In veterinary medicine, a principal requirement for fluid therapy is in the treatment of neonatal diarrhoeal diseases. Restoration and maintenance of fluid and electrolyte balance, as well as energy support, are necessary considerations. Effective fluid replacement therapy for diarrhoeic neonates should fulfil several basic needs. First, the dehydration must be reversed and adequate fluid input provided to compensate for normal body water turnover plus increased fecal losses. From the standpoint of the animal’s requirement, the best therapeutic approach would be a continuous fluid input at a rate equal to body losses, a condition best provided by continuous intravenous administration. This is not a practical approach under many conditions. In some situations subcutaneous fluid administration can also be effective in providing a sustained fluid input, as subcutaneous fluids are slowly absorbed over a period of several hours.

Currently, the major emphasis on fluid and electrolyte therapy for diarrhoeic calves is focused on the use of oral fluids. A principle basis for this approach is the outstanding success of oral glucose electrolyte therapies for the treatment of *Vibrio cholera* diarrhoea in humans. Associated with this beneficial effect is the recognition that the mechanism by which *Escherichia coli* enterotoxin induces diarrhoea is analogous to the mechanisms of *V. cholera* as well as other enterotoxigenic bacteria. Increased secretory activity is responsible for the diarrhoea, yet absorption may continue and even be increased. However, the general assumption, that during diarrhoea absorption is normal or increased as is seen with many bacterial diarrhoeas, may be fallacious when considering the spectrum of diarrhoeal aetiologies. There is also speculation regarding the role of motility in diarrhoea. Both increased motility, as with parasympathetic stimulation or flaccid paralysis, creating an open tube, can conceivably cause more rapid transit and diarrhoea. Malabsorption of sugars and lipids are well recognized in diarrhoeal diseases. It could be anticipated that viral diseases which cause significant changes in both individual epithelial cells and in villus morphology.
would be associated with inhibited absorptive capacity. Most evidence supports the thesis that decreased absorption is the basis for viral diarrhoeas \(^1,^7,^26,^31\).

The use of oral antibiotics has been linked to malabsorption syndromes (diarrhoea) in domestic and laboratory animals, and in man. Several of these antibiotics are commonly used in veterinary medicine as prophylaxis or therapy of neonatal diarrhoeas. For example, oral chloramphenicol in calves can cause a malabsorptive diarrhoea with villus atrophy and epithelial cell dysfunction \(^39,^42\). When malabsorption causes the diarrhoea, particularly if glucose absorption is limited \(^31\), then oral therapies will be ineffective, and may actually exacerbate the diarrhoea by providing an increased supply of energy-rich substrate for bacteria, particularly in the large intestine. A secondary consequence of bacterial fermentation is the creation of excessive osmolality in the lower bowel. A number of the antibiotic-induced malabsorption syndromes in humans are associated with colonic overgrowth of *Clostridium difficile* and other Gram-positive enterotoxigenic organisms \(^20\).

**DIARRHOEAL LOSSES AND IMBALANCES**

Therapy composition should be based on several factors, a major one being the extent of fluid and electrolyte loss. It can be considered that, regardless of the route of administration, diarrhoeic animals that require fluid, electrolyte and energy support will have the same requirements. As animals become diarrhoeic they also become dehydrated. It is not uncommon to find that neonatal calves have lost 6–12% of their body fluids. Therefore, therapeutic provision of large quantities of water is necessary. Water losses are not evenly distributed through body water pools \(^35\). The greatest loss is from the extracellular fluid (ECF) and blood volume is most severely depleted \(^28,^36,^38\). Blood volume may be decreased as much as 50% causing peripheral vasoconstriction and hypovolaemic shock. The rest of the ECF is also decreased but not to the same extent \(^38\). Only small changes are seen in the intracellular water pool, and it may in fact be increased in volume \(^38\), presumably due to decreased cellular metabolism and cellular swelling \(^55\). The ionic composition of oral fluid replacement therapy should, therefore, be designed to approximate the composition of the ECF \(^35\). This approach is necessary so that crystalloids will remain after energy substrates are utilized, otherwise there will not be sufficient water retention.

Significant fecal losses of sodium, potassium, chloride and bicarbonate occur during diarrhoea \(^27,^33,^34,^49,^50\). Routine clinical assessment of electrolyte status is dependent on measurement of plasma or serum ion concentrations but these values are not necessarily indicative of fecal losses. Further, they may give false impressions of whole body electrolytes. This is particularly true for potassium which may be significantly increased in the blood, reaching cardiotoxic levels \(^19,^29,^30,^35,^41,^52\), yet a whole body potassium deficit occurs during diarrhoea (Figure 11.1) \(^19,^28,^52\). Potassium accumulation in extracellular fluids is a complex phenomenon associated with developing acidosis and cellular energy imbalances. Fecal bicarbonate loss, accumulation of lactic acid in the blood, and decreased renal function are all