



Hydrocephalus in Dogs and Cats

Joan R. Coates, DVM, MS, DACVIM (Neurology)

University of Missouri-Columbia

Todd W. Axlund, DVM, MS, DACVIM (Neurology)

Auburn University

Curtis W. Dewey, DVM, MS, DACVIM (Neurology), DACVS

Long Island Veterinary Specialists
Plainview, New York

Jodi Smith, DVM

Auburn University

ABSTRACT: Hydrocephalus is a common congenital or acquired neurologic disorder in dogs and cats. Although a differential diagnosis for the disorder exists, the underlying cause of congenital hydrocephalus is often unknown. Medical treatment decreases cerebrospinal fluid volume and production but offers only temporary palliation of clinical signs. Advances in shunt technology have allowed ventriculoperitoneal shunt placement to treat congenital hydrocephalus and as an adjunct in managing secondary hydrocephalus. This article discusses the pathophysiology and medical and surgical treatment of hydrocephalus.

The term *hydrocephalus* implies the presence of an excessive accumulation of cerebrospinal fluid (CSF) within the cranial cavity with subsequent dilation of the ventricular system.^{1,2} Hydrocephalus has been characterized by the anatomic relationship of the underlying disease process and abnormal pressure differences.³ Internal and external hydrocephalus refer to increased fluid accumulation within the ventricular and subarachnoid spaces, respectively.⁴ Obstructive (i.e., noncommunicating) hydrocephalus is characterized by occlusion of CSF flow within the ventricular system rostral to the site of obstruction.

Conversely, communicating hydrocephalus occurs when there is extraventricular impedance to normal CSF circulation and absorption. Communicating hydrocephalus may also be a result of excessive CSF production, but this is rare. Compensatory hydrocephalus (i.e., hydrocephalus ex vacuo) occurs when CSF occupies space in the cranial cavity that normally would be occupied by brain parenchyma.⁵ For example, CSF replaces the area of parenchymal destruction secondary to an ischemic event or senile atrophy of the brain. Although CSF pressure measurements are rarely done in veterinary medicine, *normotensive* and *hypertensive* are descriptive terms used to further classify cases of hydrocephalus in which the CSF pressure is normal or increased, respectively.⁶

Send comments/questions via email
editor@CompendiumVet.com
or fax 800-556-3288.

Visit CompendiumVet.com for
full-text articles, CE testing, and CE
test answers.

PATHOPHYSIOLOGY

The primary function of CSF is to provide physical protection for the brain and spinal cord from mechanical forces. These forces may arise externally from traumatic blows to the head or vertebral column or internally from acute changes in intravascular volume. The CSF provides a buoyancy effect for the central nervous system because the specific gravity of CSF (i.e., 1.007) is lower than that of brain tissue (1.040).⁷ Suspension of the brain in a liquid environment reduces its weight by approximately 97%.⁸ CSF also nourishes neural tissue through distribution and movement of various proteins, molecules, and ions; maintenance of the acid–base balance; and regulation of brain volume.⁹ CSF homeostasis is maintained during normal cerebral metabolic activity through active transport or diffusion processes of chemical elements or lipid-soluble substances. CSF serves an important role of “volume buffering” (mediating) intracranial compliance in disease states to allow compensation for altered intracranial volume.¹⁰

It is estimated that approximately 60% of CSF is produced by the choroid plexuses located in the lateral, third, and fourth ventricles.⁷ Net movement of water

pulsatile flow of intracranial arterial blood that is influenced by cardiac cycle–dependent changes of vascular volume.^{15,16} The Monro-Kellie doctrine states that the total volume of the intracranial components (i.e., blood, brain parenchyma, and CSF) remains constant at all times. A volume increase in one results in a volume decrease in the others.^{8,17} Increased systolic blood volume is compensated for by caudal displacement of venous blood and CSF into the subarachnoid space.¹⁷

Most CSF absorption from the subarachnoid space has traditionally been thought to occur via one-way valvular mechanisms at collections of arachnoid villi called *arachnoid* or *pacchionian granulations* located in the dorsal sagittal sinus.⁸ It is currently believed that most CSF absorption actually occurs throughout the central nervous system through capillaries of the brain and spinal cord.^{18–20}

Causes of hydrocephalus can be congenital or acquired. In veterinary patients, congenital hydrocephalus is more common than acquired hydrocephalus. In a retrospective study²¹ of 564 cases of congenital hydrocephalus, 11 breeds (i.e., Maltese, Yorkshire terrier, English bulldog, Chihuahua, Lhasa apso, Pomeranian, toy poodle, Cairn terrier, Boston terrier, pug, Pekingese) were determined to

The cause of hydrocephalus can be congenital or acquired, and the condition requires brain imaging for accurate diagnosis.

from plasma to CSF is hypothesized to occur through passive ultrafiltration of plasma, producing a hyperosmolar solution and active secretion of ions by the choroid epithelium. The remaining 40% of normal CSF volume is derived from the ependymal tissue of the ventricles and parenchymal capillaries.⁷ CSF production is believed to occur at a constant rate, but studies^{11–13} have shown variations in human CSF production related to circadian rhythm, age, and the presence of pathologic processes such as ventriculitis.

The bulk of CSF flow occurs through the ventricular system and into the subarachnoid space. The pathway begins in the lateral ventricles, passes through the inter-ventricular foramina into the third ventricle, mesencephalic aqueduct, and fourth ventricle, and continues into the subarachnoid space by passing through the lateral apertures of the fourth ventricle.⁵ CSF flow is bidirectional toward the brain and the compliant thecal sac of the cauda equina of the spinal cord.¹⁴

The hydrodynamics of CSF flow are governed by the

be at significantly higher risk for hydrocephalus versus other breeds examined.

Congenital hydrocephalus is usually considered obstructive or noncommunicating.⁵ Congenital hydrocephalus is suspected to be associated with fusion of the rostral colliculi, causing secondary mesencephalic aqueductal stenosis.² Pre- or postnatal inflammations alter the ependymal surface of the aqueduct and cause secondary stenosis. Aqueductal stenosis has occurred in kittens exposed in utero when a queen was treated with griseofulvin²² and with exposure to feline panleukopenia virus.²³ Neuropathologic changes associated with hydrocephalus include focal destruction of the ependymal lining, compromise of cerebral vasculature, damage of periventricular white matter, and neuronal injury.²⁴ Periventricular-associated diverticula and tears are prevalent in cases of spontaneous hydrocephalus.²⁵

Malformations affecting the cerebellum have also been associated with congenital hydrocephalus. Hydrocephalus has been observed in a dog and cats with cerebellar



Figure 1. Bulldog puppy with clinical features of hydrocephalus, including a domed calvarium and bilateral ventrolateral strabismus.

vermian hypoplasia analogous to Dandy-Walker syndrome in humans.²⁶ Chiari type I malformation is a developmental disease of humans characterized by cerebellar herniation and syringohydromyelia.²⁷ The pathogenesis is related to occipital bone malformation reducing the total area of the caudal fossa and overcrowding the cerebellum and brain stem. Cerebellar herniation and abnormal brain-stem anatomy alter CSF hydrodynamics, resulting in formation of syringohydromyelia.²⁸ Chiari type I-like malformation (i.e., caudal occipital malformation syndrome) diagnosed by magnetic resonance imaging (MRI) has been identified as a significant problem in cavalier King Charles spaniels and other breeds.^{29–31} Characteristic MRI findings in affected dogs include cerebellar vermal herniation, syringohydromyelia, a kinked appearance of caudal medulla, attenuation of the dorsal subarachnoid space at the level of the foramen magnum, and hydrocephalus.^{29,31} There is no correlation between severity of neurologic signs and abnormal anatomy.³⁰

Pathophysiologic mechanisms of acquired hydrocephalus include direct or indirect obstruction, loss of brain parenchyma with subsequent ventricular enlargement (hydrocephalus ex-vacuo), or, in rare instances,

increased CSF production from a tumor of the choroid plexus.³ Examples of secondary obstruction include neoplasms, cysts, inflammation, and vascular-associated diseases such as intraventricular hemorrhage.¹ Viral infections often associated with impaired CSF absorption and causing ependymitis include parainfluenza, canine distemper, feline panleukopenia, and FIP coronavirus.^{2,32,33} Periventricular encephalitis has been described in young dogs and is thought to be a sequela of severe meningoencephalitis and hemorrhage with a possibly bacterial cause.^{34–36} Hydrocephalus ex-vacuo occurs with destruction or lack of development of cerebral tissue (hydranencephaly and porencephaly) during critical times of development or subsequent to ischemia, trauma, or senile atrophy (i.e., cognitive dysfunction).^{33,37}

CLINICAL SIGNS

Congenital hydrocephalus is typically recognized in patients 2 to 3 months of age. Clinical signs are detected, along with the salient physical features characteristic of hydrocephalus (Figure 1). Animals with congenital hydrocephalus are often smaller than their littermates. Enlargement of the cranium and open fontanelle are often evident during physical examination. The extent of calvarial distortion depends on the rate of fluid accumulation, severity of ventricular enlargement, and stage of ossification of cranial sutures.³⁸ Bilateral ventrolateral strabismus, referred to as the *setting sun sign*, is believed to occur secondary to mechanical pressure on the eyes from orbital malformation or anatomic alterations of the midbrain and oculomotor nuclei.³⁹

Neurologic signs associated with hydrocephalus are variable.^{33,39,40} Neurologic dysfunction can be severe in young animals with congenital hydrocephalus. Forebrain signs tend to predominate.³⁹ Restlessness, marked changes in behavior, head pressing, and seizures may be evident. Blindness occurs with damage to the optic radiation or occipital cortex. Gait deficits in affected animals can range in severity based on compromise of the cerebellum or brain stem. Behavior problems are the most common complaint of pet owners. Examination of a group of hydrocephalic Maltese dogs revealed that fewer than 20% had seizures (typically occurring within the first year of life).⁴⁰ Most dogs exhibited erratic behavior associated with changes in their home environment or reproductive cycle. Most of these dogs (i.e., 75%) were reported by their owners to be difficult or impossible to house train.⁴⁰

Older animals with acquired hydrocephalus more often have clinical signs that reflect abnormalities attributable to the underlying cause rather than to hydrocephalus. Complete obstruction of CSF flow causes acute onset of clinical signs as a result of loss of compensatory mechanisms that maintain intracranial pressure. Seizures can be a sequela to acquired hydrocephalus early in the disease course.³⁹ Mentation often deteriorates to stupor or coma.^{1,41}

Diagnosis of hydrocephalus is suspected based on the physical examination and signalment. Radiographic evidence suggestive of hydrocephalus includes doming of the calvarium with thinning of cortical bone, decreased prominence of normal calvarial convolutions, and persistent fontanelles.³⁸ Diagnosis is better assisted by ultrasonography through the fontanelles⁴² and confirmed with advanced imaging (i.e., computed tomography, MRI)⁴¹ (Figure 2).

MEDICAL TREATMENT

The treatment of hydrocephalus should be dictated by the underlying cause. In most instances, medical therapy offers only temporary palliation of clinical signs.⁴⁰ This is certainly true in cases of obstructive hydrocephalus in which decreased CSF production offers short-term remediation of clinical signs but has no primary effect in relieving the obstruction. In rare cases, long-term medical therapy can be considered in animals with congenital hydrocephalus for which CSF is not surgically drained.^{40,41}



Figure 2. Transverse, T2-weighted MRI brain scan of a bulldog puppy with severe bilaterally symmetric hydrocephalus.

decompensating hydrocephalus. A dose of 1 to 2 g/kg should be administered intravenously over 15 to 20 minutes and may be repeated 2 to 4 times over 24 to 48 hours when needed, depending on the extent of increased serum sodium concentration.⁴¹ Acetazolamide, which is also a diuretic, decreases CSF production by inhibiting carbonic anhydrase. A dose of 10 mg/kg PO q6–8h has been recommended.⁴⁰ Acetazolamide combined with glucocorticoids may cause potassium depletion

Medical management of hydrocephalus may be offered as palliative therapy.

Medical treatment of hydrocephalus decreases CSF volume and production through the use of diuretics and glucocorticoids.^{40,41} Caution must be exerted when using any diuretic because electrolyte depletion is a common sequela. Electrolyte loss is accelerated when diuretics are used in combination with glucocorticoids. Monitoring sodium and potassium concentrations and hydration status is important during medical management.

Furosemide is a loop diuretic that decreases CSF production in extracellular fluid by inhibiting the sodium–potassium cotransport system. Furosemide should be administered at a dosage of 0.5 to 4 mg/kg PO q12–24h and then tapered to the lowest effective dose. Mannitol, an osmotic diuretic, is used in cases of rapidly

tion at the therapeutic drug concentration, which often requires discontinuation of acetazolamide.^{40,41}

A primary mechanism of action for various types of glucocorticoids is to decrease CSF production. Prednisone is the drug of choice and is administered at 0.25 to 0.5 mg/kg PO bid.⁴⁰ This dose should be maintained for 1 month and then slowly tapered to the lowest effective dose that ameliorates clinical signs. Dexamethasone sodium phosphate has also been used to treat hydrocephalus, but long-term use should be avoided because of the higher incidence of side effects.⁴³

In general, gastroprotectants are often used concurrently with glucocorticoids to minimize gastrointestinal side effects. Omeprazole is a proton-pump inhibitor

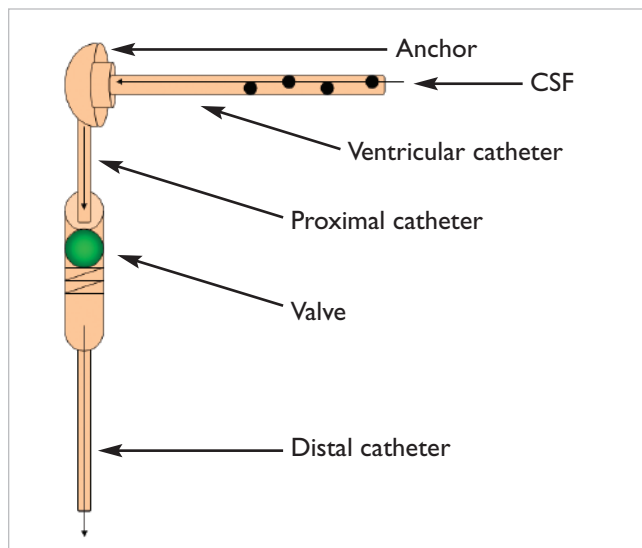


Figure 3. Schematic representation of the components of a ventricular shunt with a one-way valve.

that decreases stomach acidity. Although the mechanism of action is unclear, omeprazole has also been shown to decrease CSF production in dogs by 26%.⁴⁴ A dose of 10 mg q24h for dogs weighing less than 44 lb (20 kg) or 20 mg q24h for dogs weighing more is recommended.⁴⁴

SURGICAL TREATMENT

Shunting of CSF into another cavity is used to treat hydrocephalus and other disorders that cause secondary obstructive hydrocephalus and increased intracranial CSF accumulation.^{45,46} Commonly used cavities include the peritoneum, atrium, and pleural space. The peritoneal cavity is the most common site because of its high absorptive capacity.⁴⁷

Ventriculoperitoneal shunting is the most common type of shunting procedure.^{41,48} Ventriculoperitoneal shunting has the advantages of accommodating growth in young animals and ease of later surgical correction.⁴⁵ Disadvantages of ventriculoperitoneal shunt placement include more extensive subcutaneous dissection and additional incisions required in placing distal tubing. Ventriculoatrial shunting has also been successfully performed in dogs.^{46,49,50} However, it requires sacrifice of the jugular vein and larger patient size. Ventriculoperitoneal shunt placement is less complex than ventriculoatrial shunt placement.^{41,49}

A shunt system consists of three basic parts: a ventricular catheter, a one-way pressure cutoff valve, and a

distal catheter.⁵¹ The catheters are usually composed of radiopaque silicone tubing. The tip of the ventricular catheter has multiple small perforations for free CSF flow. The ventricular catheter connects to a unidirectional valve that allows CSF flow from the brain into the distal catheter of the shunt (Figure 3). Valve types vary according to different operating pressures (i.e., low, medium, high), and newer systems have adjustable valves.⁵¹ Appropriate operating pressures for animals with hydrocephalus have not been determined; we recommend a low-pressure valve. The distal catheter carries CSF from the valve to the peritoneal cavity. The distal catheter is fenestrated and longer than the proximal catheter.

Indications

Surgical shunting of CSF is indicated for disorders that result in obstruction and accumulation of CSF that secondarily causes severe neurologic deficits. Affected animals are usually refractory to medical therapy.^{40,41} Congenital hydrocephalus is the most common disorder for which shunt placement is advocated as treatment.⁴¹ When obstructive hydrocephalus is caused by an intracranial mass, emergency shunt placement may facilitate management of rapid elevation of intracranial pressure. The goal of CSF shunting is to halt disease progression and improve the neurologic status of the patient. We advocate early shunt placement to reduce the likelihood of severe neurologic and behavioral deficits.

Contraindications

Contraindications to implanting a ventriculoperitoneal shunt include evidence of CSF infection, an elevated CSF protein concentration, a high erythrocyte count in the CSF, or peritoneal inflammation.^{41,52} It is also important to resolve other systemic infections (e.g., urinary tract and skin infections) before scheduling ventriculoperitoneal shunting surgery.⁴¹

Surgical Technique for Ventriculoperitoneal Shunt Placement

For ventriculoperitoneal shunt placement, the patient is aseptically prepared from the cranium, extending past the thorax and caudal abdominal regions, to just over the dorsal and ventral midline of the torso. The torso of the patient is positioned in right lateral recumbency. The head is positioned in sternal or lateral recumbency and elevated approximately 30°. External pressure on the jugular veins should be avoided.

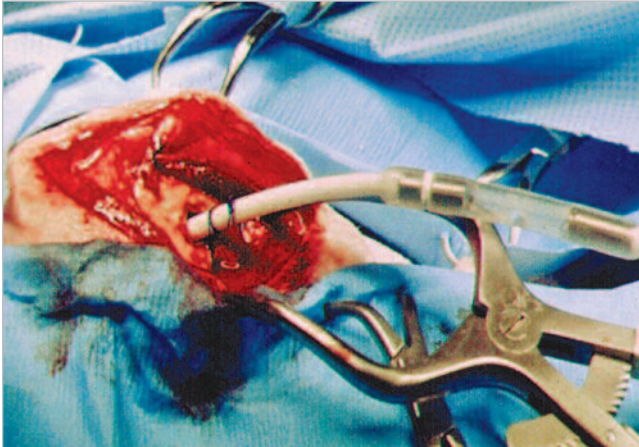


Figure 4. Intraoperative dorsal view of a caudal parietal bone approach, with the ventricular component of a shunt protruding from a burr hole. The shunt was anchored with suture instead of an anchoring device.

Ventricular Approach

A limited lateral rostromentorial craniotomy approach to the cranium is used to create a burr hole.⁵³ An incision is made lateral and parallel to the nuchal crest. The caudal aspect of the temporalis muscle fascia is incised 2 to 3 mm lateral to its origin from the nuchal crest. The temporalis muscle is elevated over the caudal and dorso-lateral portion of the parietal bone nearer to the occipital cortex. With a high-speed pneumatic drill, a burr hole just large enough to pass the shunt and seat the anchor is created in the caudal aspect of the parietal bone and lateral to the nuchal crest. The dura is incised with a blade or spinal needle. A spinal needle may be used to penetrate the cerebral tissue and estimate its thickness as well as collect CSF.⁴¹ The shunt is primed and checked for patency, and air bubbles are removed with isotonic saline before the shunt is inserted into the ventricle. The ventricular catheter is placed through the anchor. The ventricular portion of the shunt is guided through the punctured dura. The ventricular catheter is fed through the burr hole and into the ventricle until free flow of CSF is observed (Figure 4). The shunt is secured by seating the anchor into the burr hole combined with suturing it to the periosteum or overlying temporalis muscle fascia with a “Chinese finger cuff” pattern to the catheter. The shunt is tunneled through the subcutaneous fascia using straight Carmalt or Doyen forceps over the dorsolateral aspect of the cranium, dorsal to the ear pinna, and the dorsolateral cervical region. The shunt valve is positioned over the region of the middle to cra-

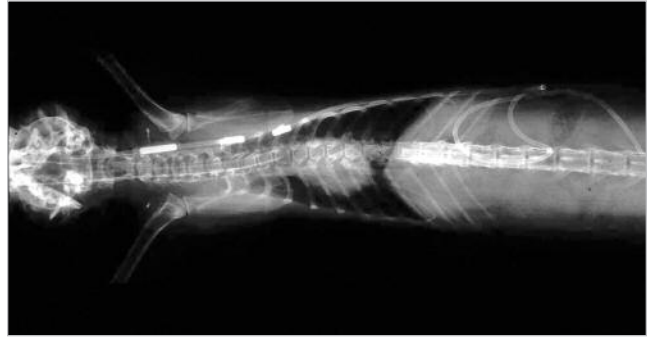


Figure 5. Ventrodorsal whole-body radiograph of a cat after placement of a ventriculoperitoneal shunt.

nial cervical spine.⁴⁸ During the tunneling procedure, excess tension on the shunt should be avoided. Extra shunt length of the proximal catheter is left in the subcutaneous tissue to accommodate for growth in young animals, being careful not to kink the tubing.⁴⁸ The shunt valve is then connected to the proximal catheter.

Flank Approach

The distal portion of the shunt is tunneled caudally through the subcutaneous fascia of the lateral abdominal region for placement into the peritoneum by an open technique.⁵⁴ A shunt passer or “introducer” can facilitate this as well as blunt dissection with long forceps. A flank approach through a small incision is performed in the midabdominal region. Three muscles are encountered: the external abdominal oblique, internal abdominal oblique, and transversus abdominal, which are separated according to their fiber direction. The transverse fascia and peritoneum are tented with thumb forceps and incised to access the peritoneal cavity. Additional fenestrations are made in the distal end of the shunt with a #11 Bard-Parker blade to lessen the chance of blockage by the omentum.⁴⁸ The remainder of the distal catheter is directed into the opening and freely placed. A purse-string suture is placed into the peritoneal fascia with 3-0 monofilament nonabsorbable suture and secured to the extraperitoneal portion of the distal catheter using a “Chinese finger cuff” suture pattern. The distal catheter is further secured to the muscle fascia. Individual muscle layers are closed separately with a simple continuous or interrupted suture pattern using synthetic absorbable suture material while keeping the distal catheter tubing superficial to the muscle layers to later be tunneled into the subcutaneous tissue to meet the proximal portion of the shunt. The subcutaneous fascia and skin are closed in

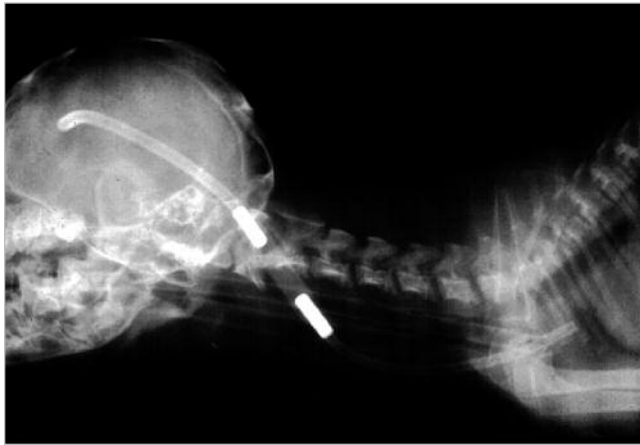


Figure 6. Lateral radiograph of the head and cervical region of a Yorkshire terrier after placement of a ventriculoatrial shunt.



Figure 7. Dorsal view of a Yorkshire terrier with skin necrosis and infection 2 months after placement of a ventriculoatrial shunt. The shunt was subsequently removed, and the dog recovered uneventfully.

routine fashion. The shunt can also be placed in the peritoneal cavity using a ventral midline approach.⁵⁴ Another recommendation is to place at least one-half of the distal catheter into the abdomen.⁴⁸

At the time of shunt placement, CSF fluid may be collected from the lateral ventricle for analysis and culture. A whole-body radiograph should be taken postoperatively to assess shunt placement (Figure 5). A

less dissection than it does with the ventriculoperitoneal shunt, the jugular vein is often too small to accommodate the size of the distal catheter tubing.

Complications of Shunt Placement

Potential complications associated with shunt placement are similar in veterinary and human medicine, but

Early ventriculoperitoneal shunt placement in animals is advocated for surgical management of hydrocephalus and other disorders with CSF accumulation.

cephalosporin antibiotic is usually administered for 24 hours after shunt placement.

Ventriculoatrial Shunt Placement

We have limited experience with placing the distal catheter into the jugular vein and atrium (Figure 6). For this procedure, the skin is incised near the region of the external jugular vein. The jugular vein is isolated and manipulated with stay sutures. The vein is incised and the distal catheter inserted toward the heart. Fluoroscopy can be used to guide the end of the distal catheter just before the jugular vein empties into the right atrium. The jugular vein is ligated distal to the bifurcation of the internal and external maxillary veins and proximal to insertion of the distal catheter.⁴⁶ The distal catheter is secured with 3-0 or 4-0 nonabsorbable sutures. Although placing the distal catheter requires

potential risk factors have been extrapolated from the human literature. In humans, causes for shunt failure include mechanical failure of the shunt, infection, and functional failure from over- or underdrainage.^{47,55,56} The failure rate in humans is as high as 50% within the first year of shunt placement.⁵⁷ Mechanical failure and infections are most likely to occur within the first 6 months after shunt surgery. Retrospective studies^{58,59} in humans also report that complications occur more frequently and were more serious with ventriculoatrial shunts than with ventriculoperitoneal shunts.

Mechanical failure involving shunt obstruction is the most common complication. The shunt system is vulnerable to obstruction at the ventricular catheter, distal catheter, and valve (reservoir).^{6,56} There does not appear to be one valve that functions the best.⁵⁷ Obstruction of CSF flow occurs in most cases at the ventricular portion



Figure 8. Midsagittal, T1-weighted MRI brain scan of a bulldog puppy with hydrocephalus and a shunt (arrow) in the occipital region of the brain.



Figure 9. Lateral view of a cat after shunt placement and intraventricular injection through a patent fontanelle of a radionuclide (^{99}Tc) to assess the patency of a ventriculoperitoneal shunt. Accumulation of radionuclide is evident in the cranial region, and patency is verified by the presence of radionuclide in the peritoneal cavity.

of the catheter.⁵⁷ Tissue or clot debris clogs the lumen of the ventricular catheter; omentum clogs the lumen of the distal catheter.⁶⁰ Other mechanical defects include kinking, breaking, or separation of catheter components.^{41,60} We have observed similar findings associated with mechanical failure of the shunt.

Shunt infection is one of the most serious complications. In humans, the infection rate varies from 4% to 10%.^{57,61} Most shunt infections occur within 3 to 6 months after the surgical procedure and result from contamination of the shunt during surgery. Other sources include wound dehiscence, skin breakdown over shunt hardware, and CSF fistula formation. Pressure necrosis of the skin from the shunt can result in external exposure of parts of the shunt (Figure 7). It is important to keep the

lous surgical technique during shunt procedures significantly reduces the rate of surgical infection in humans.⁶³

Sequelae to overshunting include intracranial hypotension and subdural hematoma, which can arise at varying times following initial shunt placement.⁵⁶ Subdural hematomas occur as a result of shearing of diploic veins as parenchymal volume is reduced. Animals with a thin cerebral cortex are especially predisposed to complications of overshunting.⁴¹

Diagnosis of Shunt Malfunction

It is important to perform serial neurologic examinations to watch for signs of improvement or deterioration. Rapid deterioration of the patient's neurologic

The goal of CSF shunting is to halt disease progression and improve the neurologic status of the patient.

patient well groomed to reduce the predilection for dermatitis. Skin flora (i.e., *Staphylococcus* spp) have been considered the probable source of early shunt infections.^{62,63} Three variables associated with increased incidence of shunt infections in humans include intraoperative CSF leakage, patient age (younger) during shunt placement, and excessive manual contact with the shunt system.⁶⁴ Surgeons should minimize direct contact with the shunt and use double gloves during shunt placement. Meticu-

status (i.e., mentation, respiration, postural reaction deficits) is suggestive of elevated intracranial pressure and shunt malfunction.

Shunt pumping has been considered a way to identify shunt malfunction.⁶⁰ If the pump is depressed easily, the shunt is patent distally. If the pump refills rapidly, the proximal catheter is patent. However, shunt pumping has poor sensitivity and specificity as a diagnostic test in determining malfunction.⁶⁵

Radiologic assessment of the patient often leads to the diagnosis.^{47,66} Survey whole-body radiographs may determine whether the shunt is disconnected or kinked. Computed tomography or MRI is useful in evaluating ventricular enlargement (if the ventricular size has been reduced), intracranial contents, and position of the ventricular shunt components (Figure 8). Scintigraphy is also useful in determining patency of the shunt and evaluating CSF flow (Figure 9).

Tapping a shunt is a consideration only when sepsis or elevated intracranial pressure is suspected.⁶⁰ Some shunts have a separate reservoir designated for this procedure. The collection site should be aseptically prepared, and fluid collection should occur slowly to avoid overdrainage. Complications of draining CSF directly from the shunt include damage to the valve and infection.⁶⁰

Prognosis of Surgically Managed Patients

Depending on the severity of the underlying disease process, the prognosis of patients that have undergone ventriculoperitoneal shunting is guarded to fair. In severe cases of hydrocephalus, neurologic improvement may be minimal. A realistic expectation is improvement of clinical signs but not complete resolution of neurologic deficits. Reconstitution of the cerebral hemispheres after shunting occurs only in the white matter and is characterized by myelin destruction, remyelination, and reactive astrocytosis.^{67–69} Results of early shunt placement in cats with experimentally induced hydrocephalus showed improvement in both cortical afferent and efferent connectivity.⁷⁰ Success rates range from 50% to 90% in dogs having undergone ventriculoperitoneal shunting.⁴¹ One study⁴⁶ of ventriculoatrial shunt placement reported a 75% success rate. Our experience has been similar. It is imperative that the surgeon has experience with shunt placement procedures and uses meticulous aseptic technique during shunt placement. Success rates vary according to the surgeon's experience.⁴⁸

Ventriculoperitoneal shunting in animals is considered an acceptable procedure for treating congenital hydrocephalus and as an adjunct to treat secondary disorders resulting in CSF accumulation.

REFERENCES

1. Dewey CW: Encephalopathies: Disorders of the brain, in Dewey CW (ed): *A Practical Guide to Canine and Feline Neurology*. Ames, Iowa State University Press, 2003, pp 99–178.
2. Summers BA, Cummings JF, de Lahunta A: Malformations of the central nervous system, in Summers BA, Cummings JF, de Lahunta A (eds): *Veterinary Neuropathology*. St. Louis, Mosby, 2000, pp 68–94.
3. Noden DM, de Lahunta A: Causes of congenital malformations, in Noden DM, de Lahunta A (eds): *The Embryology of Domestic Animals: Developmental Mechanisms and Malformations*. Baltimore, Williams & Wilkins, 1985, pp 81–91.
4. Dewey CW, Coates JR, Ducote JM, et al: External hydrocephalus in two cats. *JAAHA* 39:567–572, 2003.
5. de Lahunta A: Cerebrospinal fluid and hydrocephalus, in de Lahunta A (ed): *Veterinary Neuroanatomy and Clinical Neurology*. Philadelphia, WB Saunders, 1983, pp 30–52.
6. Reikate HL: Recent advances in the understanding and treatment of hydrocephalus. *Semin Pediatr Neurol* 4:167–178, 1997.
7. Speake T, Whitwell C, Kajita H, et al: Mechanisms of CSF secretion by the choroid plexus. *Microscopy Res Tech* 52:49–59, 2001.
8. Ganong WF: Circulation through special regions, in Ganong WF (ed): *Review of Medical Physiology*. New York, Lange Medical Books, 2003, pp 614–632.
9. Coates JR: Cerebrospinal fluid proteins, in Feldman BF, Zinkl JG, Jain NC (eds): *Schalm's Veterinary Hematology*. Philadelphia, Lippincott Williams & Wilkins, 2000, pp 917–924.
10. Davson H, Welch K, Segal MB: The proteins and other macromolecules of the CSF, in Davson H, Welch K, Segal MB (eds): *Physiology and Pathophysiology of the Cerebrospinal Fluid*. London, Churchill Livingstone, 1987, pp 583–623.
11. Nilsson C, Stahlberg F, Thomsen C, et al: Circadian variation in human cerebrospinal fluid production measured by magnetic resonance imaging. *J Am Physiol* 262:20–24, 1992.
12. May C, Kaye JA, Attack JR, et al: Cerebrospinal fluid production is reduced in healthy aging. *Neurology* 40:500–503, 1990.
13. Breeze RE, McComb JG, Hyman S, Gilles FH: CSF production in acute ventriculitis. *J Neurosurgery* 70:619–622, 1989.
14. Fishman RA, Ransohoff J, Osserman EF: Factors influencing the concentration gradient of protein in cerebrospinal fluid. *J Clin Invest* 37:1419–1428, 1958.
15. Bhadelia RA, Bogdan AR, Kaplan RF, Wolpert SM: Cerebrospinal fluid pulsation amplitude and its quantitative relationship to cerebral blood flow. *Neuroradiology* 39:258–264, 1997.
16. Enzmann DR, Pelc NJ: Normal flow patterns of intracranial and spinal cerebrospinal fluid defined with phase-contrast cine MR imaging. *Radiology* 178:467–474, 1991.
17. Greitz D, Wirestam R, Franck A, et al: Pulsatile brain movement and associated hydrodynamics studied by magnetic resonance phase imaging. *Neuroradiology* 34:370–380, 1992.
18. Greitz D: Cerebrospinal fluid circulation and associated intracranial dynamics: A radiologic investigation using MR imaging and radionuclide cisternography. *Acta Radiol Suppl* 386:1–23, 1993.
19. Greitz D, Hannerz J: A proposed model of cerebrospinal fluid circulation: Observations with radionuclide cisternography. *Am J Neuroradiol* 17:431–438, 1996.
20. Zmajevic M, Klarica M, Varda R, et al: Elimination of phenolsulphthalein from the cerebrospinal fluid via capillaries in central nervous system in cats by active transport. *Neurosci Lett* 321:123–125, 2002.
21. Selby LA, Hayes Jr HM, Becker SV: Epizootiologic features of canine hydrocephalus. *Am J Vet Res* 40:411–413, 1979.
22. Scott FW, de Lahunta A, Schultz RD, et al: Teratogenesis in cats associated with griseofulvin therapy. *Teratology* 11:79–86, 1975.
23. Csiza CK, Scott FW, de Lahunta A, Gillespie JH: Feline viruses XIV: Transplacental infections in spontaneous panleukopenia of cats. *Cornell Vet* 61:423–439, 1971.
24. Del Bigio MR: Neuropathological changes caused by hydrocephalus. *Acta Neuropathol (Berl)* 85:573–585, 1993.
25. Wunschmann A, Oglesbee M: Periventricular changes associated with spontaneous canine hydrocephalus. *Vet Pathol* 38:67–73, 2001.
26. Kornegay JN: Cerebellar vermian hypoplasia in dogs. *Vet Pathol* 23:374–379, 1986.
27. Nishikawa M, Sakamoto H, Hakuba A, et al: Pathogenesis of Chiari malformation: A morphometric study of the posterior cranial fossa. *J Neurosurg* 86:40–47, 1997.

28. Oldfield EH, Muraszko K, Shawker TH, Patronas NJ: Pathophysiology of syringomyelia associated with Chiari I malformation of the cerebellar tonsils: Implications for diagnosis and treatment. *J Neurosurg* 80:3–15, 1994.
29. Lu D, Lamb CR, Pfeiffer NE, Targett MP: Neurological signs and results of magnetic resonance imaging in 40 cavalier King Charles spaniels with Chiari type I-like malformations. *Vet Rec* 153:260–263, 2003.
30. Rusbridge C, MacSweeney JE, Davies JV, et al: Syringohydromyelia in cavalier King Charles spaniels. *JAAHA* 20:34–41, 2000.
31. Dewey CW, Berg JM, Stefanacci JD, et al: Caudal occipital malformation syndrome in dogs. *Compend Contin Educ Pract Vet* 26:886–896, 2004.
32. Kline KL, Joseph RJ, Averill DR: Feline infectious peritonitis with neurologic involvement: Clinical and pathological findings in 24 cats. *JAAHA* 30:111–118, 1994.
33. de Lahunta A: *Veterinary Neuroanatomy and Clinical Neurology*. Philadelphia, WB Saunders, 1983.
34. Higgins RJ, Vandevelde M, Braund KB: Internal hydrocephalus and associated periventricular encephalitis in young dogs. *Vet Pathol* 14:236–246, 1977.
35. Cantile C, Arispici M, Modenato M, Fatzer R: Hydrocephalus with periventricular encephalitis in the dog. *Zentralbl Veterinarmed A* 44:595–601, 1997.
36. Dewey CW: External hydrocephalus in a dog with suspected bacterial meningoencephalitis. *JAAHA* 38:563–567, 2002.
37. Noden DM, de Lahunta A: Central nervous system and eye, in Noden DM, de Lahunta A (eds): *The Embryology of Domestic Animals: Developmental Mechanisms and Malformations*. Baltimore, Williams & Wilkins, 1985, pp 92–119.
38. Becker SV, Selby LA: Canine hydrocephalus. *Compend Contin Educ Pract Vet* 2:647–652, 1980.
39. Lorenz MD, Kornegay JN: Stupor or coma, in Lorenz MD, Kornegay JN (eds): *Handbook of Veterinary Neurology*. Philadelphia, WB Saunders, 2004, pp 297–322.
40. Simpson ST: Hydrocephalus, in Kirk RW, Bonagura JD (eds): *Current Veterinary Therapy X: Small Animal Practice*. Philadelphia, WB Saunders, 1989, pp 842–847.
41. Harrington ML, Bagley RS, Moore MP: Hydrocephalus. *Vet Clin North Am Small Anim Pract* 26:843–856, 1996.
42. Hudson JA, Simpson ST, Buxton DF, et al: Ultrasonographic diagnosis of canine hydrocephalus. *Vet Radiol* 31:50–58, 1990.
43. Sorjonen DC, Dillon AR, Powers RD, Spano JS: Effects of dexamethasone and surgical hypotension on the stomach of dogs: Clinical, endoscopic, and pathologic evaluations. *Am J Vet Res* 44:1233–1237, 1983.
44. Javaheri S, Corbett WS, Simbartl LA, et al: Different effects of omeprazole and Sch28080 on canine cerebrospinal fluid production. *Brain Res* 754:321–324, 1997.
45. Hoerlein BF, Gage ED: Hydrocephalus, in Hoerlein BF (ed): *Canine Neurology: Diagnosis and Treatment*. Philadelphia, WB Saunders, 1978, pp 733–760.
46. Gage ED: Surgical treatment of canine hydrocephalus. *JAVMA* 157:1729–1740, 1970.
47. Naradzy JFX, Browne BJ, Rolnick MA, Doherty RJ: Cerebral ventricular shunts. *J Emerg Med* 17:311–322, 1999.
48. Levesque DC, Plummer SB: Ventriculoperitoneal shunting as a treatment for hydrocephalus. *Proc 12th ACVIM Forum*:891–893, 1994.
49. Gage ED, Hoerlein BF: Surgical treatment of canine hydrocephalus by ventriculoatrial shunting. *JAVMA* 153:1418–1431, 1968.
50. Few AB: The diagnosis and surgical treatment of canine hydrocephalus. *JAVMA* 149:286–293, 1966.
51. Turner MS: The treatment of hydrocephalus: A brief guide to shunt selection. *Surg Neurol* 43:314–319, 1995.
52. McNae JC: Surgical treatment and other aspects of hydrocephalus in the dog. *Aust Vet Pract* 33:132–137, 2003.
53. Bagley RS: Surgical approaches to the central nervous system: Brain, in Slatter D (ed): *Textbook of Small Animal Surgery*. Philadelphia, WB Saunders, 2002, pp 1163–1173.
54. Bellenger CR: Abdominal wall, in Slatter D (ed): *Textbook of Small Animal Surgery*. Philadelphia, WB Saunders, 2002, pp 405–413.
55. Kang JK, Lee IW: Long-term follow-up of shunting therapy. *Childs Nerv Syst* 15:711–717, 1999.
56. Madikians A, Conway Jr EE: Cerebrospinal fluid shunt problems in pediatric patients. *Pediatr Ann* 26:613–620, 1997.
57. Kestle J, Drake J, Milner R, et al: Long-term follow-up data from the Shunt Design Trial. *Pediatr Neurosurg* 33:230–236, 2000.
58. Keucher TR, Mealey JJ: Long-term results after ventriculoatrial and ventriculoperitoneal shunting for infantile hydrocephalus. *J Neurosurg* 50:179–186, 1979.
59. Smely C, Van Velthoven V: Comparative study of two customary cerebrospinal fluid shunting systems in early childhood hydrocephalus. *Acta Neurochir (Wien)* 139:875–881, 1997.
60. Rekeate HL: Shunt revision: Complications and their prevention. *Pediatr Neurosurg* 17:155–162, 1991.
61. Drake JM, Kestle JR, Milner R, et al: Randomized trial of cerebrospinal fluid shunt valve design in pediatric hydrocephalus. *Neurosurgery* 43:294–303, 1998.
62. Shapiro S, Boaz J, Kleiman M, et al: Origin of organisms infecting ventricular shunts. *Neurosurgery* 22:868–872, 1988.
63. Choux M, Genitori L, Lang D, Lena G: Shunt implantation: Reducing the incidence of shunt infection. *J Neurosurg* 77:875–880, 1992.
64. Kulkarni AV, Drake JM, Lamberti-Pasculli M: Cerebrospinal fluid shunt infection: A prospective study of risk factors. *J Neurosurg* 94:195–201, 2001.
65. Piatt Jr JH: Physical examination of patients with cerebrospinal fluid shunts: Is there useful information in pumping the shunt? *Pediatrics* 89:470–473, 1992.
66. Goeser CD, McLeary MS, Young LW: Diagnostic imaging of ventriculoperitoneal shunt malfunctions and complications. *Radiographics* 18:635–651, 1998.
67. Hale PM, McAllister JP, Katz SD, et al: Improvement of cortical morphology in infantile hydrocephalic animals after ventriculoperitoneal shunt placement. *Neurosurgery* 31:1085–1096, 1992.
68. Kriebel RM, Shah AB, McAllister JP: The microstructure of cortical neuropil before and after decompression in experimental infantile hydrocephalus. *Exp Neurol* 119:89–98, 1993.
69. Bagley RS: Intracranial surgery, in Slatter D (ed): *Textbook of Small Animal Surgery*. Philadelphia, WB Saunders, 2002, pp 1261–1275.
70. Eskandari R, McAllister JP, Miller JM, et al: Effects of hydrocephalus and ventriculoperitoneal shunt therapy on afferent and efferent connections in the feline sensorimotor cortex. *J Neurosurg Pediatr* 101:196–210, 2004.

ARTICLE #3 CE TEST



This article qualifies for 2 contact hours of continuing education credit from the Auburn University College of Veterinary Medicine. Paid subscribers may purchase individual CE tests or sign up for our annual CE program. Those who wish to apply this credit to fulfill state relicensure requirements should consult their respective state authorities regarding the applicability of this program. To participate, fill out the test form inserted at the end of this issue or take CE tests online and get real-time scores at CompendiumVet.com.

I. The choroid plexuses are not located in the

- a. lateral ventricle.
- b. third ventricle.
- c. fourth ventricle.
- d. mesencephalic aqueduct.

2. Which best describes the hydrodynamics of CSF flow?

- a. CSF moves via pulsatile flow of intracranial arterial blood influenced by changes in the cardiac cycle.
- b. CSF flow occurs only in a cranial to caudal direction.
- c. Net movement of water from plasma to CSF is thought to occur by active transport.
- d. Increased systolic blood volume is compensated for by cranial displacement of CSF.

3. Neuropathologic changes associated with hydrocephalus do not include

- a. focal destruction of ependymal lining.
- b. damage of periventricular white matter.
- c. neuronal loss.
- d. intracellular edema.

4. _____ do(es) not decrease CSF production.

- a. Mannitol
- b. Glucocorticoids
- c. Acetazolamide
- d. Omeprazole

5. Which statement regarding medical management of hydrocephalus is incorrect?

- a. Treatment of hydrocephalus is dictated by the underlying cause.
- b. Obstructive hydrocephalus can be managed in the short term.
- c. Spontaneous hydrocephalus is best managed with long-term medical treatment.
- d. Medical treatment of hydrocephalus decreases CSF volume and production.

6. Which is not considered an indication for shunt placement in managing hydrocephalus?

- a. congenital hydrocephalus
- b. secondary obstructive hydrocephalus
- c. patients refractory to medical therapies
- d. CSF infection

7. In hydrocephalic animals treated with ventriculoperitoneal shunt placement, neurologic signs

- a. resolve completely.
- b. may partially improve.
- c. usually do not improve.
- d. may initially worsen.

8. Which complication of ventriculoperitoneal shunt placement is the most common?

- a. shunt obstruction
- b. shunt infection
- c. breaking of the shunt
- d. overshunting

9. Which is the least accurate method to assess patency of a shunt?

- a. shunt pumping
- b. whole-body radiography
- c. radionucleotide scintigraphy
- d. tapping the shunt

10. Which is important for reducing the risk for postoperative shunt infection?

- a. surgical experience
- b. double gloves during shunt placement
- c. aseptic technique
- d. all of the above