

Behavioural Signs and Neurological Disorders in Dogs and Cats

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INTRODUCTION

Veterinary behavioural medicine is relatively new, and its evolution may suffer more than other specialties from an unclear identity, because so many disparate groups who are not rooted in veterinary medicine have participated in its evolution [1]. In the past, behavioural problems of companion animals were not dealt by veterinarians, but mainly by dog trainers [2]. When animal clinical psychology started to be developed as a new scientific discipline, the interest increased also in the veterinary field [3]. In 1997 in Birmingham (UK) was held the first International Meeting on veterinary behavioural medicine [4]. Later, the general approach and terminology was more in line with psychiatry than with psychology, and behavioural problems were seen as a trauma or an infection, with a physical cause to be treated in order to solve the problem [5-7]. However, blind adherence to a medical model causes serious problems when it comes to the scientific investigation of problem behaviour. In fact, the use of medical paradigms for the study of problem behaviour which seek to categorise disorders rather than focus on their underlying mechanism and evolutionary function, may restrict the development of our knowledge of these processes [8].

Behaviour is the ultimate integrator of all organ system responses, and as such, is a dynamic outcome resulting from the interactions of complex mechanisms. Understanding such systems is difficult, but progress can occur if an attempt is made to understand all the mechanistic levels that contribute to behavioural patterns and behavioural conditions [1].

Historically veterinary medicine has focused on differentiating between behavioural and physical problems; priority was given to the diagnosis and treatment of physical diseases, and only after having dealt with them the behavioural component was taken into account. A more holistic approach is desirable, dealing contemporaneously with physical and behavioural

components, in order to optimize the patient's welfare. This should be particularly true for neurology, as the boundary between a neurological disease and a behavioural problem is often unclear. For instance, all disorders involving the central nervous system, especially the forebrain, have a consequence on behaviour [9].

A strict collaboration between veterinary neurologists and behaviourists is desirable [10, 11]. Such collaboration would be beneficial for both veterinary neurologists and behaviourists to have a multidisciplinary approach. On one hand neurologists, together with the use of advanced diagnostic methods and the exclusion of metabolic and infectious diseases, may dwell also on the behavioural aspects and taking into account, when needed, the possibility of a behavioural consultation for the animal patient. On the other hand, behaviourists should take into account to perform a complete physical examination, including a neurological examination, on a subject displaying behavioural problems. However, it must be considered that a normal neurological examination does not allow to exclude the presence of neurological problems. For instance, an intracranial neoplasia can result in a normal neurological examination and the only sign, for a long time, can be a change in behaviour [11, 12].

The challenge is to determine the sensible limit to clinical investigation. Once basic medical data have been obtained (including general physical examination, neurological examination, and blood tests), the level of consistency between a particular pattern of behaviour and the environment within which it is expressed may help to decide whether there is likely to be an underlying medical condition, and whether it may be necessary to perform additional medical tests [13].

In the scientific literature there are many physical diseases (such as infections, endocrinologic disorders, neoplasia, toxins, congenital lesions, degenerative diseases and allergies)

that are recognized to be associated with behavioural signs [5, 13, 14]. As a matter of fact, often owners realize that their animal is ill when the dog or the cat changes the behaviour, even if it is not a behavioural problem and owners refer this behavioural change to the veterinarian [15]. In particular, many neurological disorders can lead to behavioural modifications; and for many abnormal behaviours there is not yet a clear distinction between neurological and behavioural disorder. This is the case of compulsive disorders, some of which can benefit from a treatment with anti-epileptic drugs while others are

solved with psychoactive drugs normally used for behavioural problems [11, 16, 17]. In addition, diseases related to perception such as blindness are responsible for clear modifications of the behaviour [18].

When approaching a differential diagnosis, surgical sieves are commonly used. Using the acronym VITAMIN-D and the available scientific literature, we created a table (Table 1) summarizing a series of behavioural problems and their possible associations with neurological disorders [10, 11, 15, 16, 19-67].

Table 1: Central nervous system diseases based on the VITAMIN-D acronym for dogs and cats. V = vascular, I = inflammatory/infectious, T = trauma, A = anomalous, M = metabolic, I = idiopathic, N = neoplasia, D = degenerative. FIP = feline infectious peritonitis, FIV = feline immunodeficiency virus infection, FSE = feline spongiform encephalopathy; n.r.= not reported.

POSSIBLE BEHAVIOURAL SIGNS	UNDERLYING NEUROLOGICAL CONDITIONS
<p>Inappropriate elimination/urogenital diseases</p>	<p>V: n.r. I: Distemper. T: Intervertebral disc disease. A: Lissencephaly, hydrocephalus, hydrosyringomyelia. M: Hepatic encephalopathy, uremic syndrome, hypercalcemia, hyperthyroidism, hyper- or hypoadrenocorticism. I: Neurogenic cystitis, idiopathic cystitis. N: Neoplasia of the basal nuclei, thalamus, cerebellum, pudendal nerve. D: Neurodegenerative conditions of the basal nuclei, thalamus, cerebellum, pudendal nerve; upper and lower motor neuron diseases.</p>
<p>Aggressive behavior</p>	<p>V: Feline ischemic encephalopathy, cerebrovascular accidents, brain hypoxia. I: Distemper, cryptococcosis, FSE, FIP, rabies, toxoplasmosis, neosporosis, Borna disease, steroid responsive arteritis-meningitis, Reflex sympathetic dystrophy. T: Brain trauma. A: Lissencephaly, hydrocephalus, porencephaly. M: Hepatic encephalopathy, uremic syndrome, hypercalcemia, hyper- or hypothyroidism, hyper- or hypoadrenocorticism, heavy metal poisoning. I: Neurogenic cystitis, idiopathic ganglioradiculoneuritis, feline hyperesthesia syndrome, idiopathic epilepsy. N: Intracranial tumors: temporal lobes, limbic system, hypothalamus (ventromedial and posterolateral areas in the cat). D: Polioencephalomalacia of the pyriform lobe and hippocampus, lysosomal storage diseases, mitochondrial encephalopathies.</p>
<p>Depression, reduced activity, somnolence, apathetic behavior</p>	<p>V: Feline ischemic encephalopathy, cerebrovascular accidents, brain hypoxia. I: Distemper, cryptococcosis, protothecosis, canine ehrlichiosis, canine encephalitozoonosis, bacterial meningoencephalitis, necrotizing meningoencephalitis, necrotizing leukoencephalitis, granulomatous meningoencephalomyelitis, steroid responsive arteritis-meningitis, FSE, FIP, FIV, Borna disease. T: Brain trauma. A: Lissencephaly, hydrocephalus. M: Hepatic encephalopathy, uremic syndrome, hypercalcemia, hyper- or hypothyroidism, hyperadrenocorticism, hypoglycemia, heavy metal poisoning, Marijuana poisoning. I: n.r. N: Intracranial tumors: thalamus, frontal lobes, tegmentum of midbrain and pons. D: Lysosomal storage diseases, spongy degeneration of the gray matter, necrotizing encephalopathy.</p>

<p>Changes in ingestive behavior</p>	<p>V: Feline ischemic encephalopathy, brain hypoxia. I: Rabies, FSE, FIV. T: n.r. A: n.r. M: Hyperadrenocorticism, hyperthyroidism, lead poisoning, hepatic encephalopathy, uremic syndrome. I: n.r. N: Intracranial tumors: thalamus, ventromedial and posterolateral hypothalamus in the cat. D: n.r.</p>
<p>Cognitive dysfunction, learning and memory deficits, dementia</p>	<p>V: Feline ischemic encephalopathy, brain hypoxia. I: Distemper, FSE, mycotic meningoencephalitis, rabies, toxoplasmosis, neosporosis, Borna disease. T: Frontal lobe trauma. A: Hydrocephalus, hydrosyringomyelia, Chiari-like syndrome. M: Organic aciduria, lead poisoning, hepatic encephalopathy, uremic syndrome. I: n.r. N: Frontal lobe tumors. D: Age-related brain lesions, lysosomal storage diseases.</p>
<p>Stereotypic and compulsive disorders</p>	<p>V: Feline ischemic encephalopathy, brain hypoxia, polycythemia. I: Rabies, Aujeszky disease, granulomatous meningoencephalomyelitis, tetanus, FIV, ehrlichiosis, necrotizing meningoencephalitis, necrotizing leukoencephalitis, protothecosis. T: Frontal lobe trauma, Reflex sympathetic dystrophy. A: Hydrocephalus, caudal occipital malformation syndrome, hydrosyringomyelia. M: Hypocalcemia, lead and thallium poisoning, Loperamide toxicity, hepatic encephalopathy, uremic syndrome, hypothyroidism, hyperadrenocorticism. I: Idiopathic ganglioradiculoneuritis, feline hyperesthesia syndrome, feline orofacial pain syndrome. N: Intracranial tumors: frontal lobes, basal nuclei (caudate nuclei). D: Spongy degeneration of the gray matter, axonal neuropathy, cauda equina syndrome.</p>
<p>Non-specific Anxiety and Fear</p>	<p>V: Feline ischemic encephalopathy, cerebrovascular accidents, brain hypoxia. I: Distemper, rabies, cryptococcosis, arthropod borne diseases, FSE, FIP, toxoplasmosis, neosporosis, Borna disease. T: Brain trauma, Reflex sympathetic dystrophy. A: Hydrocephalus, hydrosyringomyelia, Chiari-like syndrome. M: Hepatic encephalopathy, uremic syndrome, hypothyroidism, hypo- or hyperadrenocorticism, heavy metal poisoning. I: n.r. N: Intracranial tumors: frontal lobes, basal nuclei. D: n.r.</p>
<p>Anomalies of sexual behavior</p>	<p>V, I, T, A, M, I: n.r. N: Intracranial tumors: temporal lobes, limbic system, hypothalamus. D: Axonal neuropathy.</p>

In some cases the link between behavioural and neurological problems is unclear because of the lack of information. For instance, it has been found that the set of genes that contributes to narcolepsy seems to be involved also in anxiety disorder related to neurochemical system dysregulation [5]. Boundaries between behavioural conditions and medical differentials are

likely to blur more rather than less as we learn more about genomic, cellular, and subcellular effects on common conditions. These changes should lead to better treatment but may also require a paradigm shift in how we view behavioural conditions and the mechanisms that contribute to them [15].

CONCLUSION

The boundary between neurological and behavioural disorders often is not clear, and they often coexist. As a matter of fact, many neurological conditions lead to behavioural changes, which can be the only sign and the first sign complained by the owners. Sometimes it is not possible to reach a diagnosis based only on neurology or on behavioural medicine. Finally, some behavioural problems can facilitate the onset or worsening of neurological conditions.

A synergy between neurology and behavioural medicine would be beneficial for both of them, in the diagnostic procedure and in the treatment. Veterinarians of such disciplines should collaborate as much as possible.

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