

Orthomanual therapy

Non-surgical treatment
of intervertebral disc disease

Travelling with pets

Am I giving the correct advice?

Eradicating rabies

How vets can help

Uncommon bone pathologies



Also in this volume:

Murmur intensity in small breed dogs with cardiac
disease and Rabies in the consulting room.

FECAVA news, veterinary app & book reviews
... and more, online!

**FECAVA
SYMPOSIUM
INSIDE**

Contents

- | | | | |
|---|---|---|---------|
|  |  | Are we doing enough and how can vets help?
Rabies surveillance, stray dogs and disease control
<i>Luke Gamble</i> | 4 - 9 |
|  |  | Clinical aspects of rabies: What will we see in our
clinics and how should we protect ourselves?
<i>Denis Novak</i> | 10 - 14 |
|  |  | Travelling with pets: am I giving the correct advice?
<i>Cathy Tourlouse</i> | 15 - 19 |
|  |  | Uncommon bone pathologies
<i>Josep Font Grau</i> | 20 - 32 |
|  |  | Murmur intensity in small-breed dogs with myxomatous
mitral valve disease reflects disease severity
<i>Ingrid B. Ljungvall, Mark Rishniw, Francesco Porciello, Luca Ferasin
and Dan G. Ohad</i> | 33 - 41 |
|  |  | Orthomanual therapy as treatment for suspected
thoracolumbar disc disease in dogs
<i>Dorit C. Aharon, Jacha Heukels, Rosanne Buntsma</i> | 42 - 50 |

Icons

Each scientific article is classified with one or more icons.

These refer to the species (in green) of animal or the veterinary discipline (in blue) relevant for the article.



Dogs



Cats



Dogs and Cats/
Small animals



Rabbits



Less common pets



Anaesthesia



Bacterial Diseases



Behaviour



Cardiovascular



Dental



Dermatology



Diagnostic imaging



Digestive System



Ear Nose Throat



Genetics



Internal Medicine



Neurology



Oncology



Ophthalmology



Orthopaedics



Practice Management



Surgery



Urogenital



Zoonosis



FECAVA Symposium*

Are we doing enough and how can vets help? Rabies surveillance, stray dogs and disease control

Luke Gamble¹

SUMMARY

Rabies is a fatal zoonosis, mainly transmitted through bites of dogs infected with rabies. The disease occurs in over 150 countries wide, with over one third of victims (25,000) in India, in particular in children. The disease is entirely preventable by vaccinating dogs and by a solid epidemiological approach.

* This paper was presented at the FECAVA symposium on rabies held in Barcelona on 16 October 2015.
Eur J Comp An Pract, Spring 2016; (26)1:p4-p9
Go to <http://www.ejcap.org> for the interactive online presentation of this paper

The global spread of rabies

Rabies, a neurotropic lyssavirus, is a vaccine-preventable zoonotic disease, estimated to cause between 40,000 to 100,000 human deaths annually worldwide.^[1] The main cause of rabies transmission is through dog bites^[2] and the vast majority of reported deaths (84%) occur in rural areas.^[3] Over a third of deaths occur within India, the widely acknowledged global hotspot for rabies in the world, and a country which has annually reported an estimated 25,000–30,000 human deaths from rabies since 1984.^[4] The disease occurs in more than 150 countries and territories world wide and is regarded as the worlds deadliest zoonosis.

Sadly, the majority of people who die of rabies are people of poor or low-income socioeconomic status^[2], 30–60% being children under the age of 16.^[5] Without post-exposure prophylaxis, prognosis is incredibly poor with only a very small handful of humans surviving once the onset of clinical rabies is apparent. Access to treatment is often extremely limited in many global rabies hotspots

and if available, cost is also a huge issue leading to many patients not completing their treatment course or using regimens that re not recommended. Previous estimates have indicated that a full course of post exposure prophylaxis represents as much as 3.87% of the gross national income for a person in Asia and 5.80% for a person in Africa (equivalent to 51 days wage for an average African and 31 days wages for an average Asian).^[6]

In many countries where the disease is endemic, rabies is additionally not notifiable and there is no organized surveillance system of human or animal cases. The issue of effective surveillance is compounded by the fact rural families will not always insist on the cause of death being officially registered and it is incredibly difficult to ascertain the exact scale of the problem which is generally regarded as being vastly underestimated in endemic areas.

Economically, the estimated global cost of rabies is in the region of €7.6 billion and 3.7 million disability-adjusted life years (DALYs)^[3]. Approximately €1.78 billion (~40%) is annually attributable due to lost productivity after premature deaths and a further €1.42 billion is spent directly on global post-exposure prophylaxis^[6]. In India, for example, which has a population exceeding 1.02 billion, it is estimated that 15 million people are bitten by animals annually.^[7] That equates to at least one person being bitten by a dog every two seconds and the

¹ Luke Gamble, Worldwide Veterinary Service, 14 Wimborne St, Cranborne, Wimborne BH21 5PP, UK. info@wvs.org.uk

estimated annual cost of post-bite treatment in this country totals the equivalent of approximately €22.2 million.^[4]

Despite all the above, there is still very little investment in strategic dog vaccination in most endemic countries^[3] which, by targeting 70% of a resident dog population over a sustained period (2-3 years) is regarded as the most effective way to eliminate the disease.

Rabies in Europe

Rabies in Europe is predominately sylvatic rabies, with wildlife species accounting for approximately 80% of all rabies cases. Of these, more than 80% are red foxes (*Vulpes vulpes*) and thanks to a comprehensive oral rabies vaccination strategy, implemented originally in 1978 in Switzerland and then rolled out across large parts of mainland Europe, annual number of wildlife rabies cases in Europe dropped from 21,000 in 1990 to just 5,400 in 2004^[6]. Subsequently most parts of Western and Central Europe have successfully controlled and eliminated rabies with many countries such as Finland, The Netherlands, Italy, Switzerland, France, Belgium, Luxembourg, Germany, Austria and the Czech republic being declared free of terrestrial rabies.



Fig 1. Well-designed oral rabies vaccination programmes are the most effective approach to managing wildlife reservoirs

Surveillance and reporting of rabies cases in Europe is also regarded as being generally exceptional. A European rabies reporting system, hosted by the WHO collaborating Centre for Rabies Surveillance and Research at the Friedrich-Loeffler Institute in Germany, is continuously updated, and all data reported is automatically transferred to a database, summarized by an administrative unit and then aggregated

per country. More than 40 European countries report officially confirmed rabies cases in both wild and domestic animal species and in humans on a quarterly basis. The Rabies Bulletin Europe is printed quarterly, and a free version is available electronically from www.who-rabies-bulletin.org/.

That said, the risk of an animal clinically affected by rabies in any veterinary clinic in Europe is a real one and more so in light of relaxed travel requirements between countries and a laxity in border controls relating to the illegal importation of animals.

Box 1.

Key lessons learnt from Europe

- Population reduction was not the key
- Enhancing a culture of responsible pet ownership is important
- Culling may have significant detrimental effects
- Systematic 'windscreen wiper' method was effective – chasing rabies foci was not
- Comprehensive mapping of vaccination areas was essential – attack and maintenance phased approach
- Establishing reliable surveillance systems vital – involvement of public health, veterinary services, laboratories, physicians and community engagement was key

How can vets help?

Aside from the moral aspect that rabies is a neglected zoonotic disease resulting in the agonising death of at least one child every ten minutes, the best way to prevent the virus coming into our communities is to eliminate it at source.

Limited resources and a stretched public health structure in many rabies endemic countries precludes data collection and analysis which makes it difficult to establish baseline data and objectively evaluate intervention programmes, but rabies has been successfully controlled in dog populations throughout the Americas and in specific Indian States which has demonstrated that the disease can be eliminated with sufficient resource and focused endeavour. Repeated annual vaccination of 70% of a resident canine population over a three-year period, is the marker to achieve swift elimination of the virus and when coupled with active rabies

surveillance and on-going vaccination programmes, and is a sound strategy to achieving rabies free status. It stands to reason that since the reservoir host for rabies are dogs, vets should have a key role and responsibility in assisting authorities to tackle the disease.

The aim of Mission Rabies (www.missionrabies.com) is to support programmes in developing countries which are striving to eliminate rabies. The goal is to bolster local veterinary infrastructure, resources and logistical strategies to facilitate focused, epidemiologically analysed mass vaccination campaigns in accordance with WHO and OIE guidance. Concurrently, Mission Rabies seeks to help with humane dog population control through its partner charity Worldwide Veterinary Service (www.wvs.org.uk) and run concurrent community education programmes, supporting local endeavours and under local Government guidance in all circumstance. These programmes require motivated veterinary professionals, the majority of whom volunteer their time to coordinate international projects, each of which generally includes volunteers from at least ten countries marking the initiative as one of global unity amongst the veterinary profession in tackling this disease.

Box 2.

Steps of elimination to effect change - irrespective of location!

- **Establish need in targeted area** (rabies baseline incidence / children / adults / livestock losses / dog bites / access to PEP / wildlife / dog population survey)
- **Determine strategy** for implementation – solid science + public engagement – static + mobile vaccination team approach depending on country/culture/land-type
- **Deliver proof of concept** – ONE KEY AREA – 70% vaccination – show it can be done, that your strategy works (policy makers/sponsors back winners)
- **Establish ongoing rabies response / surveillance** (dedicated teams, respond to notifications with ring-fenced vaccination)
- **Present to policy makers**
- **Expand programme** regionally / nationally

In relation to mass elimination of rabies in more challenging global hotspots, there is a continuing need to develop tailored field strategies which allow vaccination

of large numbers of free roaming dogs in rural and urban areas in very differing geographical and cultural locations. One key area in which Mission Rabies has worked is the development of an improved methodology which can rapidly assess whether the vaccination coverage achieved is sufficiently high enough to result in a significant protection to a targeted dog population.

Methodology of a rabies control programme

The following protocol is outlined for the implementation of the pilot project to establish proof of concept in a given area. Once completed and lessons learned from working in a particular area, this can be rolled out across a Region, State or Country:

- Identify a defined pilot project region
- Establish infrastructure, legal framework and dedicated rabies control team
- Collect baseline data and conduct comprehensive dog population survey of the region
- Prepare logistically for implementation of rabies vaccination campaign and drive a pre launch community education/awareness programme
- Run focused mass vaccination campaign with comprehensive post-vaccination surveillance for epidemiological analysis
- Establish on-going rabies surveillance and response team
- Review impact and objectively assess benefit of the programme, correlated specifically but not exclusively against the measure of a reduction in canine rabies incidence.

Epidemiological analysis - utilisation of smartphone technology in disease control

The aim of any rabies vaccination project is to prevent the spread of rabies between dogs by creating a state of 'herd immunity' within a given dog population. Since over 99% of rabies cases are caused by dog bites, this significantly reduces the risk of rabies to humans and concurrently enhances community regard for vaccinated street dogs. Integrating the modern technologies of a smart phone app and GPS mapping to log dogs vaccinated and surveyed is crucial in providing comprehensible and solid scientific data detailing and demonstrating an intervention programme and the work performed.

Box 3.

The three steps of Rabies control

1. Strategy

- Determining strategy is crucial
- Planning is everything
- Community engagement vital
- VETS LEAD THIS – ESPECIALLY IN EUROPE!

2. Action!

Teams comprising catchers (3-4), paravet and data administrator

Once caught:

- Check for injuries
- Vaccinate– choice of vaccine is key (we use MSD)
- Mark
- Data record
- Release
- All under a minute!

3. Follow-up with humane population control

- Key component for long-term control of rabies
- NOT necessary for focused elimination of the virus
- Huge pluses in supporting local veterinary infrastructure
- Long term much easier to maintain the 70% coverage!!!
- Mission Rabies works synergistically with WVS to achieve this!



© Photo Mission Rabies

Utilising the Mission Rabies App, downloadable for both android and Apple smartphones, this unique approach allows each field team to electronically collect GPS catching and demographic data for every dog vaccinated. This allows epidemiological assessment of the coverage of each targeted area and catching teams can then be directed to ensure they cover all wards thoroughly. It also prevents any pockets of un-vaccinated dogs being overlooked. Using an integrated path tracker function,

mark-resight surveys are conducted after the catching teams have covered each geographic area to assess the percentage vaccination cover within the dog population. This further allows direction of the dog catching teams, by an epidemiology coordinator reporting to a regional coordinator, to ensure the 70% targets are reached. The dogs are data collected and GPS tagged at the point of capture – an example of the MR catching coverage in Margao, South Goa is shown in box 4.

Box 4.

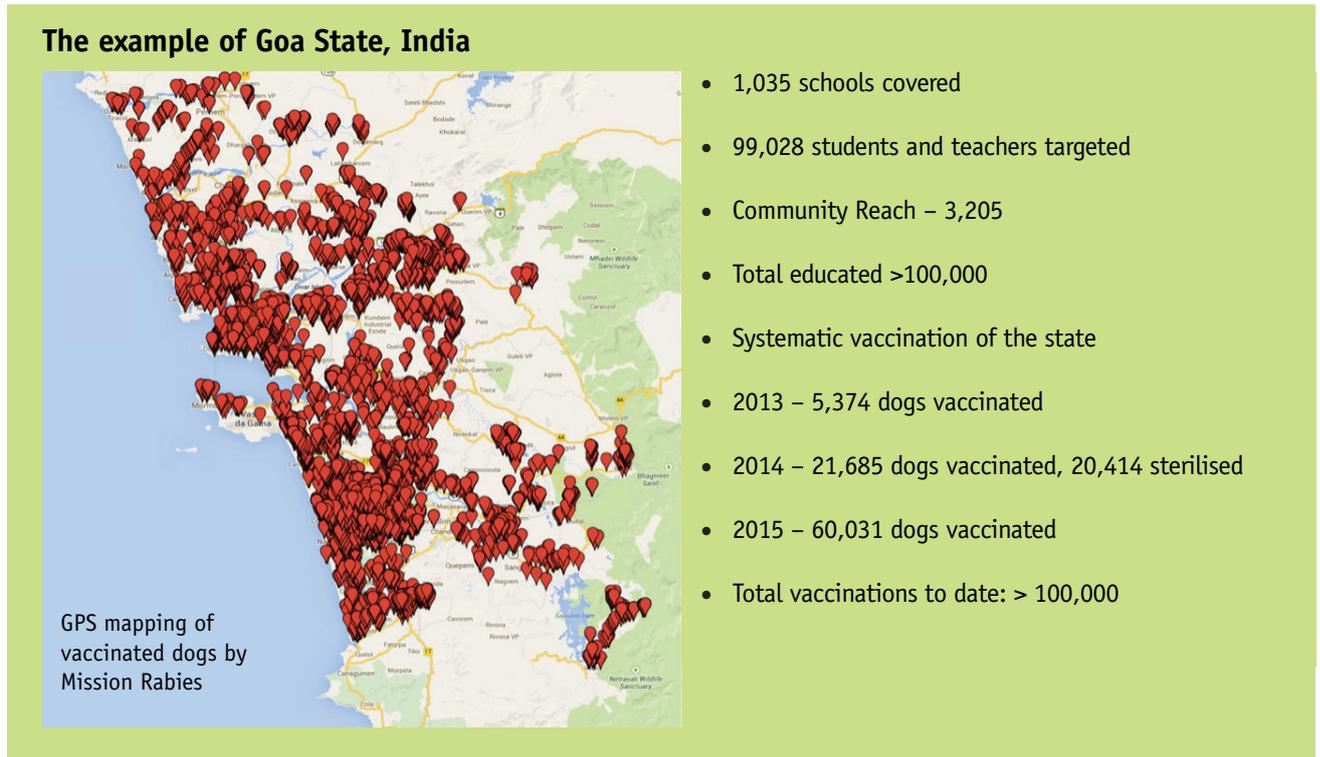
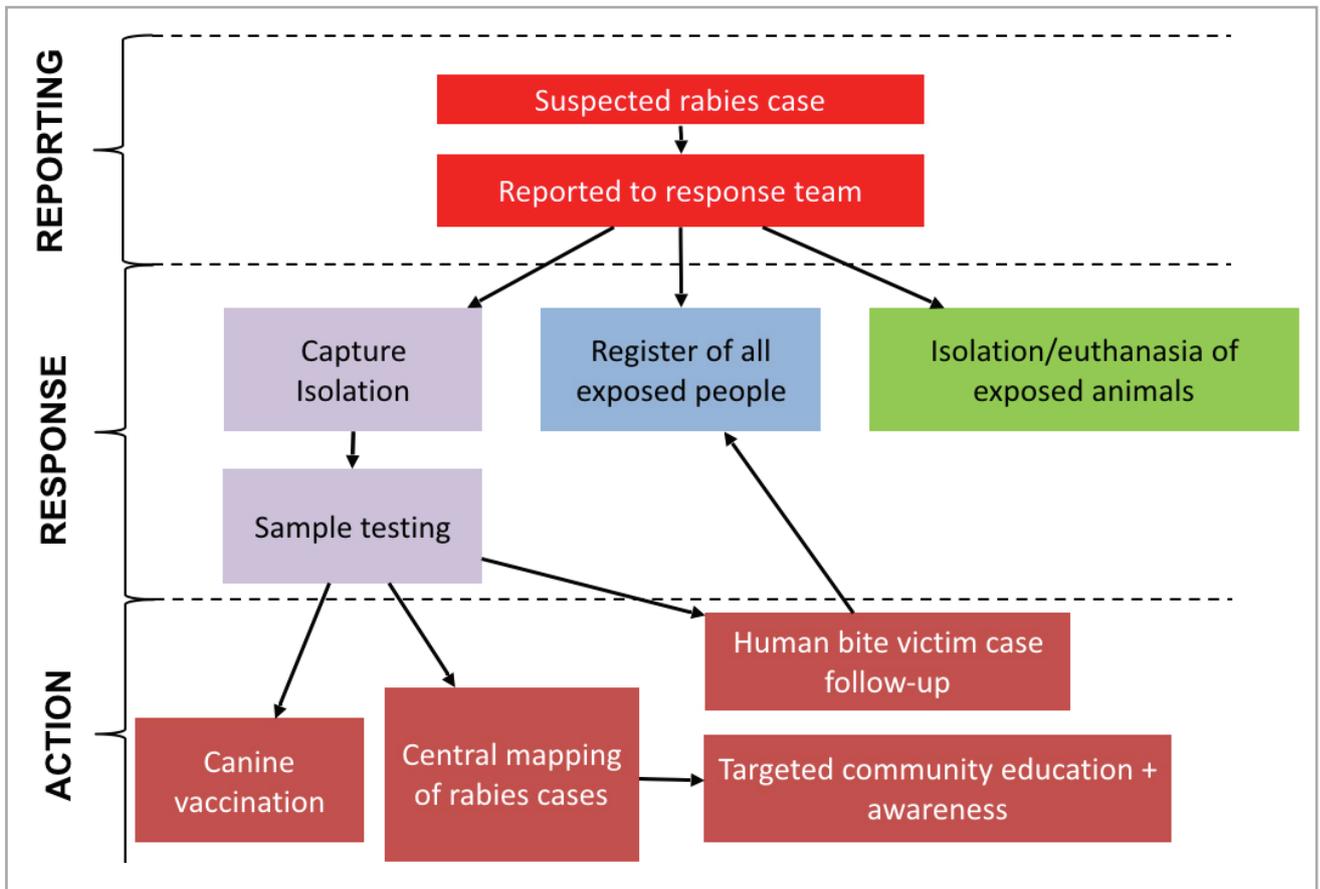


Fig 2. Steps of rabies surveillance.



Post vaccination surveys are essential to assess the vaccination coverage. The map below (fig. 3) shows an example from MR North Goa project, collected via GPS and utilising the Smartphone app, with green areas showing

coverage >70% of the population, orange indicates 60-70% requiring some repeat catching work and red indicates areas we need to re-target as the coverage is currently below 60%.

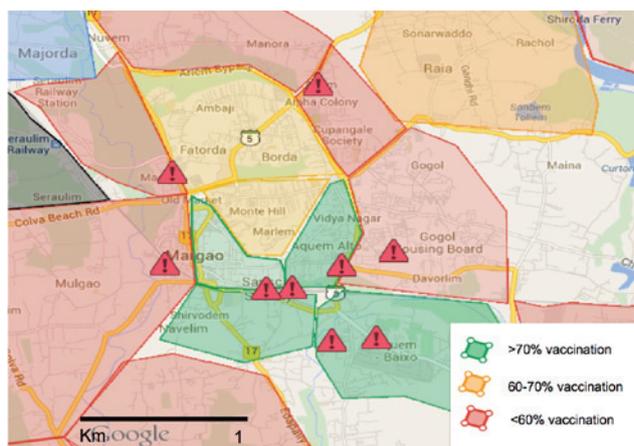


Fig. 3. Example of rabies surveillance from the Mission rabies North Goa project.

One Health

Rabies is a disease that vets need to be central in eliminating, it's totally achievable and a brilliant example of directly championing animal health and welfare for human health and welfare. The utilisation of modern technology and disease surveillance mapping has been a recent advancement in the field of rabies control and will allow increased strategic global surveillance of the disease that will hopefully help drive the elimination of the virus. Projects such as the Mission Rabies Malawi Endeavour, launched in May 2015, are a prime example of how European vets can work with colleagues globally in uniting against rabies. Blantyre Government Hospital annually records the highest incidence of child rabies from any single institution in the whole of Africa, and thanks to the international veterinary community (representatives from 12 different countries, the majority of which were European), over 35,000 dogs were vaccinated in just in 20 working days with a >70% coverage of the entire city dog population. So whilst a problem that could easily be seen within veterinary clinics throughout Europe, there is thankfully a clear pathway ahead to eliminate the virus. It just needs the profession to keep working together as an international community in seeing it through.

References

1. Knobel DL, Cleaveland S, Coleman PG, et al. Re-evaluating the burden of rabies in Africa and Asia. *Bull World Health Organisation*. 2005;83:360-368.
2. Ghosh TK. Rabies. Proceedings of the IX National Conference of Pediatric Infectious Diseases;2006;Chennai, India
3. Hampson K, Coudeville L, Lembo T, Sambo M, Kieffer A, Attlan M, et al. Estimating the Global Burden of Endemic Canine Rabies. *PLoS Negl Trop Dis*. 2015;9:e0003709. doi:10.1371/journal.pntd.0003709
4. Sudarshan MK. Assessing burden of rabies in India. WHO sponsored national multi-centric rabies survey (May 2004). *Assoc Prev Control Rabies India J* 2004;6:44-5.
5. Bulletin of the WHO; Vol 87, No 12, Dec 2009, 885-964
6. WHO Expert Consultation on Rabies 2013
7. Rozario Menezes, Rabies in India, *CMAJ* v.178(5);Feb 26, 2008

For more information:

Mission Rabies

www.missionrabies.com

World Veterinary Service

www.wvs.org.uk

Fox rabies blueprint

www.foxrabiesblueprint.org



FECAVA Symposium*

Clinical aspects of rabies: What will we see in our clinics and how should we protect ourselves?

Denis Novak¹

SUMMARY

Rabies is one of the most important zoonotic diseases in the world. The disease affects domestic and wild animals, and is spread to people through close contact with infectious material, usually saliva, via bites or scratches.

* This paper was presented at the FECAVA symposium on rabies held in Barcelona on 16 October 2015.
Eur J Comp An Pract, Spring 2016; (26)1: p10-p14
Go to <http://www.ejcap.org> for the interactive online presentation of this paper

have a long incubation period (six months and more) and symptoms may take several weeks to appear after infection. Once symptoms appear, rabies is always fatal.

Rabies is a disease listed in the World Organization for Animal Health (OIE) Terrestrial Animal Health Code and must be reported to the OIE (OIE Terrestrial Animal Health Code).

Aetiology and epidemiology

Rabies has been recognized for centuries. Louis Pasteur identified a virus as the cause of the disease in 1880s. Rabies is present on all continents with the exception of Antarctica. In Europe, foxes are the main reservoir while in North America the skunk, fox, raccoon and bat are important sources of infection. Globally, the dog is the most important reservoir, particularly in developing countries. In Asia, Africa and Latin America the main reservoir is not wildlife but stray dogs. In these areas, human infection and fatalities are more common. Once symptoms of the disease develop, rabies is nearly always fatal. The disease may be suspected based on clinical signs, but laboratory tests are required to confirm the diagnosis. Rabies is caused by a single-stranded RNA virus Lyssaviruses in the Rhabdovirus family. To date, over 15 different Lyssaviruses have been described. Rabies virus is the most important member of the genus.

Rabies virus affects the central nervous system of warm-blooded animals, including humans. The disease can

Transmission and pathogenesis

Transmission almost always occurs via introduction of virus-laden saliva into tissues, usually by the bite of a rabid animal. Virus from saliva, salivary glands, or brain can cause infection by entering the body through fresh wounds or intact mucous membranes. Saliva is infectious at the time clinical signs occur, but domestic dogs, cats, and ferrets may shed virus for several days before onset of clinical signs.

Rabies virus is highly neurotropic. After the virus enters the dog's body, it replicates in the cells of the muscles. Then virus spreads to the nearest nerve fibers, including all peripheral, sensory and motor nerves, traveling from there to the CNS. The virus travels via the peripheral nerves to the spinal cord and ascends to the brain. After reaching the brain, the virus travels via peripheral nerves to the salivary glands. If an animal is capable of transmitting rabies via its saliva, virus will be detectable in the brain.

¹ Denis Novak DVM MRCVS, Veselina Masleše 55, Beograd, Serbia. Email: d.novak@sbb.rs

Virus is shed intermittently in the saliva. Haematogenous spread does not occur. Near the end of the clinical phase, after replication in the CNS, virus may be found in nearly every innervated organ.

The incubation period varies from several days to several months. The virus usually incubates from two to eight weeks before signs are noticed. Typically, the virus remains at the inoculation site for a considerable time. Most rabies cases in dogs develop within 21–80 days after exposure, but the incubation period may be shorter or considerably longer. Transmission of the virus through saliva can happen as early as ten days before symptoms appear. Once symptoms are present, the disease is fatal for both animals and humans.

Clinical signs

Rabies virus causes acute encephalitis in all warm-blooded hosts. In dogs, the first symptoms of rabies may be nonspecific and include lethargy, fever, vomiting, and anorexia. All animals exhibit certain neurological signs as a result of rabies. Clinical signs of rabies will vary depending on the effect of the virus on the brain. Signs progress within days to cerebral dysfunction, cranial nerve dysfunction, ataxia, weakness, paralysis, seizures, difficulty breathing, difficulty swallowing, excessive salivation, abnormal behaviour, aggression, and/or self-mutilation. Typical signs include sudden behavioural changes and progressive paralysis leading to death. It is important to understand that animal may die rapidly without demonstrating significant clinical signs.

The most reliable signs, regardless of species, are acute behavioural changes and unexplained progressive paralysis. Behavioural changes may include sudden anorexia, signs of apprehension or nervousness, irritability, and hyperexcitability. The animal may seek solitude. Ataxia, altered phonation, and changes in temperament are apparent. Uncharacteristic aggression may develop and infected animal may suddenly become vicious.

The disease progresses in few stages

Prodromal form. In the first or prodromal phase the dog undergoes a change in temperament and behaviour. Early, the dog can show only mild signs of CNS abnormalities. Fever may be present. Quiet dogs become agitated and active pets become nervous or shy. Pruritus

may be present at the site of exposure. This first stage usually lasts for about 1–3 days. Most dogs will then progress to the paralytic stage, the furious stage, or a combination of the two. As said earlier, some dogs will have infection without displaying any major symptoms.

Paralytic (dumb) form. The majority of canine cases will show paralytic (dumb) phase. Animals with this form of rabies may be depressed or unusually docile. This stage is characterized by the inability to swallow, distortion of the face leading to a typical sign of foaming saliva around the mouth. This is manifest by ataxia and paralysis of the throat and masseter muscles, often with profuse salivation and the inability to swallow. Dropping of the lower jaw is common in dogs. Voice or bark changes are often noticed. Some animals may develop paralysis beginning at the hind extremities with typical signs of lower motor neuron paralysis. Disorientation, incoordination and staggering may occur, caused by paralysis of the hind legs. Eventually, complete paralysis and coma are followed by death. Owners will frequently think the dog has something stuck in the mouth or throat. Care should be taken in examination since rabies is transmitted by saliva. This form lasts 1 to 7 days, from onset of overt signs to death.

Excitative (furious) form. The prodromal stage is followed by a period of severe agitation and aggressiveness. Initially, a dog that has become infected may show extreme behavioural changes such as restlessness or apprehension and aggression. Furious rabies is characterized by extreme behavioural changes, including overt aggression and attack behaviour. Animals with this form of rabies may demonstrate sudden behaviour changes, and attack without provocation. Animals may be anxious, highly excitable and/or aggressive with intermittent periods of depression. A fever may also be present at this stage. As the virus progresses, an infected dog may become hypersensitive to touch, light and sound. The animal often bites any material. Rabid dog becomes highly excitable and displays evidence of a depraved appetite, eating and chewing stones, earth and rubbish (pica).

Infected dogs may develop a typical high barking sound during furious rabies. As the disease progresses, muscular weakness, incoordination and seizures are common.

Paralysis eventually sets in and the rabid animal may be unable to eat and drink. Hydrophobia (fear of water) is not a sign of rabies in dogs. This is a feature of human

rabies. Death results from progressive paralysis. Death may follow convulsions even without the paralytic stage of the disease. This form lasts 2 to 8 days, from onset of overt signs to death.

Diagnosis

Clinical diagnosis is difficult, especially in areas where rabies is uncommon. In the early stages, rabies can easily be confused with other diseases or with normal aggressive tendencies. Differential diagnosis that should be included in case of suspected rabies include: encephalitis: viral (canine distemper), immune mediated, pseudorabies, toxicity (e.g. lead), portosystemic shunt, hypoglycemia, neoplasia, trauma, other causes of ptyalism.

When rabies is suspected and definitive diagnosis is required, laboratory confirmation is indicated. Suspect animals should be euthanized, and the head removed for laboratory shipment. Rabies diagnosis should be done by a qualified laboratory, designated by the local or state health department in accordance with established standardized national protocols for rabies testing. Immunofluorescence microscopy on fresh brain tissue, which allows direct visual observation of a specific antigen-antibody reaction, is the current test of choice. It can establish a highly specific diagnosis within a few hours. Brain tissues examined must include medulla oblongata and cerebellum (and should be preserved by refrigeration with wet ice or cold packs). Virus isolation by the mouse inoculation test or tissue culture techniques using mouse neuroblastoma cells may be used for confirmation of indeterminate fluorescent antibody results.

Prevention and control

Rabies is a vaccine-preventable disease. The most cost-effective strategy for preventing rabies in people is by eliminating rabies in dogs through vaccination. Integrated veterinary management of local animal populations, by mass vaccination of dogs and community promotion of responsible pet ownership, is the most cost-effective, humane, long-term solution toward eliminating regional canine rabies in a One Health context.

Comprehensive guidelines for control in dogs have been prepared internationally by the World Health Organization and they include the following: 1) notification of suspected

cases, and euthanasia of dogs with clinical signs and dogs bitten by a suspected rabid animal; 2) reduction of contact rates between susceptible dogs by leash laws, dog movement control, and quarantine; 3) mass immunization of dogs by campaigns and by continuing vaccination of young dogs; 4) stray dog control and euthanasia of unvaccinated dogs with low levels of dependency on, or restriction by, people; and 5) dog registration.

Many effective vaccines, such as modified-live virus, recombinant, and inactivated types, are available for use throughout the world. Recommended vaccination frequency varies from 1 to 3 years, after an initial series of two vaccines 1 year apart.

Zoonotic aspect

Rabies has the highest case fatality of any infectious disease. When a person is exposed to an animal suspected of having rabies, the risk of rabies virus transmission should be evaluated carefully. Risk assessment should include consideration of the species of animal involved, the prevalence of rabies in the area, whether exposure sufficient to transmit rabies virus occurred, and the current status of the animal and its availability for diagnostic testing.

Any healthy domestic dog whether vaccinated against rabies or not, that exposes (bites or deposits saliva in a fresh wound or on a mucous membrane) a person should be confined for 10 days; if the animal develops any signs of rabies during that period, it should be euthanized and its brain promptly submitted for rabies diagnosis. If the dog responsible for the exposure is stray or unwanted, it may be euthanized as soon as possible and submitted for rabies diagnosis. Since the advent of testing by immunofluorescence microscopy, there is no value in holding such animals to "let the disease progress" as an aid to diagnosis.

Internationally, the World Health Organization recommends several types of cell-culture vaccines for human groups at risk.

Occupational groups regularly in contact with animals for example, veterinarians, animal control and wildlife officers should obtain protection through pre-exposure vaccination. Abattoir personnel, particularly in endemic

areas, must take preventive actions to prevent infection from saliva, salivary gland and nervous tissue of infected animals. Infection does not occur by consumption of meat from a rabid animal

Preexposure immunization is strongly recommended for people in high-risk groups. Preexposure vaccine is administered on days 0, 7, and 21 or 28. Preexposure prophylaxis alone cannot be relied on in the event of subsequent rabies virus exposure and must be supplemented by a limited postexposure regimen (two doses of vaccine, IM, on days 0 and 3). For healthy, unvaccinated patients bitten by a rabid animal, postexposure prophylaxis consists of wound care, local infiltration of rabies immune globulin, and vaccine administration on days 0, 3, 7, and 14. Modern postexposure prophylaxis assures human survival if it is provided on time and in appropriate manner.

Box

References

1. Baer GM (1991). The Natural History of Rabies, Second Edition. CRC Press, Boca Raton, Florida, USA, 620 pp.
2. Pastoret PP, Brochier A, Aguilar-Setién A, Blancou J (1997). Chapter 18 Examples of vaccination, Part 2: Vaccination against rabies. In: Veterinary Vaccinology, Pastoret, P.-P., Blancou J., Vannier P. & Verschuereen, eds. Elsevier Science, BV, Amsterdam, the Netherlands, pp 616–628.
3. Rupprecht CE, Barrett J, Briggs D, Cliquet F, Fooks AR, Lumlertdacha B, Meslin FX, Müller T, Nell H, Schneider C, Tordo N, Wandeler AI (2008). Can rabies be eradicated? *Dev. Biol. (Basel)*, 131, 95–121.
4. World Health Organization (2007). Oral Vaccination of Dogs against Rabies. WHO, Geneva, Switzerland.
5. Merck Veterinary Manual: www.merckvetmanual.com/mvm/index.jsp?cfile=htm/bc/toc_50000.htm
6. OIE Reference Laboratories for Rabies. <http://www.oie.int/en/our-scientific-expertise/reference-laboratories/list-of-laboratories/>
7. OIE web portal on Rabies: www.oie.int/en/animalhealth-in-the-world/rabies-portal/

Clinical cases: not always obvious

Clinical diagnosis is difficult, especially in areas where rabies is uncommon. In the early stages, rabies can easily be confused with other diseases or with normal aggressive tendencies, as shown by these two cases seen by the author (Serbia, early 2000s).

Clinical case n° 1

Muna, a 3-year-old intact female GSD. Owned by a breeder, living in the countryside and kennelled since puppyhood. Used as a show dog.

Presenting history

Retching, scratching neck region, very mild salivation, subtle weakness. As the dog had been fed raw fish heads, the owner was suspicious of a foreign body in the mouth/throat. The dog had been seen by a local vet, who also suspected a foreign body in the mouth. The vet had sedated the dog (ketamine only) and had explored the mouth (without gloves). Antibiotics and steroids were prescribed 'just in case' and the dog was referred for X-rays at the owner's request.

Clinical examination

T 39.5°C. Chest auscultation was unremarkable, as was abdominal palpation. Muna showed moderate salivation, muscle twitching, vocalisation, anisocoria and was semi-recumbent – some of which could also be attributed by ketamine. However, they could also be caused by rabies, which is why quarantine and observation was recommended, in particular since there was no history of rabies vaccination. However, the owner refused to leave the dog in observation and spent the night with the dog. The local veterinary inspector and head of infectious department were contacted.

The following morning, the dog showed moderate salivation, mild ataxia when walking and muscle twitching. The dog also seemed somewhat 'disorientated'. The owner insisted on further investigation to rule out the foreign body, so an X-ray was taken. Chest radiographs showed no foreign body – but gas in the oesophagus, which is consistent with paralysis of the throat and masseter muscles.

... continued on the next page



X-ray of Muna, showing gas in the oesophagus.

Follow-up

No underlying disease could be found, nor any history of exposure to rabies. The owner had become very emotional at the mere suggestion of rabies and refused quarantine and came to remove the dog. As it was physically impossible to retain the dog against the owner's will, the vet convinced the owner to a minimum agreement of placing it in isolation (kennel) at all times for observation, and to contact the vet if the dog died. The referring local vet was also informed.

No news was received for two days. On the third day, the owner called to say that the dog had died... the previous day... and had been buried! Despite winter conditions (frozen soil), the body was dug up and was sent to the Serbian Pasteur Institute (EU reference laboratory for rabies). The following day, rabies was confirmed.

Epilogue

As there was a theoretical risk of exposure due to contact with saliva in a fresh open wound, the veterinary staff all underwent post-exposure prophylaxis (five injections of hyperimmune antiserum over 28 days).

Clinical case n° 2

Presenting history

A 2-year-old female German short-haired pointer, used as a hunting dog, was referred by a local veterinarian for hind limb weakness for two days. Steroids had been prescribed but no improvement was seen.

Clinical examination

As rabies was part of the differential diagnosis, the clinical examination consisted of observation only. The dog was ataxic, but also showed moderate salivation, muscle twitching and a lowered mandible. The dog also seemed anxious, disoriented and was highly excitable.

The dog was referred to a veterinary school for isolation and observation.

Follow-up

The dog died three days later. Rabies was confirmed.



FECAVA Symposium*

Travelling with pets: am I giving the correct advice?

Cathy Tourlouse¹

SUMMARY

Since 2003, the rules to travel within Europe and to travel from a non-EU country to a European country with dogs, cats and ferrets have been harmonised. All details can be found in Regulation (EU) 576/2013 on non-commercial movement of pet animals and in Commission Implementing Regulation (EU) 577/2013.

The objective is to lay down animal health requirements for the non-commercial movement of dogs, cats and ferrets in order to prevent or minimize risks to public health mainly concerning rabies.

The Regulation of the European Parliament and of the Council of 12 June 2013 creates a frame on the non-commercial movement of pet animals repealing Regulation (EC) 998/2003.

The Commission Implementing Regulation (EU) 577/2013 of 28 June 2013 provides more detail:

- Model identification documents for the non-commercial movement of dogs, cats and ferrets
- Establishment of lists of territories and third countries
- Format, layout and language requirements of the declarations attesting compliance with certain conditions provided for in 576/2013

* This paper was presented at the FECAVA symposium on rabies held in Barcelona on 16 October 2015.

Eur J Comp An Pract, Spring 2016; (26)1:p 15-19
Go to <http://www.ejcap.org> for the interactive online presentation of this paper

Commercial versus non-commercial

First of all, it is important to distinguish between commercial and non-commercial movement of pets. A non-commercial movement of a pet animal is defined as one whereby the pet accompanies its owner and is under his direct responsibility without the purpose of sale or transfer of ownership. This move may take place up to five days before or after the movement of the owner and can be carried out under the responsibility of someone who has a written authorisation from the owner. The maximum number of pet animals that may be moved is five.

Exceptionally, this maximum number of five may be exceeded if all of the following conditions are met:

- The movement is for the purpose of participation in competitions, exhibitions or sporting events or in training for such events;
- Written evidence is provided that the animals are registered either to attend an event or with an organisation organising such events;
- The animals are more than six months old.

In all other cases, the same conditions apply as for non-commercial movement – in addition to the requirements of trade in or import into the Union: coming from registered holding (competent authorities check on animal health and animal welfare), specific health certificate, clinical exam within 48 hours before departure, notification through TRACES (Trade Control And Expert System: pre-notifies arrival to BIP – border inspection post).

¹ Cathy Tourlouse DVM, DAP Iscavets, Terhulpensteeweg 246, 3090 Overijse, Belgium
E-mail cathy.tourlouse@skynet.be

Requirements for non-commercial travelling within the EU

The basic requirements for non-commercial travelling within the EU are pretty straightforward:

- **Microchip** ISO standard 11784/11785 (tattoo accepted if applied before 3 July 2011 and readable). The implantation of microchip is an invasive procedure where complications are possible and can only be implanted by a suitable qualified person e.g. veterinarian. If a Member state allows another person to implant a transponder, it should lay down rules on the minimum qualifications required for such a person.
- **Vaccination against rabies**
- Waiting period of at least **21 days**, except after booster within validity period of the vaccine as stated in the specifications of the marketing authorization in the country where it is administered
- **Treatment against *Echinococcus multilocularis*** with praziquantel for dogs within 24 hours to 5 days prior to entering the UK, Ireland, Finland, Malta or Norway
- **European passport** duly completed and mentioning all of the above
- Written declaration signed by the owner, if the movement of the animal is carried out under the responsibility of a person who has authorisation (in writing) from the owner to move the animal on behalf of the owner, within five days of the owner's movement.

Most of these acts can only be carried out by approved veterinarians. Please note that some countries have breed specific limitations.

Young animals

Since 29 December 2014, pets must be at least 12 weeks old before being vaccinated against rabies for the purpose of travelling. Adding the 21 days' waiting period means that pets cannot travel before 15 weeks of age.

Some countries will allow animals less than 12 weeks of age without rabies vaccination. Some countries will allow 12 to 16-week-old animals that have received a rabies vaccination but not yet fulfilled the 21 days' waiting period. However, both exemptions can only be granted if:

- Either the owner or the authorised person provides a signed declaration that from birth until the time

of the non-commercial movement the pet animals have had **no contact with wild animals** of a species susceptible to rabies, or

- The pet animals are **accompanied by their mother**, on whom they still depend, and from the identification document accompanying their mother it can be established that, before their birth, the mother received an anti-rabies vaccination which complied with the validity requirements.

Format, lay out and language requirements of the above-mentioned declarations are set out in 576/2013.

Derogation from the anti-rabies vaccination

Mutual agreement to derogate from the anti-rabies vaccination is possible between certain member states of equal favourable rabies status through a joint application if certain conditions are met:

- Ongoing surveillance and reporting system with regard to rabies
- States are free of rabies and rabies is not known to have been established in wild animals for at least two years prior to joint application
- Efficient and effective control measures to prevent introduction and spread of rabies
- Justified and proportionate to the risks to public or animal health

New passport model since 29 December 2014

- Only for pets receiving a passport from 29 December 2014 onwards – no need to replace older passports
- Increased security through laminated strips to cover the page with microchip information and any treatment certified with a sticker
- Dedicated page with the details of the vet issuing the passport
- A 'valid from' date needs to be added for rabies vaccinations to simplify the compliance check procedure
- Signature of owner required

Requirements for non-commercial travelling from outside the EU to the EU

- Microchip ISO standard 11784/11785 or reader provided (tattoo accepted if applied before 3 July 2011 and readable)

- Vaccination against rabies
- Rabies antibody titration test at least 30 days after vaccination and not less than 3 months before travelling – if the test was performed in a member state before leaving the Union there is no need to wait 3 months before re-entering the Union – test result remains valid if boosters are given within the validity period
- Treatment against *Echinococcus multilocularis* with praziquantel for dogs within 24 hours to 5 days prior to entering the UK, Ireland, Finland, Malta or Norway
- Identification document duly completed, mentioning all of the above and endorsed by an official vet – model see 577/2013 – can be similar to European passport
- Fixed points of entry for most countries (others than Andorra, Switzerland, Faeroe Islands, Gibraltar, Greenland, Croatia, Iceland, Liechtenstein, Monaco, Norway, San Marino, Vatican City State) - derogation possible for military and search-and-rescue dogs if a permit (advance application) has been granted and the dogs undergo a compliance check.

The same derogations for young animals and non-vaccinated animals may apply if the pet is coming from a safe country (see box 1).

There is no need for antibody titration test if the pet comes from a safe country.

Note: When preparing an animal for departure to a non-EU country, always check the requirements of the country of destination!

Transit in a non-safe country is possible if accompanied by a signed declaration of the owner or authorized person that during transit, the pet(s) had no contact with animals of species susceptible to rabies and remain secured within a means of transport or within the perimeter of an international airport.

Pitfalls of the EU legislation

Even if plenty of consideration is made when establishing the rules (and amending them, like in 2014) there are still a lot of issues that need to be addressed.

Lack of knowledge

First of all even if the Member States are required to make the relevant provisions of the law available to the public there is a huge lack of knowledge. The increased mobility of owners with their pets and the lack of border

BOX 1

'Safe' countries Annex II of 577/2013

Part I: apply rules the content and effect of which are the same as those applied by Member States

Andorra, Switzerland, Faeroe Islands, Gibraltar, Greenland, Croatia, Iceland, Liechtenstein, Monaco, Norway, San Marino, Vatican City State

No fixed points of entry – no systematic check – similar identity document as European Passport possible – no rabies antibody titration needed to enter a Member State

Part II: territories or third countries that have demonstrated that they meet at least some criteria

Ascension Island, UAE, Antigua and Barbuda, Argentina, Australia, Aruba, Bosnia and Herzegovina, Barbados, Bahrein, Bermuda, Bonaire, Sint Eustatius and Saba (BES Islands), Belarus, Canada, Chili, Curaçao, Fiji, Falkland Islands, Hong Kong, Jamaica, Japan, Saint Kitts and Nevis, Cayman Islands, Saint Lucia, Montserrat, Mauritius, Mexico, Malaysia, New Caledonia, New Zealand, French Polynesia, Saint Pierre and Miquelon, Russia, Singapore, Saint Helena, Sint Maarten, Trinidad and Tobago, Taiwan, USA (incl American Samoa, Guam, Northern Mariana Islands, Puerto Rico, US Virgin Islands), Saint Vincent and the Grenadines, British Virgin Islands, Vanuatu, Wallis and Futuna, Mayotte

No rabies antibody titration needed to enter a Member State

controls contribute to a certain risk. Putting the initiative for the checks at the points of entry at the level of the owner is tricky. Lack of knowledge also concerns the officials charged with compliance checks and the veterinary profession. Definitely more emphasis should be put on the importance of preventing rabies and the impact of rabies on public health.

Passport

Even if the model of the European passport is fixed still too much flexibility is allowed. The purpose of the passport is to allow it to follow the pet through its entire life. Often too little space is available for consecutive owners allowing the mutation of nationality of the pet.

Age and vaccination status of puppies

Many of us also have noted an acute epidemic of a rare genetic disorder: the 'delayed teeth eruption' in puppies from certain countries... This raises the question of whether the puppy has actually been vaccinated? And if so, considering maternal antibodies, whether the puppy has achieved an appropriate antibody level lasting through the validity period of the vaccination?

Adoptable pets: commercial or not?

Commercial travel can be 'disguised' as non-commercial travel, especially for pets available for adoption from southern Europe coming to northern Europe. Some suggest creating an exception – but is this desirable? It removes the need for the countries concerned to address their animal welfare problems. It also facilitates the spread of emerging diseases. Such pets often are fearful and might not fit in well with the family they're put in. And it also reduces chances for local shelter pets.

Socialisation

Finally, even if the delayed age for vaccination can be perfectly justified for protecting human and animal health, it poses a serious threat to pet behaviour. If puppies can only travel after 15 weeks of age, and if paper work is actually accurate, a crucial period in the development of the animal is missed unless the breeder puts a lot of additional effort in his offspring which isn't likely to be compensated making sales abroad less likely. Even if socialisation at a later age is still possible it will be more

difficult. A problem which would even be worse in puppy trade if birth dates were accurate. We should also consider the negative impact of chronic unresolvable stress of environmental stimuli if no attachment is possible to a human social partner after weaning.

The case of Belgium

Since May 2013, Belgian vets have a legal obligation to notify the competent authorities if they encounter a non-conform importation of a pet originating from a country not mentioned in the OIE-list of rabies-free countries. The general practitioner makes a preliminary risk assessment. If there is a risk, the competent authority takes over to further investigate and take appropriate measures. In addition, a free hotline has been set up to notify health issues after adopting a dog. The purpose is to get an overview of frequent problems linked to the origin of the pet and to decide whether appropriate measures should be taken. One of the issues is the 'delayed teeth eruption'.

'Force majeure'

In certain exceptional circumstances for conditions relating to the owner an exception can be granted to the general rules if a permit is applied for and granted. The pet will be isolated under official supervision in a place approved by the competent authority till the conditions are fulfilled.

Derogation

Non-commercial movement of pet animals between following countries and territories may continue under the conditions laid down by the national rules of those countries and territories:

- San Marino – Italy
- Vatican – Italy
- Monaco – France
- Andorra – France
- Andorra – Spain
- Norway – Sweden
- Faeroe Islands – Denmark
- Greenland – Denmark

Compliance

Compliance is achieved by documentary and identity checks:

- Spot checks
- Travellers' point of entry: owner or authorised person contacts the competent authority at arrival for pets coming from non-EU countries not listed in part I of annex II of 577/2013

Training of staff at points of entry should be achieved by Member States and they should keep records of number of checks and instances of non-compliance.

Non-compliance

Several options are possible in the instance of non-compliance, all at the expense of the owner or the authorised person:

- Return the pet to country of origin of dispatch
- Isolate the pet under official control for the time necessary for it to comply with the rules
- As last resort: euthanasia of the animal

Record keeping for the vet

The new regulation requires vets to keep records for no less than 3 years of the passport numbers in combination with the information of section I, II and III:

- Microchip number (or tattoo where applicable), location, date of reading or application
- Name, species, breed, sex, colour, date of birth, specific features or characteristics of the animal
- Owner's name and contact details

Competent authorities need to record details of vet and the alphanumeric codes of the passports he received for at least 3 years.



Diseases don't respect borders



Additional Information

http://ec.europa.eu/food/animals/pet-movement/index_en.htm

http://ec.europa.eu/food/animal/liveanimals/pets/nat_rules_dogscatferret_en.htm

http://www.fve.org/uploads/publications/docs/044_questions_and_answers_re_new_pet_passport_regulation_0210.pdf



FECAVA Lecture*

Uncommon bone pathologies

Josep Font Grau¹

SUMMARY

In human medicine a disease is considered to be rare if its incidence is one per 2,000 individuals or less. The majority are genetic in origin and only 30% of the thousands that exist are studied. In animals this concept is not established but there are a number of pathologies that can be classified as rare because of their low incidence in daily clinical practice. We will refer only to bone diseases that pose a challenge for the clinician both when making a diagnosis using imaging techniques and/or biopsies, and when determining possible treatments. This paper will highlight multiple cartilaginous exostosis, epiphysiolysis of the calcaneus, femoral epiphysiolysis, Osgood-Schlatter syndrome, synovial osteochondromatosis, calcinosis circumscripta, low-grade osteosarcomas, secondary bone lesions to leishmaniasis and some pathologies related to growth and nutrition.

KEY WORDS: Bone, uncommon pathologies, dog

* This paper is based on the FECAVA lecture delivered at the 21st EuroCongress in Barcelona, Spain in October 2015.
Eur J Comp An Pract, Spring 2016; (26)1: p20-32
Go to <http://www.ejcap.org> for the interactive online presentation of this paper

Multiple cartilaginous exostosis

Multiple cartilaginous exostosis (MCE) is a bone disease of uncertain origin characterized by multiple, cartilage-capped bony protuberances that arise from the surfaces of bones formed by endochondral ossification⁽¹⁾ (Figures 1-4). Historically, the condition has been described by various terms (chondroma, diaphyseal aclasis, dyschondroplasia, enchondromatosis, hereditary deforming chondrodysplasia, hereditary multiple exostoses, multiple osteochondromatosis). However, a certain degree of agreement on nomenclature has currently been achieved whereby a solitary lesion is referred to as osteochondroma, and the presence of multiple lesions in an individual is defined as multiple cartilaginous exostosis⁽¹⁾. The vertebrae, ribs and long bones are most frequently

affected^(1, 2). Exostoses affecting the ribs can be found on both lateral and pleural rib surfaces and may compress pulmonary lobes⁽¹⁾. The growth of osteochondromas continues until skeletal maturity and they may remain subclinical unless enlargement causes dysfunction by the compression of a vital structure, typically the spinal cord or interference with joint function⁽³⁾. Chondrosarcomas and osteosarcomas are usually reported as a result of a malignant transformation of a single exostosis in the dog. Sequential radiographic studies and bone biopsies are required to confirm neoplastic transformation of an exostosis⁽⁴⁾. The aetiology of canine MCE is uncertain. The two main theories based on the human condition have been adopted by veterinary medicine.

The first theory explores factors such as perichondrial ring defects, or physical stresses at the margin of the growth plate, which lead to a proliferation of the growth plate in an unnatural direction, developing a growth plate-like structure at right angles to the bone shaft. The second theory considers that the defect may lie with the periosteum, which due to some unknown initiating factor may regain its perichondrial potential⁽²⁾.

¹ Josep Font Grau DVM PhD, Hospital Veterinari Canis, Pau Birol 36, 17006 Girona, Spain. Email josepfontgrau@hotmail.com

Some authors believe that both theories are not mutually exclusive. The early age of onset, the metaphyseal location of the limb lesions in the majority of cases, and the characteristic radiographic and histological patterns

allow for the differentiation between MCE from other syndromes characterized by benign polyostotic exostoses, such as canine disseminated idiopathic skeletal hyperostosis or tumoural calcinosis ⁽⁵⁾.

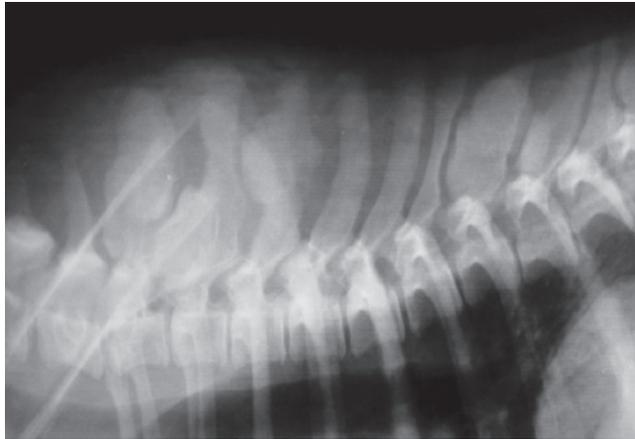


FIG 1. Cross-breed Golden Retriever, 6 months old. Lateral radiograph of the thoracic spine showing several bone masses affecting the spinous processes of the thoracic vertebrae.

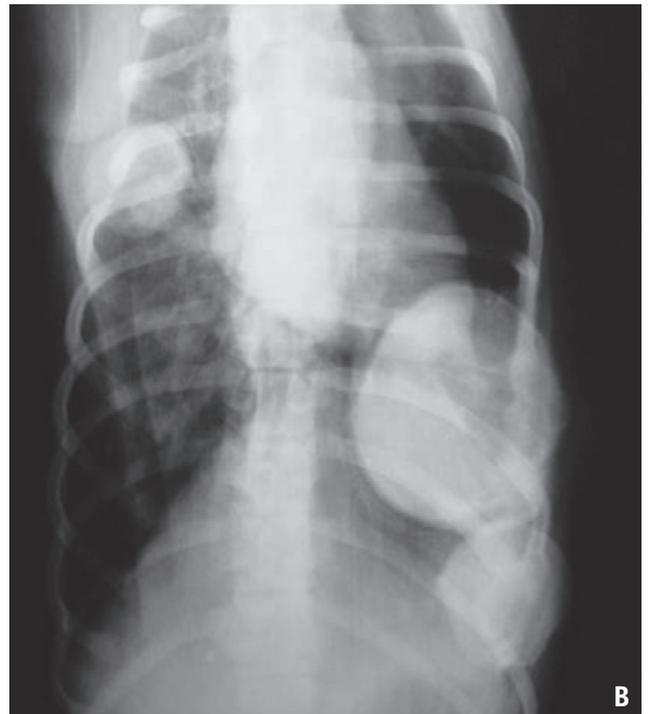


FIG 2. Lateral (2A) and dorsoventral (2B) radiographs of the thorax showing the rib exostoses.

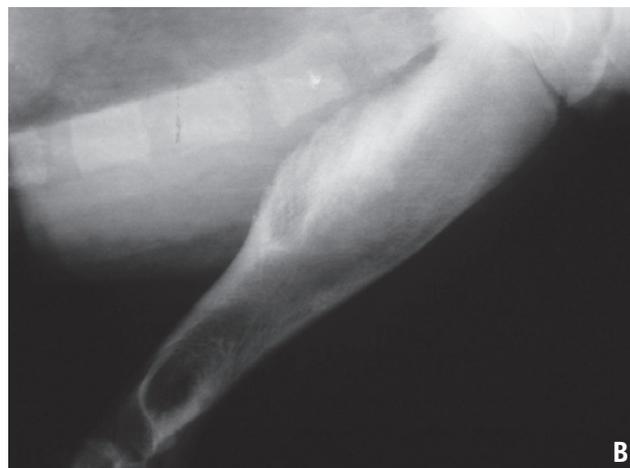


FIG 3. Mediolateral radiographic view of the forelimb showing the exostosis affecting the proximal radius (3A) and humerus (3B).

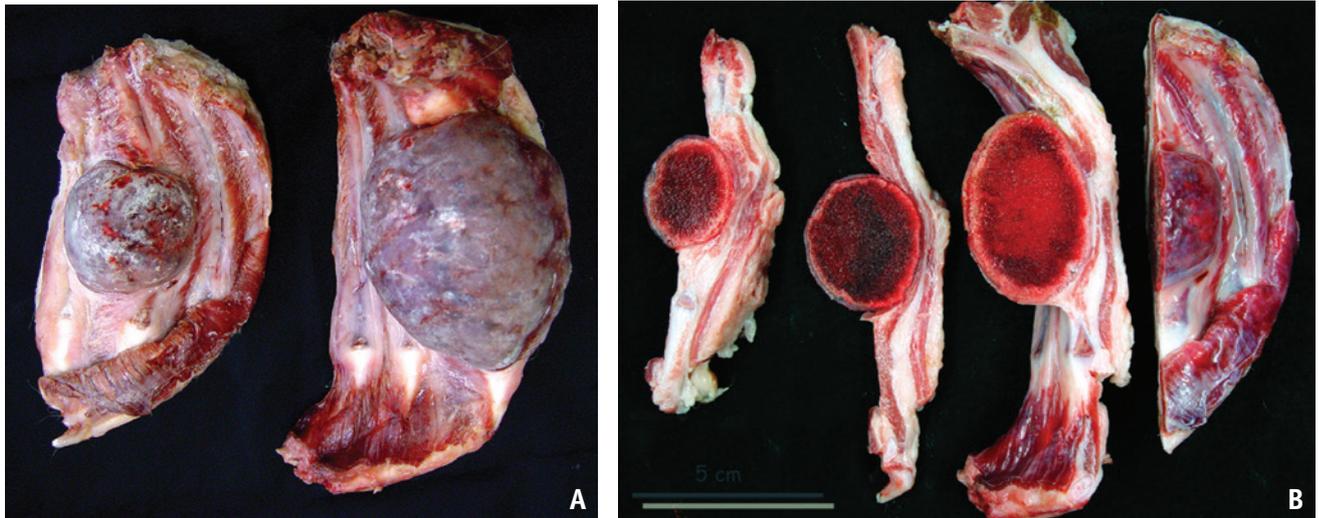


FIG 4. The costal mazes in post-mortem examination: smoothly contoured, round-shaped (4A). Once sectioned, a hard pinkish covering could be observed (4B).

Calcaneal epiphysiolysis

Partial or complete separation of an epiphysis from the diaphysis of bone is known as epiphysiolysis⁽⁶⁾ (Figures 5-7). In skeletally immature dogs, it is a common consequence of trauma resulting in Salter-Harris type I fractures^(6, 7). Separation and lysis of the femoral capital epiphysis without any history of trauma has been well described, although a few cases have been reported to affect the calcaneal epiphysis^(8, 9). There are many unproven theories explaining the aetiology of calcaneal epiphysiolysis⁽⁹⁾. The most accepted one is that this condition may be a form of osteochondrosis. Other theories include an excess of mechanical traction on the epiphysis because of an increased tibiotarsal angle and micro-trauma resulting in fractures with disruption and avulsion of the epiphysis. The epiphysis of the calcaneus is a traction epiphysis and may represent a former sesamoid in the

gastrocnemius tendon⁽⁹⁾. Physeal areas are considered to be a natural weak point during periods of rapid growth⁽⁸⁾. Sever's disease, and other similar conditions described in humans, little-leaguer's elbow and iliac apophysitis are believed to be caused by decreased resistance to shear stress at the bone-growth plate interface. Studies have indicated that traction epiphyses have a higher composition of fibrocartilage than epiphyses subjected to a higher axial load, which are composed predominantly of hyaline cartilage⁽⁶⁾. Epiphysiolysis of unknown origin in growing dogs can affect different bones. Those that involve the calcaneus bilaterally are very rare, based on the few cases reported. Histologic examinations show areas with loss of the trabecular bone structure with formation of organized and disorganized osteoid tissue associated with a large amount of fibrocartilaginous proliferation⁽¹⁰⁾. The prognosis of calcaneal epiphysiolysis is grave in view of the results obtained in the literature. More studies need to be done to provide information about the best approach for this condition⁽¹⁰⁾.

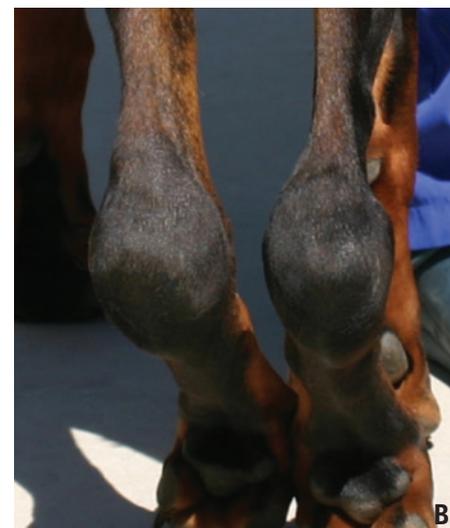


FIG 5. Doberman Pinscher, 6 months old. At rest, hyperflexion of the interphalangeal joints (5A) and swelling of the tibiotarsal joints (5B).



FIG 6. Lateral radiographic images of the right (A) and left (B) hind limbs. Non-union between the calcaneal tuber with the body of the calcaneus. Evidence of bone remodelling.



FIG 7. Lateral radiograph of the right hind limb, 3 weeks after surgery. Separation and collapse of the physis and progression of the osteolysis. The holes of the pins of the type II transarticular external fixator that remained for three weeks can be seen.

Femoral epiphysiolysis

Femoral epiphysiolysis or slipped capital femoral epiphysis (Figures 8-9) is described when separation of the capital epiphysis appears to occur in the absence of any trauma. In a retrospective review of 43 femoral fractures, three dogs had separation of the capital femoral epiphysis from metaphysis in the absence of trauma⁽¹¹⁾. This is a rare and under-diagnosed pathology, which has also been described in other species such as cats and, mainly, pigs⁽¹²⁾. In dogs, the femoral physis stays open until 9 months of age. The aetiology is not clear and is associated with rapid growth rates, manifestation of osteochondrosis mechanical factors and more probably a form of

physeal dysplasia⁽¹³⁾. Widening of the growth plate as a consequence of osteochondrosis will predispose it to biomechanical failure secondary to torsional or shearing forces⁽¹¹⁾. In children it may be associated with endocrine abnormalities such as hypothyroidism and hypogonadism, but the majority of cases are idiopathic⁽¹¹⁾. In dogs, 30% of cases occur bilaterally. Excessive stress in twisting and bending can damage the growth plate so it is important to avoid rapid growth, excess weight and excess exercise. Clinical examination, radiography and histopathology lead to a definitive diagnosis. Histopathologically, cartilaginous lesions with cavities occupied by eosinophils and chondrocytes arranged in clusters and irregular columns of osteocytes and foci of necrotic tissue⁽¹²⁾. In early cases with little displacement of the physis, stabilization may be attempted by internal fixation, although arthroplasty or complete hip replacement are the treatments of choice⁽¹³⁾.



FIG 8. Golden Retriever, 7 months old. Evolution of a case of slipped capital femoral epiphysis, on the day of diagnosis (8A), at 2 weeks (8B) and one month (8C).

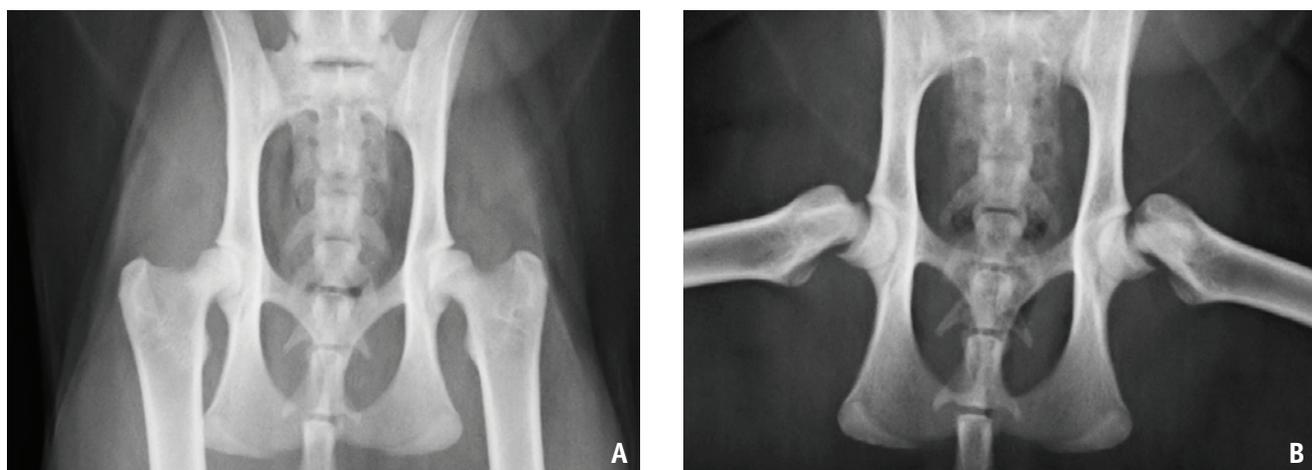


FIG 9. Case of slipped capital femoral epiphysis in a common European cat of 6 months of age. In the ventrodorsal radiography with limbs extended, the separation (9a) is seen poorly, becoming more evident in the ventrodorsal position with the limbs bent (9B).

Osgood-Schlatter disease

This is a condition affecting human adolescents in which there is partial separation of bone fragments from the tibial tuberosity at the site of insertion of the patellar ligament to the tibial tuberosity. Clinical signs in people consist of swelling and pain at the proximal part of the tibial tuberosity and around the distal end of the patellar ligament⁽¹⁴⁾. Its pathogenesis is believed to be caused by repetitive tendon/muscle strain at the insertion of the patellar tendon to the immature tibial tuberosity, which has its own secondary ossification centre. Morphologically it is characterized by chronic avulsion with incomplete separation of the tuberositas tibiae, and proximal dislocation of the patella (patella alta)⁽¹⁵⁾. The term Osgood-Schlatter disease has also been used for the canine patient⁽¹⁴⁾. However, radiographs of these patients typically show an enlarged radiolucent line at the apophyseal plate of the tibial tuberosity (Figure 10). This finding is consistent with a mild avulsion fracture of the canine tibial tuberosity. Based on the radiographic

differences between the two species, it seems more appropriate to use the term Osgood-Schlatter disease only for people⁽¹⁴⁾. There are different classification systems to describe the tibial tuberosity avulsions in growing dogs according to the displacement of the tibial tuberosity and fracture through the apophysis^(14, 16). Conservative therapy for minimally displaced avulsion fractures is usually also successful in dogs. Surgery by a tension-band-wire should be considered in significant displacement of the tibial tuberosity⁽¹⁷⁾.

Synovial chondromatosis

This disorder, also known as synovial chondrometaplasia, synovial osteochondromatosis and synovial chondrometaplasia, is characterized by the formation of chondral or osteochondral nodules in synovial joint tissue, tendon sheaths or bursa⁽¹⁸⁾. The nodules may become pedunculated and break off forming loose bodies often referred to as "joint mice" which may continue to grow because they are nourished by synovial fluid

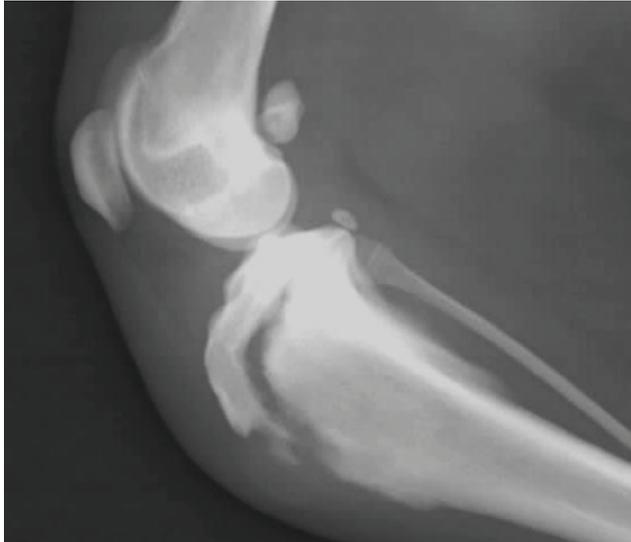


FIG 10. Lateral radiograph of the right stifle in an 8 month old Weimaraner with a lesion compatible with Osgood-Schlatter Disease, showing a displacement of the tibial tuberosity apophyseal plate.



FIG 11. Mixed breed 25 kg, 2 years old. Shoulder radiograph shows mineral density opacities in the caudal aspect (11A). Craniolateral arthrotomy allowed removal of four loose bodies (11B). The loose bodies were composed of a core of necrotic trabecular bone overlain by cartilage.

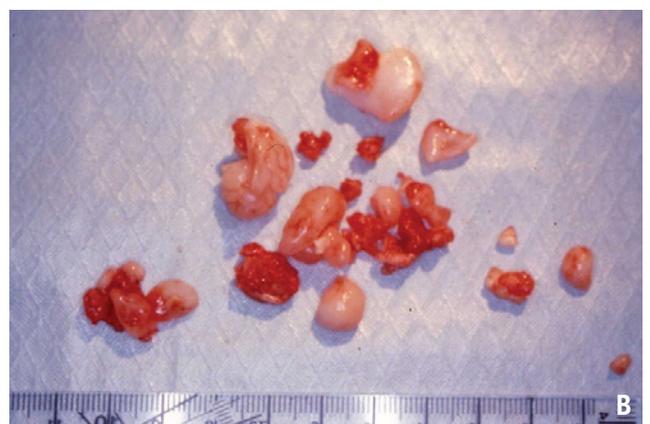


FIG 12. Four-year-old German Shepherd. Radiographic examination of the stifle revealed multiple, smooth bone densities of variable size (12A). Osteochondral bodies after removal (12B). Courtesy of Dr. Jacques Drapé.

(18). The most typical presentation is at the level of the shoulder joint (Figure 11). It is usually mono-articular and rarely affects several joints. At knee level, numerous intra-articular calcifications can form that have been defined as a “snowstorm” in appearance (19) (Figure 12). The pathogenesis is not known but in human medicine a fibroblast growth factor FGF-9 has been found, which maintains the mesenchymal cells in a proliferative state (19). Treatment includes surgical fragment removal and sometimes synovial stripping, and controversy exists regarding the removal of chondral bodies alone or associated synovectomy. Malignant transformation to chondrosarcoma is very rare and the histopathological distinction between synovial chondromatosis and synovial chondrosarcoma is not clear (20).

Calcinosis circumscripta

Calcium salts (phosphates or carbonates) are deposited on the soft tissues or in periarticular position. Some authors call it tumoural calcinosis when it affects the periarticular area and calcinosis circumscripta when it affects the subcutaneous tissue⁽²¹⁾. It mainly affects large breed animals: German Shepherds, Rottweilers and Labrador Retrievers, and generally young animals (less than 4 years old). Lesions were solitary in 82% of affected dogs, and occurred most commonly on the hind feet (50%) and tongue (23%)⁽²²⁾. With multiple lesions there was no apparent body symmetry. Microscopically, most lesions were well-defined single or multiple variably sized aggregates of amorphous to granular, lightly to darkly basophilic material with or without peripheral granulomatous reaction and surrounded by varying amounts of fibrous connective tissue⁽²²⁾. The source can be iatrogenic (Figure 13), dystrophic or idiopathic (Figure 14). Clinical signs depend on the anatomical location of the calcinosis and its relationship to vital structures⁽²³⁾. The biopsy provides the definitive diagnosis and excision is usually curative but relapses can occur⁽²²⁾. There is one reported case of calcinosis circumscripta in combination with multiple cartilaginous exostoses⁽²³⁾.



FIG 13. Ventrodorsal radiograph in a 1-year-old Beagle shows a calcinosis circumscripta due to iatrogenic aetiology.



FIG 14. Radiographic (14A) and CT (14B) images corresponding to ectopic mineralization in a tarsal area in a 3-year-old Rottweiler. The masses were covered with skin and were firm. The histopathological diagnosis was calcinosis circumscripta.

Low-grade osteosarcomas/pathological fractures

Osteosarcoma is the most common primary bone tumour of the appendicular skeleton in dogs, occurring most commonly in the metaphyseal region of long bones of large or giant breed dogs^(24, 25) (Figures 15-16). Biopsy results are inconclusive in up to 20% of cases⁽²⁶⁾. Pathologic fracture is relatively rare in human appendicular osteosarcoma. The equivalent incidence

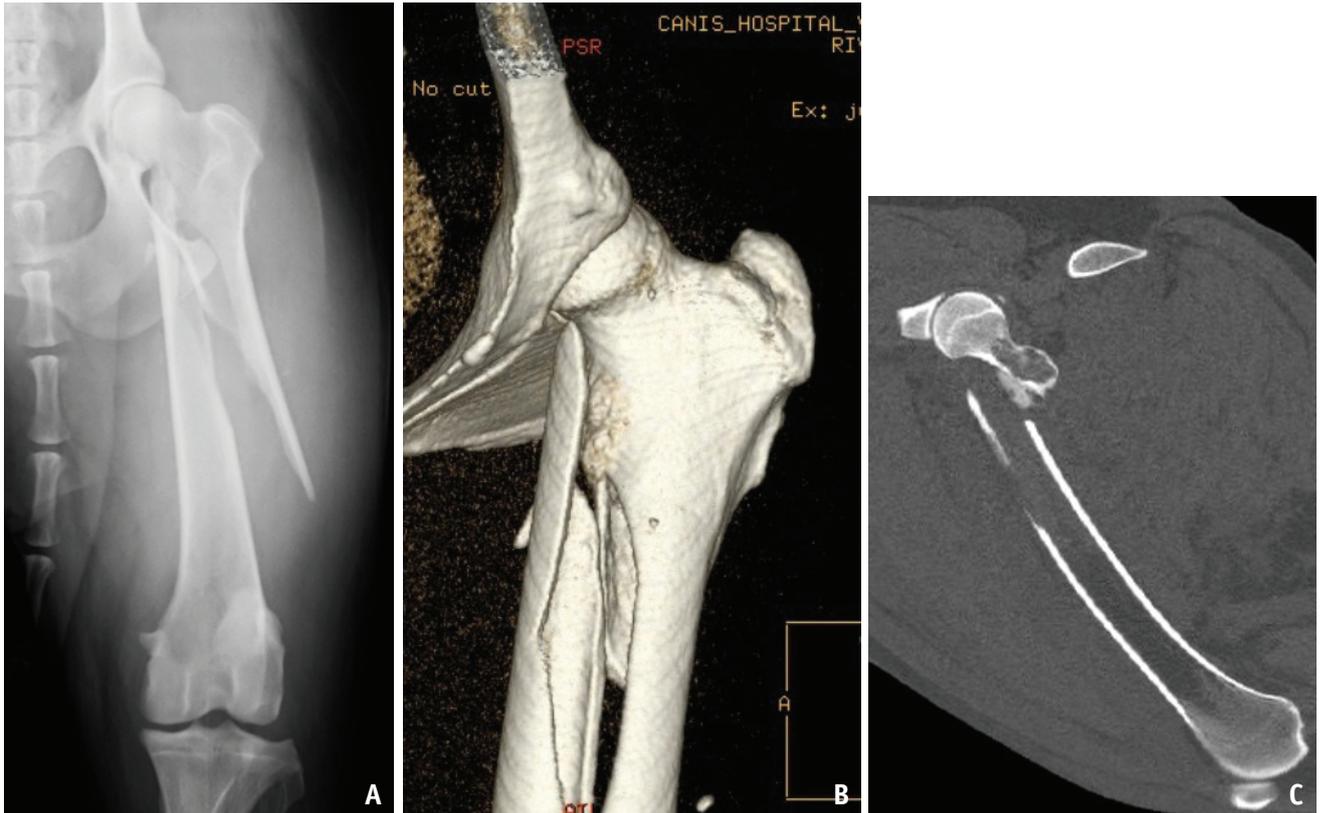


FIG 15. Ventrodorsal radiography (15A) with a spontaneous oblique fracture of the femur in a 10-year-old English Setter. In the CT (15B) and (15C) there is bone growth at the proximal focus of the fracture which was histologically compatible with an intermediate grade osteosarcoma.



FIG 16. Anteroposterior radiograph (16A) showing a distal radioulnar fracture in a 5-year-old Greyhound. A spontaneous fracture was suspected, so a biopsy was carried out that was inconclusive. Three months later, a second biopsy was performed, giving the result of an intermediate grade osteoblastic osteosarcoma. Anteroposterior radiograph (16B) and CT (16C) of the radioulnar area on the day of the second biopsy showed evidence of bone destruction.

in dogs is unknown but is suspected to be low⁽²⁵⁾. Some studies have been reported in < 3% of all long bone fractures observed in dogs. Pathological fractures are a challenge for clinicians because they are sometimes discovered intraoperatively. The medical history and case history are very important: whether there has been trauma, if the dog has limped before, etc. Deciding whether or not to repair a pathologic fracture is challenging and will depend on many factors: the owners, fracture configuration, and others. CT could be recommended before considering pathologic fracture repair^(24, 25).

Bone lesions caused by leishmaniasis

Canine leishmaniasis in the Mediterranean area is caused by the protozoan parasite *Leishmania infantum*, which is transmitted by sand-flies of the genus *Phlebotomus* or its New World synonym *Leishmania chagasi*, and is considered to be an endemic disease in more than 70 countries all over the world⁽²⁷⁾. In endemic regions the prevalence of infection is about 60% but only 10-30% of dogs become symptomatic. There is a wide variety of clinical manifestations due to different types of immune response⁽²⁷⁾. The classical signs of the disease are chronic wasting, anaemia, pyrexia cutaneous lesions, ocular lesions, renal failure and more rarely orthopaedic problems⁽²⁸⁾. Leishmaniasis should always be included as a differential diagnosis in cases of synovitis, arthritis and bone lesions in endemic areas⁽²⁸⁾. The disease is currently spreading north into the foothills of the Alps and the Pyrenees and numerous cases have been reported in northern countries of Europe. In our area of work, the seroprevalence of leishmaniasis is 13-15% and bone manifestations associated with the disease are approximately 5%. In Agut's work it was 44% and 4% in that of Sbrana^(28, 29). These differences suggested a role for genetics in the resistance to developing the disease and the immune response of dogs^(28, 30). There are different patterns of bone lesions related to leishmaniasis:

a. In the joints the radiographic pattern is osteolysis associated with soft tissue swelling caused by a synovial infection with extension into the epiphyseal spongiosa⁽³¹⁾. Arthritis is the most common bone lesion in dogs with leishmaniasis. *Leishmania* can cause arthritis by two mechanisms, one being the direct presence of the parasite within the joint, which can cause a granulomatous inflammatory reaction, and the second being a type III hypersensitivity reaction with the deposition of immune complexes

within the joint⁽²⁸⁾. Arthritis can be erosive (Figure 17) and non-erosive and at the level of the joint fluid, amastigotes of *Leishmania* spp or changes associated with osteolysis phenomena can be detected⁽²⁸⁾.

- b. In the long bones there was periosteal and intramedullary proliferation, involving the diaphysis in the area of the nutrient foramen⁽³¹⁾ (Figure 18). The periosteal proliferation may be induced by an initial infection of the marrow cavity through the nutrient canal of the bone. In more severe cases, granulomatous osteomyelitis is presented⁽³¹⁾.
- c. In the metadiaphysis in the long bones there is periosteal proliferation and an increase in endomedullary opacity and lysis (Figure 19). This pattern is uncommon and could be interpreted as the result of a chronic infection⁽³¹⁾.
- d. In anatomical sites of the insertion of ligaments and tendons we have observed cases of bone lysis and bone destruction (Figure 20).

Nutritional bone diseases

Current dietary changes for pets (substituting homemade diets for commercial diets) means that bone pathologies that were relatively frequent a few years ago are rarely seen today. They include rickets, osteoporosis and hypervitaminosis A.

Rickets

Rickets is characterised by a lack of calcification of the matrix of physal cartilage with thickening of the physis as the non-degenerated cartilage cells have not been removed. It is rare and its aetiology is thought to be due to an inadequate supply of vitamin D, insufficient endogenous production or decreased absorption related to enteric pathologies, high protein diets, or calcium/phosphorus imbalances (especially excess calcium in the diet and lack of phosphorus)⁽³²⁾. Clinically, animals manifest apathy, weakness, carpal and tarsal hyperextension and bone pain with thickening of carpal or tarsal regions or costochondral junctions. The diagnosis can be confirmed by demonstrating subnormal concentrations of 25-hydroxycholecalciferol in serum⁽³³⁾. Radiologically, osteopenia, widening and irregularity of the physis is observed due to the presence of cartilaginous islands and cup-shaped widening of the metaphyses – cupping – which is more evident in the distal physis of the ulna due to the greater ability of growth of this physis⁽³²⁾ (Figure 21).



FIG 17. Latero-lateral radiograph of a 3-year-old Labrador displaying erosive arthritis at tarsal level caused by leishmaniasis.



FIG 18. Latero-lateral radiograph corresponding to an 8-month-old Belgian Shepherd with periosteal and intramedullary proliferation at the level of the ulnar shaft due to leishmaniasis.



FIG 19. Latero-lateral radiograph of the distal part of the radius-ulna, corresponding to a Bulldog of 2 years of age diagnosed with leishmaniasis. Note an increase in endomedullary opacity and lysis at level of the metadiaphyseal area.



FIG 20. Left (20A) and right (20B) latero-lateral radiograph of a West Highland terrier of 1 year of age presented due to bilateral plantigradism. There is osteolysis and destruction of the calcaneus with common calcaneal tendon failure. The biopsy confirmed the presence of leishmaniasis by immunohistochemical techniques.



FIG 21 Anteroposterior radioulnar radiograph corresponding to a German Shepherd of 3 months of age with radiological signs compatible with rickets: enlargement and irregularity of the physis and cup-shaped widening of the metaphysis.

Osteoporosis

Osteoporosis as a consequence of secondary nutritional hyperparathyroidism occurs when there is an imbalance of calcium/phosphorus in the diet (for example, animals with an excessive intake of meat, rich in phosphorus and low in calcium). In a balanced diet, the Ca/P ratio is 1.3/1 and an exclusive intake of meat can be 1/20. The resulting hypocalcaemia produces parathyroid stimulation in an attempt to compensate calcaemia levels, and excessive resorption of calcium from bone occurs, resulting in osteopenia. Radiographic signs of osteopenia include: decreased opacity of bones, thin cortices, coarse trabeculation and, sometimes, folding fractures (Figure 22). Loss of 30 to 50 per cent of bone mass must occur before osteopenia is apparent on survey radiographs as decreased bone opacity. As osteopenia develops there is a resorption of the transverse and longitudinal trabeculae resulting in division of the cortex in two components (double line) ⁽³⁴⁾.



FIG 22. Latero-lateral radiograph of the knee of a 4-month-old Bernese Mountain Dog with osteoporosis: decreased opacity of bones, thin cortices, coarse trabeculation and folding fracture.

Hypervitaminosis A

This disorder occurs in cats that have been fed excessive amounts of products high in vitamin A (spleen, liver...) that produce a saturation of the vitamin in the body fat, and secondary hyperplasia of the sub-periosteal tissue that invades the vertebrae, elbow and knees, producing a bony ankylosis (Figures 23-24). The aetiology of this disease is not well known but it is believed to be an alteration in the synthesis of collagen ⁽³⁵⁾. Hypervitaminosis A must be differentiated from mucopolysaccharidosis, which is due to an abnormal accumulation of glycosaminoglycans, producing a chronic multisystem disease. In hypervitaminosis A, the level of serum retinol is increased. In severe cases the prognosis is reserved and lesions are irreversible.

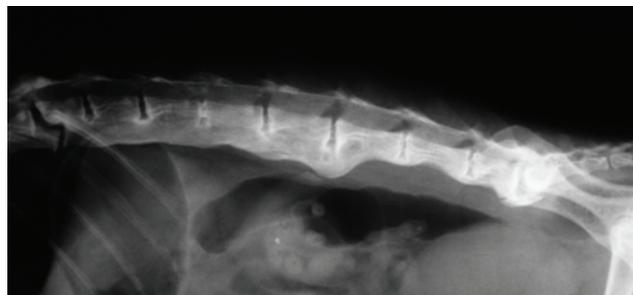


Fig 23. Lateral radiograph of the lumbar area of a 5-year-old Siamese cat which had been fed exclusively with spleen. Vertebral ankylosis compatible with hypervitaminosis A.

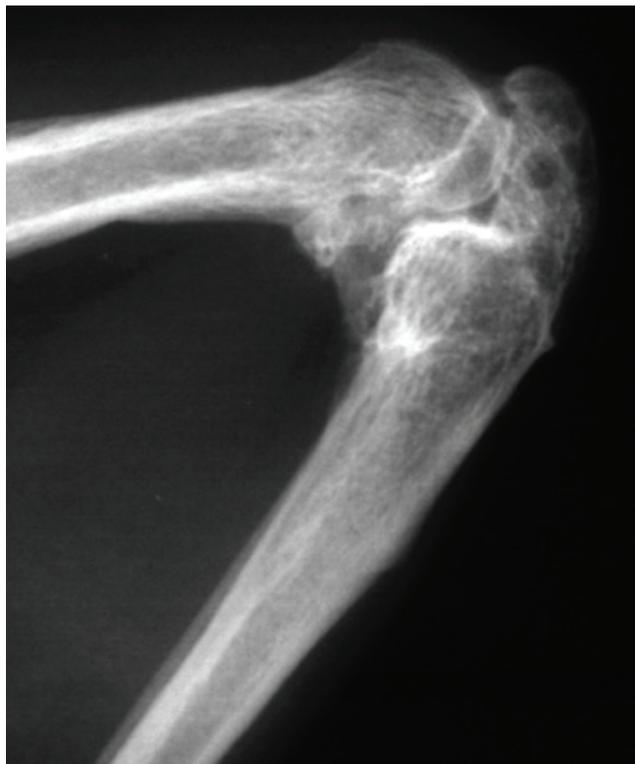


FIG 24 . Ankylosis of the stifle and postmortem appearance of the same case of a Domestic European cat of 10 years of age with a diet exclusively of spleen and liver, suggestive of hypervitaminosis A .

References

1. Franch J, Font J, Ramis A, Lafuente P, Fontecha P, Cairo J. Multiple cartilaginous exostosis in a Golden Retriever cross bred puppy. *Vet Comp Orthop Traumatol.* 2005; 3,189-193.
2. Gambardella PC, Osborne CA, Stevens JB. Multiple Cartilaginous Exostoses in the Dog. *J Am Vet Med Assoc.* 1975; 166: 761-768.
3. Quirini G, Meyer J, Herman M et al. Osteochondroma of the thoracic spine: an unusual cause of spinal cord compression. *Am J Neuroradiol.* 1996; 17: 961-964.
4. Green EM, Admas WM, Steinberg H. Malignant transformation of solitary spinal osteochondroma in two mature dogs. *Vet Radiol Ultrasound.* 1999;40:634-637.
5. Jacobson, LS Kirberger RM Canine Multiple Cartilaginous Exostoses: Unusual manifestations and a review of the literature. *J Am Anim Hosp Assoc.* 1996; 32, 45-50.
6. Salter RB, Harris WR. Injuries involving the epiphyseal plate. *J. Bone Joint Surg.* 1963; 45: 587-622.
7. Lee R. Proximal femoral epiphyseal separation in the dog. *J Small Anim Pract.* 1976; 11: 669-679.
8. Dupuis J, Breton L, Drolet R. Bilateral epiphysiolysis of the femoral heads in two dogs. *J Am Vet Med Assoc.* 1997; 210:1162-1165.
9. Van Ee RT, Walker TL, Patton CS. Bilateral calcaneal epiphysiolysis in a dog. *Vet Surg.* 1986; 15: 60-64.
10. Font J, Pelach M, Font C, Cairó J. Bilateral calcaneal epiphysiolysis in a dog. *Vet Comp Orthop Traumatol.* 2013; 26: 238-241.
11. Moores AP, Owen MR, Fews D, Coe RJ, Brown PJ, Butterworth SJ. Slipped capital femoral epiphysis in dogs. *J Small Anim Pract.* 2004; 45:602-608.
12. Dupuis J, Breton I, Drolet R. Bilateral epiphysiolysis of the femoral heads in two dogs. *J. Am Vet Med Assoc.* 1997;210: 1162-1165.
13. Gemmil TJ, Pink J, Clarke SP, McKnee WN. Total hip replacement for the treatment of atraumatic slipped capital femoral epiphysis in dogs. *J Small Anim Pract.* 2012; 53: 453-458.
14. von Pfeil DJF, DeCamp CE, Diegel KL, Gholve PA, Probst CW, Dejardin LM. Does Osgood Schlatter Disease exist in the dog?. *Vet Comp Orthop Traumatol.* 2009; 22: 257-263.
15. Nehrbass D, Arens D, Zeiter S. Spontaneous bilateral avulsion fracture of the tuberositas tibiae in a New Zealand white rabbit a counterpart to Osgood Schlatter disease in humans?. *Exp Toxicol Pathol.* 2015; 67:223-227.
16. Gower JA, Bound NJ, Moores AP. Tibial tuberosity avulsion fracture in dogs: a review of 59 dogs. *J Small Anim Pract.* 2008; 49: 340-343.
17. Ehrenborg G, Olsson S. Avulsion of the tibial tuberosity in the dog. Study with special reference to the Osgood Schlatter lesion in man. *Acta Chir Scand.* 1962; 123: 28-37.
18. Flo GL, Stickle RL, Dunstan RW. Synovial chondrometaplasia in five dogs. *J Am Vet Med Assoc.* 1987; 191:1417-1422.
19. Smith TJ, Baltzer WI, Lör C, Stieger SM. Primary synovial osteochondromatosis of the stifle in an English Mastiff. *Vet Comp Orthop Traumatol.* 2012; 2:160-166.

20. Diaz Bertrana C, Durall I, Rial JM, Franch J, Fontecha P, Ramis A. Extra and intraarticular synovial chondromatosis and malignant transformation to chondrosarcoma. *Vet Comp Orthop Traumatol.* 2010; 4: 277 283.
21. Voll, R.; Gaiga, L. H.; Voll, J.; Oliveira, R. T. de; Nóbrega, F. S.; Araújo, A. C. P. de; Ferreira, M. P.; Martins, J. M.; Cervical tumoral calcinosis in dog. *Acta Scientiae Veterinariae.* 2009; 37:53 57.
22. Tafti, A. K.; Hanna, P.; Bourque, A. C.; Calcinosis circumscripta in the dog: a retrospective pathological study. *Journal of Veterinary Medicine.* 2005;52:13 17.
23. Engel, S.; Randall, E. K.; Cuddon, P. A.; Webb, B. T.; Aboellail, T. A.; Imaging diagnosis: multiple cartilaginous exostoses and calcinosis circumscripta occurring simultaneously in the cervical spine of a dog. *Veterinary Radiology & Ultrasound.* 2014; 55:305 309.
24. Bhandal J, Boston S. Pathologic fracture in dog with suspected or confirmed osteosarcoma. *Veterinary Surgery.* 2011; 40: 423 430.
25. Boston S, Bacon N, Culp W, Bhandal J, Bruce C. Outcome after repair of a sarcoma related Pathologic fracture in dogs: A Veterinary Society of Surgical Oncology Retrospective Study *Veterinary Surgery.* 2011; 40:431 437.
26. Liptak JM. Appendicular osteosarcoma. Current treatment options. 2012; ECVS Proceedings. 163 171.
27. Kaszak I, Planellas M, Dworecka Kaszak B, Canine leishmaniasis . An emerging disease. *Annals of Parasitology.* 2015; 61: 69 76.
28. Agut A, Corzo N, Murciano J, Laredo FG, Soler M. Clinical and radiographic study of bone and joint lesions in 26 dogs with leishmaniasis. *Vet Rec.* 2003;22: 648 652.
29. Sbrana S, Marchetti V, Manciatì E, Guidi G, Bennet D. Retrospective study of 14 cases of canine arthritis secondary to Leishmania infection. *J Small Anim Pract.* 2014; 55: 309 313.
30. Baneth G, Kpoutinas AF, Solano Gallego L, Bourdeau P, Ferrer L, Canine leishmaniasis. New concepts and insights on an expanding zoonosis. *Trends in parasitology.* 2008; 24: 324 330.
31. Turrel JM, Pool R. Bone lesions in four dogs with visceral leishmaniasis. *American Veterinary Radiology Journal.*1982; 23: 243 249 .
32. Morterallo, CM, Petazzoni M, Vezzoni A. Atlas de enfermedades ortópédicas y su predisposición racial. 2001; Editorial Servet. *Raquitismo:* 77 79.
33. Malik R, Laing C, Davis PE. Rickets in a litter of racing greyhounds. *J. Small Anim. Pract.* 1997; 38:109 14.
34. Lamb CR. The double cortical line: A sign of osteopenia. *J. Small Anim. Pract.* 1990; 31:189 192.
35. Polizopolop Z, Kazacos G, Patsikas M, Roubies M. Hypervitaminosis A in the cat: A case report and review of the literatura. *J. Feline Med Surg.* 2005;7:363 367.



Reprint paper*

Murmur intensity in small-breed dogs with myxomatous mitral valve disease reflects disease severity

Ingrid B. Ljungvall¹, Mark Rishniw, Francesco Porciello, Luca Ferasin and Dan G. Ohad

SUMMARY

Objectives: To determine whether murmur intensity in small-breed dogs with myxomatous mitral valve disease reflects clinical and echocardiographic disease severity.

Methods: Retrospective multi-investigator study. Records of adult dogs ≤ 20 kg with myxomatous mitral valve disease were examined. Murmur intensity and location were recorded and compared with echocardiographic variables and functional disease status. Murmur intensities in consecutive categories were compared for prevalences of congestive heart failure, pulmonary hypertension and cardiac remodelling.

Results: 578 dogs [107 with “soft” (30 Grade I/VI and 77 II/VI), 161 with “moderate” (Grade III/VI), 160 with “loud” (Grade IV/VI) and 150 with “thrilling” (Grade V/VI or VI/VI) murmurs] were studied. No dogs with soft murmurs had congestive heart failure, and 90% had no remodelling. However, 56% of dogs with “moderate”, 29% of dogs with “loud” and 8% of dogs with “thrilling” murmurs and subclinical myxomatous mitral valve disease also had no remodelling. Probability of a dog having congestive heart failure or pulmonary hypertension increased with increasing murmur intensity.

Clinical Significance: A 4-level murmur grading scheme separated clinically meaningful outcomes in small-breed dogs with myxomatous mitral valve disease. Soft murmurs in small-breed dogs are strongly indicative of subclinical heart disease. Thrilling murmurs are associated with more severe disease. Other murmurs are less informative on an individual basis.

* This paper first appeared in the *Journal of Small Animal Practice* (2014) 55, 545–550
DOI: 10.1111/jsap.12265 *Eur J Comp An Pract*, Spring 2016; (26)1: p33-41
Go to <http://www.ejcap.org> for the interactive online presentation of this paper

INTRODUCTION

Myxomatous mitral valve disease (MMVD) is the most common acquired cardiac disease of small-breed dogs. The

disease has a long subclinical course which progresses variably to clinical disease [exercise intolerance, congestive heart failure (CHF) or syncope] (Kittleson 2010, Borgarelli et al. 2012). Disease severity, and therefore likelihood of developing CHF, is related to valvular regurgitant volume, which is reflected by the size of the left atrium (LA) and left ventricular (LV) diastolic dimension. Furthermore, LA size has been associated with clinical outcome in dogs with MMVD (Borgarelli et al. 2008, Moonarmart et al. 2010, Lord et al. 2011, Borgarelli et al. 2012).

¹ Department of Clinical Sciences, University Animal Hospital, Swedish University of Agricultural Science, SE-750 07 Uppsala, Sweden.
E-mail: ingrid.ljungvall@slu.se

A key component of the cardiovascular examination in dogs with MMVD is cardiac auscultation and classification of the murmur intensity. Murmur intensity has been classically characterised according to a 6-level scheme proposed by Levine (1933) and subsequently modified by various investigators (Haggstrom et al. 1995, Borgarelli et al. 2008, Ljungvall et al. 2009). A study in Cavalier King Charles spaniel (CKCS) dogs demonstrated an association between murmur intensity and functional class of heart disease, with soft murmurs being found only in dogs with mild MMVD (Haggstrom et al. 1995). A subsequent study found that increasing murmur intensity in small-breed dogs was associated with increasing severity of MMVD as assessed by the relative size of the LA (defined as the ratio of the LA to the aortic diameter – LA:Ao) (Ljungvall et al. 2009). Both of these studies examined sample populations of less than 100 dogs, the majority of them were CKCS, and were examined by one group of investigators. However, a larger study of a more heterogeneous population of dogs of various sizes and breeds with MMVD failed to identify murmur intensity as a predictor of disease status or survival (Borgarelli et al. 2008). Therefore, whether murmur intensity would help clinicians examining a small-breed dog with MMVD to determine the severity of the disease remains unclear. If the observations of the studies in small-breed dogs were replicated, clinicians would be able to use murmur intensity to help stratify small-breed dogs with MMVD into varying categories of severity, and, consequently, risk of developing CHF in the near future or having CHF at presentation for evaluation of clinical signs.

Additionally, no studies have examined the validity of the 6-level murmur intensity classification scheme for dogs with MMVD. This scheme has been considered problematic for use by physicians (Keren et al. 2005) and is poorly understood by many first opinion veterinary practitioners and veterinary students (personal observations). It would seem logical that the least complex scheme that stratifies murmurs while preserving clinically useful information is desirable.

Therefore, it was hypothesised that murmur intensities could be stratified into a scheme that was less complex than the currently used canonical 6-level scheme without loss of clinically useful information. Further, it was hypothesised that small-breed dogs with MMVD and soft murmurs would have mild disease, as characterised by LA:Ao close to that of healthy dogs, and that louder

murmurs would be less informative to a clinician about disease severity in any particular patient, but might provide information about probabilities of the presence of CHF. To test this hypothesis, an estimate of cardiac size (LA:Ao) to murmur intensity in a cohort of small-breed dogs with MMVD was compared. The probability that dogs with different murmur intensities would have evidence of cardiac remodelling, pulmonary hypertension or CHF at the time of examination was further examined.

MATERIALS AND METHODS

Dog selection

Dogs ≤ 20 kg that had a primary echocardiographic diagnosis of MMVD, made by a cardiologist or clinician with a focus in cardiology, were included. Dogs both with and without CHF were included, and dogs receiving treatment for heart disease were not excluded. The clinician's diagnosis of CHF as being correct was accepted, and how the diagnosis was obtained was not examined. Dogs less than five years of age were excluded as were dogs with other cardiac disease, except for concurrent, mild myxomatous tricuspid valve degeneration and those with acute mitral valve chordal rupture. Dogs with major non-cardiac co-morbidities (e.g. chronic kidney disease, severe respiratory disease of non-cardiogenic origin) were also excluded. Patient details for all dogs (e.g. age, weight, breed, sex) were recorded. Cases were selected by review of ancillary data from another study (Ohad et al. 2013a) or by sequential evaluation of case records for 2009 to 2012 by the authors. This study was not submitted for formal ethical review although advice was sought to determine if formal review was required.

Murmur classification

In all dogs, the intensity and location of the murmur were recorded, using the Levine 6-level classification scheme. Locations were described as left apical, left basilar, right-sided and other.

Echocardiographic classification

Standard echocardiographic variables were recorded, including LA, Ao and LV dimensions obtained in the right-parasternal short-axis view as previously reported (Thomas & Gaber 1993, Rishniw & Erb 2000,

Hansson et al. 2002). The LA:Ao, normalised LA (wLA) and normalised LVID (wLVIDd) (using weight-based echocardiographic ratio indices) (Brown et al. 2003) were calculated for each dog.

Cardiac remodelling was defined as an increase in at least two of the three echocardiographic variables (LA:Ao, wLA and wLVID) above previously established reference intervals (Brown et al. 2003). Dogs were assigned into one of three categories, according to the ACVIM Consensus statement on diagnosis and treatment of MMVD: those without evidence of remodelling (i.e. less than two variables greater than reference intervals) were coded as Stage B1, those with evidence of remodelling (two or more variables greater than reference intervals) but without evidence of CHF were coded as Stage B2 and those with evidence of CHF (treated or untreated) were coded as Stage C (Atkins et al. 2009).

Presence or absence of pulmonary hypertension (defined as a tricuspid regurgitation velocity >3.2 m/s) (Kittleson 2010) was recorded where possible.

Statistical analyses

Shapiro–Wilk test of the LA:Ao for each murmur category confirmed a Gaussian distribution. Levene's test for homogeneity of variance showed differences in the variances so the Welsh F-test was used to adjust the results of a one-way ANOVA, followed by the Games-Howell Test for pairwise comparisons, with the comparison-wise P-value set at 0.05. The probability of having CHF, pulmonary hypertension or subclinical cardiac remodelling in each consecutive pair of murmur categories (i.e. Grade I/VI versus Grade II/VI, Grade II/VI versus Grade III/VI, Grade III/VI, versus Grade IV/VI etc.) were compared using pairwise Chi-square tests, with Bonferroni-adjusted P-values to account for multiple comparisons and preserve the comparison-wise error rate. Statistical analyses were performed using Medcalc 12 (Medcalc Software bvba Ostend, Belgium, 2013) and SPSS Statistics (IBM 2013).

RESULTS

Five hundred and seventy-eight small-breed dogs met the inclusion and exclusion criteria. The dogs were a median of 11 years of age (range 5 to 19 years), with a median weight of 8.3 kg (range 1.5 to 20 kg); 325 were

male and 253 were female. Data were contributed by 22 clinicians, however 75% of the data were contributed by the authors.

Of the 578 dogs, 30 had Grade I/VI murmurs, 77 had Grade II/VI murmurs, 161 had Grade III/VI murmurs, 160 had Grade IV/VI murmurs, 119 had Grade V/VI murmurs and 31 had Grade VI/VI murmurs. In almost all dogs (96%), the point of maximal murmur intensity was described as left apical. In 22 dogs (all with moderate murmurs or louder), the murmur was as loud or louder on the right side as on the left apex. In six dogs, the right-sided murmur was softer than the left apical murmur, but was still reported as IV/VI. Thus, 28 dogs had at least IV/VI right-sided murmurs along with left apical murmurs.

Pairwise comparisons of consecutive pairs of murmur intensities demonstrated no differences in the proportions of dogs with CHF, pulmonary hypertension or cardiac remodelling and either Grade I/VI or Grade II/VI murmurs ($P=1.0$, 1.0 and 0.17 , respectively). Therefore, these two groups were consolidated into a single category, labelled "soft" murmurs. Differences between the proportions of dogs with Grade II/VI and Grade III/VI murmurs for two of the clinical variables ($P=0.01$ for CHF, $P<0.001$ for remodelling) and the proportions of dogs with Grade III/VI and Grade IV/VI murmurs for two of the clinical variables ($P=0.001$ for CHF and $P=0.0003$ for remodelling) were observed. Therefore, Grade III/VI murmurs were relabelled as "moderate." Differences between the proportions of dogs with Grade IV/VI and Grade V/VI murmurs for two of the clinical variables ($P=0.00001$ for CHF and $P<0.001$ for remodelling) were observed. Therefore, IV/VI murmurs were relabelled as "loud." Differences in the proportions of dogs with CHF, pulmonary hypertension or cardiac remodelling and either Grade V/VI or Grade VI/VI murmurs ($P=0.45$ for CHF, $P=0.8$ for pulmonary hypertension and $P=1.0$ for remodelling) were not observed. Therefore, these two groups were consolidated into a single category, labelled "thrilling" murmurs.

Disease severity (as determined by LA:Ao) differed between all four groups of murmur intensity ($P<0.00001$) (Fig 1).

All dogs with a soft murmur had an $LA:Ao \leq 2.0$, and 90% of dogs had an $LA:Ao \leq 1.5$ (only 8/107 dogs had $LA:Ao > 1.6$). However, 53% of dogs with moderate murmurs, 29% of those with loud murmurs and 10% of those with thrilling murmurs also had $LA:Ao < 1.6$.

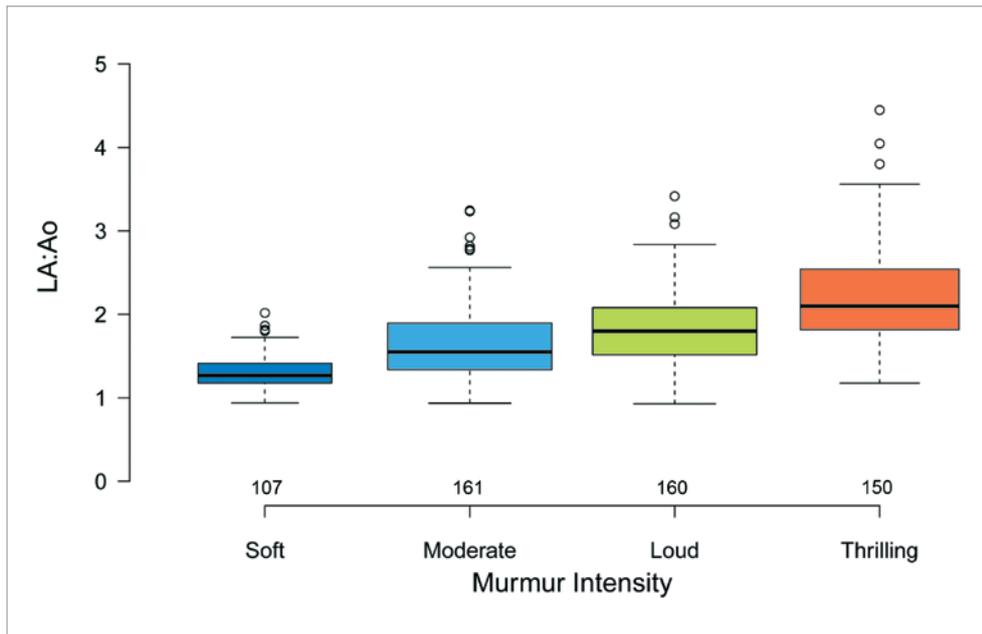


FIG 1. Increasing murmur intensity is associated with increasing left atrial size, as determined by the left-atrial-to-aortic ratio (LA:Ao). The black line denotes the median value, the box denotes the interquartile range [IQR (25th to 75th percentile)], the circles denote values greater than 1.5 times the IQR greater than the 75th percentile. Numbers above the category labels denote the sample sizes in each category. All groups were different from each other ($P<0.05$)

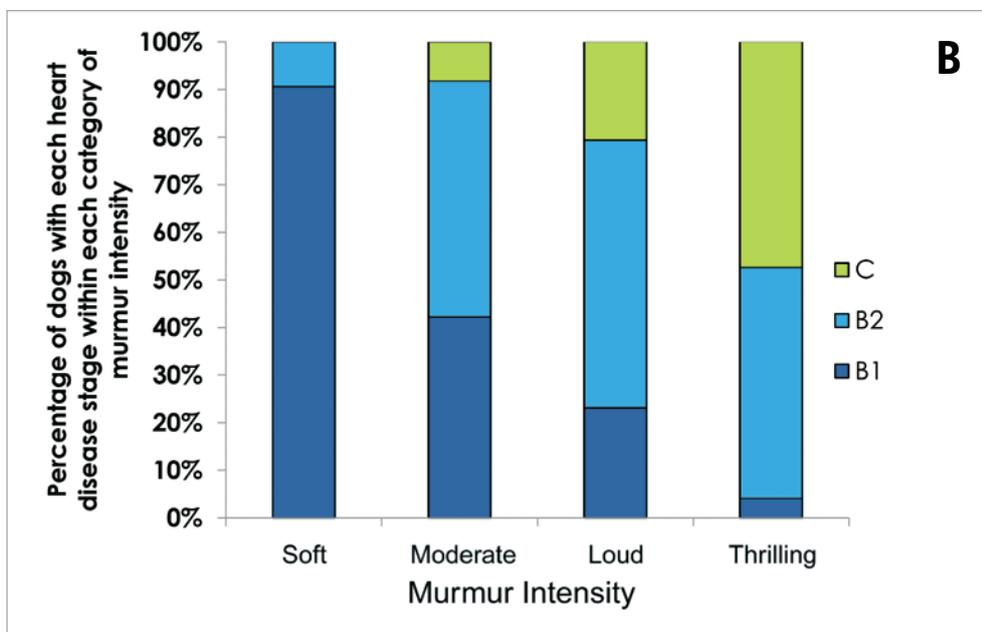
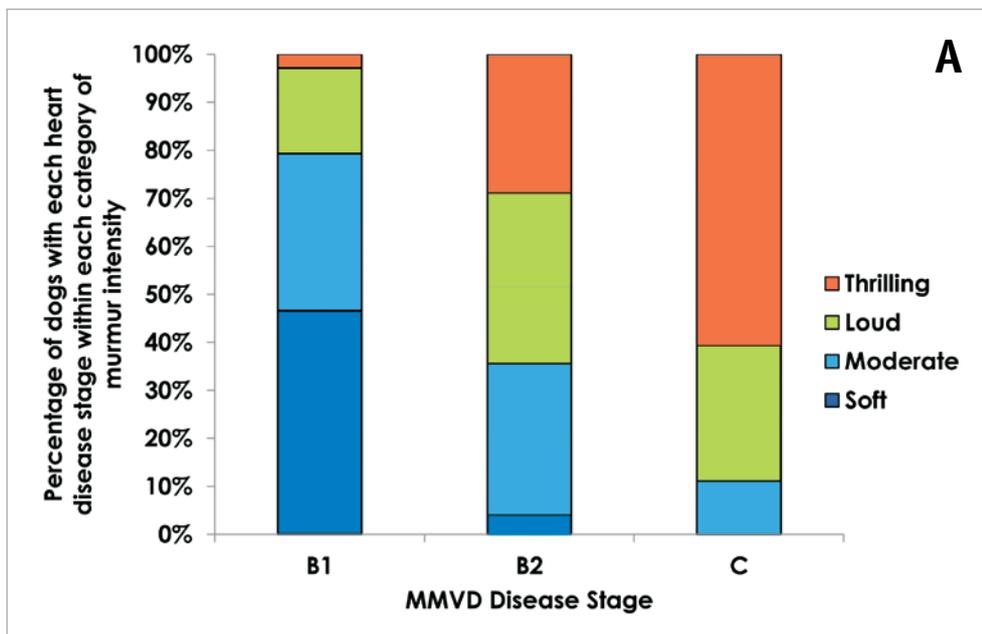


FIG 2. Increasing murmur intensity is associated with increasing probability of CHF. (A) Distribution of murmur intensity by categories of disease severity. (B) Distribution of disease severity among categories of murmur intensity. All groups were different from each other ($P<0.0001$) for the probability of having CHF. B1 – no evidence of cardiac remodelling, B2 – subclinical disease with evidence of cardiac remodelling, C – congestive heart failure

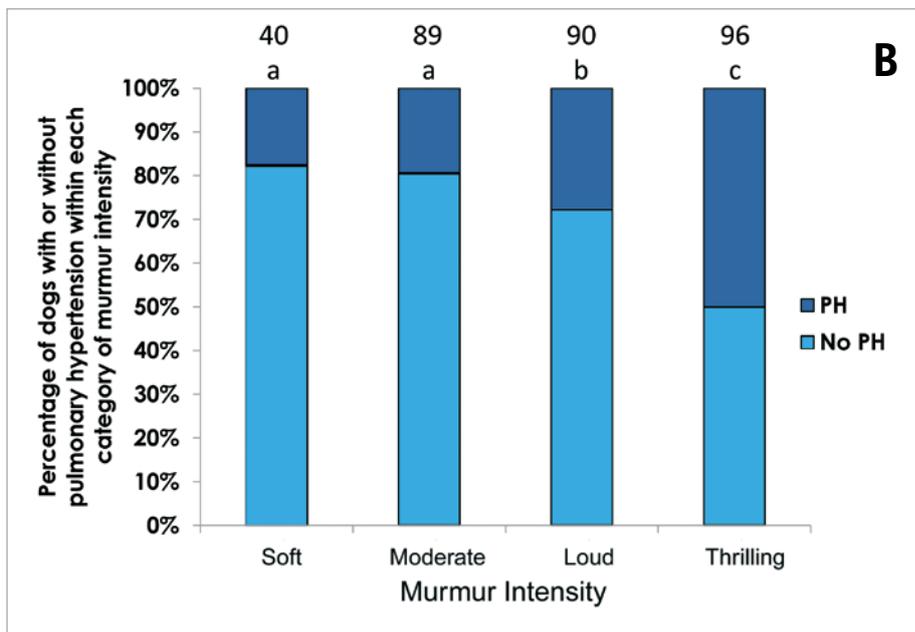
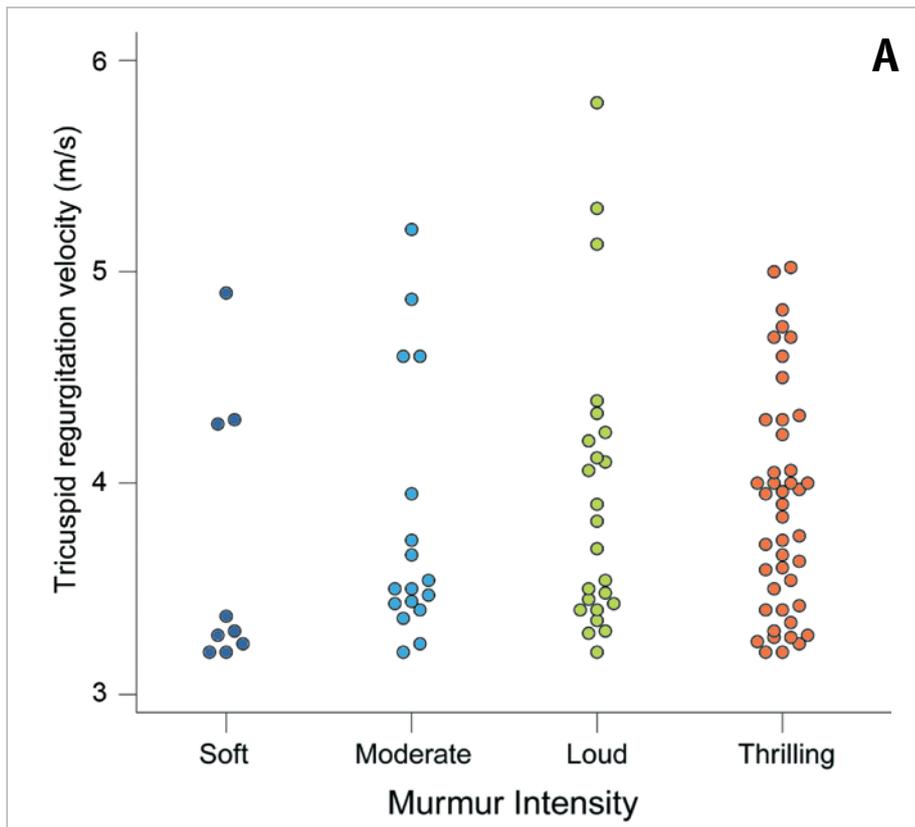


FIG 3. Loud murmurs are associated with increased probability of pulmonary hypertension. (A) Dot plot showing the number and range of tricuspid regurgitation velocities in dogs with pulmonary hypertension (velocity greater than 3.2 m/s) with different murmur intensities. (B) Proportion of dogs with each murmur intensity identified with pulmonary hypertension. Numbers above the category labels denote the sample sizes in each category. Columns with different superscripts have different proportions of pulmonary hypertension ($P < 0.05$). PH – pulmonary hypertension

Figure 2 shows the association of murmur intensity and class of mitral valve disease. Dogs with loud or thrilling murmurs were more likely to be diagnosed with CHF than those with mild or moderate murmurs, accounting for 90% of dogs with CHF (Fig 2A). Specifically, none of the 107 dogs with soft murmurs were diagnosed with CHF; 13/161 dogs with moderate intensity murmurs, 33/160 dogs with loud murmurs and 71/150 dogs with thrilling murmurs were diagnosed with CHF at the time of auscultation ($P < 0.0001$) (Fig 2B). Most of the dogs with soft murmurs had no evidence of remodelling (Fig 2B). However, soft

murmurs were present in less than 50% of dogs without evidence of remodelling (B1) (Fig 2A), with more than 50% of B1 dogs having moderate or loud murmurs. On the other hand, 90% of dogs with thrilling murmurs had evidence of either remodelling or CHF. Very few dogs with evidence of remodelling (B2) had soft murmurs, and no dogs with CHF had soft murmurs (Fig 2A).

Figure 3 shows the association of pulmonary hypertension and murmur intensity. Pulmonary artery pressure was estimated in 40/107 dogs with soft murmurs, 88/161

Table 1. Probabilities of dogs with murmurs of different intensities having congestive heart failure, pulmonary hypertension or cardiac remodelling (CHF: congestive heart failure; PH: pulmonary hypertension; B1: subclinical disease, no evidence of remodelling; CI: confidence interval)

Probability of:	Murmur Intensity			
	Soft	Moderate	Loud	Thrilling
CHF %(95%CI)	0 (0-4)	8 (5-14)	20 (15-28)	47 (40-55)
PH %(95%CI)	18 (8-33)	19 (12-29)	28 (19-38)	50 (40-60)
B1 status %(95%CI)	90 (83-95)	42 (35-50)	29 (22-38)	8 (4-16)

dogs with moderate murmurs, 90/160 dogs with loud murmurs and 96/150 dogs with thrilling murmurs. Pulmonary hypertension was identified in 7/40 (18%) dogs with soft murmurs, 17/88 (19%) dogs with moderate murmurs, 25/90 (28%) dogs with loud murmurs, and 48/96 (50%) dogs with thrilling murmurs ($P < 0.0001$). Of the 73 dogs with loud or thrilling murmurs and pulmonary hypertension, 21 had loud or thrilling right-sided murmurs (with loud or thrilling left-sided murmurs), while 52 had predominantly left-sided murmurs.

Table 1 shows the probabilities of dogs with each murmur intensity having CHF, pulmonary hypertension or, if subclinical, absence of cardiac remodelling (B1 status).

DISCUSSION

This study shows that the canonical murmur intensity scale, first proposed by Levine in 1933, has redundant murmur categories that can be compressed into a simpler, descriptive scheme without losing clinically important information about the probability of CHF, pulmonary hypertension or cardiac remodelling in dogs with MMVD. It is proposed that in small-breed dogs with MMVD, murmurs can be classified as “soft”, “moderate”, “loud” and “thrilling”. Using this classification system, the present study shows that in small-breed dogs with MMVD, murmur intensity helps predict disease status. Specifically, no dogs with soft murmurs had CHF, and most often had mild disease, without evidence of cardiac remodelling. Thus, a clinician auscultating a soft mitral murmur in a small-breed dog with suspected MMVD can reasonably conclude that the disease severity is mild and that CHF is extremely unlikely in that case, even if the dog is showing clinical signs such as tachypnoea or dyspnoea. Conversely, dogs with thrilling murmurs rarely had mild subclinical disease (without evidence of remodelling), and these dogs had the highest probability of having CHF and pulmonary hypertension. However, MMVD severity cannot be accurately determined by auscultation in dogs with moderate or loud murmurs. The results

support the hypothesis that loud murmurs in small-breed dogs with MMVD are not necessarily the proof of severe disease, but soft murmurs are strongly suggestive of mild disease. Nevertheless, the probability of having evidence of remodelling in dogs with subclinical disease or of having CHF (clinical disease) increases with increasing murmur intensity. This probability is very high, but not 100%, in dogs with thrilling murmurs. Finally, the first objective system for identifying “cardiac remodelling” in small-breed dogs with MMVD, using a combination of echocardiographic ratio indices and LA:Ao is proposed.

The present findings are similar to those in a smaller cohort of CKCS dogs ($n=59$), which had a similar association between murmur intensity and functional cardiac class (Haggstrom et al. 1995) as well as findings in a more mixed population of small-breed dogs ($n=77$) that demonstrated an association between murmur intensity and LA:Ao (Ljungvall et al. 2009). In the earlier study, all dogs with soft murmurs were classified with mild functional disease. However, as in this study, several dogs in that study with loud murmurs were also classified with mild functional disease. In the latter study, where a combination of LA:Ao and regurgitant jet area was used to determine disease severity, only four dogs with soft murmurs had evidence of more than mild MMVD, and none of these dogs had severe MMVD. This slight difference could be explained by different methods of stratifying severity – regurgitant jet area when classifying severity of disease was not examined, but echocardiographic ratio indices to define “remodelling” (B2) were used. Differentiation between “moderate” and “severe” disease was not attempted, but dogs were simply classified as having no remodelling, remodelling or CHF. Despite these minor differences in methodology, the present findings mirror those of the previous studies, but extend them to a more diverse and a larger cohort of small-breed dogs (not breed-specific), and to a larger cohort of examining clinicians. Thus, this study augments the previous studies and substantiates the critical value of auscultation in the diagnosis of MMVD.

The present findings differ from another study that found that murmur intensity was not predictive of survival or disease status (Borgarelli et al. 2008). In that study, the authors identified soft murmurs in 16% of dogs with evidence of CHF. However, these authors included large-breed dogs (up to 67 kg), where murmur intensity might not reflect disease severity (Borgarelli et al. 2004, Borgarelli & Haggstrom 2010), and dogs with atrial fibrillation, which would likely decrease murmur intensity. Additionally a different scheme for identifying CHF was used. Any or all of these factors could contribute to the differences between that study and the present findings. A simplified and descriptive 4-level murmur grading scheme, based on the original 6-level scheme proposed by Levine (1933), was developed for several reasons. First, a scheme that describes the murmur intensities clearly and intuitively is likely more easily understood and interpreted by most clinicians. Using the 6-level scheme would require the clinician to understand and remember the definitions of each level, and then interpret those into some intuitive scheme. Second, investigators have demonstrated that a simplified 3-level scheme increases accuracy of the murmur identification by attending physicians and students (Keren et al. 2005). Finally, there appear to be no studies that demonstrate a clinically meaningful utility of the original 6-level system. Levine (1933) suggested that soft murmurs (those graded I/VI or II/VI) were often clinically unimportant, but those graded III/VI or higher were more commonly associated with clinically relevant pathology. This study demonstrates the validity of these observations, as a clinically important difference between the four murmur intensities was demonstrated and, equally, no clinically meaningful information was lost by collapsing the two extreme pairs of murmur categories into two simpler categories. Thus, Ockham's approach to the classification of murmurs was adopted: "*Frustra fit per plura quod potest fieri per pauciora*" or "*It is pointless to do with more what can be done with fewer*" (Ockham circa 1323); possibly more succinctly espoused in a quote attributed to Einstein: "*Everything should be as simple as it can be, but not simpler*" (Sessions 1950).

While the ACVIM Consensus statement on MMVD classifies dogs with subclinical disease as either B1 (no remodelling) or B2 (remodelling), no criteria were provided by which such a distinction can be made (Atkins et al. 2009). Therefore, a scoring system was created that examined three variables which likely indicate remodelling: an LA:Ao

greater than 1.59, a LV internal dimension in diastole, normalised to the weight-based aortic measurement, and a left atrial dimension, normalised to the weight-based aortic measurement, as proposed by Brown et al. (2003). The present scheme could be considered conservative, requiring at least two of these measured variables to be in excess of reference intervals for a dog to be considered as having "remodelling". This scheme reduces the risk of a single measurement being erroneously large and a dog being misclassified. However, it might preclude very small changes in cardiac size from being detected. This scheme could provide collaborating researchers with a uniform method of identifying remodelling, because it relies on multiple variables to be outside the reference intervals to identify enlargement, removing some of the subjective inter-observer variability in identifying remodelling. Finally, it could provide clinicians with a reasonable and practical method of stratifying their patients and support clinical recommendations for additional diagnostic tests.

As with all studies, this study has limitations. The findings apply only to older small-breed dogs with MMVD and cannot be extrapolated to large-breed dogs with MMVD or dogs with other cardiac disorders.

The cause of pulmonary hypertension in dogs with tricuspid regurgitation velocities ≥ 3.2 m/s was not examined. In the three cases with soft murmurs where the tricuspid regurgitation velocity exceeded 4 m/s, the LA:Ao was less than 1.5, suggesting the MMVD was clinically inconsequential. Therefore, it is likely that these dogs had pulmonary hypertension unrelated to their MMVD. None of these three dogs had loud right-sided murmurs. Of the 28 dogs with loud or thrilling right-sided murmurs, 21 had evidence of pulmonary hypertension. This is consistent with recent observations that older dogs with loud right-sided murmurs often have secondary pulmonary hypertension (Ohad et al. 2013b). The drugs used in the dogs in this study were not recorded. Pimobendan could potentially increase the murmur intensity in some dogs due to its positive inotropic properties, or conversely, decrease the murmur secondary to vasodilation. Other drugs used for treating CHF might also affect murmur intensity. Therefore, it is possible that dogs with CHF, if they were being treated with pimobendan, could have had louder murmurs than they would have had otherwise.

This study demonstrates the value of a thorough physical examination in small-breed dogs with MMVD. Clinicians

can confidently exclude CHF or even severe disease (as determined by LA:Ao) in small-breed dogs with soft murmurs caused by MMVD. Therefore, if clinical signs, such as dyspnoea and/or tachypnoea, exist in these patients, clinicians should initially direct their investigations at causes other than CHF. While the probabilities of CHF and pulmonary hypertension increase with increasing murmur intensity, loud and even thrilling murmurs are not necessarily proof of severe disease. Consequently, small-breed dogs with apical murmurs caused by MMVD that are moderate or loud warrant additional investigation to better define the disease severity and clinical status.

Conflict of interest

None of the authors of this article has a financial or personal relationship with other people or organisations that could inappropriately influence or bias the content of the paper.

References

- Atkins, C., Bonagura, J., Ettinger S., et al. (2009) Guidelines for the diagnosis and treatment of canine chronic valvular heart disease. *Journal of Veterinary Internal Medicine* 23, 1142-1150
- Borgarelli, M., Crosara, S., Lamb, K., et al. (2012) Survival characteristics and prognostic variables of dogs with preclinical chronic degenerative mitral valve disease attributable to myxomatous degeneration. *Journal of Veterinary Internal Medicine* 26, 69-75
- Borgarelli, M. & Haggstrom, J. (2010) Canine degenerative myxomatous mitral valve disease: natural history, clinical presentation and therapy. *The Veterinary Clinics of North America Small Animal Practice* 40, 651-663
- Borgarelli, M., Savarino, P., Crosara, S., et al. (2008) Survival characteristics and prognostic variables of dogs with mitral regurgitation attributable to myxomatous valve disease. *Journal of Veterinary Internal Medicine* 22, 120-128
- Borgarelli, M., Zini, E., D'agnolo, G., et al. (2004) Comparison of primary mitral valve disease in German shepherd dogs and in small breeds. *Journal of Veterinary Cardiology* 6, 27-34
- Brown, D. J., Rush, J. E., Macgregor, J., et al. (2003) M-mode echocardiographic ratio indices in normal dogs, cats, and horses: a novel quantitative method. *Journal of Veterinary Internal Medicine* 17, 653-662
- Haggstrom, J., Kwart, C. & Hansson, K. (1995) Heart sounds and murmurs: changes related to severity of chronic valvular disease in the Cavalier King Charles spaniel. *Journal of Veterinary Internal Medicine* 9, 75-85
- Hansson, K., Haggstrom, J., Kwart, C., et al. (2002) Left atrial to aortic root indices using two-dimensional and M-mode echocardiography in cavalier King Charles spaniels with and without left atrial enlargement. *Veterinary Radiology and Ultrasound* 43, 568-575
- Keren, R., Tereschuk, M. & Luan, X. (2005) Evaluation of a novel method for grading heart murmur intensity. *Archives of Pediatric and Adolescent Medicine* 159, 329-334
- Kittleson, M. D. (2010) Pathophysiology of heart failure: signs of heart failure. In: *Small Animal Cardiovascular Medicine*. Eds M. D. Kittleson & R. D. Kienle. Veterinary Information Network, <http://beta.vin.com/Members/proceedings/Proceedings.plx?CID=SACARDIO&PID=11485&O=VIN>. Accessed January 19, 2014
- Levine, S. A. (1933) Systolic murmur: its clinical significance. *Journal of the American Medical Association* 101, 436-438
- Ljungvall, I., Ahlstrom, C., Hoglund, K., et al. (2009) Use of signal analysis of heart sounds and murmurs to assess severity of mitral valve regurgitation attributable to myxomatous mitral valve disease in dogs. *American Journal of Veterinary Research* 70, 604-613
- Lord, P. F., Hansson, K., Carnabuci, C., et al. (2011) Radiographic heart size and its rate of increase as tests for onset of congestive heart failure in Cavalier King Charles Spaniels with mitral valve regurgitation. *Journal of Veterinary Internal Medicine* 25, 1312-1319
- Moonarmart, W., Boswood, A., Luis Fuentes, V., et al. (2010) N-terminal pro B-type natriuretic peptide and left ventricular diameter independently predict mortality in dogs with mitral valve disease. *The Journal of Small Animal Practice* 51, 84-96
- Ockham, W. 1323. *Summa Totius Logicae*. i. 12
- Ohad, D. G., Rishniw, M., Ljungvall, I., et al. (2013a) Sleeping and resting respiratory rates in dogs with subclinical heart disease. *Journal of American Veterinary Medical Association* 243, 839-843
- Ohad, D. G., Lenchner, I., Bdolah-Abram, T., et al. (2013b) A loud right-apical systolic murmur is associated with the diagnosis of secondary pulmonary arterial hypertension: retrospective analysis of data from 201 consecutive client-owned dogs (2006-2007). *The Veterinary Journal*, 198, 690-69
- Rishniw, M. & Erb, H. N. (2000) Evaluation of four 2-dimensional echocardiographic methods of assessing left atrial size in dogs. *Journal of Veterinary Internal Medicine* 14, 429-435

Sessions, R. (1950) How a "Difficult" composer gets that way. *New York Times*, January 8, 89

Thomas, W. P., Gaber, C. E., Jacobs, G. J., et al. (1993) Recommendations for standards in transthoracic two-dimensional echocardiography in the dog and cat. Echocardiography Committee of the Specialty of Cardiology, American College of Veterinary Internal Medicine. *Journal of Veterinary Internal Medicine* 7, 247-252



Reprint paper*

Orthomanual therapy as treatment for suspected thoracolumbar disc disease in dogs

Dorit C. Aharon¹, Jacha Heukels, Rosanne Buntsma

SUMMARY

According to the principles of veterinary orthomanual medicine, vertebral misalignments are associated with intervertebral disc disease in dogs. Manual correction of these vertebral misalignments are presumed to contribute to successful recovery. The objective of this retrospective study was to evaluate the effects of veterinary orthomanual therapy (VOT) in 261 dachshunds with suspected thoracolumbar intervertebral disc disease (TLDD). Effect of treatment was assessed using a retrograde neurological status classification. From one clinic's 2003-2008 medical records, 261 dachshunds with suspected TLDD met the inclusion criteria. Individual data included signalement and history, orthomanual aspects and neurological evaluations before treatment and at 2 weeks, 3 months and 6 months. Telephone interviews with owners were conducted one year after the initial treatment. The initial neurological status according to Griffiths' grading, was grade I in 115 animals (44%), grade II in 59 animals (23%), grade III in 27 animals (10%), grade IV in 52 animals (20%) and grade V in 8 animals (3%). Two weeks after the first treatment, 111 animals (55%) with initial grade I, II or III and two animals (3%) with initial grade IV or V had improved from their initial grade to a neurologically normal state; within 6 months of the initial treatment this full recovery was observed in 154 animals (77 %) with initial grade I, II or III and 27 animals (45%) with initial grade IV or V. Of the initially non-ambulatory dogs, 82% recovered to an ambulatory state. Of the owners, 89% evaluated the treatment as successful after 1 year. Most (78%) of the animals underwent a single VOT treatment, and the most commonly misaligned vertebrae were T12, T13 and L1. Veterinary orthomanual therapy is a conservative treatment method, which is minimally stressful for the animal and inexpensive. VOT combined with cage rest seems to be effective in treating TLDD in dachshunds. The results of this study demonstrate that veterinary orthomanual therapy might be considered an adjunct modality for the non-surgical treatment of dachshunds with intervertebral disc disease. A prospective controlled clinical trial is needed to further examine its efficacy.

Keywords: complementary medicine, dachshund, intervertebral disc disease, spinal cord injury, thoracolumbar disc disease, veterinary orthomanual medicine

* This paper originally appeared in the *J Hellenic Vet Med Assoc* 2015, 66(1):31-40.
Eur J Comp An Pract, Spring 2016; (26)1: p42-50
Go to <http://www.ejcap.org> for the interactive online presentation of this paper

Introduction

Intervertebral disc disease (IVDD) is one of the most common neurological problems in chondrodystrophic dog breeds (Hoerlein, 1978; Kornegay, 1986; Oliver 1997). In IVDD, a degeneration of the intervertebral disc occurs prior to the extrusion of the nucleus pulposus or protrusion of the annulus fibrosis. Hansen type-I IVDD is characterised by chondroid degeneration followed by extrusion of the nucleus, whereas Hansen type-II IVDD is characterised by fibroid degeneration followed by protrusion of the annulus fibrosis (Wheeler and Sharp, 1993; Bray and Burbidge, 1998; Coates, 2000; Jaggy, 2010). The extrusion or protrusion may result in spinal cord and nerve root compression. Typically, Hansen type-I IVDD affects chondrodystrophic dogs (Oliver, 1997; Jaggy, 2010). The thoracolumbar region is predisposed to disc extrusion or protrusion (Wheeler and Sharp, 1993; Joaquim et al., 2010) which is referred to as thoracolumbar disc disease (TLDD). The manifestation of TLDD depends on the degree of damage to the spinal cord caused by primary and secondary trauma. The clinical manifestation of this disease varies widely between individuals and may include local pain, paraspinal muscle hypertonicity and pelvic limb neurological deficits (Anderson et al., 1982; Lorenz et al., 2011)

Different treatment methods are available for TLDD, including movement restriction (cage rest) analgesia and anti-inflammatory drugs, physiotherapy (Levine et al., 2007; Mann et al., 2007; Lorenz et al., 2011; Ingram et al., 2013), (electro)acupuncture (Janssens and Prins, 1989; Han et al., 2010; Joaquim et al., 2010) and surgery (Hoerlein, 1978; Bitetto and Kapatkin, 1989; Mckee, 1992; Sukhiani et al., 1996; Besalti et al., 2006; Mann et al., 2007; de Lahunta and Glass, 2009). Manual treatments, such as orthomanual therapy, are regularly used to treat back problems in humans and veterinary orthomanual therapy (VOT) has also been practiced for several years (Sickesz, 1986). The focus of orthomanual medicine is on normal positions of components of the skeleton and symmetry in the spine (Genee et al., 2006). Manipulative techniques are applied by fixed protocols using a fast impulse (i.e. quick pressure). This force is applied on the abnormal positioned segment of the skeleton in the direction of the natural position and function (van de Veen et al., 2005).

Orthomanual medicine presumes that the degeneration of the intervertebral disc results in an instability

of consecutive vertebrae. This instability can cause misalignment of vertebrae. As a consequence of this misalignment and disc degeneration, extrusion or protrusion of the nucleus pulposus or the annulus fibrosis results. The consequential pressure on the spinal cord, or trauma induced by the kinetic energy component of the extruded disc material may cause pain and neurological dysfunction. According to the principles of orthomanual medicine, a misalignment of skeletal components can cause loss of function, movement limitations and pain (van de Veen et al., 2005). It is theorised that correcting the misalignment of the vertebrae diminishes the pressure on the intervertebral disc and creates an environment that facilitates an improvement of the neurological state (Assendelft and Lankhorst, 1998; van de Veen et al., 2005). The realignment contributes to pain relief as well. In this study, the clinical value of VOT is retrospectively analyzed in a population of 261 dachshunds with suspected TLDD.

Materials and Methods

Medical records from 2003 – 2008 were searched for dachshunds that were treated with veterinary orthomanual medicine (VOM) for suspected TLDD. Patients with concurrent orthopaedic, systemic or neurological disease were excluded from the study. Patients with neck pain, lower back pain, and/ or vertebral misalignments other than in the thoracolumbar region were also excluded. Furthermore, cases were excluded when complete medical record data was not available. Suspicion of TLDD was based on the patients' typical history and clinical findings (Wheeler, 1995; Oliver, 1997; Levine et al., 2007). The dogs were examined by one veterinarian trained in VOM. During neurological examination the following items were evaluated: the ability to walk, spinal pain, posterior paresis or paralysis, proprioception, spinal reflexes, superficial and deep nociception and bladder function and control. Neurological grading according to a modified Griffiths' scale (Wheeler and Sharp, 1993) was used. In short this grading comprises grade I only pain, grade II posterior paresis with absent proprioception, grade III paralysis with absent proprioception, grade IV paralysis with absent proprioception and absent bladder control, grade V paralysis with absent proprioception, absent bladder control and absent deep nociception. Neurological grades were evaluated before the treatment, at mean 14 days (range: 12 – 16 days), at mean 104 days (range: 89 – 119 days) and at 6 months after the initial visit. Data

collection consisted of age, gender, weight, disease onset, period before first VOM treatment, general examination, initial and repeated neurological examination, medication prior to referral, orthomanual aspects (misaligned vertebrae), evaluation of the animal's neurological performance by the owner 1 year after the first treatment and the number of examinations and treatments.

The clinical results were expressed as a difference in neurological grade compared to day 0, using a retrograde neurological status classification (Table 1).

This classification discriminates between a completely normal neurological status and minor residual neurological abnormalities, such as delay in proprioceptive positioning or slight paresis when, for instance, lifting a hind leg to urinate in males. One year follow-up interviews were conducted by telephone, with the main criterion being whether the animal exhibited normal physical abilities.

Veterinary orthomanual therapy

In VOM special attention is given to interpretation of vertebral positions, such as (i) symmetry and vertebrae alignment (by palpation and visual observation of positions

of the thumbs), (ii) local paraspinal muscle tone, (iii) whether muscle atrophy is present. Furthermore, attention is paid to hyperaesthesia. Palpation is performed along the entire vertebral column from the tail to the head by placing the thumbs on either side of the vertebrae dorsal spinous process (Figs 1 and 2). An assistant, using both hands, fully supports the dog and positions it squarely in front of the veterinarian. The condition of the animal is considered abnormal when the vertebrae are misaligned along the longitudinal axis or with deviations in the ventro-dorsal and lateral (in VOM termed dextro-sinistral) axes. In addition, the position of a vertebra is considered misaligned if the transverse processes of the vertebrae are not parallel to each other, with difference in the level or muscle tone on either side of the vertebra. Several misaligned vertebral positions could be observed in the dogs, including 1. tipper (ventro position): rotation of the vertebra on the longitudinal axis with slight rotation in the sagittal plane to the homolateral side; 2. tumbler: rotation of the vertebra on the sagittal plane with a slight rotation to the homolateral side; 3. slight dorsal dislocation: the frontal part of the vertebra is slightly elevated in the transversal plane; and 4. lateral translation: misalignment

Table 1. Recovery classification in retrograde neurological status. Modified Griffiths' scale (Wheeler and Sharp, 1993).

Score	Definition
0	Clinically and neurological normal, unremarkable neurological examination
I	Ambulatory with slight residual ataxia or proprioceptive delay
II	Ambulatory but paretic, proprioceptive deficits
III	Non-ambulatory; posterior paralysis, intact bladder control
IV	Non-ambulatory; posterior paralysis, absent bladder control
V	Non-ambulatory; posterior paralysis, absent bladder control, absent deep nociception

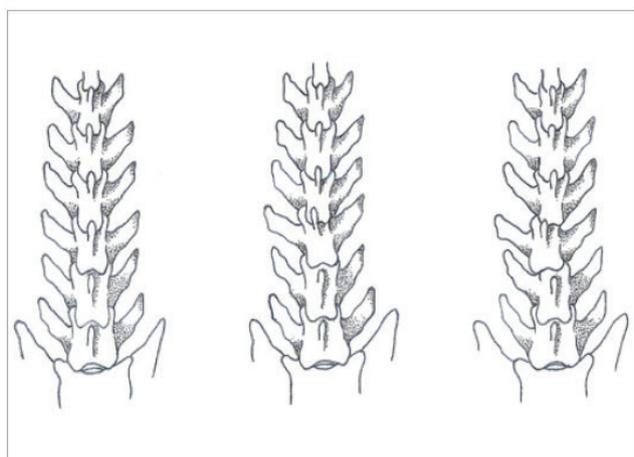


Fig. 1. Normal alignment of the vertebral column (left), tumbler (middle) and lateral translation (right).

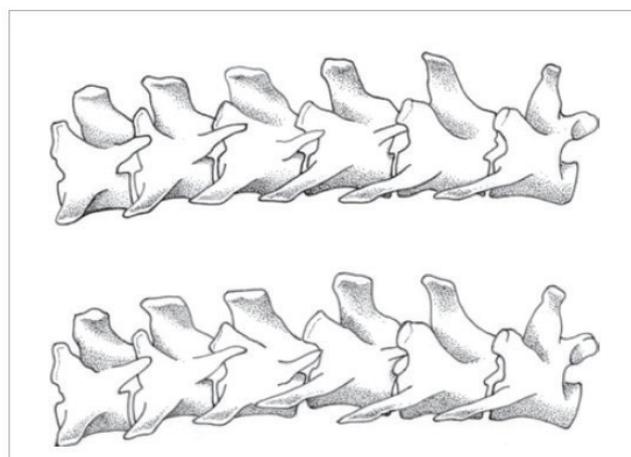


Fig. 2. Normal alignment of the vertebral column (upper illustration) and slight dorsal dislocation (lower illustration). The caudal side of the animal is on the right in the figure.

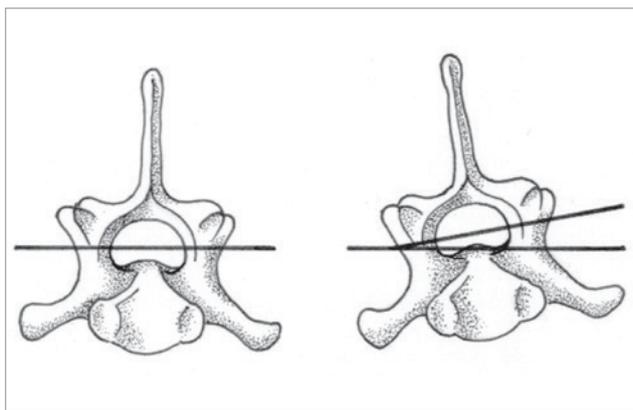


Fig. 3. Normal alignment of a vertebra (left) and a tipper (right).

along the lateral axis (Fig. 3).

The aim of VOT is to correct misaligned vertebral positions. To do this the veterinarian first slightly elevates the vertebral body with one hand and then imparts an impulse to the spinous process with the thumb of the other hand, thereby pushing it into the correct position. Misaligned vertebrae generally require little force to be pushed back into their original positions. All of the misaligned vertebrae are repositioned in a single session. After the first treatment all medications are discontinued if further analgesia is not required and the animals are confined to cage rest for two weeks (Wheeler and Sharp, 1993). After the period of cage rest limited exercise is gradually initiated by the owner according to an individual VOM revalidation protocol. Movement restriction (cage rest) and good care (nursing) are also necessary for neurological improvement (Simpson, 1992; Levine et al., 2007). General and neurological examinations are repeated during the two follow-up visits, and neurological status was graded again. Vertebral misalignments are diagnosed by inspection and palpation during the orthomanual examination of each follow-up. Any misaligned vertebrae, if present, are repositioned.

Data analysis

We calculated the median and range values for age and weight and determined prevalence of gender. Animals were sorted into groups according to their initial neurological grade. Misaligned vertebrae were determined during every visit and the type of misalignment was documented. Neurological change after each treatment was documented at each visit. The evaluation at the 1 year telephone evaluation involved placement into one of the following

categories per group of initial neurological diagnosis: good, unsuccessful, euthanasia for another reason (Euth. other) or unknown. The evaluation was considered "good" if the animal was pain free and exhibited normal physical abilities in and around the house. The evaluation was considered unsuccessful if the animal was not normal in function, had relapsed, was referred to surgery because of unsatisfactory improvement or because it remained paralytic or was euthanized because of the severe neurological state.

Results

In total, 261 Dachshunds were included; 96 (37%) were intact male, 48 (18%) intact female, 39 (15%) castrated male and 78 (30%) neutered female dogs. The mean age was 6.4 years (range: 1 to 15 years), and the mean weight was 8.2 kg (range: 2.8 to 18 kg). The initial neurological status was diagnosed as grade I in 115 animals (44%), grade II in 59 animals (23%), grade III in 27 animals (10%), grade IV in 52 animals (20%) and grade V in 8 animals (3%). 105 Out of 261 dogs (40%) had symptoms of acute onset and 156 (60%) of chronic onset. Prior to VOT, 94% of the animals were medicated before (NSAIDs or corticosteroids).

In this patient population, the most commonly misaligned vertebrae were T12, T13 and L1. The most commonly detected type of misalignment was the tipper (ventroposition).

Two weeks after the first treatment 261 (100%) dogs revisited. 111 of the 201 animals (55%) with initial grade I, II or III exhibited an improvement from their initial neurological grade to normal. A majority (58%) of the animals initially diagnosed as grade II exhibited an improvement of one grade, whereas 44% of animals initially diagnosed as grade III exhibited an improvement of two grades. Furthermore, 2 of the 60 animals (3%) with initial grade IV or V exhibited an improvement from their initial neurological grade to normal. Of the animals initially diagnosed as grade IV, 35% exhibited an improvement of one grade, whereas half of the animals (50%) initially diagnosed as grade V exhibited an improvement of two grades (Table 2). Two patients showed deterioration and were subsequently referred for further diagnostics and surgery.

At three months (mean 104 days) 197 (75%) animals

Table 2. Changes in neurological scoring at two-week follow-up visit. Numbers in red indicate deterioration.

Final Score	Start score				
	I	II	III	IV	V
0	87 (76%)	19 (32%)	5 (19%)	2 (4%)	
I	23 (24%)	34 (58%)	12 (44%)	9 (17%)	
II		4 (7%)	4 (15%)	17 (33%)	1 (13%)
III		1 (2%)	6 (22%)	18 (35%)	4 (50%)
IV		1 (2%)		6 (12%)	2 (25%)
V					1 (13%)
Total	115	59	27	52	8

Table 3. Changes in neurological scoring at three-month follow-up visit. Not all dogs were reassessed at the three-month evaluation.

Final Score	Start score				
	I	II	III	IV	V
0	75 (96%)	32 (71%)	9 (41%)	18 (40%)	1 (14%)
I	2 (3%)	13 (29%)	10 (46%)	19 (42%)	1 (14%)
II	1 (1%)		1 (5%)	6 (13%)	2 (29%)
III	2 (9%)	2 (4%)	3 (43%)	18 (35%)	4 (50%)
IV					
V					
Total	78	43	22	45	7

Table 4. Changes in neurological scoring at six-month follow-up visit. Numbers in red indicate deterioration.

Final Score	Start score				
	I	II	III	IV	V
0	106 (92%)	38 (64%)	10 (37%)	25 (48%)	2 (25%)
I	9 (8%)	17 (29%)	13 (48%)	17 (33%)	2 (25%)
II		2 (4%)	1 (4%)	7 (14%)	1 (13%)
III		1 (2%)	3 (11%)	1 (2%)	3 (38%)
IV		1 (2%)		2 (4%)	
V					
Total	115	59	27	52	8

returned for follow-up. 116 Out of the 145 animals (80%) that revisited with initial grade I, II or III exhibited an improvement from their initial neurological grade to normal. A majority (71%) of the animals initially diagnosed as grade II and 46% of initially graded III exhibited an improvement of two grades. Furthermore, 19 out of the 52 animals that revisited (37%) examined with initial grade IV or V, exhibited an improvement to neurologically

normal. 43% of the animals initially diagnosed as grade IV exhibited an improvement of three grades, and 43% of animals initially diagnosed as grade V exhibited an improvement of two grades (Table 3).

At the six months follow-up 81% of all animals had recovered to a pain free and ambulatory state with 28% exhibiting minor residual ataxia (grade I) and 53%

Table 5. Results of owner evaluation of animal condition at one-year telephone follow-up.

Evaluation	Start score				
	I	II	III	IV	V
Good	109 (95%)	54 (92%)	18 (97%)	47 (90%)	5 (63%)
Unsuccessful	1 (1%)	4 (7%)	4 (15%)	2 (4%)	3 (38%)
Euth./other	1 (1%)				
No answer	4 (4%)	1 (2%)	5 (19%)	3 (6%)	
Total	115	59	27	52	8

Table 6. Total number of orthomanual treatments in a six-month period.

Number of treatments	Start score				
	I	II	III	IV	V
1	95 (83%)	48 (91%)	19 (70%)	36 (60%)	5 (63%)
2	19 (17%)	11 (19%)	7 (26%)	5 (10%)	2 (25%)
3	1 (1%)		1 (4%)	10 (19%)	1 (13%)
4				1 (2%)	
Total	115	59	27	52	8

considered completely normal (grade 0). Of the animals initially diagnosed as grade IV or V, 98% improved at least 1 neurological grade. The 2 animals that had deteriorated and were referred for surgery at 2 weeks have been included in the 6 month table for sake of completeness (Table 4).

In the 1-year telephone evaluation 95% of the owners responded. 181 animals (90 %) with initial grade I, II or III were evaluated by the owner as "good". Furthermore, 52 animals (87%) with initial grade IV or V were evaluated as "good" (Table 5). A single patient was euthanized for reasons unrelated to spinal injury in the interval between the last visit and telephone contact for owner evaluation. Most of the animals (203, i.e. 78%) underwent a single VOT treatment during the six-month study period (Table 6). The following factors were significantly associated with and may have negatively influenced neurological outcome: 1. insufficient cage rest, 2. prior corticosteroid therapy (Levine et al., 2007) and 3. excess body weight. The following factors may have positively influenced the neurological outcome: 1. cage rest 2. prior corticosteroid therapy.

Discussion

A preliminary diagnosis of TLDD is often made based on the animal's history together with information on breed, age and

clinical signs (Wheeler, 1995; Oliver, 1997). Additional diagnostics are required to confirm the diagnosis. Normally myelography, computed tomography (CT) or magnetic resonance imaging (MRI) are performed but they tend to be reserved if surgical management is required.

Dachshunds are predisposed to develop TLDD, making them a suitable model to study the effects of VOT in TLDD (Goggin et al., 1970; Ball et al., 1982; Olby et al., 2004). In this study, the mean age of the affected animals was 6.4 years, which is in accordance with previous findings (Mckee, 1992; Scott, 1997; Nečas, 1999). The most commonly misaligned vertebrae observed in our study were T12, T13 and L1. According several studies, most disc lesions in chondrodystrophic dog breeds occur between T12 and T13, and T13 and L1 (Gambardella, 1980; Mckee, 1992; Scott, 1997; Brisson et al., 2004; Tanaka et al., 2004; Levine et al., 2009).

Orthomanual therapy in dogs with TLDD is presumed to create a condition that facilitates improvement of the neurological state by relieving pressure on the intervertebral disc and spinal cord (Assendelft and Lankhorst, 1998; van de Veen et al., 2005). The addition of cage rest to this therapy allows the animal's body to resorb a part of the extruded disc material and to strengthen the annulus fibrosus, and reduces the risk of further nucleus pulposa extrusion. Cage rest also decreases the incidence of self-trauma due to incoordination (Simpson, 1992; Mochida

et al., 1998; Doita et al., 2001; Levine et al., 2007). One study of medical conservative treatment reported successful treatment of 100% of animals with hyperpathia with or without mild neurologic deficits (i.e., grade I or II in our neurological grading) (Mann et al., 2007). A success rate of 54.7% in a patient group with initial neurological grade I to V was reported in another study of medical conservative treatment, with dogs that were completely normal and without recurrence of clinical signs (Levine et al., 2007). In a third study, success rates of 100%, 84%, 100%, 50% and 7% were reported in respectively initial grade I, II, III, IV and V, with residual paresis (i.e., at least grade I in our neurological grading) in 13% of the animals (Olby et al., 2004). In our study, patients with residual paresis were graded I-II and not considered fully recovered, which makes outcome percentages difficult to compare. In this light, success rates according to the retrograde classification for initial grades I-V were 92%, 64%, 37%, 48% and 25% respectively. When not taking residual paresis into account however, success rates were 100%, 97%, 89%, 94% and 63% respectively.

For initially non-ambulatory patients, the ability to walk voluntarily after treatment and the time to ambulation are the criteria commonly used to discern between a successful and unsuccessful outcome. Recovery rates including animals with residual paresis after decompressive surgery in non-ambulatory dogs with intact deep nociception (i.e. grade III and IV in our study) vary between 86% and 96% (Gambardella, 1980; Scott, 1997; Ne as, 1999; Davis and Brown, 2002; Ferreira et al., 2002; Brisson et al., 2004; ruddle et al., 2006; Bush et al., 2007). In this study 78% of the initial grade III and 54% of the grade IV animals were ambulatory within 2 weeks, and respectively 89% and 94% within 6 months. However, 44% of grade III and IV animals were considered completely recovered without any residual paresis.

Absent deep nociception (grade V) is often associated with severe spinal cord damage and worse prognosis (Henke et al., 2013). Reported percentages of functional recovery after surgery in dogs without deep nociception ranges from 38% to 76% (Aikawa et al., 2012). In our study 63% of the initial grade V dogs recovered to an ambulatory state and were considered successfully treated by the corresponding 63% of owners at the 1 year evaluation. Practical experience indicates that reoccurrence of vertebral misalignments, once treated, is rare. This is in

conclusion with a single treatment that was needed for the majority of patients, as shown in table 6. The neurological grade at onset is positively correlated with the number of orthomanual treatments, which suggests that the likeliness for vertebral misalignment to reoccur might be related to the severity of spinal cord compression.

The lack of diagnosis confirmation by medical imaging is a limitation of this study and it is possible that some animals had other conditions that led to the same symptoms. Also, due to lack of diagnostic imaging, no distinction was made between HNP type 1 and type 2 and this may account for some variability in outcome. Other limitations are the relatively small group of initially graded V dogs, outcomes based on telephone interviews with owners that are prone to information bias, and its retrospective nature. Due to the lack of a control group, it remains unclear what the natural course of the disease would have been, and if and to what extent VOT contributed to recovery. It is well known that many ambulatory dogs with thoracolumbar spinal lesions (grade I-II) recover with medical management and cage rest alone (Ingram et al., 2013). However, recovery rates for non-ambulatory patients (grade III-V) with conservative management alone are considerably lower in proportion to the initial neurological grade, and spontaneous recovery from grade IV and V lesions is unlikely (Lorenz et al., 2011; Wheeler and Sharp, 1993). Many dogs in our study had already been treated unsuccessfully with steroids, NSAIDs and cage rest, and improved only after VOT treatment. Also, recovery rates after VOT for grade III-V dogs were good and similar to those reported for surgical treatment.

Concluding remarks

In this study a retrograde neurological classification system was introduced with the aim of creating uniformity in describing and quantifying neurological recovery from spinal cord injury. Veterinary orthomanual therapy combined with cage rest seems to be effective in treating TLDD in dachshunds and might be considered an acceptable form of non-surgical treatment. Furthermore, VOT is minimally stressful for the animal, inexpensive and non-invasive. Success rates from this retrospective study are comparable with those reported for decompressive surgery, and long-term owner evaluations were excellent. No recidives or complications were reported in the timespan of 1 year.

This is the first study to describe the effects of orthomanual therapy in dogs with suspected intervertebral disc disease. Further research is required to study other breeds, larger groups of grades III to V, treatment mechanism and the correlation between diagnostic findings and VOM clinical findings. A prospective controlled clinical trial is the next logical step.

Acknowledgements

The authors would like to thank Henk Halsema for help with the figures, and Prof. Dr. Andre Jaggy, Dr. Luc Janssens, Dr. Clare Rusbridge and Dr. Franck Forterre for their advice.

Conflict of interest statement

None of the authors of this paper has a financial or personal relationship with other people or organisations that could inappropriately influence or bias the content of the paper.

References

- Aikawa T, Fujita H, Kanazono S, Shibata M, Yoshigae Y (2012) Long-term neurologic outcome of hemilaminectomy and disk fenestration for treatment of dogs with thoracolumbar intervertebral disk herniation: 831 cases (2000- 2007). *J Am Vet Med Assoc* 241:1617-1626.
- Anderson DK, Means ED, Waters Tr, Green ES (1982) Microvascular perfusion and metabolism in injured spinal cord after methylprednisolone treatment. *J Neurosurg* 56:106-113.
- Assendelft WJJ, Lankhorst GJ (1998) Effectiviteit van manipulatieve therapie bij lage rugpijn: geen uitsluitel in systematische literatuuroverzichten en behandelrichtlijnen. *Nederl Tijds Geneesk* 142:684-688.
- Ball Mu, McGuire JA, Swaim SF, Hoerlein BF (1982) Patterns of occurrence of disk disease among registered dachshunds. *J Am Vet Med Assoc* 180:519-522.
- Besalti O, Peckan Z, Sirin YS, Erbas G (2006) Magnetic resonance imaging findings in dogs with thoracolumbar intervertebral disk disease: 69 cases (1997-2005). *J Am Vet Med Assoc* 228:902-908.
- Bitetto WV, Kapatkin AS (1989) Intraoperative problems associated with intervertebral disc disease. *Probl Vet Med* 1:434-444.
- Bray JP, Burbidge HM (1998) The canine intervertebral disk. Part two: degenerative changes-non chondrodystrophoid versus chondrodystrophic disks. *J Am Anim Hosp Assoc* 34:135-144.
- Brisson BA, Moffatt SL, Swayne SL, Parent JM (2004) Recurrence of thoracolumbar intervertebral disk extrusion in chondrodystrophic dogs after surgical decompression with or without prophylactic fenestration: 265 cases (1995-1999). *J Am Vet Med Assoc* 224:1808-1814.
- Bush WW, Tiches DM, Kamprad C (2007) Functional outcome following hemilaminectomy without methylprednisolone sodium succinate for acute thoracolumbar disk disease in 51 nonambulatory dogs. *J Vet Emerg Crit Care* 17:72-76.
- Coates Jr (2000) Intervertebral disk disease. *Vet Clin N Am Small Anim Pract* 30:77-110.
- Davis GJ, Brown DC (2002) Prognostic indicators for time to ambulation after surgical decompression in nonambulatory dogs with acute thoracolumbar disk extrusions: 112 cases. *Vet Surg* 31:513-518.
- De Lahunta A, Glass EN (2009) Small animal spinal cord disease. In: (eds: De Lahunta A, Glass EN) *Veterinary Neuroanatomy and Clinical Neurology*, 3rd edn. Elsevier, St. Louis, pp. 243-265.
- Doita M, Kanatani T, Ozaki T, Matsui N, Kurosaka M, Yoshiya S (2001) Influence of macrophage infiltration of herniated disc tissue on the production of matrix metalloproteinases leading to disc resorption. *Spine* 26:1522-1527.
- Ferreira AJA, Correia JHD, Jaggy A (2002) Thoracolumbar disc disease in 71 paraplegic dogs: influence of rate of onset and duration of clinical signs on treatment results. *J Small Anim Pract* 43:158-163.
- Gambardella PC (1980) Dorsal decompressive laminectomy for treatment of thoracolumbar disc disease in dogs: a retrospective study of 98 cases. *Vet Surg* 9:24-26.
- Genee T, van de Bunt A, de Bruijne, JJ (2006) De standafwijkingen. In: (eds: Genee T, van de Bunt A, de Bruijne JJ et al.) *Orthomanuele Geneeskunde*, 1st edn. Nederlandse Vereniging Orthomanuele Geneeskunde, Amsterdam, pp. 54-93.
- Goggin JE, Li AS, Franti CE (1970) Canine intervertebral disc disease: characterization by age, sex, breed, and anatomic site of involvement. *Am J Vet Res* 31:1687-1692.
- Han HJ, Yoon HY, Kim JY, Jang HY, Lee B, Choi SH, Jeong SW (2010) Clinical effect of additional electroacupuncture on thoracolumbar intervertebral disc herniation in 80 paraplegic dogs. *Am J Chin Med* 38:1015-1025.

- Henke D, Vandeveld M, Doherr MG, Stöckli M, Forterre F (2013) Correlations between severity of clinical signs and histopathological changes in 60 dogs with spinal cord injury associated with thoracolumbar intervertebral disc disease. *Vet J* 198:70-75.
- Hoerlein BF (1978) Intervertebral disks. In: (ed: Hoerlein BF) *Canine neurology: diagnosis and treatment*, 3rd edn. Saunders, Philadelphia, pp. 470-560.
- Hoerlein BF (1978) The status of various intervertebral disc surgeries for the dog in 1978. *J Am Anim Hosp Assoc* 14:563-570.
- Ingram EA, Kale DC, Balfour RJ (2013) Hemilaminectomy thoracolumbar Hansen Type I intervertebral disk disease in ambulatory dogs with or without neurologic deficits: 39 cases (2008-2010). *Vet Surg* 42:924-931.
- Jaggy A (2010) Spinal cord. In: (ed: Jaggy A) *Small animal neurology*, 1st edn. Schlütersche, Hannover, pp. 351-356.
- Janssen LAA, Prins EMD (1989) Treatment of thoracolumbar disc disease in dogs by means of acupuncture: a comparison of two techniques. *J Am Anim Hosp Assoc* 25:169-174.
- Joaquim JG, Luna SP, Brondani JT, Torelli SR, Rahal SC, de Paula Freitas F (2010) Comparison of decompressive surgery, electroacupuncture, and decompressive surgery followed by electroacupuncture for the treatment of dogs with intervertebral disk disease with long-standing severe neurologic deficits. *J Am Vet Med Assoc*. 236:1225-1229.
- Kornegay JN (1986) Intervertebral disk disease. In: (eds: Kornegay JN) *Neurologic disorders – contemporary issues in small animal practice*, 1st edn. Church Livingstone, New York, pp. 21-39.
- Levine JM, Fosgate GT, Chen AV, Rushing R, Nghiem PP, Platt SR, Bagley RS, Kent M, Hicks DG, Young BD, Schatzberg SJ (2009) Magnetic resonance imaging in dogs with neurologic impairment due to acute thoracic and lumbar intervertebral disk herniation. *J Vet Intern Med* 23:1220-1226.
- Levine JM, Levine GJ, Johnson SI, Kerwin SC, Hettlich BF, Fosgate GT (2007) Evaluation of the success of medical management and prognostic factors in non-ambulatory Hansen type I intervertebral disc extrusions: 308 cases. *Vet Comp Orthop Traumatol* 19:29-34.
- Lorenz MD, Coates Jr, Kent M (2011) Confirming a Diagnosis. In: (eds: Lorenz MD, Coates Jr, Kent M) *Handbook of veterinary neurology*, 5th edn. Saunders/Elsevier, St. Louis, pp. 75-77.
- Mann FA, Wagner-Mann CC, Dunphy ED (2007) recurrence rate of presumed thoracolumbar intervertebral disc disease in ambulatory dogs with spinal hyperpathia treated with antiinflammatory drugs: 78 cases (1997 – 2000). *J Vet Emerg Crit Care* 17:53-60.
- Mckee WM (1992) A comparison of hemilaminectomy (with concomitant disc fenestration) and dorsal laminectomy for the treatment of thoracolumbar disc protrusion in dogs. *Vet Rec* 30:296-300.
- Mochida K, Komori H, Okawa A, Muneta T, Haro H, Shinomiya K (1998) regression of cervical disc herniation observed on magnetic resonance images. *Spine* 23:990-995.
- Nečas A (1999) Clinical aspects of surgical treatment of thoracolumbar disc disease in dogs. A retrospective study of 300 cases. *Acta Vet* 68:121-130.
- Olby N, Harris T, Burr J, Muñana K, Sharp N, Keene B (2004) Recovery of pelvic limb function in dogs following acute intervertebral disc herniations. *J Neurotr* 21:49-59.
- Oliver JE (1997) Pelvic limb paresis, paralysis, or ataxia. In: (eds: Oliver JE, Kornegay JN, Lorenz MD) *Handbook of veterinary neurology*, 3rd edn. Saunders, Philadelphia, pp. 133-138.
- Ruddle TL, Allen DA, Schertel ER, Barnhart MD, Wilson ER, Lineberger JA, Klocke NW, Lehenbauer TW (2006) Outcome Scott HW (1997) Hemilaminectomy for the treatment of thoracolumbar disc disease in the dog: a follow-up study of 40 cases. *J Small Anim Pract* 38:488-494.
- Sickesz M (1986) De standsafwijking van de wervels. In: (eds: Sickesz M) *klinische orthomanipulatie*, 1st edn. Ankh-Hermes bv, Deventer, pp. 7-15.
- Simpson ST (1992) Intervertebral disc disease. *Vet Clin N Am Small Anim Pract* 22:889-897.
- Sukhiani HR, Parent JM, Atilola MAO, Holmberg DL (1996) Intervertebral disc disease in dogs with signs of back pain alone: 25 cases (1986-1993). *J Am Vet Med Assoc* 209:1275-1279.
- Tanaka H, Nakamaya M, Takase K (2004) Usefulness of myelography with multiple views in diagnosis of circumferential location of disc material in dogs with thoracolumbar intervertebral disc herniation. *J Vet Med Sci* 66:827-833.
- Van de Veen EA, de Vet HC, Pool JJ, Schuller W, de Zoete A, Bouter LM (2005) Variance in manual treatment of nonspecific low back pain between orthomanual physicians, manual therapists, and chiropractors. *J Manipul Physiol Ther* 28:108-116.
- Wheeler SJ (1995) Neurological examination of the limbs and body. In: (eds: Wheeler SJ) *BSAVA Manual of small animal neurology*, 2nd edn. BSAVA, Cheltenham, pp. 27-36.
- Wheeler SJ, Sharp NJH (1993) Cervical disc disease. In: (eds: Wheeler SJ, Sharp NJH) *Small animal spinal disorders, diagnosis and surgery*, 1st edn. Mosby-Wolfe Publishers, London, pp. 85-108. for presumptive thoracolumbar intervertebral disk herniation in dogs. *Vet Surg* 36:482-491.