CASE REPORT

Syndactyly in a litter of cats

In this case report, we describe the clinical and radiographic features of a litter of kittens affected with complex syndactyly. We also provide guidelines for the diagnosis, possible treatment and prevention of propagation of this condition. This is the first report of syndactyly in a litter of kittens and syndactyly affecting both the pectoral and pelvic limbs.

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The incidence of syndactyly in human beings is approximately two or three per 10,000 live births (Kozin 2003). The mode of transmission of familial syndactyly is considered to be autosomal dominant with variable expressivity and incomplete penetrance. This means that syndactyly in human beings is propagated in family lines, may not be present in full form in each patient (phenotypic variation) and may skip a generation (Kozin 2003). It is also associated with defects of the second chromosome (Kozin 2003).

Syndactyly in human beings is associated with a mutation of the homeobox D cluster-13 (Hoxd-13) gene, fibroblast growth factor receptors, ligand Wnt-1 and transcription factor Msx-2 (Daluiski and others 2001, Arias and Stewart 2002, Gilbert 2003). In human beings, complicated complex syndactyly is seen in association with symbrachydactyly (Poland syndrome), constriction bands and acrocephalosyndactyly (Apert syndrome) (Kozin 2003).

Information about syndactyly in the veterinary literature is sparse. To the authors’ knowledge, all canine (Baum 1889, Gehring and Schröder 1961, Riser 1964, Leipold and Guffy 1973, Dallman and Brown 1980, Carrig and others 1981, Renoy and Balligand 1991, Richardson and others 1994, Schulz and Watson 1995) and feline (Howe 1902, Hays 1917, Boehringer 1975) reports of syndactyly are limited to isolated cases with the exception of one case report describing complicated complex syndactyly in a cat (Searle 1953) and one case report describing complicated complex syndactyly in a family of Australian shepherd dogs (Freeman and others 1988; Table 2). The incidence, aetiology and prognosis of feline syndactyly are unknown. In this case report, we will describe a litter of kittens with syndactyly and review clinical signs, treatment, prognosis, aetiology and possible mode of inheritance of feline syndactyly.

CASE HISTORY

Six kittens from a single litter were presented to the Purdue University Veterinary Teaching Hospital (PUVTH) for evaluation of paw deformities on multiple limbs.
The queen was obtained from the humane society by its foster owner (L. R. T.). The queen’s gestation and vaccination history were unknown. The queen, a domestic shorthair, did not have any gross skeletal deformities. The foster owner (L. R. T.) immediately noticed the paw deformities and markable and no other abnormalities besides the manus and pes deformities were detected. Physical examination findings consisted of abnormal or absent digits, abnormal shape, fusion or absence of digital pads and abnormal shape or fusion of nails. No lameness was detected in any of the kittens during the initial visit. Radiographs of affected limbs showed varying degrees of complex syndactyly. Kitten 1 had syndactyly of all four feet (Table 3 and Fig 1). Kittens 2, 3 and 6 had pelvic limb syndactyly (Table 3 and Figs 2 to 4).

One kitten died before the eight-week re-evaluation appointment. Gross postmortem findings included mesenteric lymphadenopathy and pneumonia. Virology was negative for feline panleukopenia, coronavirus and feline herpes via the fluorescent antibody test. Calici virus was isolated from the spleen and tongue via virus isolation assay. The ultimate cause of death was not determined. Physical examination for the remaining kittens was normal during the second appointment and no lameness was noted during observation.

Kitten 1 had complex incomplete uncomplicated syndactyly of both the pectoral and pelvic limbs. Orthopaedic examination and radiographs determined that kitten 1 (Fig 1) had incomplete separation of the third phalanx of the second and third digits of the right pelvic limb, incomplete separation of the third and second phalanges of the second and third digits of the left pectoral limb, and incomplete separation of the first to the third phalanges of the second to fourth digits of the right and left pelvic limbs. Both pectoral limbs and the right pelvic limb had only three digital pads; the remaining pads were normal. The digital pads of the left pelvic limb were fused into one pad. In addition, two nails were present in the pectoral limbs and right pelvic limb of the affected digits, whereas in the left pelvic limb, there was one large nail for the digits 2 to 4.

Kitten 2 had complex incomplete uncomplicated syndactyly of both pelvic limbs. Orthopaedic examination and radiographs confirmed that kitten 2 had normal pectoral limbs and demonstrated incomplete separation of the third and second phalanges of digits 3 and 4 (Fig 2). The right pelvic limb had three digital pads; the remaining pads were normal. The left pelvic limb had two digital pads; the remaining pads were normal. In addition, two nails were present in each of the affected digits.

Kitten 3 had complex incomplete uncomplicated syndactyly of the right pelvic limb and complex complete uncomplicated syndactyly of the left pelvic limb. Orthopaedic examination and radiographs confirmed that kitten 3 had normal pectoral limbs, incomplete separation of
the first to third phalanges of digits 2 and 3 of the right pelvic limb, and complete fusion of the first to third phalanges of digits 2 and 3 of the left pelvic limb (Fig 3). Each pelvic limb had three digital pads; the remaining pads were normal. In addition, two nails were present in the right pectoral limb for the fused digits and one large nail was present in the left pelvic limb for the affected digits.

Kittens 4 and 5 had normal pectoral and pelvic limbs upon physical and radiographic examination. Kitten 6 had complex incomplete uncomplicated syndactyly of the right and left pelvic limbs. Orthopaedic and radiographic examination revealed that the third and second phalanges of digits 3 and 4 were completely joined in the right pelvic limb and the first to third phalanges of digits 3 and 4 were partially joined (Fig 4). The right pelvic limb had three digital pads, whereas the left pelvic limb had four digital pads. One large nail was present in the right pelvic limb for the affected digits, while two nails were present in the left pelvic limb for the affected digits.

It has now been a year since the kittens were diagnosed with syndactyly. To date, all kittens have been successfully adopted and have been free of lameness.

### DISCUSSION

In this case report we have presented a litter of kittens with various forms of complex uncomplicated syndactyly involving both pectoral and pelvic limbs. Four of six kittens in the litter were affected. In one kitten, all four feet were affected, whereas in the other three only the pelvic limbs were affected. None of the kittens was lame, required medical or surgical treatment or had other gross skeletal anomalies.

Feline syndactyly is a rare condition, and to our knowledge, there are only four previous reports of this condition. Thus, information on the clinical presentation and concurrent anomalies, clinical and surgical management is needed.
radiographic findings and possible links to heredity and methods of prevention is limited. All reported cases were affected with complex syndactyly. In two of the previous reports, the affected cats also had ectrodactyly (Searle 1953) or a family history of polydactyly (Howe 1902). Thus, although the kittens in the present report did not have concurrent anomalies, feline syndactyly may be associated with polydactyly or ectrodactyly (complicated syndactyly). This parallels canine syndactyly, in which approximately half of the reported cases are complicated (Table 2). All affected cats in the previous four and in this report were presented without lameness and the kittens in the present report had not developed lameness at the time of writing. This is similar to canine syndactyly, where all reported cases with complex syndactyly were without lameness and only cases with simple syndactyly were presented with lameness.

Paw changes in syndactyle cats include abnormal or absent digits, abnormal shape, fusion or absence of digital pads and abnormal shape or fusion of nails. Dissection of feline syndactyle legs demonstrated that deep digital flexor muscles to fused digits were often absent (Howe 1902, Hays 1917). In general, the musculature corresponded to the skeletal anomalies, for example, fused digits corresponded to single-muscle units and some muscles were lacking in entirety. Radiographic findings consisted of complete or incomplete joining of phalanges 1 to 3 and metacarpal or metatarsal bones (Howe 1902, Hays 1917, Searle 1953, Boehringer 1975). Both the external and radiographic features of feline syndactyly are similar to those of canine complex syndactyly. However, a lack of cutaneous separation between digits without osseous abnormalities, as seen in canine simple syndactyly (Richardson and others 1994), has not been observed (Howe 1902, Hays 1917, Searle 1953, Boehringer 1975).

An inheritable form of feline syndactyly has been reported (Searle 1953). This report was from a cattery where the female was allowed to breed freely. In addition, affected kittens also had ectrodactyly deformities (Searle 1953). The proportion of kittens with either defect, in successive litters, was 0 of 5, 2 of 6, 2 of 7, 3 of 4 and 0 of 4. Both males and females were affected. It was concluded that ectrodactyly combined with syndactyly was due to the action of a heterozygous gene with a variable expression. In another report, syndactyly was reported in a colony of polydactyle cats (Howe 1902). Although the incidence of syndactyly in this colony was not reported, it may be that syndactyly was an inherited trait, just like polydactyly (Howe 1902). In the present report, environmental and family histories were not available. However, because four of the six kittens in the litter were affected, a genetic background for the syndactyly is not unlikely (Thrusfield 1988, Patterson and others 1989). Thus, it may be that almost all reported feline syndactyly cases have a genetic aetiology.

The presented litter and previous reports yield guidelines for the diagnosis, treatment and prevention of feline syndactyly. Because syndactyly may be a hereditary trait, a detailed history with questions about syndactyly in littersmates and previous litters (including male:female ratio) and other related cats should be obtained. Information on drug exposure, maternal disease or radiation exposure during the first and second trimester of gestation.
should be collected to determine possible causative environmental factors (Towle and Breur 2004). The clinician should perform a thorough general physical examination to detect other congenital anomalies. The paws should be carefully examined for abnormal or absent digits, abnormal shape, fusion or absence of digital pads and abnormal shape or fusion of nails. Radiographic changes may be dramatic and can include complete or incomplete fusion of phalanges and metatarsal or metacarpal bones. The diagnosis of complex syndactyly is based on clinical and radiographic findings. It appears that feline complex syndactyly causes minimal to no discomfort and reported patients affected with this condition have presented without lameness. Therefore medical or surgical treatment of this condition is not recommended. Because the condition may be hereditary, breeding of affected animals should be discouraged and it is recommend that affected animals be neutered or spayed.

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Acknowledgement
We thank Dr Alex Mih of The Indiana Hand Center in Indianapolis, for his help with the preparation of the manuscript.

References
ARIAS, A. M. & STEWART, A. (2002) Patterns in three dimensions. In: Molecular Principles of Animal Development. Eds A. M. Arias and A. Stewart, Oxford University Press, Oxford, pp 363-400
BAUM, H. (1889) Ein Fäll von Syndaktylie beim Hunde. Deutsche Zeitschrift für Tiermedizin 15, 81-90 (In German)
BEHRINGER, B. T. (1975) Foot deformity in a cat. Feline Practice 5, 50
CARR, C. B., WRIGHT, J. A., MOHRIS, E. L., BLENNIS, W. E., ROOT, C. R., HAYDON, G. F. & SUTER, P. F. (1981) Ectrodactyly (split-hand deformity) in the dog. Veterinary Radiology 22, 123-144
DALLMAN, M. J. & BROWN, R. E. (1980) Syndactyly in the dog. Canine Practice 1, 21-24
DALLUM, A., YI, S. E. & LYNG, K. M. (2003) Original communications: the molecular control of upper extremity development: implications for congenital hand anomalies. Journal of Hand Surgery 26A, 9-22
DAO, R. D., SHIN, A. Y., BILLEN, A., OEBE, K. C. & WOOD, V. E. (2004) Surgical treatment of congenital syndactyly of the hand. Journal American Academy Orthopedic Surgery 12, 39-48
FREEMAN, L. E., SNOWBERG, D. P. & SCHREIBER, D. G. (1988) Morphologic characterization of a heritable syndrome of cleft lip/palate, polydactyly, and tibial/tibular dysgenesis in Australian Shepherd dogs. Anatomia Histologia Embryologia 17, 81
GERRING, H. & SCHREIBER, U. (1961) Syndaktylie und Oligodonte bei einem Papillon-Rüden. Deutsche Tierärztliche Wochenschrift 68, 352-364
GRIEVE, E. (1998) Syndactyly. In: Congenital Malformation of the Hand and Forearm. Ed D. Buck-Gramcko. Churchill Livingstone, London, pp 131-141
GILBERT, S. F. (2003) Development of the tetrapod limb. In: Developmental biology, 7th edn. Sinauer Associates Inc. Sunderland, pp 523-546
HEYS, G. P. (1917) A case of syndactyly. Journal of Morphology 30, 65-82
HOEVE, F. (1932) A case of abnormality of cats’ paws. American Naturalist 36, 511-526
KUCIN, S. H. (2003) Current concepts review: upper-extremity congenital anomalies. Journal of Bone and Joint Surgery 85A, 1564-1576
LEFOLD, H. W. & GIFFTY, M. M. (1973) Syndactyly in a German shepherd dog. Veterinary Medicine/Small Animal Clinician 68, 910-911
NOGID, D. & DE LA LUNTA, A. (1985) Limb development. In: The Embryology of Domestic Animals: Developmental Mechanisms and Malformations, Eds D. M. Noden and A. de Lahunta, Williams & Williams Co, Baltimore, MD, USA, pp 196-210
OGEN, J. A. & GIOGEN, P. (1987) Prenatal skeletal development and growth of the museulcoskeletal system. In: The Scientific Basis of Orthopaedics. Eds J. A. Albright and R. A. Brandt. Appleton & Lange, Norwalk, pp 47-89
PATTERSON, D. F., AGUIRRE, G. A., FITE, J. C., GIGER, U., GREEN, P. L., HAKINS, M. E., JIZIY, P. F., NOGID, D., DE LA LUNTA, A. & MEXIER-WALENS, Y. N. (1988) Is this a genetic disease. Journal of Small Animal Practice 30, 127-139
RENY, B. F. & BILLAUD, M. (1991) Un cas de syndactylie chez le chien. Annales de Medecine Veterinaire 135, 43-44 (In French)
RICHARDSON, E. F., WOE, P. D. & HOFFMAN, L. A. (1994) Surgical management of syndactyly in a dog. Journal of the American Veterinary Medical Association 205, 1149-1151
RIGER, W. H. (1964) What is your diagnosis? Journal of the American Veterinary Medical Association 145, 169-170
SCHULTZ, V. A. & WATSON, A. G. (1995) Limb-based transitional vertebra and thoracic limb malformations in a Chihuahua puppy. Journal of the American Animal Hospital Association 31, 103-106
SMIRLE, A. G. (1953) Hereditary “split-hand” in the domestic cat. Annals of Eugenics 17, 279-282
SWANSON, A. B. (1976) A classification for congenital limb malformation. Journal of Hand Surgery 1, 8-22
THURSFIELD, M. (1988) Is it hereditary?: the cause of disease. Journal of Small Animal Practice 29, 603-609
TONG, H. A. & BREUR, G. J. (2004) Dysostoses of the canine and feline appendicular skeleton. Journal of the American Veterinary Medical Association 225, 1685-1692