Evaluation of mortality rate and predictors of outcome in dogs receiving outpatient treatment for parvoviral enteritis

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OBJECTIVE

To determine mortality rates and prognostic factors for dogs with parvoviral enteritis receiving outpatient treatment.

DESIGN

Retrospective case series and case-control study.

ANIMALS

130 client-owned dogs with a diagnosis of parvoviral enteritis between August 1, 2012, and January 31, 2015, that were treated with outpatient care.

PROCEDURES

Medical records were reviewed and data extracted regarding dog age, body weight, breed, and vaccination history; treatments administered; and short-term (≥ 3 day) outcome (determined via telephone call with owner). Treatments were administered according to clinician preference. Mortality rates were calculated overall and for various signalment and treatment groupings and compared.

RESULTS

97 (75%) dogs survived and 33 (25%) dogs failed to survive for ≥ 3 days after initial diagnosis of parvoviral enteritis. Compared with distributions in the general hospital population, Chihuahuas, German Shepherd Dogs, pit bull–type dogs, and males were overrepresented. No significant difference was identified between survivors and nonsurvivors regarding age, body weight, or sex. Dogs prescribed a caloric supplement fed every 2 to 4 hours had a mortality rate of 19% (16/85). Most of these dogs had also received fluids administered SC, an antiemetic, and antimicrobials.

CONCLUSIONS AND CLINICAL RELEVANCE

Clinicians should note the 25% mortality rate of the dogs with parvoviral enteritis that received outpatient care in this study setting when discussing treatment options with owners of affected dogs who are financially unable to pursue hospitalization. (*J Am Vet Med Assoc* 2017;251:1035–1041)

Canine parvovirus type 2 is a severe and deadly infectious disease that primarily affects young dogs[1] and was first reported in the mid 1970s.[2] Dogs with parvoviral enteritis caused by CPV-2 infection classically have mucoid to bloody diarrhea, dehydration, vomiting, and signs of depression, lethargy, and inappetence[3–5] and are highly contagious to other dogs.[6] Mortality rates as high as 90.9% have been reported for dogs that receive no treatment,[3] and those for dogs that receive treatment range from 4% to 53%, depending on the therapeutic approach.[7–11] No specific medication has been established as an effective antiviral treatment for parvoviral enteritis. Secondary bacterial infection from intestinal translocation, septicemia, systemic inflammatory response syndrome, and multiple organ dysfunction syndrome play a considerable role in the mortality rate for this disease.[5,12]

Ideally, dogs with parvoviral enteritis should be admitted to the hospital and managed with aggressive IV fluid therapy and medication (antimicrobials, antiemetics, analgesics, dextrose, electrolytes, nutritional support, and anthelmintics).[1] The high cost associated with a 24-hour hospitalization period makes treating these infections financially challenging; therefore, research is warranted into the effectiveness of outpatient treatment for dogs with parvoviral enteritis when intensive hospitalization is not feasible.

Despite the ready availability of effective vaccines, parvoviral enteritis remains a common infectious disease. Canine parvovirus type 2 is easily spread and is hardy in the environment.[6,13,14] Vaccination against CPV-2 infection can be ineffective in puppies with maternal antibody interference; thus, dogs can remain susceptible until the vaccination series

ABBREVIATIONS

CPV-2 Canine parvovirus type 2

is complete.[2,4,15] Since the initial discovery of CPV-2, new strains have emerged and have been designated as CPV-2a, CPV-2b, and CPV-2c.[1,6] Vaccines containing components of CPV-2 and CPV-2b reportedly provide protection against CPV-2c infection.[16]

Several dog characteristics (sex, body weight, age, and breed) have been evaluated for associations with clinical CPV-2 infection and prognosis. No significant associations have been identified between development of parvoviral enteritis and dog sex or body weight in other studies,[5,17–19] and dog sex does not appear to influence the probability of survival.[8] It has been suggested the mortality rate of younger dogs is higher than that of older dogs,[18,20] although a large retrospective Australian study[8] showed that age had no association with mortality rate.[8] Purebred dogs have a higher risk of developing parvoviral enteritis than mixed-breed dogs,[5,18] with Doberman Pinschers and Rottweilers overrepresented.[17] In addition to these 2 breeds, pit bull–type dogs and German Shepherd Dogs are also at increased risk of developing parvoviral enteritis.[18] The authors are aware of no studies of breed predilection for CPV-2c infection, although natural infection has been investigated in German Shepherd Dogs.[21,22]

Current recommendations are that clinically ill dogs be medically managed in the hospital setting, with treatment consisting primarily of supportive care that includes IV fluid therapy, antiemetics, nutritional support, and prevention of secondary infection. Balanced isotonic electrolyte solutions are given IV to restore perfusion and maintain hydration.[9] Fluids are modified with additives to support glucose, potassium, and oncotic pressure as needed.[9] Some clinicians have recommended consideration of plasma or whole blood transfusions,[9] although a study[23] failed to reveal an improvement in survival rate with plasma administration.

Because of the mucosal sloughing and loss of barrier function of the gastrointestinal tract, dogs with parvoviral enteritis are vulnerable to bacterial translocation, sepsis, and endotoxemia from *Escherichia coli*.[24–26] Neutropenia often accompanies the clinical syndrome in these dogs, further compromising their immune response to secondary infections.[27,28] Broad-spectrum bactericidal antimicrobials are used to reduce the risk introduced by gram-negative and anaerobic bacteria in the intestinal tract.[4,7] Antimicrobial recommendations in the past have included an aminoglycoside or enrofloxacin given with a β-lactam antimicrobial[9] or, in dogs with milder cases of disease, ampicillin, first-generation cephalosporins, or trimethoprim-sulfonamide.[9]

Provision of enteral nutrition early in the course of disease is now recommended for dogs with parvoviral enteritis, in contrast to the previous suggestion of nothing PO until vomiting resolves.[9] Pediatric patients are prone to hypoglycemia, and blood glucose concentration needs to be supported by frequent feedings, particularly if dextrose is not provided in IV administered fluids.[9] A study[29] in which urinary lactulose concentration was used as an indicator of intestinal healing in dogs demonstrated that nourishing enterocytes early (as opposed to withholding food) in the course of disease resulted in a more rapid gain in clinical improvement. Data from that study[29] also support the provision of protein sources directly to the area of inflammation to promote intestinal healing. Reduction in circulating amounts of proinflammatory cytokines such as tumor necrosis factor-α and interferon-γ combats the catabolism that occurs with malnutrition.[9,29] Various methods for enteral feeding include nasoesophageal, nasogastric, esophagostomy, and jejunal tube placement as well as forced (syringe) feeding.

The canine population susceptible to parvoviral enteritis is also susceptible to intestinal parasitism, and deworming is recommended.[9] Presence of intestinal parasites increases disease severity.[11,30] The odds of developing parvoviral enteritis reportedly increase in puppies with a history of anthelminthic treatment,[5] but no data have been reported regarding the impact of anthelmintic treatment on mortality rate.[5] At the time of writing, no specific studies had been reported regarding outpatient treatment and survival rate in dogs with parvoviral enteritis. The purpose of the study reported here was to determine mortality rates in dogs with parvoviral enteritis receiving various forms of outpatient treatment and explore relationships between disease outcomes and dog sex, body weight, breed, rectal temperature at initial evaluation, and prior vaccination status.

Materials and Methods

Case selection criteria

Medical records at a private small animal clinic were searched to identify dogs with a diagnosis of parvoviral enteritis recorded from August 1, 2012, to January 31, 2015. Dogs were included in the study if they had clinical signs consistent with parvoviral enteritis and positive results of an in-clinic fecal ELISA for CPV-2 antigen.[a] Dogs euthanized following a positive parvovirus test result without treatment were excluded. Dogs were also excluded from the study if they had been hospitalized for treatment or had incomplete or missing outcome data.

Medical records review

Medical records were reviewed and data collected regarding dog breed, body weight, age and rectal temperature at initial evaluation, sex, vaccination history (ever vaccinated in the past [yes or no]), treatments received, and outcome. For vaccine history, the records were searched for any mention of prior vaccination (rather than the timing and identity of vaccines administered), given that the timing and type of vaccines administered were not consistently recorded. Data on treatments received included any medications administered at the clinic or prescribed for administration on an outpatient basis.

Generally, dog owners were instructed to administer fluids SC at home on a daily basis, support body

temperature by housing the dog indoors in a climate-controlled area, and keep the dog clean and isolated from other dogs. All owners were instructed to force feed small amounts frequently if dogs were not eating voluntarily. If dogs were not sent home with a specific supplement, owners were instructed to feed any food that the dog would accept or to force feed small amounts of unspecified canned foods or baby foods.

Patient outcomes were documented in the record as part of a routine follow-up telephone conversation with owners 3 days after initial evaluation. Follow-up was continued via telephone until the dog was reported by the owner to be clinically normal or deceased.

Statistical analysis

Mortality rates were calculated for the whole group of included dogs and for various signalment and treatment groupings. The Fisher exact and χ^2 tests were used to compare distributions of survivors or nonsurvivors between the various groupings, and the Welsh *t* test was used to compare means of continuous variables (eg, body weight, age, and rectal temperature) between survivors and nonsurvivors. Continuous data are summarized as mean ± SD. Results were considered significant at a value of $P < 0.05$. Statistical software[b] was used for all analyses.

Results

Animals

During the 31-month study period, 160 dogs examined because of signs of gastrointestinal disease had positive results of fecal CPV-2 antigen testing. Fifteen dogs were treated with hospitalization, and 6 were immediately euthanized after diagnosis. One hundred thirty-nine dogs were treated for parvoviral enteritis on an outpatient basis, but 9 were lost to follow-up, leaving 130 dogs treated on an outpatient basis for parvoviral enteritis in the study. Overall, 97 (75%) dogs survived and 33 (25%) dogs failed to survive for ≥ 3 days after initial diagnosis.

Seventy-five (58%) of the included dogs were sexually intact males, 48 (37%) were sexually intact females, 4 (3%) were neutered males, and 3 (2%) were spayed females. Age ranged from 1 to 36 months, with 59% (77/130) of the dogs in the age range of 3 to 6 months. Compared with the sex distribution of all dogs evaluated at the hospital during the same period as the dogs with parvoviral enteritis (n = 4,744), males were significantly ($P = 0.03$) overrepresented among dogs with parvoviral enteritis. No significant difference was identified between survivors and nonsurvivors regarding sex distribution, mean age, mean body weight, or mean rectal temperature at initial evaluation **(Table 1)**.

Dogs included in the study consisted of various breeds, with more than half consisting of 3 breeds or types (Chihuahua, pit bull–type dog, and German Shepherd Dog; **Table 2**). No difference in mortality rates was identified among dog breeds ($P = 0.63$). However, the 3 most common breeds or types for the affected dogs were significantly overrepresented and mixed-breed dogs significantly underrepresented, compared with their distributions in the general canine patient population.

Thirty-two (25%) dogs had a recorded history of vaccination (at a veterinary clinic, animal shelter, breeder facility, or unnamed facility). Twenty-seven

Table 2—Breed distribution (%) of the dogs in Table 1 (n = 130), compared with the distribution in the population of all canine patients evaluated at the hospital during the same period (4,744).

Type of dog or breed	Dogs with parvoviral enteritis	All dogs	*P* value
Mixed breed	21.5	32.3	0.006
Chihuahua	20.8	9.1	< 0.001
Pit bull type	20.0	6.1	< 0.001
German Shepherd Dog	12.3	2.7	< 0.001
Labrador Retriever	8.5	7.4	0.32

See Table 1 for key.

Table 1—Characteristics of client-owned dogs with parvoviral enteritis that were treated with outpatient care and survived (n = 97) or did not survive (33) for ≥ 3 days after initial diagnosis.

Characteristic	Survivors	Nonsurvivors	*P* value
Age (mo)	5.1 ± 4.9	5.1 ± 2.6	0.92
Body weight (kg)	7.77 ± 6.18	7.00 ± 5.71	0.49
Rectal temperature (°C)	38.47 ± 0.53	38.32 ± 0.68	0.38
Sex			
Male	58 (73)	21 (27)	0.84
Female	39 (76)	12 (24)	—
History of CPV-2 vaccine			
Yes	27 (84)	5 (16)	0.17
No	70 (71)	28 (29)	—

Data reported as mean ± SD for age, body weight, and rectal temperature and number (percentage of all in category [ie, survival or mortality rate]) for sex and vaccine history.

— = Not applicable.

Values of $P < 0.05$ were considered significant (χ^2 or Fisher exact test for comparisons of proportions and Welch *t* test for comparison of means).

Table 3—Number (%) of dogs in Table 1 that received various types of treatment.

Treatment	Survivors	Nonsurvivors	*P* value
Maropitant (SC)			0.34
Yes	91 (73)	33 (27)	—
No	6 (100)	0 (0)	—
Cefovecin (SC)			0.32
Yes	55 (79)	15 (21)	—
No	42 (70)	18 (30)	—
Amoxicillin (PO)			0.83
Yes	29 (72)	11 (28)	—
No	68 (76)	22 (24)	—
Metronidazole (PO)			0.28
Yes	27 (68)	13 (32)	—
No	70 (78)	20 (22)	—
Crystalloid fluid (SC)			0.34
Yes	90 (73)	33 (27)	—
No	7 (100)	0 (0)	—
Pyrantel pamoate (PO)			0.64
Yes	75 (76)	24 (24)	—
No	22 (71)	9 (29)	—
Sucralfate (PO)			0.23
Yes	78 (77)	23 (23)	—
No	19 (66)	10 (34)	—
Caloric supplement (PO)			0.02
Yes	69 (81)	16 (19)	—
No	28 (62)	17 (38)	—
Any antimicrobial (SC or PO)			0.19
Yes	87 (73)	32 (27)	—
No	10 (91)	1 (9)	—

See Table 1 for key.

(84%) of these dogs survived with outpatient treatment for parvoviral enteritis, and 5 (16%) did not survive ($P = 0.17$; Table 1).

Treatment

Dogs were treated by 5 veterinarians during the study period. Treatments included SC administration of crystalloid fluid[c] (n = 123 [95%]), maropitant citrate[d] for nausea (124 [95%]), and cefovecin sodium[e] (70 [54%]) and oral administration of sucralfate[f] (101 [78%]), pyrantel pamoate[g] (99 [76%]), a caloric supplement[h] (85 [65%]; given every 2 to 4 hours), amoxicillin[i] or metronidazole[j] (40 [31%] each), and a vitamin supplement[k] (8 [6%]) or probiotic (5 [4%]). Only the prescribed caloric supplement (vs no such supplement) was associated with a significantly lower mortality rate **(Table 3)**.

Discussion

The present study represents the largest retrospective study in which short-term outcome was evaluated in dogs that received outpatient treatment for parvoviral enteritis. Results suggested that an overall outpatient mortality rate of 25% within 3 days after diagnosis may be expected in similar settings. Overall mortality rate was 19% for the patients treated with a prescribed caloric supplement in addition to other outpatient treatments such as SC administered fluids, antimicrobials, antiemetics, and pyrantel pamoate. In a study[31] involving dogs with parvoviral enteritis that were hospitalized and received IV fluid therapy, antiemetics, and antimicrobials, the mortality rate was 20% (16/79).

Selection bias likely influenced the results of the present study, given that clinicians may have more strongly encouraged hospitalization of dogs with severe disease. Because the study involved dogs treated at a primary care facility with a large low-income clientele, only 15 of the 160 dogs that received a diagnosis of parvoviral enteritis during the study period were treated with hospitalization. The present study was not intended as an endorsement of outpatient treatment, but it did provide information regarding prognosis for dogs with parvoviral enteritis treated on an outpatient basis for clients unable to afford in-hospital treatment.

Owing to the financial constraints of many owners and the retrospective nature of the present study, little data were available regarding comorbid diseases and infections that may have influenced the observed mortality rates. Parvovirus typing was not performed to evaluate differences in mortality or survival rates among virus strains or determine regional variation in virulence, thereby preventing the ability to predict which dogs would be expected to do well with outpatient treatment for parvoviral enteritis.

Vaccination history was analyzed for an association with mortality rate in the present study. Because of the study's retrospective nature, no standardized history taking was performed, nor was the vaccination history complete for every dog. The mortality rate for dogs with prior vaccination recorded (16%) appeared less than that of dogs with no such history recorded (29%), although this difference was not significant ($P = 0.17$). Whether the vaccination history of dogs in the present study was similar to that in other studies is unknown. Some dogs with gastrointestinal signs could have had false-positive results of fecal testing for CPV-2. Vaccination against CPV-2 infection results in fecal shedding of the vaccine virus but should not have contributed to false-positive results.[31,l] The lower mortality rate in previously vaccinated dogs may have been partially attributable to protective immunity from prior vaccinations or the prevention of comorbid diseases. None of the dogs had a recorded history indicating a complete puppy vaccination series had been received.

As in a previous study,[32] no association was identified between outcome and age, body weight, and sex of dogs. However, in contrast to previous studies[5,17–19] indicating no association between sex and parvovirus diagnoses, males were overrepresented in the present study. Likely because of the young age of most affected dogs (mean, 5.1 months), not enough dogs had been sexually altered to allow evaluation of the association between neuter status and outcome.

As in previous research,[18] German Shepherd Dogs and pit bull–type dogs were overrepresented among those with parvoviral enteritis in the pres-

ent study. Chihuahuas were also overrepresented. Mixed-breed dogs, on the other hand, were underrepresented, also as reported elsewhere.[5,18] Importantly, however, breed had no significant association with mortality rate.

Subcutaneous fluid administration was provided in the present study because the dogs were treated on an outpatient basis, but this approach has drawbacks. Severely dehydrated patients may have circulatory compromise with peripheral vasoconstriction limiting absorption, and skin necrosis is a reported risk given that affected patients are often hypovolemic and immunocompromised.[33] No adverse effects of SC fluid administration were noted for the dogs in the present study, and because a large proportion (95%) received such treatment, the power to detect an association between this treatment and outcome was reduced. It was also possible that less severely affected dogs were not clinically dehydrated and that fluids were not prioritized in the financially constrained management of cases.

The significant association with outcome of oral administration of a high-calorie supplement[h] was an unexpected finding. Although research has shown that early administration of enteral nutrition can help to accelerate the intestinal villus regeneration found in dogs with malabsorptive enteritis,[19] it is the high-protein (20% to 34%) formulations that have been recommended with specific attention given to the amount, type, and quality of protein.[34] Alternatively, the caloric supplement administered has a high carbohydrate (56%) content, including cane molasses and corn and malt syrups that provide palatability but a low amount of protein (0.68%). Perhaps carbohydrate sources of calories may have a more beneficial impact in dogs with parvoviral enteritis than previously realized, which may have more to do with hypoglycemia prevention than enteral nutrition.

Macronutrient arguments aside, the ease of feeding a high-calorie supplement could have facilitated owner compliance with at-home administration, given that its high caloric density (4 kcal/mL)[h] can be easily loaded into a feeding syringe. The degree to which owners complied with administration recommendations was not evaluated. In a previous study,[29] early clinical improvement and weight gain were identified in dogs with parvoviral enteritis that received enteral nutrition, although no significant impact on survival rate was identified. Additional research should be conducted to compare mortality rates in canine outpatients with parvoviral enteritis receiving the specific carbohydrate-based formulation used in the present study versus other nutritional options prescribed in a similar format.

Three antimicrobials (cefovecin, amoxicillin, and metronidazole) were used alone or in combination for dogs in the present study to reduce the risk of sepsis from gastrointestinal translocation of bacteria. In a previous study[24] involving 98 dogs with parvoviral enteritis, 90% had evidence of septicemic colibacillosis at necropsy.[24] For the outpatient setting, antimicrobial activity against *E coli* was the primary consideration along with safety, cost, route, and frequency of administration. Ideal antimicrobial choices for treatment of affected dogs include an aminoglycoside or enrofloxacin with a β-lactam antimicrobial, ampicillin, cephalosporins, or trimethoprim-sulfonamide.[9] An aminoglycoside was not used for dogs in the present study because of concerns about hydration status, given that dogs received SC fluid administration at home without daily reevaluation by professional staff. Enrofloxacin was not used because of cost, formulation (tablet or daily injections), and bone growth plate–related concerns. Seventy (54%) dogs received cefovecin, which is a β-lactam, third-generation cephalosporin that is not dependent on oral bioavailability because it is injected SC and has an intrinsically long half-life.[35,36] Use of cefovecin eliminated any concerns of missed doses, vomiting, or malabsorption, and the drug is effective against nonresistant strains of *E coli*.[36] Amoxicillin, which is effective against common *E coli* strains but can be inactivated by resistant strains,[37-39] was administered PO to 40 (31%) dogs. Amoxicillin is available in an inexpensive liquid formulation, facilitating oral administration to anorexic or hyporexic dogs.

Oral metronidazole administration was prescribed for some dogs in the present study, although another study[40] showed that metronidazole has no activity against *E coli* in an anaerobic environment. None of the administered antimicrobials were significantly associated with outcome. Given that each of the antimicrobials has a limited spectrum of activity, it would be interesting to determine whether other antimicrobial selections would result in better patient outcomes. The prevalence of antimicrobial-resistant *E coli* infection in dogs with parvoviral enteritis is unknown.

The present study had several limitations that should be considered when interpreting the results. This was a single-institution, retrospective study with no randomization of treatments. Rather, treatments were selected on the basis of dog characteristics and client financial status, the latter of which limited the variety of treatments offered. Few dog owners were able to afford hospitalization ($1,000 to $2,500). Too few dogs were treated with hospitalization during the study period to allow mortality rate comparisons with dogs treated on an outpatient basis. History taking and data recording were not standardized, thereby limiting options for evaluation of illness severity at initial evaluation. Another major limitation was that no laboratory data were collected for most dogs beyond the fecal ELISA for CPV-2 antigen, eliminating the ability to evaluate associations with outcome of variables such as WBC count, blood glucose concentration, serum albumin concentration, or other hematologic analytes.

A need remains for development of an optimal outpatient treatment protocol for dogs with parvoviral enteritis through randomized controlled clinical trials. A wider range of antimicrobials and various

CPV-2 subtypes need to be evaluated for associations with outcome. The strain infecting the dogs in the present study and its impact on mortality rate remain unknown, as does the impact of comorbid conditions.

We wish to emphasize that outpatient treatment of dogs with severe parvoviral enteritis would be appropriate only when hospitalization is not financially feasible. The best medical recommendation remains to manage these patients in a hospital setting with appropriate laboratory testing and treatment. At the author's clinic, hospitalization for parvoviral enteritis can cost thousands of dollars, whereas expenses incurred in the diagnosis and treatment of most outpatient cases are approximately $300 to $400. Dog owners should be advised that the outpatient option has been associated with a 25% mortality rate, as determined in this particular study setting.

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Footnotes

a. IDEXX USA, Portland, Me.
b. R, version 3.0.3, R Foundation for Statistical Computing, Vienna, Austria. Available at: www.R-project.org. Accessed Jan 7, 2016.
c. Lactated Ringer solution, Abbott Laboratories, Chicago, Ill.
d. Cerenia (10 mg/mL), Pfizer, New York, NY.
e. Convenia (80 mg/mL), Zoetis, Kalamazoo, Mich.
f. Carafate (1 g/10 mL) Aptalis Pharma US, Bridgewater, NJ.
g. Strongid T, Pfizer, New York, NY.
h. NutriCal oral gel, Vetoquinol, Princeville, QC, Canada.
i. DAVA Pharmaceuticals, Fort Lee, NJ.
j. Watson Pharma Private Ltd, Mumbai, Maharashtra, India.
k. Lixotinic, Pfizer, New York, NY.
l. Schultz RD, Larson LJ, Lorentzen LP. Effects of modified live canine parvovirus vaccine on the Snap ELISA Antigen Assay (abstr), in *Proceedings*. Int Vet Emerg Crit Care Symp 2008.

References

1. Goddard A, Leisewitz A. Canine parvovirus. *Vet Clin North Am Small Anim Pract* 2010;40:1041–1053.
2. Carmichael LE, Binn LN. New enteric viruses in the dog. *Adv Vet Sci Comp Med* 1981;25:1–37.
3. Njenga MK, Nyaga PN, Buoro IBJ, et al. Effectiveness of fluids and antibiotics as supportive therapy of canine parvovirus-2 enteritis in puppies. *Bull Anim Health Prod Afr* 1990;38:379–389.
4. Pollock RVH, Coyne MJ. Canine parvovirus. *Vet Clin North Am Small Anim Pract* 1993;23:555–568.
5. Kalli I, Leontides LS, Mylonakis ME, et al. Factors affecting the occurrence, duration of hospitalization and final outcome in canine parvovirus infection. *Res Vet Sci* 2010;89:174–178.
6. Smith-Carr S, Macintire DK, Swango LG. Canine parvovirus. Part 1. Pathogenesis and vaccination. *Compend Contin Educ Pract Vet* 1997;19:125–133.
7. Prittie J. Canine parvoviral enteritis: a review of diagnosis, management, and prevention. *J Vet Emerg Crit Care* 2004;14:167–176.
8. Ling M, Norris JM, Kelman M, et al. Risk factors for death from canine parvoviral-related disease in Australia. *Vet Microbiol* 2012;158:280–290.
9. Macintire DK, Smith-Carr S. Canine parvovirus. Part II. Clinical signs, diagnosis, and treatment. *Compend Contin Educ Pract Vet* 1997;19:291–302.
10. Kocaturk M, Martinez S, Eralp O, et al. Prognostic value of serum acute-phase proteins in dogs with parvoviral enteritis. *J Small Anim Pract* 2010;51:478–483.
11. Brunner CJ, Swango LJ. Canine parvovirus infection: effects on the immune system and factors that predispose to severe disease. *Compend Contin Educ Pract Vet* 1985;7:979–988.
12. Otto CM, Jackson CB, Rogell EJ, et al. Recombinant bactericidal/ permeability-increasing protein (rBPI21) for treatment of parvovirus enteritis: a randomized, double-blinded, placebo-controlled trial. *J Vet Intern Med* 2001;15:355–360.
13. Gordon JC, Angrick EJ. Canine parvovirus: environmental effects on infectivity. *Am J Vet Res* 1986;47:1464–1467.
14. Kennedy MA, Mellon VS, Caldwell G, et al. Virucidal efficacy of the newer quaternary ammonium compounds. *J Am Anim Hosp Assoc* 1995;31:254–258.
15. O'Brien SE. Serologic response of pups to the low-passage, modified-live canine parvovirus-2 component in a combination vaccine. *J Am Vet Med Assoc* 1994;204:1207–1209.
16. Larson LJ, Schultz RD. Do two current canine parvovirus type 2 and 2b vaccines provide protection against the new type 2c variant? *Vet Ther* 2008;9:94–101.
17. Glickman LT, Domanski LM, Patronek GJ, et al. Breed-related risk factors for canine parvovirus enteritis. *J Am Vet Med Assoc* 1985;187:589–594.
18. Houston DM, Ribble CS, Head LL. Risk factors associated with parvovirus enteritis in dogs: 283 cases (1982–1991). *J Am Vet Med Assoc* 1996;208:542–546.
19. Castro TX, Miranda SC, Larbarthe NV, et al. Clinical and epidemiological aspects of canine parvovirus (CPV) enteritis in the State of Rio de Janeiro: 1995–2004. *Arquivo Brasil Med Vet Zoo* 2007;59:333–339.
20. Godsall SA, Clegg SR, Stavisky JH, et al. Epidemiology of canine parvovirus and coronavirus in dogs presented with severe diarrhea to PDSA PetAid hospitals. *Vet Rec* 2010;167:196–201.
21. Decaro N, Buonavoglia C. A review of epidemiological and diagnostic aspects, with emphasis on type 2c. *Vet Microbiol* 2012;155:1–12.
22. Decaro N, Desario C, Campolo M, et al. Clinical and virological findings in pups naturally infected by canine parvovirus type 2 Glu-426 mutant. *J Vet Diagn Invest* 2005;17:133–138.
23. Bragg RF, Duffy AL, DeCecco FA, et al. Clinical evaluation of a single dose of immune plasma for treatment of canine parvovirus infection. *J Am Vet Med Assoc* 2012;240:700–704.
24. Turk J, Miller M, Brown T, et al. Coliform septicemia and pulmonary disease associated with canine parvoviral enteritis: 88 cases (1987–1988). *J Am Vet Med Assoc* 1990;196:771–773.
25. Kreeger TJ, Jeraj KP, Manning PJ. Bacteremia concomitant with canine parvovirus infection in a pup. *J Am Vet Med Assoc* 1984;184:196–197.
26. Otto CM, Drobatz KJ, Soter C. Endotoxemia and tumor necrosis factor activity in dogs with naturally occurring parvoviral enteritis. *J Vet Intern Med* 1997;11:65–70.
27. Goddard A, Leisewitz AL, Christopher MM, et al. Prognostic usefulness of blood leukocyte changes in canine parvoviral enteritis. *J Vet Intern Med* 2008;22:309–316.
28. Macartney L, McCandlish IA, Thompson H, et al. Canine parvovirus enteritis 1: clinical, haematological and pathological features of experimental infection. *Vet Rec* 1984;115:201–210.
29. Mohr AJ, Leisewitz AL, Jacobson LS, et al. Effect of early enteral nutrition on intestinal permeability, intestinal protein loss, and outcome in dogs with severe parvoviral enteritis. *J Vet Intern Med* 2003;17:791–798.
30. McCaw DL, Hoskins JD. Chapter 8: Canine viral enteritis. In: Green CE, ed. *Infectious diseases of the dog and cat.* 3rd ed. St Louis: Saunders Elsevier, 2006;63–73.
31. Decaro N, Crescenzo G, Desario C, et al. Long-term viremia and fecal shedding in pups after modified-live canine parvovirus vaccination. *Vaccine* 2014;32:3850–3853.
32. McClure V, van Schoor M, Thompson PN, et al. Evaluation of the use of serum C-reactive protein concentration to predict outcome in puppies infected with canine parvovirus. *J Am Vet Med Assoc* 2013;243:361–366.
33. Brown AJ, Otto CM. Fluid therapy in vomiting and diarrhea. *Vet Clin North Am Small Anim Pract* 2008;38:653–675.
34. Marks SL. The principles and implementation of enteral nutrition. In: Ettinger SJ, Feldman EC, eds. *Textbook of veteri-*

nary internal medicine, diseases of the dog and cat. 6th ed. Philadelphia: Elsevier Saunders, 2005;596–597.
35. European Medicines Agency. Convenia: scientific discussion 2006. EMEA/CVMP/215997/2006. Available at: www.ema.europa.eu. Accessed Sep 18, 2015.
36. Stegemann MR, Passmore CA, Sherington J, et al. Antimicrobial activity and spectrum of cefovecin, a new extended-spectrum cephalosporin, against pathogens collected from dogs and cats in Europe and North America. *Antimicrob Agents Chemother* 2006;50:2286–2292.
37. Bywater RJ, Palmer GH, Buswell JF, et al. Clavulanate-potentiated amoxicillin: activity in vitro and bioavailability in the dog. *Vet Rec* 1985;116:33–36.
38. Grnvold AR, L'abée-Lund TM, Sørum H, et al. Changes in fecal microbiota of healthy dogs administered amoxicillin. *FEMS Microbiol Ecol* 2010;71:313–326.
39. Procter TD, Pearl DL, Finley RL, et al. A cross-sectional study examining the prevalence and risk factors for anti-microbial-resistant generic *Escherichia coli* in domestic dogs that frequent dog parks in three cities in south-western Ontario, Canada. *Zoonoses Public Health* 2014;61:250–259.
40. Pendland SL, Jung R, Messick CR, et al. In vitro bactericidal activity of piperacillin, gentamicin, and metronidazole in a mixed model containing *Escherichia coli, Enterococcus faecalis*, and *Bacteroides fragilis. Diagn Microbiol Infect Dis* 2002;43:149–156.

From this month's *AJVR*

Effect of track surface firmness on the development of musculoskeletal injuries in French Trotters during four months of harness race training

Nathalie Crevier-Denoix et al

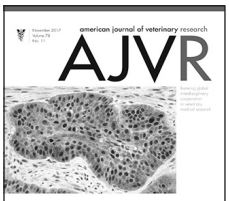
OBJECTIVE
To evaluate the effect of track surface firmness on the development of musculoskeletal injuries in French Trotters during 4 months of race training.

ANIMALS
12 healthy 3-year-old French Trotters.

PROCEDURES
Horses were paired on the basis of sex and body mass. Horses within each pair were randomly assigned to either a hard-track or soft-track group. The counterclockwise training protocol was the same for both groups. Surface firmness of each track was monitored throughout the training period. Radiography, ultrasonography, MRI, and scintigraphy were performed on all 4 limbs of each horse before and after 2 and 4 months of training. Lesions were described, and lesion severity was classified with a 5-point system, where 0 = no lesions and 4 = severe lesion.

RESULTS
86 lesions were identified, of which 46 (53.5%) were classified as potentially clinically relevant (grade, ≥ 2). Of the 18 moderate and severe lesions, 15 were identified in horses of the hard-track group, and 10 of those were in forelimbs. Moderate to severe tendinopathy of the superficial digital flexor tendon of the forelimb developed in 3 of the 6 horses of the hard-track group but none of the horses of the soft-track group. Metatarsal condyle injuries were more frequent in horses of the hard-track group than horses of the soft-track group. Severe lesions were identified only in left limbs.

CONCLUSIONS AND CLINICAL RELEVANCE
Results indicated that track surface firmness is a risk factor for musculoskeletal injuries in horses trained for harness racing. (*Am J Vet Res* 2017;78:1293–1304)



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