nwosu and Wachy, 1998).

BITING AND NUISANCE INSECTS

There are about 3000 species of tabanid flies, the mouth parts of which are designed both for biting and lapping. These flies are stout often with colored, spotted or banded eyes. The flies are capable of transmitting a number of pathogens. These are considered to be more efficient vectors for transmission of Trypanosoma evansi than Chryosops and Hematopota. The flies cause a lot of disturbance to camels and other animals.

The other flies in this group are Muscidae, which are medium sized hematophagus blood-sucking stable flies and are associated with stabled animals. Other smaller horn flies 'Hemobia" are associated with pastured animals. This family also includes common houseflies.

The stable fly, the horn fly, and the buffalo fly are included in this group. The stable fly, Stomoxys calcitrans, bites the legs and lower parts of the body of camels. Its peak feeding activity is early morning and evening, a pattern common to many diurnal insects. The irritation and disturbance caused by stable flies may decrease production and performance.

The horn fly (H. irritans irritans) and buffalo fly (H. irritans exigua) are both hemotophagus flies, which rest on their hosts and take frequent small blood meals. They cause considerable disturbance and reduction in performance of animals. There is another blood sucking genus of flies. It contains about 200 species. Among these H. camelina is well adapted to its parasitic association with the camel and large number of flies are more or less permanantly associated with their hosts. Haemobia maculata is also reported to be an occasional camel parasite. The sand flies (Hippo phlebotomus) are important in the epidemiology of Leishmaniasis. They are believed to be associated with a form of lymphangitis in camels known as "Elephant Foot" (Higgins, 1986).

Treatment and Control

Routine seasonal treatment for mange control using diazinon will offer protection against fly strike for few weeks. For valuable herds regular spraying with synthetic pyrethroids (permethrin or cypermethrin) to repel adult flies is recommended. The wounds should be treated with insecticides. For the wounds already containing larvae it is advisable to manually remove the larvae and wounds treated by insecticides mixed in vaseline. Negasunt manufactured by Bayer is very useful.

For nasal myiasis, Kunichkin (1975) recommended to irrigate nostrils of camels with coumaphos, trichlorophen or trichlorometaphos-3 for successful removal of larvae from nostrils. Administration of trichlorophan to camels in drinking water (0.03-0.05%) is reported to be good in the treatment as well as prophylaxis (Charyev, 1982). Oxfendazole has also been reported to have some activity against O. ovis . Rafoxamide, and nitroxynil are also effective in treating nasal myiasis. Ivermectin may also be helpful in treating myiasis (Lumsden, 1992).

LICE AND FLEAS

Within the phylum Insecta, order Phthiraptera (lices) contains about 3000 species. The species specific, obligatory parasitic sucking–louse of camel (Hematopinus cameli) is generally a problem only in temperate regions where the hosts grow a long winter coat.

Siphonaptera or fleas, of which there are about 1800 species, are a problem in cooler climatic zones. The intense irritation caused by lice and fleas leads to rubbing and biting which may affect the performance of animals.

Larvae of beetle "Ergaster faber" can also be encountered occasionally in the nasal cavity of camels.

The insecticides mentioned for controlling flies, ticks and mites are effective in treating and controlling lice and flea infestation.

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PROTOZOA

ANAPLASMOSIS

Causative agent of bovine anaplasmosis is Anaplasma marginale. Anaplasmosis is primarily a disease of cattle, but other domestic and wild ruminants including camels have been reported susceptible to natural infection (Ristic and Krerier, 1974; Ristic, 1977). Presence of antibodies against A. marginale have been documented by Ajayi et al., (1984) from Nigeria. They reported incidence of 10.7% in camels. There is no report on the occurrence of clinical anaplasmosis in dromedary camels.

THEILERIOSIS

Theileria camelensis is an intra-erythrocytic protozoan parasite infecting camels. Its presumed vector is Hyalomma dromedarii (Boid et al., 1985).

Theileriosis was reported by Yakimoff (1921) from camels in Turkestan. It has also been reported from USSR, Egypt and Somalia (Barnett, 1977). Occurrence of theileriosis (Theleria dromedarii) in camels has been reported by Rao et al. (1988). The latter authors have reported biochemical changes occurring in this disease. Nassar (1992) found erythrocytes infected with different species of Theilaria in 60 out of 200 camels (30%). The camels examined by him were apparently healthy. According to the author the camels examined were either chronically infected or the species of Theileria were non-pathogenic.

BALANTIDIUM

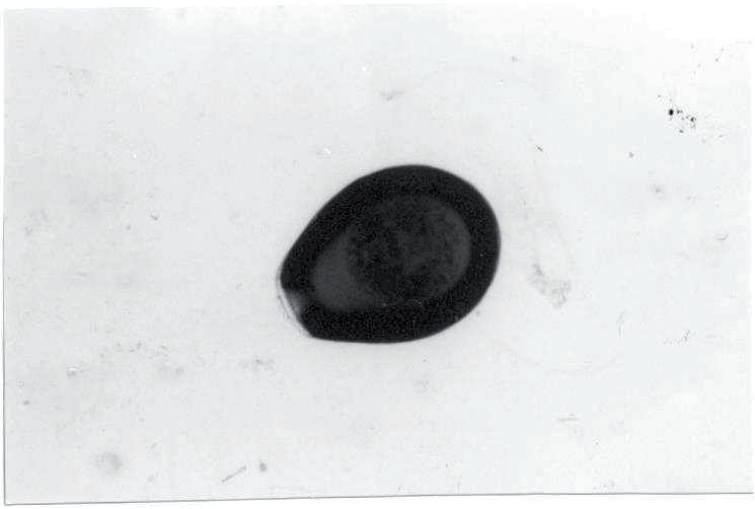
Occurrence of Balantidium coli in the camel feces has been reported by many workers throughout the world (Gill, 1976). It is usually present in the gut without showing any symptoms.

Some times the organism invades the gut mucosa and can cause severe diarrhea in camels (Vasdingh and Vanniasingham, 1969; Ali and Abdelaziz, 1982). The factors which, result in the invasion of intestinal wall by the protozoan are not clearly understood. The use of sulfa drugs and metronidazol may prove useful in treating the diarrhea caused by B. coli infection.

COCCIDIOSIS

Coccidiosis is caused by infection of intestines with Eimeria and Isospora species in all domestic animals, including camel (Kasim et al., 1985). There can be subclinical infection or there may be diarrhea and dysentry. The disease is not so important in camels, however, Kinne and Wernery (1997) have reported severe out break of coccidiosis in UAE. Chineme, (1980), and Hussein et al. (1987) have also reported severe coccidiosis and mortality up to 10% in the camels. Coccidia tend to be host specific. A few species of the genus Eimeria have been described in camels. The most ubiquitous are Eimeria cameli and Eimeria dromedari. The other species which are also widespread but are not identified easily are E. rajasthani and E. bactriani. These have been reported in dromedary and bactrian camels. Eimeria pellerdyi has been reported from India

(Rangarao and Sharma, 1997). The life cycle of coccidia includes both sexual and asexual phases. The asexual phase is called schizogony or merogony. Sporulated oocysts are ingested by the animals, and pass along the digestive tract to the small intestines. The oocyst frees sporozoites, which invade the epithelial cells. The sporozoite changes shape and become a trophozoite, which, in turn, grows larger



Eimeria dromedarii

and forms a schizont (merozont). Within the schizont, merozoites form and ultimately rupture the cell and escape to infect other cells. This process may be repeated two to three times. The sexual cycle is called gametogony. The merozoites produced by the last schizogony cycle infect a cell and develop into either male (microgametocyte) or female (macrogametocyte) gamonts. The male gamont fertilizes the female gamont while it is still in the cell, producing a zygote. The zygote matures to become an oocyst, which ruptures the host cell, and is shed in the feces. The oocysts, in 1-2 days, become infective.

Coccidia infections are usually self-limiting because sexual reproduction is repeated, 2-3 times. Normally only one cycle of development can occur, however, in contaminated environment, reinfection frequently occurs. Ruminants do have immune response. Whether camels have this response is not known. The coccidia are frequently seen, while making fecal examination by floatation method (Blood and Rhadostits, 1989).

Clinical Findings

Incidence of coccidiosis increases in humid season (Kawasmeh and El Bihari, 1983). Coccidia invade and rupture the mucosal epithelial cells, causing enteritis and diarrhea. The inflammation may be catarrhal or hemorrhagic depending on the severity of the condition. Stepanova (1982) found that young camels up to one year old were most susceptible to infection with coccidia and they exhibit signs of general weakness, failure of appetite, diarrhea, and

emaciation. Kinne and Wernery (1997) found severe sickness with abdominal pain and inappetance for 3-7 days and bloody feces with diarrea in camels suffering from coccidiosis. Chineme (1980) documented lesions of coccidiosis in a camel. He demonstrated developmental stages of the parasite in the lamina properia of the jejunum. The main pathological changes observed, by Kinne and Wernery (1997) were severe hemorrhages in the inflammed abomasum, jejunum, and ileum. They also found numerous coccidial stages (oocysts, macroschizonts and merozonts) located in the mucosa of the jejunum and ileum. Severe edema of the villi and a moderate to severe infiltration of the mucosa with eosinophilic granulocytes, and a few macrophages were also observed.

Treatment

Numerous coccidiostats are used in ruminants, which can be used in camels. Salinomycin / monensin should not be given to camels. It may cause poisoning (Chaudhry et al. 1998; Wernery et al. 1998). It is also important to give fluid therapy to take care of dehydration. Sanitary conditions at the farms should also be taken care of.

CRYPTOSPORIDIUM

Cryptosporidiosis (Cryptosporidium muri) can also cause diarrhea in young camels. It is difficult to be seen microscopically in fecal preparations. Staining by Zeil Nielson may reveal the protozoan in fecal smears.

SARCOCYSTIOSIS

Sarcocystis species belong to genus Coccidia. It has an interesting two-host life cycle. The primary host is carnivore, in which gametogony, fertilization, and sporulation occurs. Camels act as intermediate host. Sarcocytis cameli is the only species of Sarcocystis reported from camel (Hussein, 1991). Ghaffar et al (1979) have suggested, on the basis of ultra structural studies, that more than one species of Sarcocytis may infect camels. The camels become infected after the ingestion of sporulated oocysts passed in the feces of carnivores, which are the final hosts. The sporocysts rupture in the gut of camels and release sporozoites. The sporozoites multiply in the gut wall and eventually migrate to the striated muscles to form the characteristic sarcocysts. The lifecycle is completed when the sarcocyst containing infective bradyzoites is ingested by the final host which, in the case of S. cameli has been shown to be the dog (Hilali and Mohammed, 1980; Kuraev, 1981). After a period of multiplication involving asexual and sexual reproduction sporulated oocysts are passed in the feces of the final host.

Clinical Signs

No signs may be noted in light infections. In high numbers, the schizonts in the endothelial cells may produce acute febrile disease, resulting in abortion and death. The presence of sarcocysts in the muscles may interfere with muscle functions. Fatani et al. (1996) inoculated camels with sarcocysts of dog origin and recorded symptoms and pathological changes. They observed high fever and anemia, and suggested that some unknown toxic factors or metabolites released from schizonts may be responsible for this. They also noticed decreased total serum proteins, albumin and globulins.

Infection with S. cameli is often not pathogenic, being diagnosed while making postmortem examination (Hilali et al., 1995). The oocysts are usually seen in the muscles of the heart, diaphragm and esophagus. El-Etreby (1970) found nonsuppurative, focal, interstitial myocarditis in camels infected with S. cameli. Infection rate of 25% has been reported by Sherkov et al. (1976) in

Jordan, 52% by Rahbari et al., (1981) in Iran, 52% by Kuraev (1981) in Kazkastan and 8% by Ginawi and Shommein (1977) in Sudan. Occurrence of Sarcocystis in camels has been also reported from India (Rao et al., 1997).

Treatment

No specific treatment is suggested. One can try sulfa drugs.

TOXOPLASMOSIS

Toxoplasmosis is a contagious disease of all species of animals including camels. Clinically it is manifested by abortions and stillbirths in ewes and in all species by encephalitis, pneumonia and neonatal mortality (Mc Cabe and Chirurgi, 1993). The most common method of spread in ruminants is the ingestion of feed contaminated by cat feces, which contains infective oocysts. Toxoplasmosis is a true zoonosis occurring naturally in man and in domesticated and wild animals and birds (Buxton, 1993). The latter act as intermediate host, in which the protozoan under goes a period of asexual multiplication, resulting in the production of long-lived pseudocysts, in a number of host tissues. Both intermediate and final host acquire infection with T. gonadii by the ingestion of sporulated oocysts from the feces of the final host, ingestion of cyst contained in animal tissues, milk containing multiplicate forms or by transplacental transmission. Ingestion of pulmonary and nasal secretions from infected animals, particularly of rodents, is also thought to be a cause (Rifaat et al., 1964).

On the basis of serological examination, the incidence of toxoplasmosis is quite high, throughout the world (Beyer and Shevkunova, 1986). The surveys in Egypt revealed prevalence between 6% and 3% (Raffat et al., 1964). Sharma and Gautam (1974) reported infection rate of 11.19% in camels in India. Incidence of 65% was reported in camels by Kozojed et al. (1976) from Afghanistan. Hussein et al (1988) recorded incidence of 16% in Saudi Arabia and Afzal and Sakir (1994) observed the incidence of 30.9% and 36.4%, respectively, by direct agglutination and indirect haemagglutination in UAE. We found the incidence to be 18% in racing camels in UAE. The presence of such a high number of camels bearing antibodies against T. gonadii reflects the degree of contact between the camel and the final host. The symptoms associated with acute toxoplasmosis, however might be confused with those of other diseases, in particular Surra. Hilali et al. (1995) isolated tissue cysts of toxoplasma from camel meat from Saudi Arabia.

It was found by Chaudhary et al. (1996) that there was significant decrease in total WBC count and eosinophilia in camels bearing antibodies against T. gonadii. Manefield and Tinson (1996) reported one clinical case of a 6 years old camel showing signs of dyspnea and pyothorax. Twenty-four liters of turbid fluid was drained from the pleural cavity. Toxoplasma tachyzoites were found within macrophages in smears. The fluid had a titer of 1:20,000 for T. gonadii. It is speculated that T. gonadii may be a cause of abortion in animals.

Treatment

Administration of pyrimethamine and sulfadiazine together is reported to be highly effective in man and mice (Blood and Radostits 1989). Sulfadimidine, sulfamerazine and sulfadiazine are most effective amongst the sulfa drugs. The drugs are effective against the proliferating parasites in the acute stage of the disease but will usually not eradicate infection and they have limited activity on the organisms in tissue cysts.

As has been stated earlier sporulated oocysts in cat feces are the source of infection, so the control programs should be devised to eliminate the source of

infection. The risk of infection is more when the cats are young, as at this time they inhabit and defecate in hay, grains or other feed storage area. Fecal contamination of the pastures is also important. Risk of infection may be reduced by eliminating the cats from the farm environment.

GLOBIDIOSIS

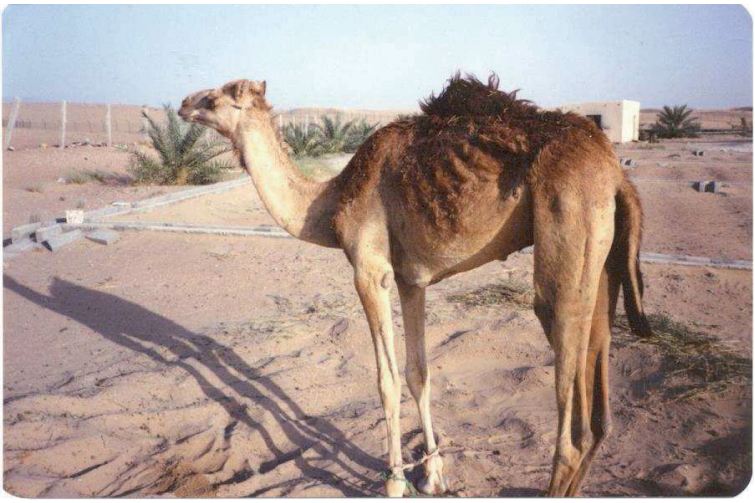
Globidium belongs to Genus Eimeria. It occurs as pinhead size cysts in the skin and alimentary tract of camels. Considerable thickening of the skin may be seen, especially over inside of the thigh (Manefield and Tinson, 1996).

TRYPANOSOMIASIS (SURRA)

Trypanosoma evansi causes the most serious protozoal disease of camels (Burn et al., 1998). It is an acute, subacute or chronic disease of horses and camels. It is characterized by fever, emaciation, edema, nervous signs and death. The incidence of the disease is more in wet season, when population of flies increases. Infection can also occur with T. brucei, T. congolanse and T. vivax (Luckins, 1992). The surra is prevalent in large areas of the world including Africa, Middle East, Asia and Central and South America (Llemobade, 1971). The protozoan is injected in the camels by the flies (Dia et al., 1997). The parasites multiply in the blood, and body fluids including cerebrospinal fluid. Trypanosoma evansi can also cause disease in cattle, buffalo, sheep and goats.

The disease in camels is usually chronic. Rarely it can be acute. Trypanosomes appear in the peripheral blood in high numbers initially, but thereafter, parasite numbers are often low and sometimes difficult or impossible to be detected. In chronic infections, parasites are very difficult to identify. The principal clinical signs are recurrent fever accompanied by a progressive anemia and poor body condition. Mostly the appetite is unaffected. Camels can die in advanced stage of emaciation. Chronic infection may lead to abortion, premature birth and inability to produce milk. Calves born after full term are usually very weak. The course of disease is variable among individual animals. Some die within few months of infection, others develop chronic disease lasting for years. Spontaneous recovery sometimes occurs. The most pronounced lesion in trypanosomiasis is anemia. Typically, it is macrocytic and hemolytic anemia (Raisanghanis et al. 1981). Accompanying this there is decrease in erythrocytes, an increase in lymphocytes, neutrophils, eosinophils and monocytes. Infection is accompanied by progressive changes in the serum protein concentration, with IgM levels increasing five-fold during the course of infection. Alterations in some of the enzymes have also been reported especially AST and ALT (Boid et al., 1985). No pathognomonic clinical signs of the disease have been reported.

It has been reported by Ouma et al. (1998) that there is a rapid increase (47%) in hemolytic complement levels, following infection with trypanosomiasis and activation of complement is responsible for immunodepression observed in trypanosomiasis. Nijiru et al. (1997) observed enhancement of phagocytic capacity of polymorphonuclear cells in dromedary camels during experimental infection with T. evansi. They also found reduction in respiratory burst and capability to adhere to the endothelial surfaces of polymorphonuclear cells, suggesting overall suppression of immune system.



Emaciation in a five year old camel suffering from chronic Surra

Diagnosis

Control of disease depends how efficiently the disease is diagnosed. Various diagnostic techniques for T. evansi are evaluated by Butt et al. (1998), Pathak et al., (1997) and Raisinghani and Lodha (1986).

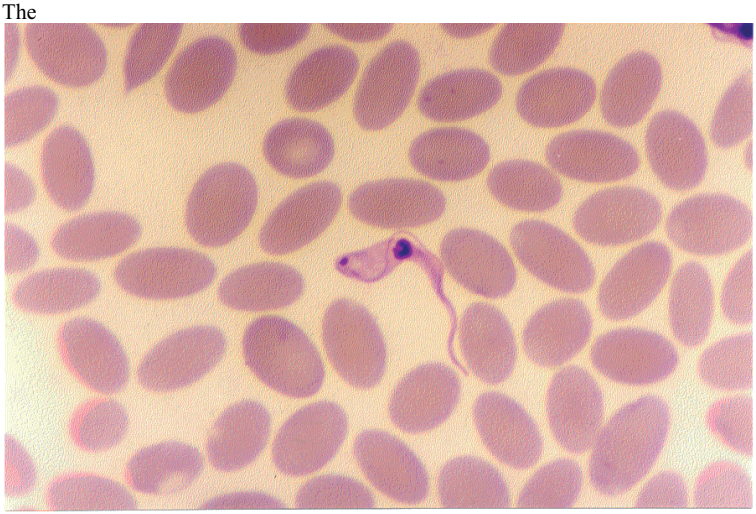
The simplest and most reliable technique for detecting trypanosomiasis is demonstration of trypanosomes in the blood of infected camel. However, this method can only detect 60% of the infected animals. Centrifugation may help to concentrate the blood increasing chances of detection of trypanosomes.

The number of trypanosomes present in the peripheral blood of infected animals is affected by many factors. Less number of circulating trypanosomes are present in animals recovered from acute disease and in chronically affected animals. Physiological stress increases parasitemia. Localization of trypanosomes in the tissues result in decreased parasitemia, which usually cause failures in detecting parasites in smears made from peripheral circulation.

Inoculation of laboratory rodents with blood from camels suspected of harboring trypanosomes is a very sensitive technique for detecting non-patent infection.

A number of serological diagnostic methods have been developed to detect humoral response against trypanosomiasis. Although these techniques are very sensitive but are not able to differentiate active infection and post infection situation, as the antibodies persist in the circulation even after three months of successful treatment of trypanosomiasis. Increase in the levels of globulins, which occur in trypanosomiasis, can be detected by flocculation assay e.g. formal gel test and mercury chloride test (Chand and Singh, 1970). These tests are non-specific and are inconsistent. The complement fixation test was one of the first tests used to diagnose surra. But this test never gained popularity being very complicated. Indirect haemagglutination also had the same limitations. Indirect fluorescent technique is also being used with similar inherent difficulties. With the availability of enzyme-linked immunosorbent assay (ELISA) there has been a great success in the diagnosis

of camel trypanosomiasis. The test is easy, specific and sensitive (Rae et al. 1989). The



Trypanosoma evansi in blood smear

invention of monoclonal antibody technology has been a major breakthrough in the diagnosis of trypanosomiasis. There are no chances of cross reactivity between the other species of trypanosomes or other hematoparasites (Nantulya et al., 1989).

Recently developed tests that detect circulating trypanosomal antigens (Rae and Luckins, 1984; Nantulya, et al.,1989; Ohalo-Mukani, 1989) have significantly improved the diagnosis of trypanosomiasis. These tests are more sensitive than conventional parasitological ones and the current infection can easily be distinguished from cured ones. Latex agglutination test is now commercially available to detect trypanosomal antigens (Nantulya, 1994). Diagnosis of Trypanosoma evansi from dromedary camels has been attempted by isoenzyme electrophoresis (Al-Taqi, 1989). Diagnosis of trypanosomiasis is also being practiced by smelling the odor of urine by Baduoin (Hunter, 1986). Experiments are underway to establish the sensitivity of PCR for the detection of T. evansi in animals (Ijaz et al., 1998; Diall et. al., 1993). Demonstration of antibodies against trypanosome tyrosine amino-transferase for the diagnosis of trypanosomiasis may also be helpful (El-Sawaly and Seed, 1998).

Treatment

Toxicity of trypacidal drugs and development of resistance by the trypanosomes against these drugs is a challenge to veterinarians (Pathak et al., 1997). Comparatively new drug melarsenoxyde cysteamine (Cymelarsan), which is trivalent arsenical gives good response against trypanosomiasis in camels (Nyang'ao et al., 1995). The dose of this drug is 0.25 mg / kg body weight given as intramuscular injection. Quinapyramine sulfate (Trypacide) or quinapyramine sulfate plus chloride (Trypacide prosalt) are also being used with varying success (Wesongah et al., 1997). Berenil which, is being used in other species with success is reported to be toxic to camels, hence not recommended in this species.

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HELMINTHIC PARASITES

Outbreaks of clinical helminthiasis are not frequently observed in camels. Camel racing is an important sport in the Arabian Gulf, performance may not be achieved if the camels are stressed due to parasitic infestations. Although the climatic conditions in the areas, where most of the camels habitat, are not ideal for the survival of parasitic larvae or ova, it is not uncommon for the camels to harbor helminthic parasites.

El Bihari (1985) has done a good review on helminthiasis in camels. Many studies on the prevalence of helminthic infestation in camels have been published (Steward, 1950; Selim and Rehman, 1972; Altaif, 1974; Daynes and Richard, 1974; Burgemeister et al., 1975; Barus et al., 1976; Lodha and Raisinghani, 1979; El-Bihari and Kawasmch, 1980; Fadl et al., 1989; Kayum et al., 1992; Sharrif et al., 1997; Hayat et al., 1998).

Prevalence rates of various species of helminths vary widely from region to region as well as from season to season within the same region. Usually mixed infestation with several species at a time is encountered.

NEMATODES

Nematodes are the most common of the camel parasites. A large number of the nematodes in camels are located in the gasterointestinal tract. Majority of GI tract parasites produce a protein-losing gastro-enteropathy. In severe cases, hypoalbuminemia may develop (Partani et al., 1998). Gill (1972) emphasized on zoonotic importance. Ghadirian and Arfan (1973) have observed zoonotic infestation due to Trichostrongylosis.

Nematodes are non-segmented usually cylindrical and elongated worms. They possess alimentary canal and usually are separate sexes. Their life cycle may be direct or require an intermediate host.

Three families of nematodes commonly affecting camels are:

1). Trichostrongyloidea: Short esophagus. Buccal capsule absent or weakly developed.

2). Strongyloidea: Short esophagus. Well developed buccal capsule present.

3). Trichuroidea: Very long esophagus extending at least 1/3 of body length.

Family Trichostrongyloidae

Mostly small forms in which the buccal capsule is absent or very small and is devoid of leaf-crowns, and usually bears no teeth. The male bursa is well developed, with large lateral lobes and small dorsal lobe.

GENUS HAEMONCHUS

Members of this genus possess a small buccal cavity with slender tooth or lancet; cervical papillae are prominent; the bursa is large; especially the lateral lobes, and dorsal lobe is small and asymmetrical in this genus. The vulva is posterior in the

female and knobes, flaps or linguiform processes are present or absent in the vulvar region. The following species of this genus are found in camels:

Haemonchus longistipes: This is blood sucking nematode, which habitats abomasum of camels (Selim and Rehman, 1972; Altaif, 1974; El-Bihari and Kawasmch, 1980; Arzoun et al., 1984). The female of this parasite possesses well developed linguiform vulval flap, which is knob like in H. controtus females. According to Arzoun et al. (1984) the camels infested with H. longistipes show emaciation, anemia, edema of the lower limbs, eosinophilia, hypoproteinaemia, hyperglobinaemia, and elevated blood urea nitrogen. They also found sand in the rumen of camels infested with this parasite at necropsy. It has been reported by El Bihari and Kawasmel (1980) from Saudi Arabia. The egg production by this parasite peaks from October to February i.e. during the rainy winter season.

Following ingestion, infective L-3 larvae penetrate the intestinal mucosa, where they molt to L-4 and L-5. L-5 larvae, leave the mucosa and become adults in the lumen of the small intestine. The prepatent period is 15 days under optimal circumstances. Adult parasites live for only a few weeks. When the larvae are embedded in the intestines, they may cause enteritis and diarrhea.

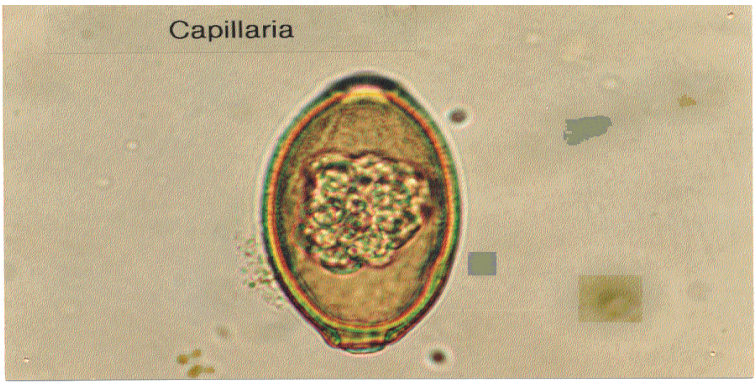
Haemonchus contortus: It is occasionally found in the abomasum of camels which are kept in close association with cattle, sheep, and goats.

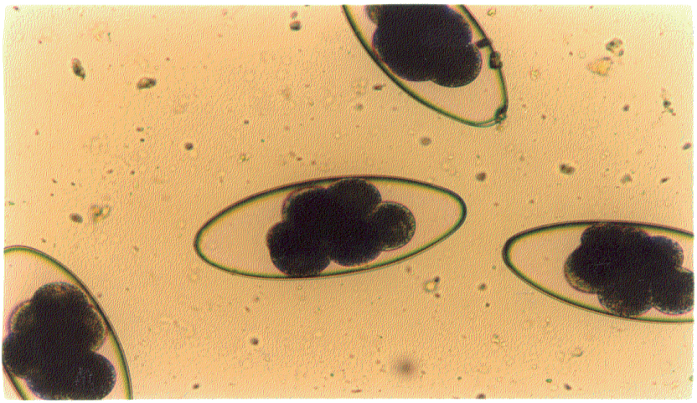
GENUS NEMATODIRUS

The anterior part of the body is thinner than the posterior part. The male bursa has elongated lateral lobes which are covered internally by rounded or oval cuticle bosses, while the dorsal lobe with its supporting rays is split in two and each half is attached to the lateral lobe. Dorsal lobe is symmetrical. The tail of the female is short and truncate, with a slender terminal appendage. The vulva opens at the posterior third of the body. The eggs are so large of this genus that their size readily distinguish them from those of other trichostrongyl species. Eggs passed in the feces contain about eight cells. The following species of genus Nematodirus have been reported from the small intestines of camels.

Nematodirus spathiger: Adults of this nematode are located in the small intestines. Eggs are passed in the feces and undergo slow development, over 2-3 months.
Other species of genus Nematodirus found in the small intestines of the camels are the following:

Nematodirus helvetianus
Nematodirus abnormalis
Nematodirus mauritanicus





Nematodirus ova

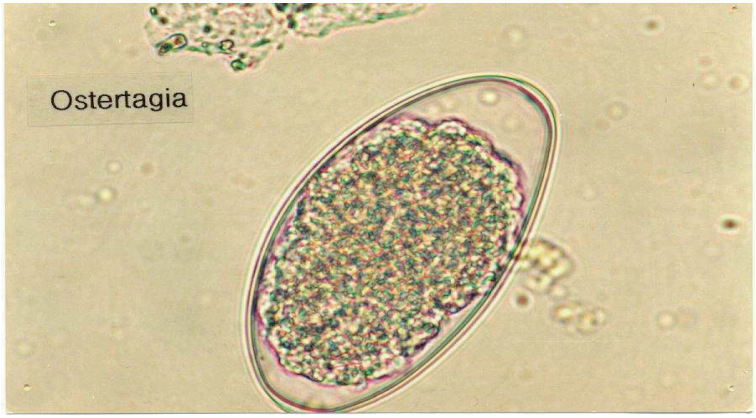
GENUS CAMELOSTRONGYLUS

This genus resembles Ostertagia except that the bursa has two large lateral lobes; the spicules are equal, long, narrow, and denticulated.

Camelostrongylus mentulatus: It is a common worm of abomasum in camels of the Middle East. The life cycle is similar to that of Ostertagia. The eggs are 75-85 x 40-50 um. The infestation produces morphological and functional changes in the abomasum similar to those caused by Ostertagia.

GENUS OSTERTAGIA

The species of this genus occur in the abomsum and small intestines of camels, sheep, goats, cattle and other ruminants. They are commonly known as brown stomach worms, because of their color when fresh.



Ostertagia circumcincta: This species is occassionally found in camels. Mostly it occurs in the abomasum of sheep and goats. Other species of the genera occasionally seen in camels are **O. ostertagi**, and **O. lyrata.**

There are two types of life cycle in this species. Type I is typical of a trichostrongyl life cycle, in which the larvae mature without passing through stage of arrest. In type II cycle larvae become arrested in the mucosa of the stomach, for a few months, where they form greyish white nodules. There is only one larva in each nodule.

GENUS MARSHALLAGIA

It is found in the abomasum and rarely in the duodenum of camels, sheep, goats, and wild ruminants. Wild ruminants serve as an important host of the parasite.

The worms are slender. The cuticle of the anterior extremity may be slightly inflated, transversely striated, and the head is not more than 25 um wide. The rest of the body cuticle bears 25-35 longitudinal ridges and has not transverse striations. The male bursa has lateral and dorsal lobes and an accessory bursal membrane situated anteriorly on the dorsal side. The spicules are pigmented brown, relatively short, and end posteriorly in two or three processes. A small anterior flap may cover the vulva of the female.

Marshallagia (Ostertagia) marshalli: It has similar life cycle as of Ostertagia. When ingested, the larvae penetrate into the gastric mucosa of abomasum and

produce nodules 2-4 mm in diameter. Nodules, in this species, harbor more than one larvae, which mature in 15-18 days. There may be arrested development in this species also. The eggs of this species are large and frequently confused with Nematodirus species eggs.

GENUS TRICHOSTRONGYLUS

The species of this genus are small, slender, pale reddish brown without a specially developed head-end. There is no buccal capsule. The excretory pore is usually situated in a conspicuous ventral notch near the anterior extremity. The male bursa has long lateral lobes, while the dorsal lobe is not well defined.
The spicules are stout, ridged and pigmented brown, and a gubernaculum is present. The eggs are oval, thin shelled and segmented when they are laid.

Trichostrongylus probolurus: Occurs in the small intestines of camels and other ruminants and accidentally, in man. Spicules are equal and 0.126-0.134 mm long.

Trichostrongylus colubriformis: Found in the anterior portion of the small intestine and some times also in the abomasum of camels and other ruminants and antelopes. Males are 4-5.5 mm long and female 5-7 mm. Spicules equal, 0.135-0.156 mm long. The eggs measure 79-101 x 39-47 um.

Trichostrongylus vitrinus: T. vitrinus have been reported from intestines and rarely abomasum of camels, small ruminants, pigs, rabbits, and man from all over the world. Spicules are equal and 0.16-0.17 mm long. Eggs measure 93-118 x 41-52 um. Males are 4-7 mm long and females 5-8 mm.

GENUS COOPERIA

Cooperides are relatively smaller trichostrongyles and live in the small intestines of ruminants and camel throughout the world. The life cycle and epidemiology are similar to those of Trichostrongylus sp.

Cooperia onchophora: It occurs in small intestines of camels, cattle, sheep, and rarely in horses. Males are 5.5-9 mm long and females 6-8 mm. Spicules are 0.24-0.3 mm long.

Cooperia pectinata: It has been reported in small intestines of camels, cattle and rarely in sheep. The male is 7 mm long and the female 7.5-9 mm. Spicules are 0.24-0.28 mm long.

GENUS NEMATODIRELLA

This genus resembles to Nematodirus. The anterior parts of the body are narrow; the spicules are equal, very long, up to half the length of the body. Three species have been reported in ruminants: N. longispiculata, N. cameli, and N. dromedari. The eggs of all of these species are large, 230-270 by 110-140 um. Nematodirella dromedari: Lodha and Raisinghani (1979) have reported occurrence of this species in camels.

Life cycle of Trichostrongyloidae

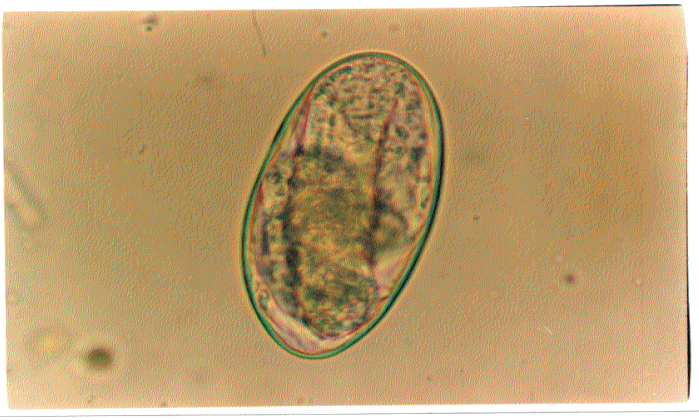
The eggs which are passed in the feces of the host are strongyle-like, being thin-shelled and in the 8 to 32 cell (blastomere) stage. Thus the infective third-stage larva is produced in four to six days under optimal conditions (27ºC, O_2, H_2O). Migration of larvae on blades of grass occurs when light intensity is about 62 foot-candles; moisture favors migration but more than 0.12 ml of water per square

centimeter of soil hinders movement. Consequently the greatest numbers of larvae are on blades of grass in the early morning and early evening when temperature, humidity and light intensity are favorable.

Infection is by ingestion of infective larvae on herbage. Following ingestion of infective larvae the development and survival of the free-living stages of the Trichostrongylus species is dependent on the weather and on pasture conditions. In general, the infective stage is produced in four to six days under optimal conditions at 27ºC. The minimum temperature for development is between 10 and 15ºC. The most rapid development occurs in summer with peak larval burdens on pasture being reached in six to eight weeks, resulting in heavy infestations.

Family Strongyloidae

Members of this family exist both as parasitic and free living. Parasitic generation have markedly elongated cylindrical esophagus and are heterogenic free living generation possesses esophagus with valvulated pole.



Strongyloides ova

GENUS STRONGYLOIDES

The parasitic forms of this genus are parthenogenetic. Their eggs may give rise, out side the host, directly to infective larvae of another parasitic generation or to a free-living generation of males and females. The esophagus in the free-living generation is rhabdiform. The vulva is near the middle of the body, the eggs are few but large, and have thin shells. This nonparasitic generation produces a parasitic generation. The esophagus of parasitic generation is not rhabditiform but is cylindrical, without a posterior bulb. The infective larvae of the parasitic generation are able to penetrate the skin of the host and pass with the blood to the lungs, thence up the trachea to the pharynx and on to the intestines.

Strongyloides papillosus: Occurs in the small intestines of sheep, goats, cattle, camels, and other ruminants. It is 3.5 to 6 mm long and 0.05-0.06 mm thick. The eggs have rather blunt ends and thin shells. They measure 40-60 x 20-25 um and contain fully developed embryos when passed in the feces of the camel.

Family Trichuroidae

Members of the family are medium to large worms, the posterior part of the body being much thicker than the anterior. Genus of importance is Trichuris.

GENUS TRICHURIS

The eggs reach the infective stage after about three weeks under favorable conditions; however, development may be much more prolonged at lower temperatures (e.g. 6-20ºC), since development is related to soil moisture and temperature. Infective eggs may remain viable for several years. The host acquires the infection by ingesting the eggs, and the larvae penetrate the anterior small intestine for two to ten days before they move to the cecum where they develop to adults.

The worms belonging to this genus are generally known as "whip-worms", since the anterior part of the body is long and slender, while the posterior part is much thicker. The hind end of the male is curled and there is one spicule surrounded by a protrusible sheath, which is usually armed with fine cuticular spines. The vulva is situated at the beginning of the wide part of the body.

Trichuris ovis: The male is frequently seen in the cecum of camels and other ruminants. Trichuris ovis is 50-80 mm long; the anterior end constitutes three quarters of the length. The female is 35-70 mm long, of which the anterior end forms 2/3 – 4/5. The fully evaginated spicule is 5-6 mm long. The sheath bears an oblong swelling a short distance from its distal extremity and is covered with minute spines, which decrease in size towards the distal extremity. The eggs are brown, barrel shaped, with a transparent plug at either pole and measure 70-80 x 30-42 um, including the plugs. They contain an unsegmented embryo when laid.

Trichuris globulosa: Seen in the cecum of camels and other ruminant species. The male is 40-70 and female 42-60 mm, the anterior part constituting about 2/3 –3/4 of the length. The spicule measures 4.2 – 4.8 mm and its sheath bears a terminal, spherical expansion on which the spines are larger than on the remaining portion. The eggs measure 68 x 36 um and are similar to those of T. ovis.

Trichuris cameli: It is found in the large intestines of camels.
T. tenuis, **T. raoi**, and **T. skrjabini** have also been reported in camels.

Some other nematode parasites of relatively less significance which have been reported in camels are:

Physocephalus cristatus: This nematode belongs to family Thelaziidae. Eggs of the parasite are passed in the feces of the host where they are swallowed by caprophagous beetles. Large number of beetles are known to act as intermediate host. The larvae develop in these to the infective stage in about 28 days. Camels become infested by eating these beetles. They inhabitat abomasum of camels.

Chobetia ovina: This nematode belongs to family "Trichonematidae". It occurs in the large intestines of camels and other ruminants. Males are 13-14 mm long and females 17-20 mm long. The anterior end is curved slightly ventral and the large buccal capsule opens anterio-ventrally. The oral aperture is surrounded by a double row of small cuticular elements representing the leaf-crown. There is a shallow ventral cervical groove, and anterior to it is a slightly inflated cephalic vesicle. The male bursa is well developed and the spicules are 1.3 – 1.7 mm long, with a

gubernaculum. The vulva of the female opens about 0.4 mm from the posterior extremity. The eggs measure 90-105 x 50-55 um. Life cycle is direct.

Oesophagostomum spp. These belong to family "Tichonematidae" and are the nematodes of rare occurrence in camels. Members of this genus have a cylindrical buccal capsules, usually narrow. Leaf – crown are present. There is a ventral cervical groove near the anterior end, anterior to which the cuticle is dilated to form a cephalic vesicle. They inhabit large intestines of camels. These nematodes are often referred to as nodular worms, owing to the fact that several species cause nodule formation on the walls of the intestines.

Bunostomum spp. These parasites belong to family Ancylostmatidae and sub family Necatorinae. Two species of this genera have been reported rarely in camels; B. trigonocephalum and B. phlebotomum. The former parasite is a hook worm which occurs in the small intestines (the male is 12-17 mm long and female 19-20 mm). The latter species also habital small intestine (the male is 10-18 mm long and female 24-28 mm. The eggs measure about 106 x 46 um. They have bluntly rounded ends and darkly pigmented embryonic cells.

Pathogenesis of Gastrointestinal Nematodes

The pathology caused by gastrointestinal parasites is varied. Infection with Ostertagia spp. is associated with morphological and functional destruction of the gastric glands of the abomasum. The primary pathology caused by Haemonchus contortus is marked hemorrhage through wounds in the abomasal mucosa. Infestation with as Trichostrongylus spp. and Nematodirus spp. cause villous atrophy and adults of Oesophagostomum spp. in the large intestine cause ulceration and hemorrhage.

In general, anorexia and thus reduced feed intake are observed in parasitized animals. There is extensive proliferation of epithelial cells in the parasitized gastrointestinal tract, which results in replacement of differential functional cells with immature non-functional cells with imperfectly formed intercellular junctional complexes. This results in leakage of macromolecules through the mucosa into the intestine. Increased urea synthesis, possibly metabolized from ammonia arising from the deamination of amino acids, elevated levels of plasma urea and increased excretion of urinary nitrogen contribute to the inferior nitrogen balance of the parasitized animals (Soulsby, 1982).

Absorption of calcium and phosphorus is depressed, causing in turn, arrested skeletal development in the young camels. Selenium uptake is also retarded. Because of the debilitating effect of parasitism the body loses the ability to resist minor infectious agents, and a secondary infection may take the animal's life. Animals infested with helminths show inappetence, leading to anorexia and combined with poor food utilization, become emaciated (Fahmy et al., 1983). Anemia may be seen in animals with loads of parasites (Arzoun et al., 1984), even with parasites that are not blood suckers. The cutting mouth parts used for attachment may result in leakage of plasma and cells from capillaries. The signs of the disease are usually an expression of a mixed infestation, and are evident probably only when a large number of worms are present (Partani et al., 1998). El-Abdin et al (1975) reported increase in some of the enzyme activities of nematode infested camels.

CESTODES (TAPEWORMS)

The tapeworms, which infest camels, belong to class "Eucestoda" order Anoplocephalidea and family Anoplocephlae. Two genera have been reported to

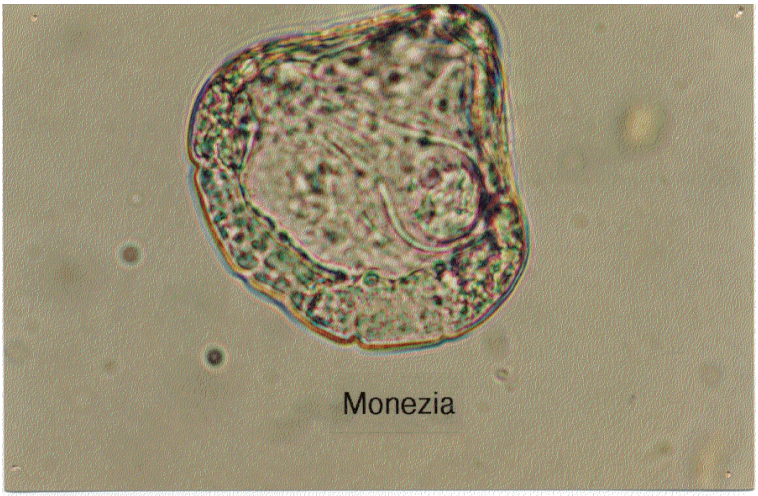
infest camels, i.e. Monezia and Stilezia (Kasim and Shawa, 1984; Rao and Sharma, 1995).

Mature proglottids separate from the rest of the tapeworm and, along with the eggs, are passed with the feces. Cysticercoid development occurs in oribatid mites, which are a true intermediate host. Infective stages develop in approximately 4 months. Camels acquire the infection from the mites. Further maturation to the adult tapeworm in the small intestines takes 37-40 days (Soulsby, 1982). Heavy infestation impair nutrition and cause debility or even intestinal obstruction. There may be a seasonality of infestation, depending upon degree (Prokopic et al., 1976).

Monezia expensa: Many workers, throughout the world have recovered Monezia expensa from camels (Kasim and Al Shawa, 1984). An intact tapeworm may reach 600 cm in length and be 1.6 cm wide. The scolex is 0.36-0.8 mm and has prominent suckers and no hooks. The eggs are somewhat triangular in shape, containing a well developed pyriform apparatus and measure 56-67 um in diameter.

Stilezia vittala: This anoplocephalid is very common in the intestine of Arabian camels.

Stilezia hepatica: It occurs in the bile ducts of sheep, goats, cattle and wild ruminants and rarely in camels. Adults are 20-50 cm long and up to 3 mm wide. The scolex has prominent suckers and there is a broad neck. The proglottids are short. The genital organs are single and the genital pores alternate irregularly. The uterus is long transverse and dumbell-shaped and the eggs pass into two paruterine organs,



each containing about 30 eggs. The ovoid eggs have no pyriform apparatus. They measure 26 x 16-19 um. The life cycle is not known, but may involve mites.

TREMATODES

The prevalence of trematodes is very low in Arabian penisula, however, Fasciola hepatica has been reported in many countries by various workers. High prevalence of F. hepatica have been reported by Magzoub and Kasim (1978) in the Eastern province of Saudi Arabia. Occurrence of paramphistoma has also been reported in camels by Sey (1977).

The circulatory system may be infested by Trematodes. Schistosoma bovis, S. indicum, S. nasalis, and Ornithobilhargia turkestanic have been reported infesting camels (Manefield and Tinson, 1996).

Schistosoma bovis: It can occur in the portal and mesenteric veins of camels and some other species of animals. The males of S. bovis are 9-22 mm long and 1-2 mm wide, depending on the degree of folding of the lateral edges. The female is 12-28 mm long . The suckers and the body of the male behind the suckers are armed with minute spines, while the dorsal surface of the male bears small tegumental tubercles.

Schistosoma indicum: It occurs in portal and mesenteric veins of camels, ruminants, and equines in India and Pakistan. Adult measures 5-22 mm and the eggs in the feces measure 57-140 X 18 – 72 um.

Schistosoma nasalis: Adults measure 5-11 mm in length. Eggs are boomerang shaped and 350-380 by 50 –80 um. The parasite develops in the veins of the nasal mucosa of camel and other animals in Pakistan and India (Soulsby, 1982). Adult parasites cause dilatation and thrombosis of the vein. The nasal mucosa is studded with granulomas and abscesses containing eggs.

The life cycle of Schistosoma species include:

The ovigerous female penetrates deeply into the small vessels of the mucosa or submucosa of the intestines, lays eggs in the capillaries, from where the eggs pass through the intestinal wall into the intestinal lumen and out in the feces. When laid the eggs are not fully mature and they continue their development as they pass out in the feces. The eggs hatch after contact and dilution of the feces with water. Agitation ensure optimal hatching. Miracidia infect aquatic snails, which are the intermediate host. The miracidia penetrate the tissues of the snail and two generations of sporocystes develop, the second forming the cercariae, which emerge from the snail and swim about the water. Emergence of cercariae from the snail is periodic. Infection of the definitive host is through active skin penetration, although cercariae may penetrate the walls of the rumen when swallowed with water. The cercariae transform into schistosomula, which are transported to the lungs via the circulation within 4-7 days. They are then carried to the liver, presumably via the blood stream, and from 8 days onwards schistosomas are found in the portal veins of the liver. Pairing of the worms takes place in the portal veins before they leave the liver to reach maturity in the mesenteric veins.

Praziquantil seems to be the drug of choice, 60 mg/kg as single dose or three doses of 20 mg/kg given 4 hours apart. Niridazole, Hycanthone, Oxamniquine, furaprodieum, tartar emetic, antimosany and stibophen can also be given.

Ornithobilharzia turkestanicum: Occurs in the mesenteric veins of camels and other animals in Russia, Turkestan, Kazakstan, Mongolia, Iraq and France. Intermediate host is snail (Lymnaea euphratica). The length of male is 4.2 – 8 mm and that of the female 3.4 – 8 mm. The eggs measure 72-77 X 18-26 um (Soulsby, 1982).

Eurytrema pancreaticum: It is of rare occurrence in camels and resides in the pancreatic ducts and sometimes in the bile ducts and duodenum. It measures 8-16 x 5-8.5 mm. Snails and grass hoppers are intermediate hosts.

Different Methods of Fecal Examination for Detection of Gastro-Intestinal Parasites

Unless adult parasites have already been seen in the feces, fecal examination should be conducted to begin the process of diagnosis. Methods of fecal examination include direct smear, sedimentation, Mc Master, Lanes floatation, and Willies floatation (Soulsby, 1982). According to Kayum et al. (1992) the Willis floatation is the most suitable method for the detection of gastro-intestinal nematodes in camel feces, however, Blantidium species can only be seen in the direct smears and sedimentation method. Hence they recommend Willis floatation method for routine fecal examination, supplemented with direct smear examination of diarrhea samples. Making eggs count is good to suggest therapy.

Treatment and Control

Information on the treatment of gastrointestinal nematodes in camels is extrapolated from experience of cattle and small ruminants (El-Bihari, 1985). Trials with some drugs have been done by El Molla et al. (1981), Lodha et al. (1977), Michael et al. (1980), Selim et al. (1983), Saleh et al (1987), Sharrif et al. (1997), Partani et al (1995). The latter workers have observed some variation in the effectiveness of some anthelmintics. They studied the efficiency of 5 anthelmintics and found Ivermectin to be most effective drug in camels (Boyce et al., 1984) followed by albendazole, levamisole, fenbendazol and tetramisol. Albendazol and fenbendazole did not show useful effect against Trichuris, but levamisole and tetramisole were effective against this parasite. Morantel tartrate also gives good results in camels. The parasites can often acquire resistance against the drugs. Care must be taken to avoid frequent use of and under dosage of drugs. The therapy should be given only to those animals having sufficient egg counts. It is also useful to change the drugs .

Praziquanet (Droncit) at a dose of 2.5–10 mg/kg, given orally or as an injection may be effective treating tapeworm infestation. Fenbendazole at 10-15 mg/kg is effective in cattle and sheep.

EXTRA GASTROINTESTINAL PARASITES

ONCHOCERCARIASIS

Three species of Onchocerca have been reported from the camels. The species of genus Onchocerca are elongated filariform worms. They live in the connective tissue of their host often giving rise to firm nodules in which they lie coiled up. The cuticle is transversely striated and, in addition, bears characteristic spiral thickenings which are usually interrupted in the lateral fields. Microfilariae are found in the skin, in lymph spaces, and connective tissue spaces. The intermediate hosts are insects of the families Simuliidae and Ceratopogonidae.

Onchocerca fasciata: have been reported by Bain and Nasher (1981) in camel populations. According to Cheema et al. (1984) 20% of 478 camels had subcutaneous and nuchal nodules of this parasite. The disease is characterized by the presence of nodules (worm nests) in the subcutis (head and neck region) and less often in the fascia of the nuchal ligament. The nodules contain live degenerated or calcified worms and inflammatory cells. The microfilaries, 245-294 um long are always in the skin of the head and neck regions; they are particularly abundant in the

anterior crest of the neck and around the lower eye lids. The vector of O. fasciata is unknown but Cuticoides species may play a role in transmission since black flies, the vector of Onchocerca species are few in camel habitats.

Onchocerca gutturosa: (El Sinnary and Hussain, 1981). It is of rare occurrence and is found in the ligamentum nuchae and other parts of the body such as on the scapular cartilage and in the hip, stifle, and shoulder regions. Holdsworth and Moorhouse (1985) have reported it from Australia. Males are 2.9-3.0 cm long and females 60 cm or more. The microfilariae, 200-230 um, are found in the skin of the host but the predilection site varies and they can be found virtually all over the body. The adult parasite are non pathogenic. The parasites occur in tunnels and nodules in the connective tissue, with unusually little reaction.

Onchocerca armilata: It is found in tortuous tunnels and nodules in the aorta. In early stages raised tunnels and a few nodules are seen. In old lesions walls of the aorta become thickened and the intima shows numerous tortuous tunnels and numerous nodules containing yellow caseous or slimmy fluid and coiled worms. In more chronic condition calcification of the nodules occur, the aortic wall becomes thinner and aneurysms may be seen (Cheema and Ivoghli, 1978). Adult parasite usually causes no clinical signs. But excessive number of microfilaraiae results in epileptic fits in cattle. El Mannan et al. (1988) reported vascular onchocerciasis in camels from Sudan.

There is usually no clinical evidence for the presence of microfilariae in the skin. However, seasonal (summer), sporadic dermatitis given various names such as allergic dermatitis etc. is seen throughout the world (Hussein et al, 1988). There is papular or exudative dermatitis with alopecia and severe pruritis. The microfilaria may or may not be present in this condition. Thus many authors consider this condition to be due to allergic reaction to the bites of Ceratopogonid or Simulid insects. It is possible that the etiology of dermatitis in animals is two fold, being associated with an immunological reaction to dead and dying microfilariae in the skin of some animals and with an allergic reaction to the insect bites in other animals.

Adult onchocerca in the ligaments can only be detected at postmortem examination. The parasites residing in the nodules in skin can be palpated. Microfilariae will be found in the subcutaneous lymph spaces. A skin biopsy taken from the site of predilection site will reveal microfilariae, if present. Fluid expressed from the affected lymph nodes may reveal the presence of microfilariae. The nodules of onchocerca must be differentiated from the nodules of tuberculosis (Michael and Saleh, 1977).

Diethylcarbamazine is microfilaricidal and is used both in man and animals at the dose rate of 5-8 mg/kg given daily for 21 days. Corticosteroides should be applied topically. Metrifonate suppresses microfilaremia and trichlorophon has also been used with success. Ivermectins are also valuable in treating onchocerciasis (both adult and microfilarae).

DIPETALONEMATOSIS

Dipetalonema evansi is an important hemoparasite of dromedary (El-Bihari, 1986; Butt et al. 1996). It has been reported from the pulmonary and spermatic arteries and lymph nodes of camels in Egypt, Far East, South East Asia, and Eastern USSR. This is a fairly large filarid, the male being 75-90 mm and the female being 170-215 mm. The microfilariae measure 250-300 um. Upto 80% of camels may be infected in the eastern republics of USSR and it is suspected that the mosquito Aedes detritus is an intermediate host. Butt et al. (1998) reported 12.5 –

14.5% incidence of microfilariae of Dipetalonema evansi in camels. Infected camels may suffer from arteriosclerosis and heart insufficiency. A parasitic orchitis may develop or aneurysms may be present in the spermatic vessels (Youssef, 1976).

The mature parasite may cause respiratory symptoms (dyspnea) when they are present in large numbers in the lungs. A large number of male and female worms collected together to form a golf-ball – sized mass in the right auricle of camel have been reported by Nagaty (1947). Saleh and Abdin (1982) have conducted biochemical studies in camels infested with D. evansi.

Diagnosis is made by examination of wet or stained thick film or by concentration of microfilariae in the blood by Knott's technique or similar suitable methods. The antigens of the parasites were used to diagnose the condition by Rifaat et al (1973).

LUNGWORMS

Dictyocaulus viviparous, D. filaria, and D. cameli have been reported occasionally infecting camels. The worms reside in trachea, bronchi and bronchioles. Incidence is reported to be more in bacterian camels than in dromedary. Dictyocaulus cameli is considered to be synonym of D. viviparus (Priadko and Baitursinov, 1984).

Dictyocaulus viviparus: It occurs in the bronchi of camel and some other ruminants throughout the world. The male is 4-5.5 cm long and female 6-8 cm. The eggs measure 82-88 x 33-38 um.

Dictyocaulus filaria: Reside in the bronchi of sheep, goats, camels and some other wild ruminants throughout the world. The male is 3-8 cm and the female 5-10 cm long. The worms have a milky white color and the intestine shows a dark line. The eggs measure 112-138 x 69 – 90 um.

The eggs may hatch in the lungs, but are usually coughed up and swallowed, and first stage larvae hatch while pass through the alimentary tract of the host. Some eggs may be expelled in the nasal discharge or sputum. The first stage larvae pass in the feces (0.55-0.58 mm). After one or two days the larva reaches the second stage, but does not cast the old cuticle until the third or infective stage is reached. Under ideal conditions infective stage is reached in 6-7 days. Infestation of the host occurs per os. Larvae penetrate into intestinal wall within three days and pass via the lymph vessels to the mesenteric lymph nodes, where they develop and perform the third ecdysis about 4 days after infestation. The worm can now pass via lymphatics and blood vessels to the lungs, where they are arrested in the capillaries and break through into the air passages. Development to maturity within the host takes 4 weeks. The life cycle of D. vivparous and D. cameli also resemble the life cycle of D. filaria.

Ivermectin, tetramisole, levamisol, albendazole, fenbendazole, oxfendazole, morantel, methylridrnic, diethyl carbamazine, and cycinacethydazine are used with varying degree of success. Wundersee and Tschener (1976) treated D. filaria infestation successfully by mebendazole.

EYEWORM

Parasites of genus Thelazia are found in the conjunctival sac or lachrymal duct of mammals and birds.

Thelazia leesi: Round (1962) has reported from dromedary in USSR and Asia. Its occurrence is very rare.

HYDATED DISEASE

Ecchinococcus granulosus: It is found in the small intestines of carnivore animals (particularly dog). Its intermediate state (hydated cyst) is found very frequently in man and large number of animals, including camels (Aboudaya, 1985; Abu Damir et al., 1985; Abdul Salam et al., 1988; Abdel Hafez et al., 1986; Afshar et al., 1971; Dada, 1978; El Ridi, 1983; Hassounah and Behbehani, 1976; Lodha et al., 1982).

Ecchinococcus granulosus passes eggs in the feces of the carnivores, they are immediately infective on ingestion by ungulates. The oncospheres penetrate the intestinal venules or lymphatics to reach the liver or lungs or some other organ (Mahamud, 1984). The hydated cysts develop slowly over several months. Hydatid cysts are commonly 5-10 cm in diameter or larger. A cyst 50 cm in diameter and containing about 16 liters of fluid has been recorded. The hydatid cyst is usually uniocular and is composed of a fairly thick outer concentrically laminated membrane. From this, brood capsules, each containing protoscolices, develop about five months after infection. At this time the cyst is infective for the definite host. The brood capsules may become detached and float free in the cyst fluid, being called hydatid sand. Occassionally daughter cysts develop with in the hydatid cyst. If a cyst gets ruptured, proscolices and brood capsules can develop into other external daughter cysts. External daughter cysts may also be formed if a piece of germinal membrane becomes enclosed in a laminated layer. The life cycle is completed when a dog ingests protoscolices. These evaginate, penetrate deeply between the villi into the crypts of Lieberkuhn and develop to maturity in about 47 days. Dogs may remain infested for about 2 years. Protoscolices are not produced by all of the hydatid cysts. Ones, which do not produce protoscolices, are sterile.

The prevalence of cysts in camels varies at different places. Rates upto 49% have been reported (Babero et al., 1963). It depends upon the association of camels with dogs. Mostly, the cysts are diagnosed at the time of postmortem examination. Al Abbasy et al. (1984) evaluated haemagglutination test for diagnosis of hydatidosis in camels. The condition is also being attempted to be diagnosed by DNA markers (Zhang et al., 1998). Treatment of pet dogs for tapeworms and eradication of stray dogs is indicated.

Coenurus cerebralis: It is the larval stage of tapeworm, Taenia multiceps of the dog. It has been also reported in camel (Burgmeister et al, 1975). It may cause neurological condition “gid”.

Cysticercus dromedarii has been reported to occur in camels by El Sergany et al (1970).

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