

## Polyphagia

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**P**olyphagia is the consumption of food in excess of normal caloric intake. Hunger and satiety and, consequently, feeding behavior are primarily controlled by certain regions in the central nervous system (CNS), but many factors affect the function of these areas. Thus polyphagia can be classified as *primary* (i.e., a CNS abnormality) or *secondary* (i.e., a systemic problem affecting the CNS). Secondary polyphagia is by far more common and usually is accompanied by clinical signs of the underlying disease. Determining whether weight gain or loss has occurred should be the first step in formulating a list of differential diagnoses and a diagnostic plan.

### PHYSIOLOGY

Food intake is controlled by a variety of factors, including gastrointestinal, environmental, and CNS phenomena. The CNS, mainly the hypothalamus, controls eating behavior. The lateral hypothalamic nuclei represent the “feeding center”; their stimulation causes an animal to eat, and their destruction results in severe, fatal anorexia. Conversely, the ventromedial nuclei are the “satiety center,” because their stimulation causes a refusal to eat even highly appetizing food, and their ablation leads to polyphagia and obesity. The feeding center is constantly active unless inhibited by the satiety center (e.g., postprandially). Lesions of the amygdala or paraventricular nuclei also can increase feeding behavior.

Gastrointestinal components that affect feeding include gastric distention, the rate of gastric emptying, the release of gastric hormones, and absorption of nutrients, such as fatty acids, glucose, and amino acids. The gut hormones can act locally on the gastrointestinal tract and centrally on the CNS. Insulin, glucagon, and cholecystokinin secreted in response to a meal decrease feeding signals from the CNS. Leptin, a polypeptide released from adipose tissue, may also help to create a sense of satiety. Decreased serum concentrations of glucose, amino acids, or lipid metabolites cause hunger by stimulating neural centers so as to re-establish normal levels. Feeding behavior also can be incited by increased nutrient utilization (i.e., an elevated metabolic rate).

Normal control of feeding, therefore, is complex and works to maintain energy stores and body weight through an interplay of central and peripheral inputs. Pathologic conditions that affect the CNS can increase feeding behavior even in the presence of normal energy stores (*primary polyphagia*). *Secondary polyphagia* exists when feeding behavior is stimulated by non-neural factors and can be caused by an increased metabolic rate or decreased nutrient supply (Box 34-1). An augmented metabolic rate can be physiologic (e.g., pregnancy) or pathologic (e.g., hyperthyroidism) in origin. Diabetes mellitus is an unusual case of decreased nutrient supply. Due to an inability to respond to or a lack of insulin, the body does not recognize glucose and reacts to a perceived hypoglycemia. Certain diseases (e.g., hyperadrenocorticism

and liver disease) lead to polyphagia by unknown mechanisms. Secondary polyphagia can also be caused by certain drugs.

### HISTORY

Any change in body weight is an important differentiating feature of the various causes of polyphagia (Figure 34-1). Primary or drug-induced polyphagia typically results in weight gain, because nutrients are adequate and feeding is inappropriately increased. Pathologic secondary polyphagia is more commonly associated with weight loss, because the nutrient supply usually does not meet physiologic demands. However, some causes, such as acromegaly, hypoglycemia caused by an insulinoma, sudden acquired retinal degeneration syndrome (SARDS), and hyperadrenocorticism (HAC), lead to weight gain. Physiologic polyphagia can result in weight gain (e.g., pregnancy, growth) or maintenance of weight (e.g., lactation, cold environment, increased exercise). An animal with HAC or in the early stages of any of these states, however, may show no weight change.

Certain causes of polyphagia may be diagnosed on the basis of the history. The possibility of exposure to a cold environment, increased exercise and, for intact females, pregnancy and lactation should be ascertained. Polyphagia is commonly associated with anticonvulsant and glucocorticoid therapy but has been observed with other medications as well (see Table 34-1). Psychogenic polyphagia has been noted after introduction of a more palatable diet or in response to a stressful event, most commonly introduction of a new pet into the household. Feeding of a low-calorie diet may also be diagnosed on the basis of a complete dietary history.

An animal with primary polyphagia caused by destruction of the satiety center may have a history of trauma or clinical signs associated with CNS disease. Depending on the extent of a hypothalamic lesion, upper motor neuron signs may be seen in all four limbs or unilaterally. A midbrain lesion often leads to incessant pacing, circling, and blindness; polyuria/polydipsia may also be present. Disorders caused by diffuse or multifocal CNS disease will have other clinical signs as well, depending on the areas affected.

Perturbation of hypothalamic control of the pituitary can lead to reproductive, thyroidal, and adrenal hypofunction and associated clinical signs. Hypothyroidism secondary to pituitary dysfunction is clinically identical to primary thyroidal failure. If adrenal insufficiency occurs secondary to a lack of adrenocorticotropic hormone (ACTH), vague, nonspecific signs of lethargy and gastrointestinal disease usually are seen. Serum electrolyte concentrations (e.g., sodium and potassium) are normal because aldosterone secretion is not affected by pituitary disease. This type of hypoadrenocorticism is referred to as *atypical*.

Historical findings associated with secondary polyphagia can be highly varied. Animals with diabetes mellitus,

## Box • 34-1

**Differential Diagnoses of Polyphagia****Primary Polyphagia**

- Destruction of satiety center
  - Trauma
  - Mass lesion (e.g., neoplasia)
  - Infection
- Psychogenic causes
  - Stress
  - Introduction of a more palatable diet

**Drug-Induced Polyphagia**

- Glucocorticoids
- Anticonvulsants
- Antihistamines
- Progestins
- Benzodiazepines
- Amitraz
- Cyproheptadine

**Reported Specific Disorders Associated with Polyphagia**

- Feline infectious peritonitis
- Lymphocytic cholangitis (feline)
- Spongiform encephalopathy (feline)
- Foreign body encephalitis (feline)

**Secondary Polyphagia**

- Physiologic increase in metabolic rate
  - Cold temperature
  - Lactation
  - Pregnancy
  - Growth
  - Increased exercise
- Pathologic increase in metabolic rate
  - Hyperthyroidism
  - Acromegaly
- Decreased energy supply
  - Diabetes mellitus
  - Malassimilation syndromes
    - Pancreatic exocrine insufficiency
    - Infiltrative bowel disease
    - Parasites
    - Lymphangiectasia
- Decreased intake
  - Megaesophagus (congenital)
  - Low-calorie diet
  - Hypoglycemia
- Unknown
  - Hyperadrenocorticism
  - Portasystemic shunt/hepatoencephalopathy
  - Sudden acquired retinal degeneration (SARDS)

acromegaly, HAC, SARDS, and hyperthyroidism usually are polyuric and polydipsic. Feline acromegaly is seen in middle-aged to older males, and naturally occurring canine acromegaly is seen almost exclusively in intact bitches. In dogs of either sex, progestin administration can lead to acromegaly, and use of this medication should be historically evident. Owners may note inspiratory stridor or a change in body conformation, such as increased interdental spaces, skin folds, or head size in acromegalic animals. It should also be noted that progestin administration to dogs and cats can increase appetite without causing acromegaly. A multitude of historical details can be associated with HAC, including abdominal enlargement, persistent panting, failure to regrow hair after clipping, lethargy, and muscle weakness. Animals with SARDS typically have the presenting complaint of sudden-onset blindness. Hyperthyroidism commonly leads to increased activity but can be associated with depression and lethargy. Gastrointestinal signs (e.g., vomiting and diarrhea) may also be present.

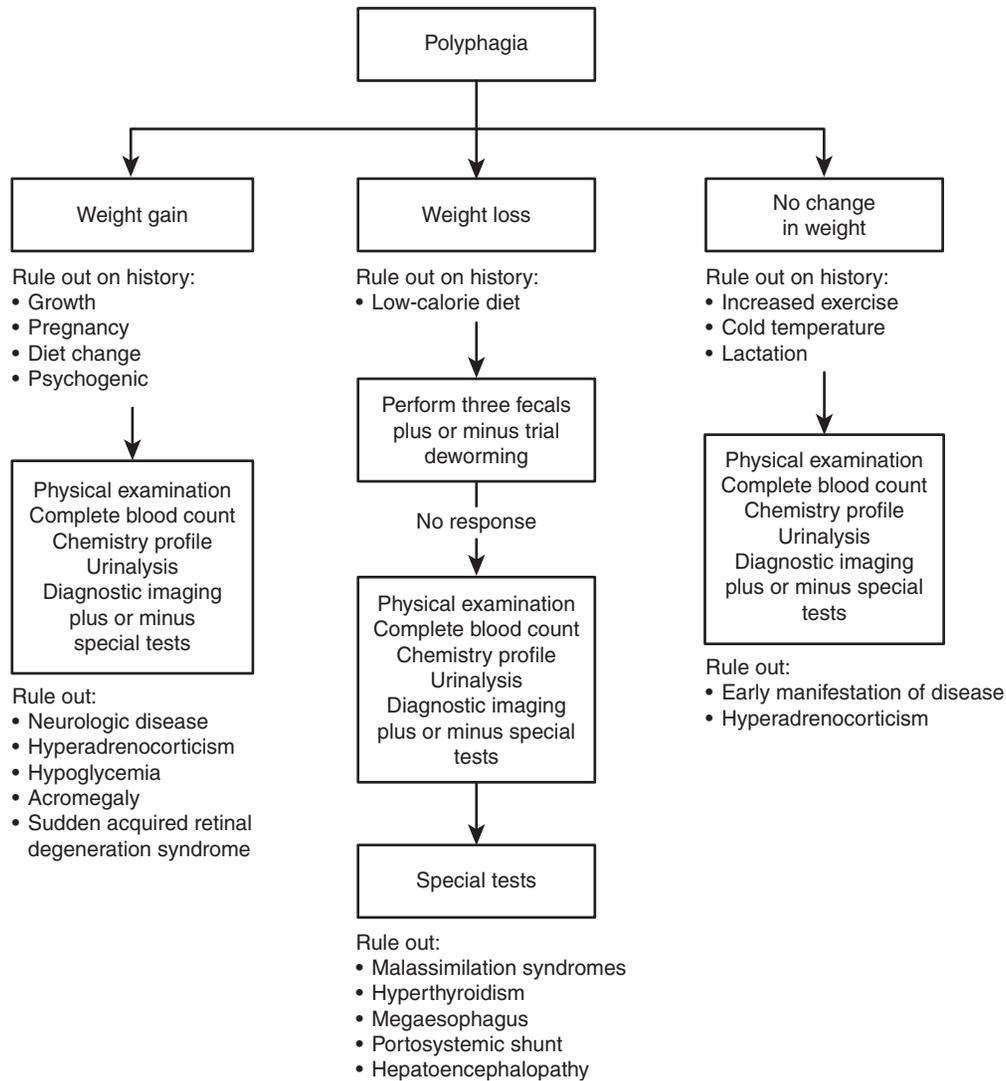
Hypoglycemia has a number of causes. Of these, insulinoma is the most likely to lead to polyphagia, but other neoplasias and insulin overdose may also do so. Hypoglycemic patients may exhibit weakness, trembling, ataxia, disorientation and, possibly, grand mal seizures. Malassimilation can be caused by a number of problems such as pancreatic exocrine insufficiency (PEI), infiltrative bowel disease, parasites, and lymphangiectasia. Malassimilation syndromes and PEI generally cause large-volume, malodorous, soft stools. PEI is more common in younger dogs (i.e., those less than 2 years of age), and the German shepherd breed shows a predisposition for this disorder. In older dogs and cats, PEI is rare but, if seen, is most commonly associated with chronic pancreatitis. The category of infiltrative disease encompasses processes such as inflammatory bowel disease, neoplasia, and infections such as histoplasmosis. Historical details vary according to the underlying disease.

Acquired esophageal disease often leads to anorexia, but animals with congenital megaesophagus may be polyphagic and typically have a history of regurgitation. Although anorexia is more common in animals with a portacaval shunt, polyphagia has been reported in approximately 10% of cases. Depression, vomiting, weight loss, polydipsia, and neurologic signs may also be noted. Polyphagia has been reported rarely in cases of hepatoencephalopathy; other clinical findings are the result of hepatic failure and may be similar to those in an animal with a portasystemic shunt.

**PHYSICAL EXAMINATION**

Physical examination findings in polyphagic animals vary, depending on the underlying disease. With primary polyphagia, neurologic abnormalities such as ataxia and proprioceptive deficits may be present. A complete neurologic and fundic examination should be performed. With acute causes of central blindness, however, the fundus appears normal.

If unclear from the history, pregnancy potentially can be diagnosed by abdominal palpation and lactation by inspection of the mammae. Approximately 80% of cats with hyperthyroidism have a palpable thyroid nodule, and approximately 50% have tachycardia or a gallop rhythm. Hyperthyroidism is rare in dogs, and a cervical mass usually is palpable. Hyperadrenocorticism can have a variety of physical examination findings, including abdominal and hepatic enlargement, muscle wasting, bilaterally symmetric alopecia, cutaneous hyperpigmentation, areas of poor hair regrowth or calcinosis cutis. Even when not noted by an owner, the physical changes associated with acromegaly can be documented on physical examination; a degenerative polyarthropathy may also be present.



**Figure 34-1** Algorithm for diagnostic approach to polyphagia.

Examination findings in a dog with SARDS may be unremarkable, because in the early stages of the disease, the retinas appear normal on fundic examination. Dogs or cats with PEI, an insulinoma, megaesophagus, hepatoencephalopathy, a portosystemic shunt, or a malassimilation syndrome may have no abnormal physical findings other than the associated weight change. In rare cases, polyneuropathies may accompany an insulinoma. Aspiration pneumonia may be present in animals with megaesophagus. Neurologic abnormalities may be detected in an animal with a portosystemic shunt, and ascites is noted in approximately 20% of afflicted dogs. Neurologic findings associated with hepatoencephalopathy may be episodic, and other examination findings vary with the cause of liver disease. Depending on the cause of malassimilation, the intestines may feel thickened. Lymphangiectasia may lead to ascites.

Occasionally, polyphagia may be a clinical sign of a disease with which it is not usually associated. For example, one cat with feline infectious peritonitis (FIP), one with foreign body encephalitis, and one with spongiform encephalopathy have been reported as being polyphagic, as have 18 cats in Great Britain with lymphocytic cholangitis. Other historical and clinical signs are present depending on the cause.

## DIAGNOSTIC PLAN

The first step in diagnosis is to ascertain what change has occurred, if any, in the animal's weight (see Figure 34-1). After as many differential diagnoses as possible have been ruled out on the basis of the history, further testing is warranted. In all cases a minimum data base (MDB), including a serum biochemistry profile, complete blood count (CBC), and urinalysis, should be submitted.

For dogs and cats with weight gain, pregnancy must be ruled out. To diagnose primary polyphagia, a complete neurologic examination should be performed, any abnormalities localized, and appropriate tests obtained. A cerebrospinal fluid analysis or diagnostic imaging, such as radiography, computed tomography (CT), or magnetic resonance imaging (MRI), may be necessary.

Hypoglycemia caused by an insulinoma usually can be diagnosed by measuring paired blood glucose and insulin serum concentrations when the animal is hypoglycemic. Rarely, provocative testing may be required (see Chapter 240). The diagnosis of SARDS can be made on the basis of appropriate history, physical examination findings, an MDB that rules out other causes and, if necessary, an electroretinogram (ERG).

Although certain changes in the MDB are typical of hyperadrenocorticism, they do not confirm the diagnosis and further adrenal testing must be performed; an ACTH stimulation test or low-dose dexamethasone suppression test should be performed to confirm the diagnosis (see Chapter 242). Diagnosis of acromegaly can be difficult because of the lack of a commercial assay for growth hormone, but measurement of insulin-like growth factor-I (IGF-I) may be helpful (see Chapter 235). The history, together with conformational changes, can provide evidence of the underlying disease. Acromegalic cats consistently have insulin-resistant diabetes mellitus, and imaging of the pituitary may reveal a tumor.

If weight loss is associated with polyphagia, the MDB should be preceded by three fecal examinations. If the results of these are negative, the MDB does not provide the diagnosis, and the animal is stable, trial therapy with antiparasitics may be warranted. If deworming does not resolve the problem, additional tests must be done. Hyperthyroidism often can be diagnosed on the basis of a single serum thyroxine measurement; however, other tests, such as measurement of the free thyroxine concentration by equilibrium dialysis, may be required (see Chapter 238).

Malassimilation syndromes cover myriad differential diagnoses (see Table 34-1). Protein-losing enteropathies can be associated with hypoalbuminemia and hypoglobulinemia. Depending on the suspected cause, measurement of serum folate or cobalamin, assessment of fat absorption, abdominal radiography or ultrasonography, and/or biopsy either by endoscopy or exploratory surgery may also be considerations. For verification of PEI, serum trypsin-like immunoreactivity (TLI) should be determined. Thoracic radiographs with a positive contrast esophagram should be used to diagnose megaesophagus; this imaging may also aid in the determination

of the cause. Determination of preprandial and postprandial serum bile acid concentrations can document hepatic dysfunction, but a biopsy may be required to document the cause of hepatic failure. Ultrasonography or a radionuclide scan may be used to identify a portacaval shunt.

If the disease is in the early stages, weight change may not yet have occurred, and the list of differentials may be difficult to narrow. However, a good history and physical examination combined with an MDB can eliminate many possibilities. Although animals with HAC may not have a weight change, abdominal enlargement may create the impression of weight gain. All diseases suspected as possible differential diagnoses in this situation should be diagnosed as discussed above.

## MANAGEMENT

The management of polyphagia depends on the cause. Physiologic causes of polyphagia are transient. If the condition is drug induced, the polyphagia may be temporary, as is usually seen with anticonvulsants. Psychogenic polyphagia may be corrected by removing the instigating element, if possible, or by behavioral therapy (e.g., paying more attention to the animal). If the polyphagia persists with ongoing drug therapy or if the inciting agent (stress or medication) cannot be removed, food intake should be limited to that necessary to satisfy caloric requirements. Low-calorie, high-fiber foods, such as carrots, can be added to the diet to assuage hunger and prevent obesity. Polyphagia caused by dietary factors can be managed as needed. In cases of SARDS, the polyphagia usually is self-limiting. For all other conditions, appropriate therapy should be initiated to resolve the underlying disease.

# CHAPTER • 35

## Ptyalism

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**P**tyalism is the excessive production and secretion of saliva. *Pseudoptyalism* is the drooling or dribbling of saliva because of an inability or reluctance to swallow, which results in an overflow of saliva from the oral cavity.

### PATHOPHYSIOLOGY

Saliva is continuously produced by the salivary glands. The production of saliva may be increased by excitation of the salivary nuclei, located in the brain stem, after taste and tactile stimuli are received from the tongue and other areas in the oral cavity. Higher centers in the central nervous system (CNS) may also have an excitatory or inhibitory affect on the salivary nuclei. Increased production of saliva is a normal physiologic occurrence associated with imminent feeding, hyperthermia, and purring in cats.

Oral lesions and central nervous system disorders may stimulate an increase in the production of saliva. Diseases of

the pharynx, esophagus, and stomach also may stimulate the production of excessive amounts of saliva. In some cases this excess may be clinically evident not as drooling but as frequent episodes of swallowing.

Anatomic abnormalities of the oral cavity may cause saliva to drip from the mouth even when normal amounts of saliva are produced. Drooling is evident with diseases in which the animal is unable or reluctant to swallow because of pain associated with swallowing.

### HISTORICAL FINDINGS AND THEIR MEANINGS

#### Age and Breed

Young animals with congenital anomalies, including portosystemic shunt and megaesophagus, may hypersalivate. Jaw abnormalities, such as severe retrognathism, may cause excessive drooling. In some giant breed dogs, the shape and size of the lower lip may predispose to drooling. Young animals are