Outcome of ventriculoperitoneal shunt implantation for treatment of congenital internal hydrocephalus in dogs and cats: 36 cases (2001–2009)

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**Objective**—To examine outcome data for cats and dogs with congenital internal hydrocephalus following treatment via ventriculoperitoneal shunting to determine treatment-associated changes in neurologic signs, the nature and incidence of postoperative complications, and survival time.

**Design**—Retrospective multicenter case series.

**Animals**—30 dogs and 6 cats with congenital internal hydrocephalus (confirmed via CT or MRI).

**Procedures**—Medical records for dogs and cats with internal hydrocephalus that underwent unilateral ventriculoperitoneal shunt implantation from 2001 through 2009 were evaluated. Data collected included the nature and incidence of postoperative complications, change in clinical signs following surgery, and survival time. To compare pre- and postoperative signs, 2-way frequency tables were analyzed with a 1-sided exact McNemar test.

**Results**—8 of 36 (22%) animals developed postoperative complications, including shunt malfunction, shunt infection, and seizure events. Three dogs underwent shunt revision surgery. Thirteen (36%) animals died as a result of hydrocephalus-related complications or were euthanized. Following shunt implantation, clinical signs resolved in 7 dogs and 2 cats; overall, 26 (72%) animals had an improvement of clinical signs. After 18 months, 20 animals were alive, and the longest follow-up period was 9.5 years. Most deaths and complications occurred in the first 3 months after shunt placement.

**Conclusions and Clinical Relevance**—Results indicated that ventriculoperitoneal shunt implantation is a viable option for treatment of dogs or cats with congenital hydrocephalus. Because complications are most likely to develop in the first 3 months after surgery, repeated neurologic and imaging evaluations are warranted during this period. (J Am Vet Med Assoc 2013;242:948–958)

Internal hydrocephalus is the most common congenital anomaly of the nervous system in dogs.​1,2 A mismatch between production and absorption leads to accumulation of CSF, dilation of the ventricular system, and subsequent increases in intraventricular and intracranial pressures.​3,4 Obstructive or noncommunicating hydrocephalus is characterized by an occlusion within the ventricular system and entrapment of CSF rostral to the site of obstruction.​5,6 The blockage of CSF flow occurs most often in the mesencephalic aqueduct and is usually caused by congenital abnormalities, inflammatory diseases, intraventricular hemorrhage, or the development of tumors.​7,8,10,11 Inadequate absorption or increased production of CSF without impairment of outflow is referred to as communicating hydrocephalus.​5,12,11 An overproduction of CSF is rare and can be the consequence of a choroid plexus neoplasia.​12,13 The underlying cause of the decreased rate of CSF absorption has not been identified yet and is referred to as idiopathic normal-pressure hydrocephalus in human medicine.​8,14 In veterinary medicine, congenital hydrocephalus seems to be more common than the acquired form and is detected predominantly in small and toy breeds of dog.​1,4,5 The increased CSF pressure in patients with hydrocephalus eventually leads to focal destruction of the ependymal lining, compromise of cerebral vessels, damage to periventricular white matter, neuronal injury, and severe white matter atrophy.​2,16,17

In affected dogs and cats, attempts to reduce CSF production through the use of glucocorticoids and diuretics offers only temporary improvement of clinical signs, and surgical treatment is indicated in most cases.​1,4,10 Implantation of silicone-based shunt tubes provides CSF drainage from the cerebral ventricles to the peritoneal cavity or right atrium and compensates for the impaired absorption.​10 Although this surgical procedure in companion animals was established 40 years ago, reports concerning the long-term outcome following shunt implantation in dogs with hydrocephalus are scarce.​10 It is often quoted that shunt failure attributed to plugging by the choroid plexus, glial tissue, and proteinaceous mater-
rial is common and a major factor in patient death, but the actual prevalence of these complications in small animals remains unknown. There is also little information about the improvement of neurologic signs after surgery in dogs and cats. Few case reports in which successful shunt placement and outcome of individual dogs are described have been published. In 1 study, treatment via ventriculoperitoneal shunting of 5 dogs with idiopathic hydrocephalus and 9 dogs with acquired hydrocephalus was analyzed. The aim of the study reported here was to examine outcome data for cats and a larger number of dogs with congenital internal hydrocephalus following treatment via ventriculoperitoneal shunting to determine treatment-associated change in neurologic signs, the nature and incidence of postoperative complications, and survival time. It was anticipated that the study findings would enable veterinarians to provide treatment recommendations and prognostic estimations to owners of dogs or cats with hydrocephalus.

**Materials and Methods**

**Case selection**—The clinical records and CT and MRI findings of dogs and cats with internal hydrocephalus that underwent ventriculoperitoneal shunting in 2001 through 2009 were retrospectively evaluated. For inclusion in the study, each animal had to have a complete medical record, presence of progressive neurologic signs related to internal hydrocephalus, no other underlying neurologic disorders (determined on the basis of results of clinicopathologic and CSF analyses and CT or MRI examinations), diagnosis of internal hydrocephalus via MRI or CT, implantation of a ventriculoperitoneal shunt, and available follow-up information. Findings of MRI that supported the diagnosis of hydrocephalus included presence of distended lateral ventricles with periventricular edema indicated by periventricular hyperintensities on fluid-attenuated inversion recovery images, presence of mass effects with compression of caudal fossa structures, and subjectively increased ventricular volume, compared with findings for breed-matched dogs and cats that were examined via MRI for reasons unrelated to neurologic disorders. Findings of CT that supported the diagnosis of hydrocephalus included identification of distended lateral ventricles and increased ventricular volume, compared with findings for breed-matched dogs that were examined via CT for reasons unrelated to neurologic disorders. The animals were examined at the Small Animal Clinic, Justus Liebig University Giessen; the Department of Clinical Veterinary Medicine, University of Bern; and at the Tierklinik Haar, Haar, Germany. The focus of the study was the examination of patients with congenital internal hydrocephalus; animals with other intracranial lesions (eg, choroid plexus tumors or feline infectious peritonitis), although associated with hydrocephalus, were excluded from the cases reviewed.

**Medical records review and follow-up information**—Data collected included signalment, type and duration of clinical signs, diagnostic findings, preoperative medical treatments, nature and incidence of postoperative complications, postoperative clinical signs, need for revision surgery, postoperative medical treatments, survival time, and cause of death or euthanasia. Information regarding the survival time and development of clinical signs was obtained during follow-up examinations or via telephone interviews with owners. Animals that underwent shunting at the Small Animal Clinic, Justus Liebig University Giessen, were scheduled for regular neurologic reexamination at 2 weeks, 2 months, and 6 months after surgery. A recheck MRI evaluation was scheduled at 2 months after surgery. Animals from the other hospitals had clinical examinations planned at 3 and 6 months after surgery; owners of these cats and dogs were also invited to bring their pet to the Small Animal Clinic, Justus Liebig University Giessen, for reexamination and follow-up MRI evaluation.

**Surgical procedure**—For the surgical procedure, dogs were premedicated with diazepam (0.5 mg/kg [0.23 mg/lb], IV) and l-methadone hydrochloride (0.5 mg/kg, IV). For cats, premedication with midazolam hydrochloride (0.5 mg/kg, IV) and fentanyl (0.006 mg/kg [0.0027 mg/lb], IV) was chosen. For each animal, anesthesia was induced with propofol (4 mg/kg [1.8 mg/lb], IV) and maintained after endotracheal intubation with isoflurane in oxygen. For analgesia, fentanyl was administered as continuous IV drip infusion (0.02 mg/kg/h [0.009 mg/lb/h]) to each animal.

Each patient was placed in lateral recumbency with the head slightly elevated. In most instances, the dog or cat was placed on its right side and the shunt was inserted in the left ventricle. However, if the dilatation of the right ventricle was pronounced, the dog or cat was placed in left lateral recumbency during surgery.

For each animal, the hair on the lateral side of the cervical region, thorax, abdomen, and the whole head was clipped and those areas were aseptically prepared for surgery. A rostrotentorial approach was chosen, and a hole (diameter, 5 to 7 mm) was created in the skull in the region of the caudal portion of the ectorosylvian or suprasylvian gyrus with a pneumatic drill. The exact localization was determined via multiplanar MRI or CT. The dura was opened with a scalpel blade or punctured with an injection needle. Venous hemorrhage was thoroughly controlled by bipolar cautery. The cerebral cortex was perforated with a 20-gauge IV catheter or a ventricular catheter with an introduction stylet, yielding a gush of CSF. The angle and depth of needle insertion needed was determined by examination of MRI or CT images. A commercially available shunt system was inserted. A deflector in the burr hole allowed bending of the shunt without obliteration of its lumen. The proximal catheter was passed beneath the mastiatory muscles and then SC to the destination site at the lower dorsolateral cervical area. A CSF port with a titanium base and a silicone dome for transcervical CSF sample collection was connected to the ventricular catheter and a distal peritoneal catheter that was inserted in the peritoneal cavity via a small incision caudal to the last rib. Depending on the animal’s size, the inserted tube length was 5 to 10 cm, and the catheter was attached to the abdominal wall by a Chinese finger-trap suture. A CSF-flow control valve was not used in all of the cases, but a gravitational ball valve with active opening pressure adjustment was used in animals treated at Tierklinik Haar or at the University of Bern.
All dogs and cats received an antimicrobial (cefazolin sodium or amoxicillin–clavulanic acid) at induction of anesthesia and every 2 hours during surgery. In all but 2 cases, administration of an antimicrobial (cephalexin or amoxicillin–clavulanic acid) was continued after surgery for 1 week. No long-term antimicrobial treatment was required after discharge from the hospital. Opioids or NSAIDs (carprofen and firocoxib) were administered for pain management during hospitalization.

Statistical analysis—All statistical analyses were performed with software. A Kaplan-Meier survival plot was created to describe the outcome of dogs and cats that underwent ventriculoperitoneal shunt implantation. For data analysis, animals lost to follow-up were censored at their last known survival time; nonsurviving animals were defined as cats and dogs that died because of unresolved clinical signs related to hydrocephalus after surgery or were euthanized. A Cox proportional hazard model regression was used to check the impact of age at the time of surgical intervention, seizure events, occurrence of shunt occlusion, and development of infection on survival time. In that analysis, age at the time of surgery was logarithmically transformed because the data distribution was skewed to the right.

Cox regression was also applied to analyze the relationship between duration of preoperative clinical signs and survival time. The duration of clinical signs was logarithmically transformed. To compare clinical signs before and after surgery, 2-way frequency tables were created and analyzed with a 1-sided exact McNemar test. Spearman rank correlation coefficient was used to check a possible relation between duration of clinical signs before surgery and the interval after surgery to resolution of clinical signs as well as between duration of clinical signs before surgery and improvement in clinical signs. In all analyses, a value of $P \leq 0.05$ was considered significant.

Results

Animals—In 2001 through 2009, 33 dogs and 6 cats with congenital internal hydrocephalus were treated by ventriculoperitoneal shunt implantation. Three dogs with congenital hydrocephalus were excluded from the study because of missing follow-up information. A diagnosis of hydrocephalus was made via MRI for 25 dogs and 6 cats and via CT for 5 dogs. Compared with findings for animals without hydrocephalus, distended lateral ventricles with variable degrees of periventricular edema (determined via MRI) and mass effects with compression of caudal fossa structures were identified in all dogs and cats, although the interpretation remains merely subjective. In 5 dogs, results of CT examination only were available; in those images, distended lateral ventricles that appeared to be enlarged, compared with findings for in dogs without hydrocephalus, were evident.

All of the cats and dogs for which data were collected had idiopathic internal hydrocephalus without visible obstruction of the ventricular passage; the hydrocephalus was presumed to be congenital. Among the 6 cats, there were 3 European Shorthairs and 1 each of Maine Coon, British Shorthair, and Persian. Among the 30 dogs, there were 7 Chihuahuas, 3 mixed-breed dogs, 2 West Highland White Terriers, 2 Poodles, and 1 each of Pekingese, Kuvasz, Bernese Mountain Dog, Tibetan Spaniel, Maltese, Boxer, Russian Tsvetnaya Bolonka, Dachshund, Papillon, Cavalier King Charles Spaniel, Australian Shepherd Dog, Bull Terrier, Groenendael, Jack Russell Terrier, Golden Retriever, and Pointer. Nineteen animals were male (2 of which were neutered) and 11 were female (2 of which were neutered); information regarding sex was not available for 6 ani-
mals. Median age at the time of surgical intervention was 8 months (range, 2 to 97 months).

Preoperative clinical signs—Blindness with absent menace response in both eyes and no reaction to moving items was the most common clinical sign (17 dogs and 4 cats [58% of all animals]). In these 21 animals, the pupillary light reflex was intact, and post-tectal blindness was diagnosed. Seventeen dogs and 2 cats (53% of all animals) had ataxia in all 4 limbs. In 11 dogs and 2 cats (36% of all animals), reduced postural reactions were evident; 4 animals were affected in all limbs, 5 animals were affected in the pelvic limbs, 2 dogs were affected in the thoracic limbs, and 2 dogs were affected in 1 limb only. Eleven dogs and 2 cats (36% of all animals) were obtunded, and their reaction to optic and acoustic stimuli was reduced. For 8 dogs and 4 cats (33% of all animals), owners reported seizure events, which were classified as generalized tonic-clonic in all instances. These 12 animals underwent full evaluation to determine the cause of the seizures, including MRI evaluation, CSF analysis, CBC, serum biochemical analysis, assessment of basal ammonia and electrolyte concentrations, bile acids stimulation testing, and routine urinalysis. No other underlying cause for seizures other than internal hydrocephalus was identified. In a 6-month-old Poodle, seizure activity was the only clinical sign, and consequently, idiopathic epilepsy could not completely be excluded. Eleven dogs (31% of all animals) had circling behavior, and for 7 dogs and 1 cat (22% of all animals), the owners complained about behavioral changes (abnormal movements of the tongue, aggressiveness, signs of anxiety, and scratching). Six dogs and 1 cat (19% of all animals) had spontaneous ventrolateral strabismus. Other signs, such as anorexia and signs of pain during neck manipulation, were observed sporadically. The median duration of clinical signs until surgical treatment was 4 months (range, 1 week to 36 months). Most animals that had clinical signs for > 6 months had seizures and were treated with anticonvulsants for idiopathic epilepsy by the referring veterinarian.

Surgery and complications—In all 36 animals, a unilateral ventriculoperitoneal shunt was implanted. The surgical procedure was completed successfully in all instances. One Chihuahua died a few hours after surgery of an unknown cause. All other animals were hospitalized for a median period of 4 days (range, 1 to 9 days) and subsequently treated by the referring veterinarian. For 8 (22%) animals, complications developed after surgery. Two dogs had a combination of 2 complications. With regard to all 8 animals that developed complications, the owners were strongly advised to return their animals for clinical and neurologic examinations, clinicopathologic and CSF analyses, and brain MRI. In 3 of these cases, the owners refused reevaluation and decided to euthanize their animals (2 dogs with a shunt infection and 1 dog with shunt occlusion). Three dogs had a shunt revision surgery. One dog developed progressive seizures 10 days after surgery and died as a result of status epilepticus 3 weeks after shunt placement (before initiation of anticonvulsant treatment). Postmortem examination of that dog was not permitted by the owner.

Shunt occlusion was detected in 4 dogs (11% of all animals) after a median postoperative interval of 5 months (range, 2 to 13 months), and 3 dogs (8.5% of all animals) developed shunt-associated infections at 2, 13, and 21 months after surgery. Infection resulted in the occlusion of 2 shunts, and the owners decided to euthanize their pets at 2 and 13 months after surgery. In these 2 dogs, the shunt was occluded with infectious debris and purulent meningitis was detected during necropsy. Bacterial culture of samples from the ventricular catheter from both dogs yielded positive results, but no additional details of culture results were available from the medical records. One dog that developed a postoperative infection underwent successful shunt replacement 21 months after the first surgical intervention. For that dog, examination of a CSF sample

Figure 2—Transverse CT (A) and T2-weighted MRI (B) images of the brain at the level of the mesencephalon in 2 dogs with hydrocephalus. In both images, dilated lateral ventricles can be seen.
revealed neutrophilic pleocytosis with karyolysis, on the basis of which a diagnosis of shunt infection was made. No bacteria were cultured from the CSF sample or ventricular catheter. One dog with shunt occlusion underwent revision surgery 7 months after shunt implantation, and another was euthanized 2 months after surgery. One dog (2.8% of all animals) died percutely 2 weeks after surgery because of shunt overdrainage and brain collapse, which was detected via postmortem MRI examination. In that dog, MRI revealed collapsed ventricles and a subdural hematoma. Another complication in a dog was a disconnected catheter, which occurred 2 months after shunt implantation; revision surgery was performed successfully.

Outcome—Among the 36 animals, median follow-up time after shunt implantation was 6 months (range, 1 day to 118 months). Animals were lost to follow-up because reevaluation examinations were not performed or telephone updates were not obtained (9 cases). Three dogs were lost to follow-up at 3 or 4 weeks after surgery, 1 cat and 1 dog were lost to follow-up at 3 months after surgery, 1 cat and 1 dog were lost to follow-up at 6 months after surgery, 1 dog was lost to follow-up at 1 year after surgery, and 1 dog was lost to follow-up at 2 years after surgery.

Seven dogs and 1 cat died or were euthanized in the first 2 to 3 months after surgery before the first scheduled reexamination. Of the 19 remaining animals, 14 had complete follow-up examinations. Five owners declined reexaminations of their pets and agreed to provide telephone updates only.

At the time of data analysis, 12 dogs and 1 cat (36% of all animals) had died as a result of hydrocephalus or were euthanized at the owner's request because of a lack of resolution of the clinical signs associated with hydrocephalus. Ten dogs and 2 cats (33% of all animals) were alive, 7 dogs and 2 cats (25% of all animals) were lost to follow-up, and 1 dog and 1 cat (6% of all animals) was euthanized or died of causes unrelated to hydrocephalus. With regard to the latter 2 animals, the cat died of hypertrophic cardiomyopathy and the dog was euthanized because of chronic renal insufficiency. The Kaplan-Meier survival curve revealed that > 80% of all animals were alive at 1 month after surgery; 66% of all animals were alive at 3 months after surgery, and 55% of all animals were alive at 18 months after surgery. One dog lived for > 9.5 years (118 months) after ventriculoperitoneal shunt implantation (Figure 3). The latest recheck examination at which shunt patency and correct shunt placement in this dog were verified via MRI was performed 9 years after the initial surgery.

One cat and 12 dogs died or were euthanized due to unresolved clinical signs related to hydrocephalus after surgery (nonsurviving animals). Three dogs were euthanized because of shunt failure, and 1 cat and 4 dogs were euthanized because of neurologic deterioration or persisting clinical signs. Five dogs died, either of unknown causes (1 day, 10 days, and 6 weeks after surgery) or as a result of status epilepticus (n = 1) or shunt overdrainage (1).

The Cox proportional-hazards regression analysis did not reveal a significant relationship between the animal’s survival time and the age at time of surgery ($P = 0.26$), the occurrence of seizures ($P = 0.93$), shunt occlusion ($P = 0.29$), or shunt infection ($P = 0.85$). No significant ($P = 0.77$) relationship between the duration of preoperative signs and survival time was identified.

Improvement in clinical signs was apparent at a median time of 4 months after surgery. Twenty-one dogs and 5 cats (72% of all animals) had an improvement without further long-term medication, and in 9 dogs and 1 cat, clinical signs did not resolve. One dog developed seizures, which it did not have before surgery. After surgery, 12 animals had improvement in 1 clinical sign, 4 animals had improvement in 2 or 3 clinical signs, and 1 dog had improvement in 4 clinical signs. Seven dogs and 2 cats (25% of all animals) were free of signs after shunt implantation without further medication.

In 2 dogs and 1 cat, ventrolateral strabismus resolved after surgery; vision returned in 4 dogs. In 5 dogs and 2 cats, an improvement of gait abnormalities was detected. Four dogs ceased circling, and postural reactions normalized in 2 dogs. Six dogs and 1 cat returned to full consciousness. In 5 dogs, behavioral abnormalities resolved. Seizure activity was not apparent after surgery in 10 of 12 animals. Four dogs and 2 cats had no detectable seizure events without any further medication (3 of them were administered anticonvulsant medication before shunt implantation); in 3 dogs and 1 cat, seizure activity reoccurred after a clinical sign–free period of 3 to 22 months. For that cat, photo-
barbital treatment was initiated; the cat was seizure free at 15 months after surgery. In 2 dogs, medical treatment of seizures was not necessary. However, before shunt implantation, those dogs were not administered anti-convulsant medication. One dog was euthanized at 3 months after surgery because of increased frequency of seizures.

With regard to pre- and postoperative clinical signs, statistical analysis revealed a significant surgery-related improvement in gait abnormalities ($P = 0.016$), impairment of consciousness ($P = 0.008$), and behavioral changes ($P = 0.031$). Changes in other clinical signs were not significant. On the basis of the calculated Spearman rank correlation coefficient, there was no significant ($r = -0.39; P = 0.066$) correlation between duration of preoperative clinical signs and the interval after surgery to resolution of clinical signs. Also, there was no significant ($r = -0.38; P = 0.07$) correlation between the duration of preoperative clinical signs and surgery-related improvement in clinical signs. Although each of those correlations was not significant, there were indications that such relationships likely existed.

**Discussion**

Compared with other breeds, toy breed dogs with brachycephalic skulls have a higher risk of congenital hydrocephalus.\(^{1,10,23}\) The disorder is not commonly encountered in cats.\(^{1,4,30}\) Nevertheless, in Siamese cats, an autosomal recessive inheritance of hydrocephalus is presumed.\(^{27}\) In the present study, data were obtained for a heterogeneous group of affected dog breeds; however, toy breeds (in particular, Chihuahuas) predominated, in contrast to the distribution of breeds in another recent study.\(^{20}\) Among the affected cats in the present study, a breed predisposition was not apparent but was difficult to evaluate because of the small number of cases. Most of the animals were sexually intact because of their young age.

Unlike another retrospective study,\(^ {20}\) only data from animals with suspected congenital hydrocephalus were included in the present study to rule out complications related to other underlying diseases and to be able to compare survival times without bias. Congenital hydrocephalus was diagnosed in these animals because no underlying cause for the disorder could be identified via neurologic examination, diagnostic imaging, and clinicopathologic and CSF analyses. Moreover, no dog or cat had a history of trauma or inflammatory or infectious disease. At the time of surgery, the median age of the animals in the present study was 8 months, which supported the assumption of a congenital disorder in most cases. Although 7 animals were $> 2$ years old and 4 of them were $> 5$ years old, we proposed a congenital cause of hydrocephalus because, according to published reports, many affected dogs and cats do not develop clinical signs until adulthood\(^ {24}\) and in an epizootiological study,\(^ {4}\) 205 of 564 (36%) of dogs classified as having congenital hydrocephalus were $> 2$ years old. Moreover, 6 of these 7 dogs in the present study were a brachycephalic breed, which is prone to congenital hydrocephalus. Nevertheless, we cannot rule out that an underlying cause for a hydrocephalic disorder remained undetected in some animals in the present study, particularly because some animals were breeds or species (cats) that are only rarely develop congenital hydrocephalus.

In the present study, the clinical signs of the animals were consistent with those described in the veterinary medical literature.\(^ {3,4,20,24,26}\) However, the incidences of the signs differ from those of a recent report.\(^ {20}\) Among the 36 hydrocephalic animals in the present study, 21 (58%) had an absent menace response and deficits of vision, and 19 (53%) had gait abnormalities, whereas obtunded mentation was evident in only 13 (36%). However, among 14 dogs included in the study by de Stefani et al,\(^ {20}\) 10 (approx 70%) had obtunded mentation at the time of hospital admission and 8 (57%) had seizure activity. Only 6 dogs (43%) had absent menace response or gait abnormalities.\(^ {20}\) Perhaps the discrepancy between results of that study and the present investigation is attributable to different pathophysiologic mechanisms in the development of hydrocephalus, given that more than two-thirds of dogs had an acquired form of the disorder in the previous study.\(^ {20}\)

Prior to surgery, seizure events occurred in 12 of 36 (33%) animals, which is comparable to findings in humans with hydrocephalus.\(^ {28,29}\) One-third of hydrocephalic children also have epileptic seizures; other common signs are lethargy, headache, strabismus, vomiting, drowsiness, and papilledema, and blindness occurs occasionally.\(^ {28,30}\) The dogs and cats in the present study generally had more severe neurologic deficits than those described for affected humans. The reason might be a more sophisticated medical care for children, compared with that generally provided for companion animals, which allows diagnosis and treatment of a congenital disorder in an earlier stage. Whether there are differences in pathophysiologic mechanisms in children and companion animals with congenital hydrocephalus remains unclear.

Among the 36 animals in the present study, clinical signs associated with hydrocephalus improved after ventriculoperitoneal shunt implantation in 26 (72%), and 9 (25%) became totally free of clinical signs. These results were similar to those of other studies,\(^ {3,4,31}\) in which success rates for ventriculoperitoneal shunting and ventriculoatrial shunting were 50% to 90% and 75%, respectively. In dogs and cats with a severe hydrocephalus, one can usually expect an improvement of clinical signs after surgery but not complete resolution.\(^ {4}\) The results of the present study supported this conclusion, and neurologic improvement was evident at a median time of 4 months after surgery. This time span was considerably longer than that described elsewhere\(^ {20}\) and might be related to the fact that we assessed only animals with congenital hydrocephalus in which brain damage occurred at an early age. In animals with acquired hydrocephalus, the sudden increase in intracranial pressure causes clinical signs to develop more rapidly; shunting is often performed in the early stages of the disease, whereas in animals with congenital hydrocephalus, the intracranial pressure is assumed to be more chronic and the intermittent changes in intracranial pressure lead to gradual atrophy of the
brain's structure. Moreover, congenital hydrocephalus occurs at a critical point during neonatal development (ie, at the time when neuronal communications are being established) and, therefore, brain damage might be comparatively more severe. In kittens with experimentally induced hydrocephalus, neurologic improvement was evident within 1 week after ventriculoperitoneal shunt placement. A limitation of the present retrospective study was the incomplete follow-up data for some animals and the fact that information regarding postoperative clinical improvement for 5 cases was obtained via telephone interview only. Thus, the owners assessed the degree of neurologic improvement, which is certainly not as reliable a determination as that achieved via clinical examination and could have biased the data.

Gait abnormalities, obtundation, and behavioral changes were clinical signs that significantly improved after ventriculoperitoneal shunt implantation in the present study. Some animals had an improvement of other neurologic deficits as well, but the proportion was not significant. In contrast to results of another retrospective study, seizure activity was not detected after surgery in 10 of 12 dogs in the present study. However, only 6 of those dogs remained seizure free without medication, and in 4 dogs, seizure activity reoccurred, despite the fact that shunt-related problems could not be detected. Moreover, 1 dog developed seizures for the first time after shunt implantation and died as a result of status epilepticus some days later. In children who undergo ventriculoperitoneal shunt placement, postoperative development of shunt epilepsy has been identified in 7.2% (5/69) to 24% (19/78) of cases. A cortical injury at the time of shunt placement in this dog could have been a factor in the development of seizures. Because the owners refused postmortem MRI and necropsy, a potential cortical alteration could not be identified in this dog.

The number of animals in which blindness resolved was very low in the present study. Of 21 animals with postsectal blindness, vision was reestablished after surgery in only 4 dogs. Reparative effects in the cerebral hemispheres is reported to occur mainly in the white matter by removal of myelin debris, remyelination of axons, and reactive astrocytosis. Neuronal repair in the cortical area is incomplete or absent. These processes may explain why ventriculoperitoneal shunting cannot reverse brain damage and why many treated patients have an incomplete resolution of clinical signs. Because tissue destruction correlates with the duration of hydrocephalus, surgical intervention should be performed as early as possible.

In the present study, the diagnosis of hydrocephalus via CT and MRI was achieved mainly on the basis of the assessment of the ventricular size in affected animals versus that expected in breed-matched animals. Additionally, the existence of periventricular edema and a mass effect was evaluated. To date, there is no generally accepted grading system with which to assess the extent of morphological alterations in hydrocephalic patients via diagnostic imaging. Therefore, evaluation remains often merely subjective. Some investigators have provided quantitative measurements of the ventricle size determined via MRI and ultrasonography in dogs with suspected hydrocephalus and in clinical sign–free dogs and cats. By means of such a quantitative method to evaluate the ventricular volume, it is likely that the diagnosis of a hydrocephalic disorder could have been achieved more objectively in the animals included in the present study. However, ventriculomegaly and ventricular asymmetry are common findings in subclinically affected dogs and cats. Also, severity of clinical signs is not associated with the extent of hydrocephalus in diagnostic imaging. Therefore, diagnostic imaging findings should be judged in the light of clinical signs, and the diagnosis of hydrocephalus cannot be solely on the basis of ventricular size.

For the animals in the present study, median follow-up period was only 6 months, whereas the mean follow-up period was 14.5 months. The reason for this rather short median follow-up period was the large portion of animals that died in the first 3 months after surgery (8/36 animals) and the numerous reevaluation examinations in the first 6 months after surgery that owners did not pursue for their pets (7 cases).

In the present study, 13 dogs and cats died or were euthanized, often because of unresolved clinical signs after surgical treatment of internal hydrocephalus with ventriculoperitoneal shunt implantation. One dog died peracute without any obvious cause 1 day after surgery; unfortunately, the owners refused postmortem examination. Reasons for euthanasia included shunt failure (3 animals) and neurologic deterioration or persisting clinical signs (5 animals). Five dogs died of unknown causes (3 dogs) or as a result of status epilepticus (1 dog) or shunt overdrainage (1 dog). There was no significant association between the animal's survival time and the age at time of surgery, occurrence of seizure events after surgery, shunt occlusion, or shunt infection. However, the statistical analysis may have been influenced by the small number of animals in which shunt occlusion or infection developed; thus, these data must be interpreted with caution.

On the basis of the Kaplan-Meier survival curve, 66% of animals in the present study were still alive at 3 months after surgery; at 18 months after surgery, 55% animals were still alive. The survival times of the animals in the present study were longer than described elsewhere. This fact might be due to the decision to include only animals with congenital hydrocephalus because additional underlying disease did not influence the prognosis for these animals. In humans, studies have revealed that approximately 13% to 16% of children with hydrocephalus who are treated surgically die of causes related directly or indirectly to the original disease or its treatment. Most of those deaths occur within the first 24 months after surgery, with a mortality rate of 11% during the first postoperative year. In children, the incidence of a fatal outcome because of shunt failure has been reported to be as low as 1.03%, whereas mortality rates associated with shunt infection can be as high as 35%. In more recent studies, mortality rate was 1.4% within 5 years after surgery and 4% within 36 months after surgery. However, the authors emphasize the difficulties attributing a death solely to hydrocephalus, considering that numerous general postoperative complications or
non–shunt-related deaths can occur. Thus, in human medicine, most studies concentrate more on long-term shunt survival and patency than on the outcome in patients following shunt implantation. Furthermore, in veterinary medicine, survival time is strongly influenced by an owner’s decision to have his or her pet euthanized because of persisting clinical signs or to decline repeated surgical intervention. Therefore, it is difficult to compare survival data for people with survival data for companion animals.

In 8 of the 36 (22%) animals that underwent ventriculoperitoneal shunt implantation in the present study, complications developed following surgery. Slightly higher rates have been reported for dogs and for people, 30% to 50% of ventriculoperitoneal shunts fail in the first year after implantation. However, the data obtained in the present study may have been biased because a few animals were lost to follow-up early after surgery and not all animals with postoperative deterioration of neurologic signs underwent clinical reevaluation. In humans, possible causes for shunt malfunction are over- and underdrainage, mechanical mismatch, occlusion, valve failure, growth effects in children, infection, catheter migration, and other less common complications. In the animals in the present study, shunt-related complications included shunt occlusion, infection, overdrainage, and disconnection and occurrence of seizure events. In humans, the probability of shunt malfunction in a combined review of 12 years of experience was 81%. Pediatric neurosurgical centers generally have a higher failure rate in the first 6 months after shunt implantation, followed by a lower rate during the next 2 years. If shunt failure occurs, a revision surgery becomes necessary.

Shunt occlusion developed in 4 dogs (11% of all animals) in the present study. Two dogs had concurrent shunt infection. In contrast, approximately half of all shunt complications that develop in children are obstructions, and in most cases, the ventricular catheter is affected. Investigators have assumed that the reason for shunt obstruction is an accumulation of tissue or clot debris, blood, or protein; the ingrowth of the choroid plexus; or immune reactions. Cellular and clot debris appear to accumulate particularly in association with an ongoing shunt infection. In children, the risk for shunt occlusion is highest during the immediate postoperative period, but delayed occlusions can develop up to 2 years after implantation and even later. The rate of shunt occlusion in the animals in the present study was lower than that reported for humans. Other than an infectious cause, ingrowth of choroid plexus in the ventricular catheter is the most common reason for shunt occlusion in humans. Perhaps there are anatomic differences of the ventricles and choroid plexus between humans and companion animals that could explain this discrepancy, or perhaps the decrease in ventricle size in humans after shunt implantation is more pronounced, which predisposes humans to shunt occlusion by the choroid plexus ingrowth. The reason for the difference between findings for humans and the animals in the present study remains unclear, and further evaluation of animals in which shunt occlusion develops is necessary to make that determination.

However, in the present study, infection was probably the cause for shunt failure in 2 dogs, and those obstructions developed at 2 and 13 months after surgery, findings that are in accordance with other reports. Most infections develop within 2 months after surgery; 45% of infection-related shunt failures occur within the first month after surgery. Only 6% of shunt failures that occur after 2 years are typically caused by infection. The organisms most commonly isolated from infected shunts are staphylococci. Because of the short period of time in which infections develop after surgery, the infecting agent is most likely introduced at the time of shunt insertion. Three (8.5%) dogs had a shunt infection 2, 13, and 21 months after surgery in the present study; 2 of them were euthanized at the owner’s request because of associated shunt occlusion. The postmortem examination of both dogs revealed purulent meningitis and inflammatory changes in the tissues surrounding the ventricular catheter. Results of bacterial culture of samples of the ventricular catheter were positive in both cases. In the third dog with a shunt infection, in which neutrophilic pleocytosis with evidence of karyolysis was detected in a CSF sample, the ventriculoperitoneal shunt was replaced successfully. Although the results of bacterial culture of the CSF from that dog were negative, the neutrophils with karyolysis in the CSF sample were suggestive of an infectious cause. Nevertheless, immune-mediated inflammation could be a possible cause of the shunt occlusion and neutrophilic pleocytosis. To avoid further development of complications, the ventricular catheter was replaced in that dog. Unfortunately, the specific organisms isolated via bacterial culture for the euthanized dogs were not documented. Therefore, it is difficult to assess whether the reason for shunt infection was contamination with skin flora during the initial surgery, which is likely, or some other cause. These 3 dogs did not have a history of trauma, infectious disease, or sepsis. The late onset of inflammation is in contrast to the reported findings for humans who undergo implantation of ventriculoperitoneal shunts but in agreement with observations made in a recent study involving dogs. Two of the animals that underwent surgery in the present study only received antimicrobials perioperatively because of a change in the regimen of antimicrobial administration in noncontaminated surgical interventions; neither dog developed shunt complications.

In humans, prevention of shunt infection includes several procedures aimed at risk reduction, such as restriction of operating room personnel, vigorous asepsis, performance of shunt implantation first thing in the morning, minimal skin exposure, use of double gloving, and prophylactic administration of antimicrobials. Similar measures should be considered in veterinary medicine to reduce shunt infection rates.

In the present study, mechanical shunt failure occurred in 1 dog as a result of catheter disconnection at the valve. It is probable that the connection between the shunt components was not properly secured during the initial surgery. The disconnection was corrected without complications during a short revision surgery.
In human medicine, the implantation of a valve-controlled shunt catheter for treatment of hydrocephalus is the standard procedure.67,68 Most veterinary surgeons and neurologists use a valve-controlled shunt catheter as well.35 To save costs for the owners and because of a report69 of successful results with implantation of a valve-free catheter system, in 9 animals (8 dogs and 1 cat) in the present study, a ventriculoperitoneal shunt without a pressure-control valve was implanted. It was not possible to compare the outcomes of animals with different shunt types in the present study because of the low number of animals and because in some cases, surgical intervention was quite recent. However, 1 dog (2.8% of all animals) died percutaneously because of overdrainage. In that dog, a valve-free catheter had been implanted. None of the other animals that received a valve-free catheter had problems with overdrainage. Two of 3 dogs that died of unknown causes had received a catheter with a valve; thus, collapse of a ventricle is unlikely, but not excluded, in those dogs. Animals with largely dilated ventricles and thin cerebral cortex seem to be especially predisposed to overshunt syndrome.70 The incidence of overdrainage and slit ventricle syndrome, which involves ventricle collapse due to relative negative pressure within the lateral ventricle,5.6.9 The incidence of overdrainage and slit ventricle syndrome in humans is reported to be 1.2% to 5.7%, which is comparable to the results of the present study, despite use of pressure-control valves in all patients.45,50 As in other animals, humans with very large ventricles, in which the volume is reduced too rapidly, are at greater risk. The cerebral cortex can collapse, and subdural tearing of blood vessels with accumulation of blood or fluid can occur.71 In addition, in young children, shunts with low-pressure valves are often used to prevent ventriculomegaly and to encourage suture closing. Pressure ratio changes and overdrainage can occur with maturation and development, so valve replacement or implantation of an antisiphon device may be required.18 In dogs and cats, siphoning does not seem to be a major concern because the shunt is in a more horizontal orientation, compared with the orientation in humans that sit and walk in an upright position.73

Because of its retrospective nature, the present study had several limitations, especially because it relied on the accuracy of the medical records reviewed and because it involved multiple centers. It was not possible to have completely standardized approaches to surgical preparation, anesthesia, or surgical technique, including consistent implantation of shunt systems with or without a pressure-control valve. The assessment of neurologic signs by owners is an inherent limitation as well as the fact that many owners did not bring their pets for follow-up appointments or refused recheck MRI evaluations because of the necessity for anesthesia. Furthermore, the inclusion of 2 species could have biased data analysis. However, it is likely that pathophysiologic mechanisms involved in the development of hydrocephalus in dogs and cats are similar. Moreover, the clinical signs in dogs and cats with hydrocephalus seem comparable.

On the basis of the results of the present study, the implantation of a ventriculoperitoneal shunt appears to be a viable option for treatment of dogs and cats with congenital hydrocephalus. It is considered a simple surgical procedure and may prolong survival time as well as provide a better quality of life. However, important facts have to be considered before surgery. The prognosis for reestablishment of vision is poor in animals with postseptal blindness, whereas seizure activity may resolve. The prognosis for the improvement of forebrain signs is excellent.

Data obtained in the present study indicated that postoperative complications, including shunt malfunction, shunt infection, and seizure events, may develop in approximately one-quarter of small animals that undergo ventriculoperitoneal shunt implantation. It appears that complications are most likely to develop in the early postoperative period, especially in the first 3 months after ventriculoperitoneal shunt placement; therefore, repeated neurologic and MRI evaluations seem essential during this time. Early diagnosis of complications provides the opportunity to intervene immediately. Owners should be informed in detail about the potential for shunt revision surgery and the expected clinical outcome before shunt implantation. A thorough education of owners and early detection of developing complications would most likely improve the survival rate of hydrocephalic animals treated via ventriculoperitoneal shunt implantation.

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Comparison between manual aspiration via polyethylene tubing and aspiration via a suction pump with a suction trap connection for performing bronchoalveolar lavage in healthy dogs

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Objective—To compare the diagnostic quality of bronchoalveolar lavage (BAL) fluid acquired from healthy dogs by manual aspiration via polyethylene tubing (MAPT) and via suction pump aspiration (SPA) with a suction trap connection.

Animals—12 healthy adult Beagles.

Procedures—BAL was performed with bronchoscopic guidance in anesthetized dogs. The MAPT was performed with a 35-mL syringe attached to polyethylene tubing wedged in a bronchus via the bronchoscope’s biopsy channel. The SPA was performed with 5 kPa of negative pressure applied to the bronchoscope’s suction valve via a suction trap. The MAPT and SPA techniques were performed in randomized order on opposite caudal lung lobes of each dog. Two 1 mL/kg lavages were performed per site. Samples of BAL fluid were analyzed on the basis of a semiquantitative quality scale, percentage of retrieved fluid, and total nucleated and differential cell counts. Results were compared with Wilcoxon signed rank tests.

Results—Percentage of BAL fluid retrieved (median difference, 16.1%), surfactant score (median difference, 1), and neutrophil count (median difference, 74 cells/µL) were significantly higher for SPA than for MAPT. A higher BAL fluid epithelial cell score was obtained via MAPT, compared with that for samples obtained via SPA (median difference, 1).

Conclusions and Clinical Relevance—Results indicated that in healthy dogs, SPA provided a higher percentage of BAL fluid retrieval than did MAPT. The SPA technique may improve the rate of diagnostic success for BAL in dogs, compared with that for MAPT. Further evaluation of these aspiration techniques in dogs with respiratory tract disease is required. (Am J Vet Res 2013;74:523–529)