

prevent the excess accumulation of interstitial fluid and development of frank edema.<sup>6</sup> For example, lymph flow increases with the increased filtration associated with increased capillary hydrostatic pressure. Thus a larger volume of fluid enters and is removed from the interstitial space. The interstitial protein concentration decreases as increased fluid flow washes protein out of the interstitial space. Reduced interstitial space protein concentration increases the oncotic gradient, inhibiting fluid efflux from the capillary, and decreases the rate of movement of fluid from the capillary to the interstitial space.<sup>6</sup>

### DIAGNOSTIC APPROACH TO THE PATIENT WITH EDEMA

Edema is not, of itself, a disease; rather, it is a sign of a disease process. Therefore, the diagnostic approach to the patient with edema is based on an understanding of the pathogenesis of edema and a knowledge of the diseases likely to be involved (Table 3-5). The diagnostic approach to an animal with edema should not be any different than for any other sign of disease. A clinical examination, including history and physical examination, will permit the development of a list of potential diagnoses and dictate the appropriate subsequent steps in confirming the diagnosis. The reader is referred to those sections of the text that deal with specific diseases for a description of the appropriate diagnostic aids.

When taking the history of a horse that presents with edema, one should focus on acquiring those facts that have the greatest diagnostic utility in differentiating among those diseases that have edema as a sign. Housing and season and geographical region should be considered. Vaccine and parasiticide administration should be determined. Exposure to other horses and diseases present within the herd should be ascertained. The duration of the edema, its distribution, and the presence of any other clinical signs should be questioned. The remainder of the history should be investigated depending on the responses to initial questions.

The physical examination should begin with a visual evaluation of the horse's attitude and physical condition. The temperature, pulse, and respiration should be recorded. While the physical examination should be complete, particular attention should be paid to those body systems that the preliminary examination indicated may be involved in the disease process. The physical examination will reveal the distribution and severity of edema. Edema that is localized to one extremity or is not bilaterally symmetric is more likely to be caused by local factors (e.g., lymphangitis or venous obstruction) than by systemic disease. Conversely, edema that involves several areas of the body and has a symmetric distribution is likely to be associated with systemic disease (e.g., the ventral edema of congestive heart failure).

Subsequent to the initial clinical examination, the clinician will have developed an ordered list of potential diagnoses. Confirmation, or elimination, of these diagnoses is dependent on subsequent diagnostic procedures, including the response to therapy. Refer to the sections of this text dealing with the specific disease processes for appropriate diagnostic procedures.

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## 3.4

### Changes in Body Weight

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An unwelcome or unexpected change in a horse's body weight is a commonly encountered problem in equine practice. Although obesity may be a more common problem, weight loss often represents a more serious situation, with potentially severe consequences. Normal or acceptable body weight is also in the eye of the beholder, because a horse with a given body weight might look overweight as an endurance horse, appropriate as a Thoroughbred racehorse, or too thin as a show hunter.

Whether dealing with a problem of weight loss or weight gain, the veterinarian should always investigate the feeding practices of the horse. It is not uncommon to have the owner

report that the horse is receiving 3 lb of grain twice daily when the actual measuring device (usually the everyday coffee can) differs in net grain weight once the volume of the measuring device and grain density are taken into account. It may be necessary to observe firsthand the feeding practices of the stable in order to document that the horse is actually getting the reported amount of grain two or three times daily. Hay should be examined for type, quality (color, texture, leafiness, steminess), mold, weeds, and potentially toxic plants. The horse in question should be observed eating both hay and grain to ensure that it really does consume the amounts reported by the owner or feeder.

Nursing foals should also be observed by the veterinarian when suckling. The mare's udder should be examined before and after nursing to ensure that the mare is actually producing sufficient milk and that the foal is actually nursing the mare completely until her udder is empty. The milk itself should be examined from both halves of the udder to see that it appears grossly normal (no evidence of mastitis). The foal's nostrils should be examined after nursing to determine the presence of milk reflux due to dysphagia, esophageal obstruction, or gastric reflux associated with gastrointestinal ulcers.

### DECREASED BODY WEIGHT

Losses in body weight are usually insidious and chronic in nature but may be surprisingly rapid in the face of acute overwhelming systemic infections (Table 3-6). Causes have been variously classified as gastrointestinal, nutritional, infectious, or hypoproteinemic.<sup>1,2</sup> Differential mechanisms include decreased feed intake, decreased absorption of nutrients, decreased nutrient utilization, and increased loss of energy or protein leading to a catabolic "sink."<sup>1-3</sup>

Decreased feed intake may be caused by management factors, poor dentition, dysphagia, or esophageal obstruction. Management factors leading to weight loss may be multifactorial and include inadequate amounts of feed, inadequate quality of feed, or inability of the horse to eat the proper amounts of the feed given. A horse with severe lameness (e.g., chronic laminitis) may not be able to ambulate to the feed source. A horse low on the pecking order in a pasture hierarchy may be unable to eat because it cannot approach the feed without the other horses bullying it and fending it away. The feed must be palatable and digestible. Appropriate amounts and types of concentrates must be fed considering the horse's work schedule or pregnancy status. Proper investigation of stable feeding practices is described earlier.

Poor dentition may cause the horse to fail to eat some or all of its grain or hay. Parrot-mouthed horses, or aged horses with receding incisor teeth (more than 25 years old), may have difficulty in tearing off grass when grazing. A horse with one or more oral sores from a poorly fitting bit or from sharp cheek teeth may exhibit partial or complete inappetance due to pain associated with chewing. Sharp cheek teeth, wavemouth, or stepmouth may lead to poor digestion and incomplete absorption of nutrients due to inadequate mastication of hay leading to poor fiber utilization during the hindgut (cecum) fermentation process.

Dysphagia has many causes, including abnormal prehension, chewing, or swallowing.<sup>4</sup> Abnormal prehension can be

**Table 3-6. Mechanisms and Differential Diagnoses for Decreased Body Weight**

Decreased Dietary Intake
Inadequate diet
Lameness
Pecking order
Poor dentition
Dysphagia
Esophageal obstruction
Maldigestion/Malabsorption
Lactose intolerance
Gastrointestinal ulceration
Parasitism
Diarrhea
Inflammatory intestinal disease
Granulomatous enterocolitis
Eosinophilic enterocolitis
Lymphocytic/plasmacytic enterocolitis
Gastrointestinal neoplasia
Inappropriate Hepatic Utilization
Inadequate Circulation/Respiration
Heart failure
Chronic obstructive pulmonary disease
Increased Rate of Protein/Energy Loss
Infection
Pneumonia
Pleuritis
Peritonitis
Equine infectious anemia
Protein-losing enteropathy
Diarrhea
Gastrointestinal ulceration
Parasitism
Inflammatory intestinal disease
Gastrointestinal neoplasia
Renal disease (glomerular)
Increased metabolic energy utilization
Chronic pain
Secondary hyperadrenocorticism

due to tongue lacerations; dental, mandibular, or maxillary fractures; damage to nerves supplying the tongue or facial musculature (local trauma, equine protozoal myelitis [EPM], polyneuritis equi); or central neurologic disease (EPM). Basal ganglia lesions due to poisoning by ingestion of yellow star thistle or Russian knapweed prevent normal prehension in the pharynx.<sup>5</sup> Swallowing abnormalities may be due to neurologic (EPM, viral encephalitis, guttural pouch infection), muscular, or physical obstructions such as strangles, abscesses, or guttural pouch distention.<sup>4</sup> Muscular causes include hyperkalemic periodic paralysis in Quarter Horse foals, vitamin E or selenium deficiency in neonates, botulism in neonates and adults, and local trauma subsequent to laryngeal surgery (laryngoplasty).

Esophageal obstruction usually presents acutely because an apparently dysphagic horse regurgitates food from its nostrils while attempting to eat or drink. Chronic choke, or anorexia related to painful swallowing due to partial esophageal obstruction, may lead to weight loss without the owner

realizing that the horse is not eating adequately. Esophageal endoscopy is usually diagnostic, but positive contrast radiography may be helpful and is sometimes necessary to establish an accurate diagnosis.

If the horse with weight loss has been observed to fully ingest adequate amounts of good-quality hay and grain, then decreased feed absorption must be considered to be a reason for weight loss. Maldigestion and malabsorption are not easily confirmed diagnoses, but tests based on luminal absorption of simple sugars (xylose or glucose tolerance tests) have been used to document malabsorption syndromes.<sup>3, 6, 7</sup> These tests are described in greater detail in this text in the section on small intestinal diseases (see Chapter 12.3). Malabsorption may be caused by parasitism, diarrhea, and inflammatory or neoplastic intestinal disease.

Gastrointestinal parasitism results in weight loss due to several mechanisms.<sup>2</sup> Parasites may compete directly for nutrients within the lumen of the bowel. Malabsorption may result from a lack of mucosal integrity, a decrease in intestinal villi size and number (and subsequent decrease in mucosal absorptive surface area), and a decrease in digestive enzymes that originate in the mucosa. Competition of parasites for protein sources may result in decreased availability of amino acids for production of digestive enzymes or mucosal transport proteins. Increased mucosal permeability due to leakiness in mucosal intercellular bridges may result in mucosal edema and increased transudation of intercellular fluid and its associated electrolytes, amino acids, and sugars into the lumen of the intestine.

Chronic diarrhea results in partial or complete anorexia, which contributes directly to weight loss. More rapid (decreased) gastrointestinal transit time results in increased losses of incompletely digested dietary feedstuffs. Malabsorption may result from decreased transit time and from villus blunting due to specific pathogens, such as in viral diarrhea (see the malabsorption section in small intestinal diseases). Bacterial pathogens may be in direct competition for luminal nutrients. Mucosal invasion by both viral and bacterial pathogens may cause mild-to-severe degrees of mucosal sloughing (ulcers), which result in maldigestion, malabsorption, and increased mucosal losses of intercellular fluid (e.g., in parasitism).

Given that the horse has adequate feed intake and absorption, inappropriate hepatic utilization of amino acids and sugars must be considered as a differential for weight loss. Chronic liver disease may result in weight loss due to inappetence, maldigestion (due to inadequate bile acid production), and inadequate or improper processing of amino acids into normal plasma proteins in the liver. These abnormalities may result in lowered concentrations of serum albumin, liver-dependent clotting factors (factors II, VII, IX, and X), and total plasma or serum protein. Lowered circulating proteins (especially albumin) may result in decreased plasma colloid osmotic pressure and thus may manifest as peripheral dependent edema in the distal limbs, pectoral region, and ventral midline. This peripheral edema may mask further weight loss by making the horse's torso appear to be heavier than it actually is. Decreases in clotting factors may result in bleeding diatheses. Hyperlipemia, hyperlipidemia, fatty liver syndrome, and ketosis may be seen in poorly fed ponies and in miniature horses with acute anorexia or overwhelming energy demands, such as pregnancy or lactation.<sup>8</sup>

Increased loss of protein or energy is a common cause of decreased body weight in horses. Luminal losses of fluid, electrolytes, and nutrients were described earlier for intestinal parasitism and diarrhea. Acute inflammatory protein losses may occur into major body cavities in overwhelming infections such as pleuritis or peritonitis. Chronic abscessing pneumonia, pleuritis, and peritonitis often result in increased, rather than acutely decreased, serum total protein due to increased  $\gamma$ -globulin production in response to chronic antigenic stimulation from the chronic infection. These chronic infections also usually have weight loss as an additional clinical sign due to the continuing catabolic processes associated with the infection itself. Equine infectious anemia (EIA) is a type of persistent systemic infection which, in its symptomatic form, may result in chronic weight loss and varying levels of anemia.<sup>9</sup> Asymptomatic EIA carriers may have no weight loss or other obvious clinical signs but can infect pasture mates via vector transmission.

Protein-losing enteropathy (PLE) is not a definitive diagnosis but is rather a group of diseases, each of which results in luminal losses of fluid, electrolytes, plasma proteins, and nutrients. Mechanisms of protein and fluid loss were described earlier for intestinal parasitism and diarrhea. Gastrointestinal ulcers have been reported to result in lowered serum total protein and weight loss.<sup>10</sup> One of the early indications of nonsteroidal anti-inflammatory drug toxicity is the detection of a lowered serum total protein. Such horses may also manifest varying degrees of inappetence and colic, especially associated with the immediate postprandial period. Intestinal neoplasia (usually lymphosarcoma) often manifests as a PLE with weight loss.<sup>11</sup>

Acute or chronic renal diseases, especially involving glomerulonephritis, can result in urinary protein loss and subsequent body weight loss.<sup>12</sup> Such horses may have polyuria and polydipsia as associated clinical signs. Polyuria is often reported by owners or handlers as increased wetness in stall bedding. Owners should be questioned thoroughly regarding the horse's water intake. The veterinarian may need to observe stable watering habits, often including actually measuring the volume of the subject's water buckets in order to definitively establish the presence of polydipsia. It may be necessary to turn off automatic waterers in the subject's stall or pasture and to offer the horse measured volumes of water from additional buckets in order to establish a diagnosis of polydipsia. Urine puddles in stalls, or collected urine samples, may foam excessively due to increased protein concentrations. Increased urinary protein concentrations can be quickly diagnosed on the farm with the proper interpretation of urine dipstick protein indicators.

Neoplasia or abscesses within the thorax or abdomen serve as catabolic energy and protein "sinks," resulting in chronic weight loss.<sup>11, 13</sup> Chronic pain, such as that associated with severe, unresponsive laminitis, results in increased catabolism and weight loss, probably as a result of chronically elevated systemic catecholamine levels. Increased circulating epinephrine and norepinephrine levels result in a whole-body catabolic state with increased breakdown of stored energy sources and ultimately result in chronic weight loss. Similar weight loss due to systemic catabolism can be the result of chronically elevated serum cortisol associated with pituitary adenoma and secondary hyperadrenocorticism.

Heart murmurs and resultant heart failure can cause

weight loss due to inefficiency of circulation of nutrients and oxygen to peripheral tissues. Chronic obstructive pulmonary disease (COPD) or heaves may result in weight loss due to an increase in the work of breathing and due to poor oxygenation of peripheral tissues. While ventral abdominal musculature may hypertrophy and result in a "heave line," weight loss will be manifested by increased depth between the ribs and decreased muscular thickness and definition along the dorsal midline. Suckling foals with severe pneumonia may manifest weight loss if they become inappetent due to decreased suckling related to their severe dyspnea.

### Approach to the Horse with Decreased Body Weight

An appropriately taken history should document the type, amount, and quality of feed and hay being provided daily. Documentation of deworming products used and intervals of administration is critical. The history may also document the presence of anorexia, depression, polyuria, polydipsia, diarrhea, or other important historical signs that may point more quickly toward a specific cause of the weight loss.

The physical examination should reveal the presence of weight loss, a cardiac murmur, pneumonia or pleuropneumonia (increased lung sounds), COPD (increased abnormal expiratory lung sounds), dental abnormalities, peripheral edema, urine staining on the hindlimbs, diarrhea, icterus, nasal discharge (dysphagia, pneumonia), fever, or hirsutism (secondary hyperadrenocorticism). The rectal examination may document the presence of intra-abdominal masses (abscess, neoplasia), enlarged left kidney, thickened intestinal or rectal wall, colonic displacement, gritty peritoneal surfaces (peritonitis), gritty feces (sand impaction), or diarrhea.

Fecal flotation may serve as an adequate screening tool to determine whether there is any evidence of parasitism. In the event of a positive fecal flotation, Baermann's sedimentation may be necessary to quantitatively determine the severity of the patent parasitic load in the horse with weight loss. Fecal occult blood may be positive with gastrointestinal ulceration or neoplasia, but parasites or a recent rectal examination may also result in positive results.

Routine hematology (complete blood count, fibrinogen) should assist in diagnosing infectious conditions such as pleuritis or peritonitis. Decreased serum or plasma total protein and albumin concentrations are evidence of hypoproteinemia and make the following conditions more likely: severe malnutrition, PLE (diarrhea, parasitism, ulceration, intestinal neoplasia, inflammatory intestinal disease), glomerular disease, acute pleuritis or peritonitis, or chronic liver disease. Increased total protein concentrations, especially  $\gamma$ -globulins, make chronic closed cavity infections such as abscesses, peritonitis, or pleuritis more likely. Increased  $\beta$ -globulin fractions suggest the presence of parasitism.

Routine serum biochemistries should aid in diagnosing renal (renal azotemia, electrolyte abnormalities) and liver disease (increased gamma-glutamyltransferase, aspartate aminotransferase, serum alkaline phosphatase, lactate dehydrogenase). Urinalysis should reveal increased protein levels on dipstick or quantitative analysis in the event of glomerular protein losses. Metabolic alkalosis may be evident in the aftermath of salivary bicarbonate losses due to dysphagia or esophageal obstruction.

Endoscopy may aid in the diagnosis of causes of dysphagia or esophageal obstruction. Lengthy endoscopes are necessary for examination of large adult horses for suspected gastrointestinal ulcers, but shorter endoscopes may suffice for foals or shorter-necked adults (e.g., Arabians, ponies).

Peritoneal fluid analysis will document the presence of a transudate (equivocal infection) or exudate (probable infection).<sup>14, 15</sup> Both aerobic and anaerobic peritoneal fluid cultures should be performed if intra-abdominal infection is suspected. Exfoliative cytology may rarely document the presence of neoplastic cells due to intra-abdominal neoplasia.<sup>11, 16</sup>

Non-routine tests should be performed only as indicated and include oral absorption tests (see the section on small intestinal diseases) (Chapter 12.5) and biopsies of the liver, kidney, or intestinal wall. Abdominal or thoracic ultrasonography should help to rule in or out abnormalities of the liver or kidneys and may document the presence of abnormal fluid (peritonitis, pleuritis) or masses (abscesses, neoplasia). Cardiac ultrasound should be definitive in the event of a murmur and suspected heart failure. Radiography may also be helpful to document the presence of thoracic masses or COPD, but increased pleural fluid will obscure visualization of other intrathoracic structures.

### INCREASED BODY WEIGHT

Overfeeding may be the most common cause of obesity in horses and may also be the easiest to correct. The stable's feeding practices and feed and hay sources should be investigated thoroughly. It is not uncommon for novice horse owners, single horse owners, and pony owners to overfeed their animals.

Ponies seem to be particularly susceptible to obesity, perhaps because their size renders them to be more easily overfed. However, at least one author has proposed that this tendency toward obesity in ponies receiving modern confinement diets may be due to their having evolved in the inhospitable ice age climates of northern Europe.<sup>17</sup> In that era, the lack of readily available grazing feedstuffs might have placed greater selection pressure on survival of ponies with more efficient dentition and better nutrient and fluid absorption from the gastrointestinal tract. It is argued that those ponies that had greater feed conversion efficiency would have been stronger, had longer lives, and been more available for breeding. Current illustrations of this theory may lie in the Welsh and Connemara pony breeds that still thrive and flourish in the wild in the relatively inhospitable north Atlantic climates of the western coasts of Wales and Ireland, respectively.

Pregnancy in mares is a normal physiologic event that leads to increased body weight. Surprisingly, many new owners of mares may not know that their new purchase is pregnant. It is not uncommon for an earlier negative pregnancy diagnosis to have been in error. Any mare that is gaining weight in an unexpected manner should be examined rectally, and ultrasonographically if necessary, for a possible pregnancy.

Hypothyroidism has been reported to be associated with weight gain and failure to become pregnant in broodmares.<sup>18</sup> Evidence for both hypothyroid-associated weight gain and

infertility was lacking in surgically created hypothyroid pony<sup>19</sup> and quarter horse<sup>20</sup> subjects. There is, however, an abundance of field experience to infer a relationship between obesity, hypothyroidism, and infertility in mares.<sup>17</sup> Documentation of hypothyroidism must be by performance of a thyroid-stimulating hormone or thyroid-releasing hormone stimulation test,<sup>21, 22</sup> since resting thyroid levels vary diurnally<sup>23</sup> and are not truly reflective of thyroid function. It must also be remembered that only 5 days of normal phenylbutazone (PBZ) therapy results in abnormally low resting serum thyroid levels due to direct competition of PBZ with thyroid hormone for serum protein-binding sites.<sup>21</sup> The diagnosis and treatment of hypothyroidism is described in greater detail elsewhere in this text.

### Approach to the Horse with Increased Body Weight

Differential diagnoses for increased body weight include overfeeding, pregnancy, hypothyroidism, and other conditions that result in abdominal distention, such as bloat, ascites, uroperitoneum, fetal hydrops, and rupture of the prepubic tendon or abdominal wall musculature. The latter conditions are described in greater detail in the following section titled Abdominal Distention.

Feeding practices should be investigated and observed firsthand if necessary. A positive pregnancy status should be an easy historical and rectal diagnosis. Most hematologic and biochemical tests will be normal in the pregnant or simply overweight horse. Thyroid status should be assessed appropriately, not by simple resting thyroid hormone concentrations, but by thyroid-stimulating hormone or thyroid-releasing hormone stimulation tests that have been described previously and that are presented elsewhere in this text.<sup>21, 22</sup>

Education of the client is important with regard to feeding practices, especially if it is determined that the overweight horse has simply been overfed by a novice owner. Dangerous consequences, including colic and laminitis, should be explained to the client.

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## 3.5

### Abdominal Distention

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Increases in body weight due to overeating or pregnancy must be distinguished from increases in body girth due to bloat, ascites, uroperitoneum, fetal hydrops, or ruptured prepubic tendon. In each of these conditions, body weight may actually be increased due to fetal growth or fluid accumulation. More important, however, there is a perceptible change to the shape of the abdomen of the horse.

Bloat is usually associated with colic signs in horses and is due to gaseous intestinal distention secondary to ileus or simple obstruction of the large, or rarely small, intestine.