Congenital and inherited neurologic diseases in dogs and cats: Legislation and its effect on purchase in Italy

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Abstract

Many of the congenital neurologic diseases can result in incapacity or death of the animal. Some of them, such as idiopathic epilepsy and hydrocephalus, exhibit breed or familial predisposition and a genetic basis was proved or suggested. Some diseases can be presumptively diagnosed after a detailed signalment (breed predisposition), history (e.g. family history because many of these defects have familial tendencies), and through physical exam; other diagnostic methods (radiography, computed tomography, magnetic resonance, electrophysiologic tests, etc.) can provide supportive evidence for the congenital defect and help to confirm the diagnosis. Some cases can lead to civil law-suits when the lesions are congenital, but not easily recognizable, or when the lesions are hereditary but tend to became manifest only after some time (more than 12 months after the date of purchase, e.g., after the vice-free guarantee period has expired). Moreover, quite frequently an early diagnosis is not made because there are delays in consulting the veterinarian or the general practitioner veterinarian does not perceive subtle signs. This study was designed to focus on the medico-legal aspects concerning the buying and selling in Italy of dogs and cats affected by congenital and hereditary neurologic diseases that could constitute vice in these animals. While adequate provisions to regulate in detail the various aspects of pet sale have still to be drawn up by legislators, it may be helpful to involve breeders, by obliging them by contract to extend guarantees in the case of hereditary lesions, including neurologic diseases.

Keywords: buy/sell, cat, dog, hereditary disease, nervous system.

Introduction

To most European citizens, companion animals become more than just animals in the home. Cats, dogs, and other companion animals often occupy the status of the beloved family member. Despite this status which most of these animals gain in the home, they are legally considered mere goods [1] or commodities during the sales process (purchase). Indeed, companion animals have no independent status or personhood in the legal world. In the eyes of the law, animals are property: They are goods to be bought and sold, acquired, and maintained.

The term “goods” is meant to afford buyers and sellers certain rights and responsibilities in the transaction. The terms “possession” and “property” are often used synonymously. Nevertheless, there is an important difference. The term “property” (dominium, proprietas) refers to the right to dispose of a thing, while the term “possession” (possessio) designates the actual power over a thing.

The Italian Civil Code (article 812) at present considers animals as res, i.e., a thing (as property) as opposed to a person, who has rights [2,3].

The purchase (emptio venditio) is a contract that obliges one party (venditor) to provide a thing (a commodity) and the other party (emptor) to provide payment. It is a bilateral contract since both parties are creditors and debtors at the same time - the venditor owes the commodity and the emptor owes the money.

The seller is always liable for defects of the commodities (including animals). For redhibitory defects (vice), however, he/she is only liable if he kept them secret on purpose or if he guaranteed a faultless product.

In veterinary legal medicine, the illness must be considered a vice [4] in those cases where it renders an animal unsuitable for its specified use or significantly reduces its value.

An animal, which is unsuitable for its specified use, must be affected with a behavioral character defect or illness. The illness causes a disturbance in normal organic functioning, which may be localized or generalized, following an anatomic alteration which will inevitably cause an appreciable permanent or temporary impairment.

To qualify as vice, however, illness must be [4,5]:

1. Pre-existing or have a pre-existing cause. It is clear that congenital and hereditary neurologic diseases can come under this heading since most of them are typically primary; for this reason, it is difficult to demonstrate that environmental factors have contributed to their phenotypization;
2. Hidden. This means that it cannot be discovered
by an ordinary inspection or examination; or rather, it is not easily recognized at the moment of purchase, and it cannot be detected using the normal due diligence.

3. Serious or chronic so as to affect the use of the animal or such that, if the buyer knew of the disease, he/she would not enter into the contract. In fact, defect renders the thing sold unfit for the use for which it is intended or diminishes its fitness for the intended use to such an extent that the vendee would not have bought or would have given a lower price if he had been aware of the defect.

In dogs and cats, there are many neurologic conditions of proven or suspected hereditary origin, both congenital and non-congenital, which may constitute vice in the buying and selling of these animals.

These cases would be lead to civil law-suits when the lesions are congenital, but not easily recognizable, or when the lesions are hereditary but tend to become manifest only after some time (more than 12 months after the date of purchase, i.e., after the vice-free guarantee period has expired). Moreover, an early diagnosis quite frequently is not made because there are delays in consulting a veterinarian or the general practitioner veterinarian does not perceive subtle signs.

This study will present an overview of general contract law related to sales of dogs and cats as well as the rights and remedies of buyers under Italian law.

Finally, the study will highlight some concerns facing buyers, especially when purchasing companion animals (dogs and cats) affected by congenital and hereditary neurologic diseases.

**Current Italian Law**

In Italy, the purchase of animals is regulated by the Civil Code (article 1470 and subsequent) and dated back to 1942. These articles regulate the purchase of real property, and they are also adopted in the field of the purchase of animals.

In fact, the only article concerning animal purchase in Italy is article 1496 of the Italian Civil Code. It states that the guarantee against vices in the sale of animals is regulated by special laws or by local usage or, if these are lacking, by article 1490 of the Civil Code and subsequent. The latter reads as follows: “The seller is obliged to guarantee that the object sold is immune from vices which make it unsuitable for the usage to which it is destined or that decrease the value of it in an appreciable way.”

In Italy, only when the animal is affected by a serious or chronic vice that is pre-existing and not easily recognizable, will the buyer be able, with respect to the terms of expiry and to rules contained in article 1495 of the Italian Civil Code, to exercise one of the two legal actions foreseen by the Code, namely, redhibitory action or estimatory action (remedies of the vendee). “Redhibitory action” (actio redhibitoria) means rescission of sale, and “estimatory action” (actio estimatoria or quanti minoris) means reduction of the price (article 1492 Civil Code).

The time limits for rescission or exaction of proportionate reduction of the purchase price against the vendor under article 1495 are 8 days from the day of delivery and 1 year from the date of purchase.

**Congenital and Inherited Neurological Diseases**

Physiologic nervous system development may be affected by inherited or congenital factors, e.g., in utero exposure to viral (parvovirus-induced cerebellar hypoplasia) or teratogenic substances (cerebral bifidum, spina bifida, abnormal atlanto-occipital articulation, exencephaly, and hydrocephalus associated with griseofulvin therapy) [6,7].

Many kinds of anomaly result from pathologic nervous system development:

i. Macroscopic malformations (hydrocephalus, cerebellar hypoplasia, lissencephaly, hydromyelia, syringomyelia, spinal dysraphism) [8-15];

ii. Microscopic lesions (involving the inner ear in congenital deafness and vestibular syndrome and the gray or white matter in degenerative diseases) [9,16,17];

iii. Alterations involving molecular structures only (decreased number of acetylcholine receptors in congenital myasthenia gravis, enzymatic deficiency in storage diseases, abnormalities of neurotransmitters or their receptors in narcolepsy) [9,18-24].

In some cases, the involvement of the nervous system results from malformations of the skull or the spine (meningocele and myelomeningocele in association with spina bifida, spinal cord compressions caused by hemivertebrae, butterfly vertebrae, transitional vertebrae, and malformation of the dens of the axis) [9,10,12,25-30].

In many cases, the clinical signs are apparent at birth and there is no progression of the disease, but sometimes, e.g., in degenerative diseases, the onset of symptoms occurs in the first few months or years of life, and they are slowly progressive and lead to the animal’s death (Table-1). Clinical signs related to idiopathic epilepsy and spinal cord compression due to congenital vertebral anomalies can develop in adult animals (Table-1) [25,31]. Mild neurologic signs, although present at birth, can be mistaken for the normal awkwardness of the puppies by their owner.

Some congenital diseases may be diagnosed by physical examination because there are typical signs (e.g., hydrocephalus, myelomeningocele, and meningoencephalocele). Appropriate diagnostic tests are required for many congenital neurologic diseases (Table-1). In some cases, the diagnosis can be made...
### Table-1: Breeds affected by inherited neurologic diseases [9,10,34-38].

| Disease                                           | Dog                                                                 | Cat            |
|---------------------------------------------------|----------------------------------------------------------------------|----------------|
| **Abiotrophies**                                  | American Staffordshire Terrier, Australian Kelpie, Beagle, Bernese Mountain Dog, Blue Terrier, Bobtail, Border Collie, Breton Spaniel, Cairn Terrier, Chow Chow, Cocker Spaniel, Doberman Pinscher, Fox Terrier, German Shepherd Dog, Golden Retriever, Gordon Setter, Kerry Samoyed, Labrador Retriever, Lapland, Pointer, Poodle, Rhodesian Ridgeback, Saluki, Wire-haired Great Dane | Domestic, Siamese |
| Incidence: Rare                                   |                                                                      |                |
| Prognosis: Guarded to poor (there are different degrees of the disease, which is slowly progressive) |                                                                      |                |
| Age of onset of clinical signs: 6 weeks to 5 years |                                                                      |                |
| Clinical signs: Cerebellar ataxia, paresis/paralysis |                                                                      |                |
| Diagnosis ante mortem: Yes (American Staffordshire Terrier); in other breeds only post mortem |                                                                      |                |
| **Ceroid lipofuscinosis**                         | Australian Cattle Dog, Blue Heelers, Blue Heelers, Border Collie, Chihuaha, Cocker Spaniels, Dachshund, English Setter, Salukis, Tibetan Terrier, Yugoslavian Sheep Dog | Domestic, Siamese |
| Incidence: Rare                                   |                                                                      |                |
| Prognosis: Guarded to poor                        |                                                                      |                |
| Age of onset of clinical signs: 4 months to 9 years (most animal<2 years) |                                                                      |                |
| Clinical signs: Personality change, visual impairment, ataxia, seizures |                                                                      |                |
| Diagnosis ante mortem: Yes (genetic, histopathology of skin biopsies) |                                                                      |                |
| **Congenital myasthenia gravis**                  |                                                                      |                |
| Incidence: Rare                                   |                                                                      |                |
| Prognosis: Guarded                                |                                                                      |                |
| Age of onset of clinical signs: 6-8 weeks         |                                                                      |                |
| Clinical signs: Weakness worsening after exercise  |                                                                      |                |
| Diagnosis ante mortem: Yes (electrodiagnostic tests, pharmacological tests, muscle biopsy) |                                                                      |                |
| **Demyelinating diseases of peripheral nerves**   |                                                                      |                |
| Incidence: Rare                                   |                                                                      |                |
| Prognosis: Poor                                   |                                                                      |                |
| Age of onset of clinical signs: 3 weeks to 3 months |                                                                      |                |
| Clinical signs: LMN paresis/paralysis             |                                                                      |                |
| Diagnosis ante mortem: Yes (peripheral nerve biopsy) |                                                                      |                |
| **Fucosidosis**                                   |                                                                      |                |
| Incidence: Rare                                   |                                                                      |                |
| Prognosis: Poor                                   |                                                                      |                |
| Age of onset of clinical signs: 6 months to 2 years |                                                                      |                |
| Clinical signs: Various, predominantly behavioral and motor |                                                                      |                |
| Diagnosis ante mortem: Yes (enzyme analysis, genetic tests) |                                                                      |                |
| **Gangliosidosis**                                |                                                                      |                |
| Incidence: Rare                                   |                                                                      |                |
| Prognosis: Poor                                   |                                                                      |                |
| Age of onset of clinical signs: 2 months to 1.5 years |                                                                      |                |
| Clinical signs: Vision deficits, lethargy, gait disturbances |                                                                      |                |
| Diagnosis ante mortem: Yes (blood cytology, genetic tests) |                                                                      |                |
| **Globoid cell leukodystrophy**                   |                                                                      |                |
| Incidence: Rare                                   |                                                                      |                |
| Prognosis: Poor                                   |                                                                      |                |
| Age of onset of clinical signs: 5 weeks to 2 years |                                                                      |                |
| Clinical signs: Cerebellar ataxia, paraparesis/plegia amaurosis, behavior disturbance |                                                                      |                |
| Diagnosis ante mortem: Yes (genetic tests)        |                                                                      |                |
| **Glucocerebrosidosis**                           |                                                                      |                |
| Incidence: Rare                                   |                                                                      |                |
| Prognosis: Poor                                   |                                                                      |                |
| Age of onset of clinical signs: 6-8 months        |                                                                      |                |
| Clinical signs: Ataxia                            |                                                                      |                |
| Diagnosis ante mortem: Yes (enzyme assay)         |                                                                      |                |
| **Glycogenosis**                                  |                                                                      |                |
| Incidence: Rare                                   |                                                                      |                |
| Prognosis: Poor                                   |                                                                      |                |
| Age of onset of clinical signs: 5 months to 1.5 years |                                                                      |                |
| Clinical signs: Two forms: Early neonatal death or progressive neuromuscular weakness |                                                                      |                |
| Diagnosis ante mortem: Yes (genetic test in IV type in the cat) |                                                                      |                |

(Contd...)
Table-1:  

| Disease                                                                 | Dog                                                        | Cat                                      |
|------------------------------------------------------------------------|------------------------------------------------------------|------------------------------------------|
| **Hemivertebrae, Butterfly vertebrae**                                  |                                                            |                                          |
| Incidence: Common                                                      |                                                            |                                          |
| Prognosis: Good to poor                                                |                                                            |                                          |
| Age of onset of clinical signs: Variable                                |                                                            |                                          |
| Clinical signs: Asymptomatic or ataxia, paresis/paralysis due to acute or chronic spinal cord compression |                                                            |                                          |
| Diagnosis ante mortem: Yes (RX, CT)                                    |                                                            |                                          |
| **Hydrocephalus**                                                      |                                                            |                                          |
| Incidence: Common                                                      |                                                            |                                          |
| Prognosis: Guarded                                                     |                                                            |                                          |
| Age of onset of clinical signs: Shortly after birth                    |                                                            |                                          |
| Clinical signs: Enlargement of the skull, open sutures, and fontanelles, behavioral changes, depression, seizures, amaurosis, ataxia |                                                            |                                          |
| Diagnosis ante mortem: Yes (ultrasonography, TC, MR, EEG)              |                                                            |                                          |
| **Hypomyelinating diseases**                                           |                                                            |                                          |
| Incidence: Rare                                                        |                                                            |                                          |
| Prognosis: Guarded (not progressive and improvement can occur)         |                                                            |                                          |
| Age of onset of clinical signs: Shortly after birth                    |                                                            |                                          |
| Clinical signs: Tremors                                                |                                                            |                                          |
| Diagnosis ante mortem: No (post mortem histopathology)                 |                                                            |                                          |
| **Idiopathic epilepsy**                                                |                                                            |                                          |
| Incidence: Very common in dogs, rare in cats                           |                                                            |                                          |
| Prognosis: Guarded                                                     |                                                            |                                          |
| Age of onset of clinical signs: 6 months to 5 years                    |                                                            |                                          |
| Clinical signs: Seizures                                              |                                                            |                                          |
| Diagnosis ante mortem: Yes (by excluding other causes of seizures. No positive diagnostic signs can substantiate the diagnosis) |                                                            |                                          |
| **Inherited deafness**                                                |                                                            |                                          |
| Incidence: Common                                                      |                                                            |                                          |
| Prognosis: Good quod vitam                                             |                                                            |                                          |
| Age of onset of clinical signs: Shortly after birth                    |                                                            |                                          |
| Clinical signs: Deafness                                              |                                                            |                                          |
| Diagnosis ante mortem: Yes (electrodiagnostic tests)                   |                                                            |                                          |
| **Leukodystrophies**                                                  |                                                            |                                          |
| Incidence: Rare                                                        |                                                            |                                          |
| Prognosis: Poor                                                        |                                                            |                                          |
| Age of onset of clinical signs: Few weeks to few years                 |                                                            |                                          |
| Clinical signs: Ataxia, paresis/paralysis, seizures                    |                                                            |                                          |
| Diagnosis ante mortem: No (post mortem histopathology)                 |                                                            |                                          |
| **Lissencephaly**                                                     |                                                            |                                          |
| Incidence: Rare                                                        |                                                            |                                          |
| Prognosis: Guarded                                                     |                                                            |                                          |
| Age of onset of clinical signs: Shortly after birth-12 months          |                                                            |                                          |
| Clinical signs: Behavioral changes, seizures, amaurosis, visual deficit |                                                            |                                          |
| Diagnosis ante mortem: Yes (CT, MR)                                   |                                                            |                                          |
| **Malformations of the dens of the axis**                              |                                                            |                                          |
| Incidence: Common                                                      |                                                            |                                          |
| Prognosis: Guarded                                                     |                                                            |                                          |
| Age of onset of clinical signs: <1 year                                 |                                                            |                                          |
| Clinical signs: Cervical pain, tetraparesis/paralysis                  |                                                            |                                          |
| Diagnosis ante mortem: Yes (RX, CT)                                   |                                                            |                                          |
| **Mannosidosis**                                                      |                                                            |                                          |
| Incidence: Rare                                                        |                                                            |                                          |
| Prognosis: Poor                                                        |                                                            |                                          |
| Age of onset of clinical signs: 2-7 months                             |                                                            |                                          |
| Clinical signs: Facial dysmorphism, ataxia, tremors, altered behavior, seizures |                                                            |                                          |
| Diagnosis ante mortem: Yes (genetic tests)                            |                                                            |                                          |
| **Mucopolysaccharidosis**                                              |                                                            |                                          |
| Incidence: Rare                                                        |                                                            |                                          |
| Prognosis: Poor                                                        |                                                            |                                          |
| Age of onset of clinical signs: 3-10 months                            |                                                            |                                          |
| Clinical signs: Progressive paresis                                   |                                                            |                                          |
| Diagnosis ante mortem: Yes (urinary biochemical tests, genetic tests (in mucopolysaccharidosis VI of the cat) |                                                            |                                          |

(Contd…)

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**Table-1: (Continued)***

| Disease                                    | Dog                                      | Cat                                      |
|--------------------------------------------|------------------------------------------|------------------------------------------|
| **Narcolepsy**                             | Doberman Pinscher, Labrador Retriever    |                                          |
| Incidence: Rare                            |                                          |                                          |
| Prognosis: Good quaod vitam (the severe forms can be invalidating) |                                          |                                          |
| Age of onset of clinical signs: <6 months  |                                          |                                          |
| Clinical signs: Recurring sudden attacks of sleep and/or loss of muscle tone |                                          |                                          |
| Diagnosis ante mortem: Yes (clinical diagnosis, electrodiagnostic tests, genetic tests) |                                          |                                          |
| **Peripheral axonopathies**                | Boxer, German Shepherd Dog, Rottweiler   |                                          |
| Incidence: Rare                            |                                          |                                          |
| Prognosis: Guarded to poor                 |                                          |                                          |
| Clinical signs: Tetraparesis/paralysis     |                                          |                                          |
| Diagnosis ante mortem: Yes (peripheral nerve histopathology) |                                          |                                          |
| **Sphingomyelinosis**                      | Boxer, Poodle                            |                                          |
| Incidence: Poor                            |                                          |                                          |
| Prognosis: Poor                            |                                          |                                          |
| Age of onset of clinical signs: 2-4 months |                                          |                                          |
| Clinical signs: Ataxia, tremors, paresis/paralysis |                                          |                                          |
| Diagnosis ante mortem: Yes (enzyme assay)  |                                          |                                          |
| **Spina bifida/meningomyelocele**          | English Bulldog                          |                                          |
| Incidence: No common                       |                                          |                                          |
| Prognosis: Good (spina bifida only) guarded to poor (spina bifida with meningomyelocele) |                                          |                                          |
| Age of onset of clinical signs: Shortly after birth |                                          |                                          |
| Clinical signs: Spina bifida can be asymptomatic, meningomyelocele: Incontinence, paraparesis/paralysis |                                          |                                          |
| Diagnosis ante mortem: Yes (clinical signs, RX, CT) |                                          |                                          |
| **Spongiform degeneration of gray or white matter** | Bull Mastiff, Cocker Spaniel, Labrador Retriever, Rottweiler, Saluki, Samoyed, Silky Terrier | Birman, Egyptian Mau |
| Incidence: Rare                            |                                          |                                          |
| Prognosis: Poor                            |                                          |                                          |
| Age of onset of clinical signs: 1-6 months |                                          |                                          |
| Clinical signs: Tremors, ataxia, behavioral changes, mental status alterations, visual deficit |                                          |                                          |
| Diagnosis ante mortem: No (histopathology post mortem) |                                          |                                          |

Note: LMN=Lower motor neuron, CT=Computed tomography, EEG=Electroencephalogram, MR=Magnetic resonance

only by excluding other causes (e.g., idiopathic epilepsy) or only after the death of the animal because it requires anatomic and histopathologic evaluations (e.g. degenerative diseases) (Table-1) [9,10].

Inherited neurologic diseases usually affect specific breeds (Table-1).

**An Approach to the Problem: Proposals**

The authors propose to make eradication plans for hereditary and/or congenital neurologic disease official and, indeed, obligatory for legal and ethical reasons. Genetic selection giving preference to certain characteristics can result in hereditary neurologic defects, which may cause problems of varying entity and this shows a lack of respect animals as sentient beings [32]. In this context, there are emotional and psychological implications for the owner who is made aware of the fact that his animal suffers from hereditary and genetic neurologic defects.

To reduce the incidence of congenital and/or hereditary neurologic disease in pets, it would also be useful that:

a. Practicing veterinarians discourage reproduction in animals with neurologic alterations for which inherited etiology is recognized or suspected.

b. Breeders and geneticists work together on eradication of hereditary anomalies. Genetic tests, in particular, make possible a rapid, accurate, and early confirmation of diagnosis in sick animals even before the clinical signs are evident; the carriers can thus be removed from breeding programs [33]. When genetic tests are not available, detailed information on pedigrees will make identification of carriers possible so as to carry out selective and rational breeding.

Another useful development could be to enhance breeders’ responsibility by enforcing the inclusion of a lengthening of guarantee time in the case of hereditary defects, including neurologic defects, which may only become evident after the age of 12 months (Table-1), that is, beyond the validity of the guarantee.

**Conclusions**

Cooperation among dog breeders, researchers, prospective purchasers, and purebreed dog organizations at all levels is essential if genetically healthy dogs are to become a reality.

Breeders should understand the implications of genetic diseases recognized as affecting their breeds and take steps to breed only those dogs/cats that will minimize the propagation of unwanted characteristics.

Prospective buyers should be made aware of the genetic diseases related to the breed they are considering. They should also ask a physical exam and test
results or genetic histories for the animals they are planning to purchase.

Veterinarians should inform owners, breeders, and prospective breeders about congenital/hereditary neurologic diseases.

Authors’ Contributions

AP and MM generated the concept, collected materials, draft, and revised the manuscript. Both authors read and approved the final manuscript.

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Competing Interests

The authors declare that they have no competing interests.

References

1. Favre, D. (2010) Living property: A new status for animals within the legal system. Marq. L. Rev., 93: 1021-1071.
2. Passantino, A. and De Vico, G. (2006) Our mate animals. Biol. Forum, 99(2): 200-204.
3. Passantino, A. (2007) The legal protection of human feeling for animals. Aracne publisher, Roma, Italy, p 1-108.
4. Passantino, A. (2006) Medico-legal considerations of canine leishmaniasis in Italy: An overview of an emerging disease with reference to the purchase. Rev. Sci. Tech., 25(3): 1111-1123.
5. Quartarone, V., Quattrocchio, M., Cristarella, S. and Passantino, A. (2012) Technical consultation in purchase of animals: Case report. Veterinaria, 26(5): 45-53.
6. Scott, F.W., de Lahunta, A., Schultz, R.D., Bistner, S.I. and Riis, R.C. (1975) Teratogenesis in cats associated with griseofulvin therapy. Teratology, 11(1): 79-86.
7. Stuetzer, B. and Hartmann, K. (2014) Feline parvovirus infection and associated diseases. Vet. J., 201(82): 150-155.
8. Green, C.E., Vandevelde, M. and Braund, K. (1976) Lissencephaly in two Lhasa Apso dogs. JAVMA, 169(4): 405-410.
9. Bernardini, M. (2010) Neurology of the dog and cat. 2nd edition. Poletto publisher, Milan, Italy.
10. Lorentz, M.D., Coates, J.R. and Kent, M. (2011) Handbook of Veterinarian Neurology. 5th ed. Saunders, Elsevier, St. Louis.
11. Thomas, W.B. (2010) Hydrocephalus in dogs and cats. Vet. Clin. N. Am. Small., 40(1): 143-159.
12. Berlanda, M., Zotti, A., Brandazza, G., Poser, H., Calò, P. and Bernardini, M. (2011) Magnetic resonance and computed tomographic features of 4 cases of canine congenital thoracic vertebrae anomalies. Can. Vet. J., 52: 1334-1338.
13. Lee, K.I., Lim, C.Y., Kang, B.T. and Park, H.M. (2011) Clinical and MRI findings of lissencephaly in a mixed breed dog. J. Vet. Med. Sci., 73(10): 1385-1388.
14. MacKillop, E. (2011) Magnetic resonance imaging and multislice computed tomography for the detection of cervical syringomyelia in dogs. J. Vet. Intern. Med., 29: 1354-1359.
15. Lee, M. (1993) Congenital vestibular disease in a German shepherd dog. Vet. Rec., 113(24): 571.
16. Branis, M. and Burda, H. (1985) Inner ear structure in deaf and normally hearing Dalmatian dogs. J. Comp. Pathol., 95(2): 295-299.
17. Oda, K., Lamb, E.H., Lennon, V.A. and Palmer, A.C. (1984) Congenital canine myasthenia gravis: I. Deficient junctional acetylcholine receptors. Muscle Nerve, 7(9): 705-716.
18. Lin, L., Faraco, J., Li, R., Kadotani, H., Rogers, W., Lin, X., Qiu, X., de Jong, P.J., Nishino, S. and Mignón, E. (1999) The sleep disorder canine narcolepsy is caused by a mutation in the hypocretin (orexin) receptor 2 gene. Cell, 98(3): 365-376.
19. Masucci, M. (2008) Narcolepsy in dogs: Etiology, pathogenesis and clinical management. Summa animali da compagnia, 3: 11-20.
20. Hemsley, K.M. and Hopwood, J.J. (2010) Lesson learnt from animal models: Pathophysiology of neuropathic lysosomal storage disorders. J. Inherit. Metab. Dis., 33(4): 363-371.
21. Shelton, G.D. (2010) Routine and specialized laboratory testing for the diagnosis of neuromuscular diseases in dogs and cats. Vet. Clin. Path., 39(3): 278-295.
22. Mignot, E.J.M. (2014) History of narcolepsy at Stanford University. Immunol. Res., 58: 315-339.
23. Lin, L., Faraco, J., Li, R., Kadotani, H., Rogers, W., Lin, X., Qiu, X., de Jong, P.J., Nishino, S. and Mignón, E. (1999) The sleep disorder canine narcolepsy is caused by a mutation in the hypocretin (orexin) receptor 2 gene. Cell, 98(3): 365-376.
24. Shelton, G.D. (2010) Routine and specialized laboratory testing for the diagnosis of neuromuscular diseases in dogs and cats. Vet. Clin. Path., 39(3): 278-295.
25. Mignot, E.J.M. (2014) History of narcolepsy at Stanford University. Immunol. Res., 58: 315-339.
26. Dewey, CW., Marino, D.J. and Loughin, C.A. (2013) Cranio cervical junction abnormalities in dogs. N. Z. Vet. J., 61(84): 202-211.
27. Charalambous, M., Jeffery, N.D., Smith, P.M., Goncalves, R., Barker, A., Hayes, G., Ives, E. and Vanhaesebroeck, A.E. (2014) Surgical treatment of dorsal hemivertebrae associated with kyphosis by spinal segmental stabilization, with or without decompression. Vet. J., 202(2): 267-273.
28. Guiterrez-Quintana, R., Guevar, J., Stalin, C., Faller, K., Yeamans, C. and Penderis, J. (2014) A proposed radiographic classification scheme for congenital thoracic vertebral malformations in brachycephalic “screw-tailed” dog breeds. Vet. Radiol. Ultrason., 55(6): 585-591.
29. Song, R.B., Glass, E.N., Kent, M., Sánchez, M.D., Smith, D.M. and de Lahunta, A. (2014) Surgical correction of a sacral meningomyelocele in a dog. J. Am. Anim. Hosp. Assoc., 50(6): 436-443.
30. Voorbij, A.M.W., Meij, B.P., van Bruggen, L.W.L., Grimw, G.C.M., Stassen, Q.E.M. and Kooistra, H.S. (2015) Atlanto-axial malformation and instability in dogs with pituitary dwarfism due to an LHX3 mutation. J. Vet. Intern. Med., 29: 207-213.
31. De Risio, L., Bhatti, S., Muñana, K., Penderis, J., Stein, V., Tipold, A., Berendt, M., Farquiør, R., Fisher, A., Long, S., Mandigers, P.J.J., Miatisek, K., Packer, R.M.A., Pakozdy, A., Patterson, N., Platt, S., Modell, M., Potschka, H., Purnama Batlle, M., Rusbridge, C. and Volk, H.A. (2015) International veterinary epilepsy task force consensus proposal: Diagnostic approach to epilepsy in dogs. BMC Vet. Res., 11: 148.
32. European Community. (2007) Treaty of amending the treaty establishing the European Union and the treaty establishing the European community, signed at Lisbon, 13 December 2007. Off. J. EU., C306: 1-271.
33. Sargan, D.R. (2007) Inherited metabolic disease in companion animals: Prospects for their diagnosis and elimination in the next decade. Vet. J., 174(2): 222-224.
34. Abitbol, M. (2009) Cerebellar ataxia of American staffordshire terrier. Summa animali da compagnia, 3: 6-8.
35. Braund, K.G. (2003) Storage disorders. In: Vite, C.H., editor. Braund’s Clinical Neurology in Small Animals: Localization, Diagnosis and Treatment. International
36. deLahunta, A. and Glass, E. (2009) Veterinary Neuroanatomy and Clinical Neurology. Saunders Elsevier, St. Louis.
37. LeCouter, R.A. (2007) Genetic markers in the diagnosis and prevention of neurological diseases. Proceedings of the 32nd WSAVA Congress, Sydney, Australia, 19-23 August; 2007.
38. Penderis, J. (2008) Genetic advances in neurological disease. Proceedings of the 33rd WSAVA Congress, Dublin, Ireland, 20-24 August; 2008. p489-491.

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