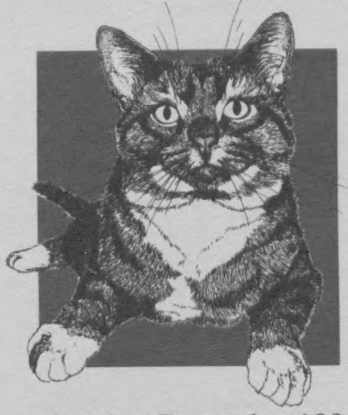

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Spinal Magnetic Resonance (MR) Imaging

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Editors Note: This article is an adaptation of Dr. Rodney S. Bagley's presentation at the 1998 American Association of Feline Practitioners and the Academy of Feline Medicine's Fall Meeting. Some of the terms used in this article were defined in "Advanced Neuroimaging" published in Volume 14 Number 3 of Feline Health Topics.

Spinal disease is a common cause of neurologic dysfunction in animals. As the spinal cord is not seen with survey radiographs, additional, more advanced imaging is needed to clarify most problems involving the spinal cord. Advanced diagnostics for spinal imaging include myelography, computed tomography (CT), and magnetic resonance (MR) imaging. Of the three, only the latter provides for superior anatomical imaging of neural tissue without the risk of radiation exposure to the patient. As MR is used more frequently to image the spinal cord of animals, it becomes obvious that this form of imaging has signifcant advantages over both myelography and CT. We have had the opportunity to image the spinal cord of over 200 dogs and cats with spinal disease in the last 3 years. This article summarizes some of our experience with MR imaging in dogs and cats.

Spinal MR imaging provides images of vertebral bodies, intervertebral disks, spinal cord, exiting spinal nerves and paraspinal anatomy. Sagittal images are initially performed to evaluate the spinal cord. Abnormal areas are reimaged in a transverse plane for further clarification. Cortical bone is dark or hypointense on both T1- and T2-weighted images. The MR appearance of the vertebral column depends mainly on the signal from bone marrow. Medullary bone, rich in fat and blood, is moderately intense (gray) on the T1- and less intense on the T2-weighted images. The ultimate appearance of marrow, however, depends upon the composition of the numerous components that may be present. The spinal cord tends to be isointense to the medullary bone of the vertebral bodies in the T1-weighted study and is typically surrounded by epidural fat which is hyperintense to the spinal cord. This hyperintense outline can be seen on the T1-weighted sagittal and axial images. Epidural fat is easily displaced and hence may not be visible in conditions that result in spinal cord swelling. The intervertebral disks on T1-weighted images are slightly hypointense to isointense to the medullary cavity of the vertebral bodies.

The spinal cord on T2-weighted image is also isointense to the vertebral bodies, however the CSF surrounding the spinal cord emits a bright white

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(hyperintense) signal. By intensifying the CSF signal on a T2-weighted study, a "myelogram effect" can be achieved. This, in essence, provides for an exclusive image of the subarachnoid space. The normal anatomy of the intervertebral disk is best appreciated on a T2-weighted sagittal MR image. Due to its high water content, the normal nucleus pulposus is bright (hyperintense) while the outer layers of the normal annulus fibrosis are homogeneously dark (hypointense).

Diseases

Intervertebral Disk Disease

Following a neurologic examination, a generalized lesion localization is made and imaging is performed in the area of the suspected lesion. T1- and T2-weighted sagittal images are performed to evaluate for signs of an extradural compression of the spinal cord. Disk degeneration is easily seen on the T2-weighted sagittal and transverse (axial) images of the spinal column. Normal hydrated nucleus pulposus has a hyperintense signal compared to the anulus fibrosus. As the nucleus pulposus loses hydration, (seen in chondroid metaplasia or Hansen type I disk disease), the signal becomes less intense and may appear iso- or hypointense to the anulus fibrosus. Most acute disk extrusions of this type appear as a hypointense lesion (disk) on the T2-weighted sagittal image. The T2-weighted sagittal images may also be used to create the "myelogram effect" which is of use in evaluating compromise of the dural sac by the disk (an extradural compression). This is rarely necessary, however, during routine evaluation. Once a lesion is identified on the sagittal image, a transverse image is performed over the area for further clarification of asymmetry.

In instances of lateral disk extrusion, a ventral extradural compression may not be seen with myelography. In these situations, however, the degenerative disk is generally identifiable on the T2-sagittal image. The transverse (axial) image often confirms the laterally extruded disk impinging on a nerve root.

Syringomyelia and hydromyelia

Syringomyelia and hydromyelia are diseases where there are abnormal fluid cavities within the spinal cord.[1] The cystic abnormality with syringomyelia is within the spinal cord parenchyma, where as with hydromyelia, the fluid is within an enlarged central canal. These cystic spinal disease may occur congenitally, due to trauma, or from a tumor. Magnetic resonance imaging is superior in the detection of these intramedullary processes compared to myelography or even CT. Definitive diagnosis is afforded, however, only with histopathologic analysis.

Most syringomyelic or hydromyelic cysts enlarge the spinal cord so that detection by myelography or by intrathecally enhanced CT is feasible. Magnetic resonance imaging may reveal small traumatic cysts that do not enlarge the spinal cord, which therefore may escape detection by other techniques.[1] Cysts typically have discrete, smooth, well-defined borders and a uniform signal intensity which is isointense with respect to cerebrospinal fluid. Although both cysts and tumors may enlarge the cord, the combination of sharply defined margins, homogeneity, and isointensity with cerebrospinal fluid are seldom found in tumors.[1]

Neoplasia

Tumors affecting the spinal cord are grouped as intramedullary (e.g. astrocytomas, oligodendromas,

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and ependymomas); intradural extramedullary (e.g. nerve sheath tumors such as neurofibroma, Schwannoma or meningioma); and extradural (e.g. osteosarcoma, fibrosarcoma, lipoma, lymphoma, and multiple myeloma). Intramedullary tumors generally cause the spinal cord to be expanded. On T1-weighted images, most intramedullary neoplasms have diminished signal intensity with respect to the cord. On T2-weighted images, they usually have a brighter signal than cord. Most tumors have a non-homogenous signal intensity and indistinct margins between tumor and surrounding normal cord. Tumors are typically vascular and invasive, disrupting the normal integrity of the blood brain barrier. Thus, intravenous injection of a contrast agent generally results in some degree of either diffuse or focal enhancement in the area affected by tumor.[2-6] Contrast enhancement does not precisely define the tumor borders; neoplastic cells are generally found outside the enhanced portion of the mass.[1]

Although contrast enhancement can not identify the type of tumor, particular enhancing characteristic have been seen with various tumors. Intradural/extramedullary tumors may be more difficult to identify because these tumors may have little contrast with respect to the adjacent spinal cord. Sagittal images may not detect these tumors as they are often lateral to the spinal cord. Intravenous gadolinium-DTPA (Magnevist, Berlex Laboratories, Cedar Knolls, NJ), enhances these tumors by increasing their signal intensity by 200-500% on T1 weighted images.[1] Coronal and transverse contrast enhanced images are the images of choice to detect this category of tumors.

Extradural tumors may be primary or metastatic. With the exception of lymphoma, most epidural tumors invade the surrounding vertebral body before impinging on the spinal cord .[1] Thus bony destruction of the vertebrae may be a clue as to where to look for the extradural compression. As a rule of thumb, on T1-weighted images marrow signal should be roughly equivalent to disk material.[1] In cases of diffuse marrow involvement, the disk spaces will show a greater signal intensity than the vertebral marrow. Gadolinium is not of help in identifying tumors that involve the vertebral bodies, since gadolinium-enhanced osseous lesions may have an appearance similar to that of normal marrow.

Trauma

Survey radiographs are the quickest and least expensive way of accessing the spinal column for gross fractures and displacement. Computed tomography is an excellent study for detecting small fractures missed on survey radiographs. A clinical study documented the advantages of MR in the areas of ligamentous or severe soft tissue injury, intervertebral disk herniation and spinal cord injury.[7] Magnetic resonance imaging was less useful, however, in the identification of fractures.[7] T2-weighted images are more sensitive than T1- weighted images for the detection of both spinal cord and paraspinal soft tissue injury.[8,9] Despite its inability to delineate bone injury, MR clearly establishes the presence of associated acute spinal pathology. When examination with MR is abnormal, the area affected may then be accessed by CT if further clarification of bone involvement is necessary.

Diskospondylitis

Diskospondylitis is an inflammation of the vertebral bodies and associated intervertebral disk. It is usually a septic condition from a hematogenous source, but occasionally is associated with an adjacent tissue infection or foreign body migration. It is extremely uncommon in cats. *Staphylococcus aureus* is the most common organism cultured from the lesion, blood, or urine. Other organisms associated with diskospondylitis include *Corynebacterium diphtheroides*, *Streptococcus canis*, *Brucella canis*, and *Nocardia*. The neurologic deficits associated with the disease result most often from extradural compression arising from associated inflammatory reaction in the tissue. Radiographic features of diskospondylitis include a collapsed disk space, sclerotic end plates, and prolifera-

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Table 2. Parenchymal hematoma[12]

Stage	Time	Hemoglobin	T1	T2
Hyperacute	<24 hrs	Intracellular oxyhemoglobin	Med.	Med.
Acute	1-3 days	Intracellular deoxyhemoglobin	Long	Short
Subacute				
Early	3+ days	Intracellular methemoglobin	Short	Short
Late	7+ days	Extracellular methemoglobin	Short	Long
Chronic	14+days	Extracellular hemichromes	Med.	Med.
		Intracellular hemosiderin	Med.	Short

tive and lytic lesions of the adjacent vertebral bodies. Vertebral osteomyelitis and disk space infection produce a characteristic imaging appearance in involved structures on MR studies, producing a high degree of sensitivity and specificity (exceeding 90%) in several studies.[10,11] On the T1-weighted image, the involved intervertebral disk space and adjacent vertebral bodies emit a decreased signal. The rough irregular edges of the vertebral bodies are easily seen. On the T2-weighted images, an increase in signal is seen in the involved vertebral bodies. Disruption of the low signal end-plates on both T1 and T2 images is a hallmark of diskospondylitis/osteomyelitis that enables distinction of this entity from neoplasms, which rarely breach the end-plates but frequently and extensively involve the marrow.[1] The progress of treatment can be monitored with MR as the eventual return of signal intensity to normal in both the body and disk space is seen with successful therapy.[1] Disk space degeneration, however, is irreversible.

Hemorrhage

With CT, acute hemorrhage usually becomes hyperdense within an hour as the clot retracts. This hyperdensity lasts a few days and then fades to isodensity and eventually hypodensity.[12] With MR, hemorrhage less than 12 to 24 hours old can not be differentiated from vasogenic edema. In the circulating blood, hemoglobin alternates between the oxyhemoglobin state and the deoxyhemoglobin state. The heme iron in both oxy and deoxy is in the ferrous Fe2+ state. When hemoglobin is removed from the high-oxygen environment of the circulation, the heme iron undergoes oxidative denaturation to the ferric state (Fe3+), forming methemoglobin. Continued oxidative denaturation forms ferric hemochromes (hemosiderin). As red blood cells break down, the various forms of hemoglobin have changing paramagnetic properties influencing the appearance of the clot on the various images (T1- and T2-weighted). Besides the form of hemoglobin present, the signal intensities of a blood clot may vary depending on the operating field strength, the type of signal the operator chooses, and on the technique (T1- vs T2-weighted). Hemorrhage may also vary in appearance depending on where the bleeding occurred (e.g. in the brain, subdural versus intraparenchymal or subarachnoid spaces). The appearance of the varying stages of hemorrhage also depends upon the strength of the magnet.

Table 2 is a guide to the appearance of parenchymal hematomas.

Contraindications

Acutely traumatized patients are not good candidates for MR imaging as life support systems can not safely be transported into the magnetic field. Pacemakers may malfunction in the magnetic field. Ferromagnetic implants may absorb sufficient energy to cause discomfort from local heating. Ferromagnetic hemoclips

maybe removed by the magnetic field. Other metallic implants are usually safe; however, they may cause local image degradation. In veterinary medicine, personnel are required to transport the animal to and from the MR scanner. The above mentioned precautions also apply to these personnel.■

References

1. Stark D.D., Bradley W.G.; Magnetic Resonance Imaging 2nd Ed.; Mosby Year Book; Philadelphia, 1992, Vol 2.

2. Dillon WP, Norman D, Newton TH, et al : Intradural spinal cord lesions: Gd-DTPA-enhanced MR imaging, Radiology 153:189, 1984.

3. Parize PM, Baleriaux D, Rodesch G, et al: Gd-DPTA enhanced MR imaging of spinal tumors, AJNR 10:249, 1989.

4. Sze G, Krol G, Aimmerman RD, and Deck MDF: Intramedullary disease of the spine: diagnosis using Gd-DtPA-enhanced MR imaging, AJNR 9:847, 1988.

5. Sze G: MR of the spine: contrast agents, Radiol Clin North Am 26:1009, 1988.

6. Sze G, Stimac G, Bartlett C, et al: Multicenter study of Gadopentetate Dimeglumine as an MR contrast agent: evaluation in patients with spinal tumors, AJNR 11:967, 1990.

7. Orrison W.W. Jr.; MR and CT of Spinal Trauma: 7th Annual Advances in Mid and Low Field Magnetic Resonance Imaging at 0.064 Tesla; Sept. 1995, Florida.

8. Beers GJ, Raque GH, Wagner GG, Shields CB, Nicholos GR, Johnson JR, Meyer JE: MR imaging in acute cervical spine trauma. J Comp Assist Tomogr 12(5):755-761, 1988

9. Tarr RW, Drolshagen LF, Kerner TC, Allen JH, Partain CL, James AE: Mr imaging of recent spinal trauma. J Comp Assist Tomogr 11(3):412-417, 1987.

10. Modic MT et al: Vertebral osteomyelitis: assessment using MR, Radiology 157:157, 1985

11. Sharif HS, Clark DC, Aabed MY, et al : Granulomatous spinal infections: MR imaging, Radiology 177:101, 1990.

12 Bradley W.G.: Hemorrhage and Brain Iron: In: Stark D.D., Bradley W.G.; Magnetic Resonance Imaging: 2nd ed. Mosby Year Book: V.1, 24: 1992

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Notice of upcoming meeting.....

The Department of Animal Science at Cornell University, in conjunction with The Cat Fanciers' Association, is developing an interactive distance-education course on feline genetics intended for cat breeders, veterinarians, and cat enthusiasts. As part of the development process, the course material will be piloted in a one time only workshop to be held at Cornell University on June 12 and 13, 2000. For more information about the workshop, see *http://www.ansci.cornell.edu/cat/fgsym/cats.html* or call Susan Herbert at (607) 255-4416.

Research Briefs

Comparison of serum fructosamine and blood glycosylated hemoglobin concentrations for assessment of glycemic control in cats with diabetes mellitus

(Authors: D.A. Elliott, R.W. Nelson, C. E. Reusch, E.C. Feldman, L.A. Neal)—This longitudinal cohort study correlates serum fructosamine concentrations with established measures of glycemic control and compares serum fructosamine and blood glycosylated hemoglobin (GHb) concentrations as a means for assessing glycemic control in diabetic cats.

Twenty-six healthy cats, 5 cats with stress-induced hyperglycemia, 15 untreated diabetic cats, and 36 treated diabetic cats were assessed. Control of glycemia was classified and monitored and serum fructosamine and blood GHb concentrations were measured for 12 poorly controlled diabetic cats before and after improving glycemic control, 8 well-controlled treated diabetic cats before and after glycemic control deteriorated, and 5 cats with diabetes mellitus before and after onset of stress-induced hyperglycemia.

The results showed mean serum fructosamine and blood GHb concentrations were significantly higher in untreated diabetic cats compared with healthy cats, and in 24 poorly controlled diabetic cats compared with 12 well-controlled diabetic cats. Mean serum fructosamine and blood GHb concentrations decreased significantly in 12 poorly controlled diabetic cats after improving glycemic control and increased significantly in 8 well-controlled diabetic cats after glycemic control deteriorated. A significant stress-induced increase in mean blood glucose concentration was evident 12 hours after insulin administration, but not in 5 docile diabetic cats that became fractious. Serum fructosamine and blood GHb concentrations are clinically useful tools for monitoring control of glycemia in cats with diabetes mellitus. *(Resource: J. Amer. Vet. Med. Assn. 214: 1794-1798, 1999)*

Primary hyperaldosteronism in two cats

(Authors: S.M. Flood, J.F. Randolph, A.R.M. Gelzer, K. Refsal)—A condition of primary hyperaldosteronism resulting from an adrenal tumor in two cats was characterized by hypertension, hypokalemia, inappropriate kaliuresis, low normal plasma renin activity, and markedly increased serum aldosterone concentration. One of the two cats underwent a laparotomy, and in this case hypertension and hypokalemia resolved following the removal of an adrenal tumor. *(Resource: J. Amer. Anim. Hosp. Assn. 35:411-416, 1999)*

Ulcerative facial and nasal dermatitis and stomatitis in cats associated with feline herpesvirus-1

(Author: A.M. Hargis, P.E. Ginn, J.E.K.L. Mansell, R.L. Garber)—Ulcerative dermatitis of the nasal planum or haired skin of the face associated with intranuclear inclusion bodies compatible with herpesvirus was identified in nine cats. Clinically, lesions were ulcerative and crusted, and often persistent. A tenth cat had focal proliferative ulcerative stomatitis, also associated with intranuclear inclusion bodies. Microscopically, there was necrosis and ulceration associated with prominent eosinophilic inflammation. Intranuclear inclusion bodies were noted in all cases within the surface or adnexal epithelium. Ultrastructural examination of skin from two cats revealed virions morphologically compatible with a herpesvirus. Polymerase chain reaction (PCR) specific for feline herpesvirus-1 on DNA extracted

from fresh-frozen or formalin-fixed paraffin- embedded biopsy samples and/or consensus primer PCR with DNA sequencing performed on DNA extracted from formalin-fixed paraffin-embedded biopsy samples from seven cats revealed that the virus was indistinguishable from feline herpesvirus-1. PCR was negative in one of eight cats tested. *(Resource: Vet. Dermatol. 10: 267-274, 1999)*

Fecal shedding of feline coronavirus in adult cats and kittens in an Abyssinian cattery

(Authors: L.M. Harpold, A.M. Legendre, M.A. Kennedy, P.J. Plummer, K. Millsaps, B. Rohrbach)—This prospective observational study tried to determine patterns of fecal shedding of feline coronavirus (FCV) by cats, age at which kittens first began to shed FCV in their feces, and whether there was any relationship between fecal shedding of FCV and serum antibody titers in adult cats or kittens.

Fifteen adult cats and 18 kittens from a single cattery were observed. Blood and fecal samples were collected from adult cats every other month for 13 months. Serum FCV antibody titers were measured by use of an indirect immunofluorescence assay. A reverse-transcriptase, nested polymerase chain reaction assay was used to detect FCV in feces. Blood and fecal samples were collected from kittens at approximately two-week intervals from 3 weeks to 15 weeks of age.

Adult cats shed FCV intermittently. All adult cats shed virus in their feces at least once during the year, and 4 of 15 shed virus > 75% of the time. Serum antibody titer was not significantly associated with shedding of FCV. For the kittens, median age at the time FCV was first detected in feces was 67 days (range, 33 to 78 days). All except one of the kittens was found to be shedding virus in their feces before or at the time of seroconversion.

Results suggest that serum FCV antibody titers are not a good indicator of shedding of FCV in the feces. Kittens may shed FCV in their feces before they seroconvert, and all kittens in a cattery in which FCV infection is endemic may be infected before 12 weeks of age. *(Resource: J. Amer. Vet. Med. Assn. 215: 948-951, 1999)*

Hip dysplasia: A feline population study

(Authors: G.G. Keller, A.L. Reed, J.C. Lattimer, E. A. Corley)—The study population consisted of cats presented to the University of Missouri-Columbia Veterinary Medical Teaching Hospital from January 1, 1991 through December 31, 1995. Ventrodorsal radiographs including the pelvic region were evaluated for radiographic evidence of hip dysplasia. Each radiograph was evaluated independently by three board-certified veterinary radiologists and a consensus normal of dysplastic evaluation was determined. There were 684 cats from 12 breeds. The data derived from this study indicate the frequency of feline hip dysplasia in this population to be about 6.6% (45/684) and that the incidence appears to be breed dependent. Also, the radiographic appearance of hip dysplasia in cats is different than in dogs. A shallow acetabulum with remodeling and proliferation involving the cranio-dorsal acetabular margin were the most common radiographic signs. Minimal remodeling of the femoral neck was seen. *(Resource: Vet. Radiol. Ultrasound 40:460-464, 1999)*

In-vitro anti-proliferative effects of some anti-tumor drugs on feline mammary tumor cell lines

(Authors: J.S. Muleya, M. Nakaichi, Y. Taura, R. Yamaguchi, S. Nakama)—Six anti-tumor drugs, namely doxorubicin, mitoxantrone, vincristine, cisplatin, recombinant human tumor necrosis factor alpha (rh-TNF alpha) and recombinant feline interferon gamma (rf-IFN gamma) were singly evaluated for their anti-proliferative effects on two feline cell lines (FRM and NAG) derived from mammary adenocarcinoma and grown as monolayers. The investigators obtained concentration response curves that enabled the determination of the

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concentration inhibiting growth by 50 per cent (IC50) for the chemotherapeutic agents with vincristine exhibiting exponential- plateau curves. Differences in anti-proliferative effects of drugs to a given cell line and between the cell lines were also observed. NAC cells were relatively more resistant compared with FRM cells. The relative resistances for NAC cells were 4.19, 12.96, 0.05 and 2.10- fold to doxorubicin, mitoxantrone, vincristine and cisplatin, respectively. FRM cells were more resistant to vincristine at lower concentrations compared with NAC cells. The cells appeared, at least in vitro, least sensitive to rh-TNFa and rf-IFN gamma. Rh-TNF alpha and rf-IFN gamma were 23 and 29 per cent inhibitory to FRM cells and only 13 and 15 per cent inhibitory to NAC cells, respectively. *(Resource: Res.Vet. Sci. 66: 169-174, 1999)*

Pulmonary thromboembolism in cats: 29 cases (1987-1997)

(Authors: C.R. Norris, S.M. Griffey, V.F. Samii)— This retrospective study tries to determine clinical signs, clinicopathologic abnormalities, radiographic findings, histologic abnormalities, and predisposing factors or diseases in cats with pulmonary thromboembolism (PTE).

PTE was confirmed at necropsy in 29 cats. Information on signalment, body weight, history, results of physical examination, results of CBC and serum biochemical analyses, whether PTE was suspected prior to death, type of indwelling venous catheter and duration of venous catheterization, results of thoracic radiography, and whether cats had any concurrent diseases was obtained from medical records. PTE was identified in cats of various ages (median, 8.7 years), weights (median, 4.1 kg), and breeds. The most common owner-reported problems included lethargy (17 cats), anorexia (14), weight loss (10), and difficulty breathing (8); physical abnormalities included lethargy (21), tachypnea or dyspnea (16), and dehydration (13). Clinicopathologic abnormalities reflected concurrent or underlying diseases. Common radiographic abnormalities included pulmonary vessel abnormalities (11), pleural effusion (8), and peripheral noncircumscribed consolidations (6). Underlying or predisposing conditions, including cardiac disease (12), neoplasia (10), corticosteroid administration (8), disseminated intravascular coagulation (5), protein-losing nephropathy (4) or enteropathy (4), immune-mediated hemolytic anemia (2), and sepsis (2) were identified in all cats.

The results suggest that PTE can develop in cats of any age, sex, or breed. Because PTE is a serious, potentially life-threatening disease, it should be suspected in cats with thoracic radiographic changes suggestive of uneven distribution of blood flow between lung lobes, especially in cats that have predisposing factors or diseases. *(Resource: J. Amer. Vet. Med. Assn. 215:1650-1654, 1999)*■

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