

Thirteen cases of tetanus in dogs

S. ADAMANTOS, A. BOAG

The records of 13 dogs with tetanus were reviewed, 12 of the dogs survived and were discharged, but the other died as a result of the acute onset of hyperthermia. Long-term follow up was available for 10 of the survivors of which nine were reported to be normal. One case was euthanased after a cervical spine fracture four months after it was discharged. Complications encountered during management included aspiration pneumonia in three cases, urinary tract infection in two cases, and upper respiratory tract obstruction, hiatal hernia, coxofemoral luxation, seizures and respiratory arrest in one case each. None of the dogs required ventilatory support, and the complications were managed successfully. The dogs were nursed intensively and monitored carefully.

TETANUS is an uncommon neurological problem in dogs (Greene 2005) and has rarely been reported in the literature. This retrospective study describes the clinical signs, treatment, complications during hospitalisation and outcome in 13 dogs with tetanus.

Tetanus is caused by an infection with the bacterium *Clostridium tetani* and the release of tetanospasmin toxin. *C. tetani* is a Gram-positive anaerobic spore-forming bacillus that is widely distributed in the environment. Following infection and in an anaerobic environment the spores vegetate and release their toxins: tetanolysin and tetanospasmin. Tetanospasmin enters the peripheral nerves distally at the neuromuscular end plate and then moves in a retroaxonal manner to the central nervous system where it binds irreversibly to presynaptic sites on inhibitory neurons and inhibits the release of glycine and γ -aminobutyric acid (GABA), leading to sustained muscle spasms. Recovery is dependent on the production of new axonal terminals (Sanford 1995). Dogs are much less susceptible to tetanus than other species; and are 600 times more resistant to tetanospasmin than horses (Greene 2005). It is thought that their resistance is related to the insensitivity of their peripheral nervous tissue to penetration by the toxin. Clinical cases may develop either localised or generalised muscle spasms although the generalised form seems to be more common in dogs. Treatment consists of supportive therapy including nursing, feeding, sedation and muscle relaxation, together with specific therapy consisting of antibiotics to prevent clostridial growth and toxin production and the administration of tetanus antitoxin to minimise further binding of toxin to nervous tissue. Ultimately, recovery depends on the successful support of the animal while new axonal terminals form.

MATERIALS AND METHODS

The medical database and computer records of the Queen Mother Hospital for Animals (QMHA) were searched for cases of tetanus in dogs between January 2000 and February 2006. The records were reviewed and included if the dog had an appropriate history and clinical signs consistent with tetanus (Greene 2005) and if diagnostic tests had not identified another cause.

The records included the dog's characteristics, its clinical signs and their course, diagnostic tests, drug treatment, management details, complications during treatment and outcome. A telephone follow-up was made if possible at the time of the review. The records were entered into a statistical package for analysis (SPSS v 14.0). Numerical results were reported as mean (sd) when normally distributed, and median and range when not.

RESULTS

Thirteen dogs were identified with tetanus as a primary diagnosis; there were five labrador retrievers, two golden retriev-

ers, two Staffordshire bull terriers, one boxer, one German short-haired pointer, one border collie and one crossbreed. Their median age was 1.7 years (three months to 10 years) and seven of them were less than two years of age. Nine of the dogs were male, of which one was neutered, and four were female of which two were neutered.

Initial clinical signs

The referring veterinary surgeons reported a variety of clinical signs (Table 1). Facial abnormalities (including third eyelid protrusion, abnormal ocular position, trismus, erect ear carriage and an abnormal facial expression) were reported in 10 of the cases. Salivation and dysphagia were reported in five cases, but stiffness and ataxia were reported in only four cases. All the cases had either facial abnormalities or dysphagia.

Clinical course

In seven cases a wound had been observed 10 to 14 days before the dog was examined, but in the other six cases no wound was seen although one dog had been lame 10 days before it showed clinical signs. In another dog a broken canine tooth was found and one dog had two damaged deciduous upper canines. The wounds were minor in six of the cases and required no further treatment, but the other case had a severe nail-bed infection that necessitated amputation of the digit.

The signs had been present for between two and seven days (median three days) before the dogs were referred to the QMHA. Three cases had signs localised to the head, but the other 10 had generalised signs. Two of these had only mild signs and were able to walk throughout the course of the disease; however, the other eight were unable to walk at some point during the course of the disease.

Diagnostic tests

A number of mild non-specific haematological and biochemical abnormalities were observed in 12 of the 13 cases: there was neutrophilia in six, lymphopenia in six, monocytosis in three, severe thrombocytopenia in one and severe anaemia in one. Biochemical abnormalities included high activities of creatine kinase in seven cases (range 1599 to 18405 U/l [reference range 61 to 394 U/l]) and moderate hypoalbuminaemia in one case. Clotting profiles were performed in two cases and were markedly abnormal in one case, with a prothrombin time of 59 seconds (reference range 12 to 17 seconds) and an activated partial thromboplastin time (APTT) of more than 299 seconds (reference range 71 to 109 seconds). The dog in this case also had severe thrombocytopenia, anaemia and gastrointestinal haemorrhage. An analysis for the presence of rodenticides in its serum was negative and the ratio of vitamin K₁ 2,3-epoxide: vitamin K₁ was normal, ruling out exposure to coumatrin as the cause of the coagulopathy. Faecal analysis was negative for parvovirus, *Giardia* species and bacterial causes. The cause of the coagulopathy was never identi-

Veterinary Record (2007)
161, 298-303

S. Adamantos, BVSc,
CertVA, MRCVS,
A. Boag, BA, VetMB,
MRCVS,
Queen Mother Hospital
for Animals, Hawkshead
Lane, North Mymms,
Hatfield AL9 7TA

TABLE 1: Initial and most severe clinical signs observed in 13 dogs with tetanus, the interval before they were referred, period in hospital and intensive care unit (ICU) and final outcome

Case	Initial signs	Most severe signs	Initial signs to referral (days)	Hospitalisation (days)	ICU (days)	Outcome
1	Trismus, hypersalivation	Trismus, hypersalivation	3	13	3	Lost to follow-up
2	Dysphagia	Generalised extensor rigidity and recumbency	3	21	14	Alive and normal
3	Dysphagia, abnormal facial expression	Generalised extensor rigidity and recumbency	3	13	4	Lost to follow-up
4	Ptyalism, stiffness	Generalised extensor rigidity and recumbency	7	7	5	Died
5	Abnormal facial expression, stiffness	Generalised extensor rigidity and recumbency	3	42	35	Alive and normal
6	Third eyelid protrusion	Third eyelid protrusion, mild dysphagia	2	8	0	Alive and normal
7	Stiffness, abnormal facial expression	Generalised extensor rigidity and recumbency	2	18	17	Alive and normal
8	Abnormal facial expression	Generalised extensor rigidity and recumbency, upper respiratory tract obstruction	2	22	20	Discharged alive, euthanased 4 months later
9	Abnormal facial expression	Generalised stiff gait	6	13	0	Alive and normal
10	Abnormal facial expression	Generalised extensor rigidity and recumbency	2	23	16	Alive and normal
11	Abnormal facial expression, dysphagia, stiffness	Generalised extensor rigidity and recumbency	2	14	11	Alive and normal
12	Dysphagia, ataxia, ptyalism	Generalised stiff gait, ataxia	4	7	2	Alive and normal
13	Abnormal facial expression	Startled expression, third eyelid protrusion	7	0	0	Alive

fied but it resolved within two days of introducing vitamin K therapy; vitamin K was discontinued after 14 days once rodenticide exposure was ruled out and repeat monitoring showed no recurrence of the coagulopathy.

Thoracic radiographs were taken in eight cases to evaluate for aspiration pneumonia. Transtracheal washes were performed in three cases in which pneumonia was suspected on the basis of the clinical signs and radiography. Bacterial culture was positive in all three cases. Urinalysis was performed daily on three dogs with indwelling urinary catheters, and samples were cultured from two cases with signs consistent with a urinary tract infection. Neurodiagnostic tests were performed in two cases, including analysis of cerebrospinal fluid, electromyography and measurements of nerve conduction velocity; they were normal in both cases. Blood gas analyses were carried out in cases managed in the intensive care unit. No evidence of hypoventilation was identified in any of them, and in no case was a venous or arterial carbon dioxide level above 45 mmHg recorded.

Treatment

Tetanus antitoxin (Tetanus Antitoxin Behring; Intervet UK) was administered intravenously to nine of the 13 cases. The dose administered was between 33.3 and 556 iu/kg (median 100 iu/kg). In four cases test doses were administered intradermally before the full dose. Four cases were not given antitoxin. In one case the signs had already been present for seven days and it was thought that the amount of unbound toxin was likely to be low. In two cases the signs were mild and it was thought that the antitoxin would have little effect. In the fourth case a test dose resulted in a wheal at the injection site and no further dose was administered.

Six of the dogs received 20 mg/kg penicillin G (Crystapen; Britannia Pharmaceuticals) every six hours, and nine received 10 mg/kg metronidazole (Metronidazole; MacoPharma) every 12 hours for the specific treatment of *C. tetani*; three of the dogs received both these drugs and one dog received neither. This dog received 10 mg/kg enrofloxacin (Baytril; Bayer) every 24 hours and 20 mg/kg ampicillin (Penbritin; GlaxoSmithKline) every eight hours. Other antibiotics including cefalexin (Ceporex; Schering-Plough Animal Health),

potentiated amoxicillin (Augmentin; GlaxoSmithKline), amikacin (Amikin; Bristol-Myers Pharmaceuticals) and enrofloxacin were administered at standard doses for aspiration pneumonia, urinary tract infections and the management of wounds on the basis of the results of culture and sensitivity tests.

Sedative and muscle relaxing drugs were administered to 11 of 13 cases: acepromazine (ACP; Vericore) to nine, benzodiazepines (Diazemuls; Alpharma, Hypnovel; Roche) to eight, dantrolene (Dantrium; Procter & Gamble Pharmaceuticals) to three, butorphanol (Torbugesic; Fort Dodge Animal Health) to two, pentobarbitone (Sagatal; Merial Animal Health) to two, propofol (Propofol; Abbott Laboratories) to one and guaiphenesin (Myolaxin; Vetoquinol) to one. In most cases a combination of the drugs was used to obtain optimal sedation and muscle relaxation. The use and route of administration of these drugs is summarised in Table 2. Bolus doses were given at variable time intervals, depending on the clinical signs. One dog with localised cephalic signs received neither muscle relaxing nor sedative drugs. All the recumbent dogs received a benzodiazepine; in these cases diazepam was used initially as a bolus of 0.2 mg/kg repeated as necessary to effect.

The dogs were kept in hospital for between less than 24 hours and 42 days, with a median of 13 days (Table 1). One dog was not hospitalised because its signs were extremely mild. Three dogs were not kept at any time in the intensive care unit; those that did spent between two and 35 days there, with a median of 12 days (Table 1).

Case management

Recumbent animals were initially managed in the intensive care unit with frequent monitoring, which included heart rate, respiratory rate, electrocardiogram, direct arterial blood pressure and blood gas analysis, urine output, electrolytes, packed cell volume and total blood solids. They were turned every four hours if unable to move or maintained in sternal recumbency to help prevent the formation of decubital ulcers.

Nutritional support was introduced as soon as practically possible. Feeding tubes were placed in the 10 dogs

showing signs of dysphagia or dysphonia: seven received a percutaneous endoscopically placed gastrotomy tube, one a naso-oesophageal tube, one an oesophageal tube and one a nasogastric tube. Normal feeding was started once the clinician managing the case felt that the risk of aspiration due to dysphagia was low. The 10 dogs received all their nutrition by tube for between five and 17 days, with a median of 11 days.

Intravenous fluid therapy with balanced isotonic crystalloid solutions was provided to prevent or correct dehydration and electrolyte abnormalities. The dogs were maintained in a quiet environment to minimise stimulation. Once there was some improvement in their spastic signs, physiotherapy was initiated applying mainly passive motion to improve joint mobility and prevent joint hyperextension; in the most severe cases this was assisted by bolus injections of intravenous benzodiazepines.

Complications during management

Three of the 13 dogs developed signs consistent with aspiration pneumonia (pyrexia, dyspnoea, coughing and/or crackles on lung auscultation). Tracheal washes were taken in these cases and *Escherichia coli* sensitive to amikacin was cultured in one case, *E coli* and *Klebsiella pneumoniae* sensitive to enrofloxacin in the second case and *E coli* sensitive to cefalexin in the third. In one case an upper respiratory obstruction secondary to laryngeal spasm required the placement of a tracheostomy tube; this relieved the obstruction and remained in place for eight days by which point there was a reduction in the degree of muscle spasm. One dog suffered a respiratory arrest on two occasions, once in relation to the aspiration of food and once for an unknown reason; on both occasions it responded rapidly to intubation and positive pressure ventilation for less than five minutes. One dog suffered seizures; it had generalised mild signs and no previous history of seizures. The cause of the seizures was not identified, but they were controlled initially by medication with 1 mg/kg phenobarbitone twice daily, the dose being tapered over six months, with no recurrence of the seizures. Two of the dogs developed a urinary tract infection associated with the presence of indwelling urinary catheters for more than seven days. *E coli*, *Enterococcus faecalis* and *Proteus* species were cultured in one case with all the isolates being sensitive to enrofloxacin. *E faecalis* and *Klebsiella* species sensitive to enrofloxacin, amoxicillin/clavulanate, oxytetracycline and sulphonamide/trimethoprim were cultured in the second case.

One dog began to regurgitate two weeks after its clinical signs were first observed. Thoracic radiographs revealed a soft tissue opacity in the caudal thorax consistent with a hiatal hernia. The dog continued to feed normally while being kept in an upright position, and no further regurgitation occurred. Its clinical and radiographic signs had resolved seven days later when it was discharged.

One dog developed coxofemoral luxation while in hospital; the femoral head and neck were excised after it had recovered from tetanus. All these dogs recovered uneventfully.

None of the dogs required ventilatory support for hypoventilation, but four were given oxygen for hypoxaemia.

One dog died six days after it was admitted, after an acute episode of hyperthermia. No cause of death was identified at postmortem examination. The dog had been improving well and had been discharged from the intensive care unit to general wards.

Outcome

Twelve of the 13 dogs recovered and were discharged from the hospital (Fig 1). Telephone follow-up was available for 10 of them, ranging from two months to four years. One case

TABLE 2: Summary of the drugs used to manage the tetanic signs in 13 dogs

Drug	Number of dogs	Route of administration	Dose range bolus or oral (mg/kg)	Frequency of bolus dosing	CRI (mg/kg/h)
Acepromazine	9	Intravenous bolus, intramuscular, oral	0.005-0.05	Every 6-8 hours	No
Diazepam	7	Intravenous bolus, oral	0.2-0.5	As required	No
Midazolam	1	Intravenous bolus, CRI	0.2	As required	0.6-1.8
Butorphanol	2	Intravenous bolus	0.2-0.4	Every 4-6 hours	No
Dantrolene	3	Oral/via feeding tube	1-1.5	Once daily	No
Propofol	1	Intravenous bolus, CRI	1-2	As required	0.5-2
Pentobarbitone	2	Intravenous bolus, CRI	2-3	As required	1
Guaiphenesin	1	Intravenous bolus, CRI	50	NA	5

CRI Constant rate infusion, NA Not applicable

developed atlantoaxial dysplasia and cervical displacement four months after it was discharged and was euthanased due to complications with the management of this problem. The other nine dogs were reported to be normal.

DISCUSSION

Tetanus is a potentially devastating disease that is uncommon in dogs. Tetanus toxin binds irreversibly to axonal terminals resulting in disinhibition and muscular spasms. Recovery is dependent on the synthesis of new presynaptic components and their transport to the distal axon, typically leading to a two-week delay before signs of clinical improvement (Bleck and Brauner 1997). In small animals two forms of the disease are recognised, a local form in which the signs are limited to a region, for example, a limb, and a generalised form in which the entire body is affected. The form that develops is probably determined by the site and route of administration of the toxin, that is, whether it enters by intramuscular or subcutaneous inoculation, or haematogenously (Greene 2005). The rate of progression and the severity of the clinical signs are both variable (Table 1).

In human beings the generalised disease has a high mortality of 11 to 28 per cent (Hsu and Groleau 2001) with most deaths occurring in the acute phase of the disease. The mortality in small animals seems to be lower, and the only death among these 13 dogs occurred in the late stage of the disease.

The disease is uncommon in the UK, and there are only single case reports and letters in the veterinary literature (Bark 1980, Hanson 1982, Ogden 1983, Edwards and others 1989, Ratcliffe 1989, Toolan 1989, Bhadwal and Wazir 2005). Reports from the Czech Republic and Greece have described cases with similar characteristics to those described here, with the majority being entire young purebred dogs. Wurst and Komarek (1987) reported that 90 per cent of their cases were less than six months old, 69 per cent were entire males and 72 per cent were German shepherd dogs; the fact that the majority were entire is probably related to the relative youth of the cases. Previously reported survival rates of 55 per cent for generalised, 100 per cent for localised tetanus (Wurst and Komarek 1987) and 83 per cent for generalised tetanus (Soubasis and others 2002) are lower than that reported in the present study, possibly owing to the more intensive nature of the treatment provided or to the severity of the disease. However, the data show that tetanus is a treatable disease with a good prognosis, although intensive monitoring and nursing is required in the majority of cases.

No definitive diagnostic test for tetanus is readily available. The cases were included if they had compatible clinical signs and history (Greene 2005) and if diagnostic tests did not reveal an alternative diagnosis. Tests that can support the diagnosis include electromyography and nerve conduction



FIG 1: (a) Four-month-old female labrador (case 8) and (b) six-month-old male border collie (case 5) exhibiting their most severe signs of tetanus, and during recovery (c and d, respectively)

studies, but they are neither sensitive nor specific for tetanus and were not applied routinely. It is also possible to measure serum antibody titres to tetanus toxin although the reliability of this is questionable (Dewey 2003). It is often not possible to isolate *C. tetani* from wounds (Greene 2005). In all these 13 cases the recovery of the dogs to full function supported the initial diagnosis.

The portal of entry has previously been reported to be via wounds, broken or deciduous teeth or surgical sites and there have been several cases of tetanus after an ovariohysterectomy (Rubin and others 1983, Bagley and others 1994, Ganssbauer and others 2000). Seven of the 13 cases had wounds identified and two had broken or damaged teeth that could have provided a portal of entry. Only one had evidence of an infection and required surgical treatment of its wound. If there is evidence of active infection or necrosis, the wound should be debrided as soon as practically possible.

The treatment of tetanus is controversial. The efficacy of the antitoxin is unproven and the optimal dose is not well defined; as it acts by binding to unbound toxin it is only likely to be useful in the peracute stage of disease. It is considered that the load of toxin, rather than the animal's bodyweight determines the optimal dose (Hsu and Groleau 2001). A study in human beings demonstrated that doses of 500 iu and 3000 to 10,000 iu were equally effective (Blake and others 1976) and

the lower dose is now recommended. The half-life of tetanus antitoxin in dogs is approximately 14 days, so that only one dose is usually required (Steihm 1979). Intrathecal administration has been described in dogs (Soubasis and others 2002) and horses (Steinman and others 2000) and has been recommended in severe cases because the antitoxin does not cross the blood-brain barrier. However, intrathecal administration is no longer recommended in human practice because a number of studies and meta-analyses have shown that it has no benefit over parenteral administration (Vakil and others 1964, Abrutyn and Berlin 1991). None of these 13 dogs were given antitoxin intrathecally, and owing to the small number of cases it was not possible to determine whether the administration of antitoxin affected their recovery times.

Intradermal testing is recommended before the administration of the antitoxin because it is derived from horses and may therefore be associated with a hypersensitivity reaction. The prevalence of hypersensitivity reactions in dogs is unknown. One of the dogs had a mild reaction to an intradermal injection and did not receive a full dose. No hypersensitivity reactions were observed in the five dogs that did not have an intradermal test dose.

Antibiotic therapy is recommended in all cases of tetanus and should be aimed at elimination of *C. tetani*. Penicillin G has been recommended because it is effective against anaer-

obic Gram-positive bacteria, especially clostridial species. Recent data from human beings suggest that metronidazole may be a better choice because it penetrates tissue with vascular compromise, including abscesses, more effectively. Its use has been associated with shorter recovery times and lower mortality (Ahmadsyah and Salim 1985). Furthermore, penicillin has GABA-antagonistic properties that may potentiate the effects of the tetanospasmin. A large randomised study of treatments with penicillin or metronidazole showed that the group treated with metronidazole required smaller doses of muscle relaxants and sedatives (Farrar and others 2000) possibly owing to a reduction in antagonism to GABA. Approximately equal numbers of these 13 dogs received these drugs without any noticeable difference in outcome.

Sedative and muscle relaxant drugs were administered in the majority of cases. Extreme muscle spasms are painful and in clinical cases of tetanus can be severe. Complete muscle relaxation can be difficult to achieve without general anaesthesia, but long-term anaesthesia may be associated with complications. Benzodiazepines are the initial drug of choice because they are GABA-agonists and have some sedative properties. In the majority of the cases several different drugs were used to provide muscle relaxation while minimising overt sedation. When possible the aim was to achieve a plane of sedation at which the dog was rousable only by noxious stimuli.

Complications during treatment occurred relatively frequently, but with early recognition and appropriate treatment there was a good response to specific therapy. Respiratory complications included aspiration pneumonia, respiratory arrest and upper respiratory tract obstruction, conditions that are not unexpected in patients with laryngeal/pharyngeal dysfunction. One case suffered a coxofemoral luxation; its cause is not known, but the dog had particularly severe muscle spasms that were difficult to control and they may have contributed to the luxation. Vertebral and femoral neck fractures have been reported in the human literature (Bohrer 1965, Schweitzer and Naidoo 1972) as complications of tetanus but have not been described in the veterinary literature. No reports of joint luxations could be found in the medical literature. The risk of urinary tract infection increases with increasing duration of catheterisation, especially after three days (Smarick and others 2004); both cases that developed urinary tract infections had urinary catheters in place for more than seven days. Hiatal hernias have been reported as an uncommon complication of tetanus; other reported cases have had successful outcomes with medical management (Dieringer and Wolf 1991, van Ham and van Bree 1992, Acke and others 2004). The problem resolved completely in three of these dogs.

Twelve of the 13 dogs survived and were discharged. The other died six days after it was admitted, although it had been improving and was able to walk. It became hyperthermic (41°C) before it died but no other abnormalities were reported. A postmortem examination did not reveal the cause of death; there was no evidence of sepsis or infective processes. Dogs with tetanus should be monitored closely for hyperthermia because the increased muscle activity is a risk factor; in the event of hyperthermia the dog should be cooled quickly to prevent life-threatening sequelae.

The results of the treatment of these 13 dogs show that tetanus has a good prognosis for recovery to full function, although intensive nursing and care are required for a good outcome. Complications are likely to occur during its management with the most common being urinary tract infections and aspiration pneumonia. Careful monitoring makes it possible to identify these complications early and treat them successfully.

References

- ABRUTYN, E. & BERLIN, J. A. (1991) Intrathecal therapy in tetanus. A meta-analysis. *Journal of the American Medical Association* **266**, 2262-2267
- ACKE, E., JONES, B. R., BREATHNACH, R., MCALLISTER, H. & MOONEY, C. (2004) Tetanus in the dog: review and a case-report of concurrent tetanus with hiatal hernia. *Irish Veterinary Journal* **57**, 592-597
- AHMADSYAH, I. & SALIM, A. (1985) Treatment of tetanus: an open study to compare the efficacy of procaine penicillin and metronidazole. *British Medical Journal* **291**, 648-650
- BAGLEY, R. S., DOUGHERTY, S. A. & RANDOLPH, J. F. (1994) Tetanus subsequent to ovariohysterectomy in a dog. *Progress in Veterinary Neurology* **5**, 63-65
- BARK, H. (1980) Canine tetanus. *Canine Practice* **7**, 25-34
- BHADWAL, M. S. & WAZIR, V. S. (2005) Tetanus in a dog. *Indian Veterinary Journal* **82**, 446-447
- BLAKE, P. A., FELDMAN, R. A., BUCHANAN, T. M., BROOKS, G. F. & BENNETT, J. V. (1976) Serologic therapy of tetanus in the United States, 1965-1971. *Journal of the American Medical Association* **235**, 42-44
- BLECK, T. P. & BRAUNER, J. S. (1997) Tetanus. In *Infections of the Central Nervous System*. Eds W. M. Scheld, R. J. Whitley, D. T. Durack. 2nd edn. Philadelphia, Lippincourt-Raven. pp 629-653
- BOHRER, S. P. (1965) Spinal fractures in tetanus. *Radiology* **85**, 1111-1116
- DEWEY, C. W. (2003) Myopathies: disorders of skeletal muscle. In *A Practical Guide to Canine and Feline Neurology*. Ed C. W. Dewey. 1st edn. Ames, Iowa State University Press. pp 444-448
- DIERINGER, T. M. & WOLF, A. M. (1991) Esophageal hiatal hernia and megaesophagus complicating tetanus in two dogs. *Journal of the American Veterinary Medical Association* **199**, 87-89
- EDWARDS, G. T., EVANS, P. M. & EVANS, W. T. (1989) Tetanus in the dog. *Veterinary Record* **125**, 117
- FARRAR, J. J., YEN, L. M., COOK, T., FAIRWEATHER, N., BINH, N., PARRY, J. & PARRY, C. M. (2000) Tetanus. *Journal of Neurology and Neurosurgical Psychiatry* **69**, 292-301
- GANSSEBAUER, B., KRAMER, S., MAYER-LINDBERGEN, A. & NOLTE, I. (2000) Tetanus following ovariohysterectomy in a dog. *Tierärztliche Praxis* **28**, 225-229
- GREENE, C. E. (2005) Tetanus. In *Infectious Diseases of the Dog and Cat*. 3rd edn. Ed C. E. Greene. Missouri, Elsevier Saunders. pp 267-273
- HANSON, C. J. (1982) Tetanus in a dog: a case report. *Veterinary Record* **110**, 336-337
- HSU, S. & GROLEAU, G. (2001) Tetanus in the emergency department: a current review. *Journal of Emergency Medicine* **20**, 357-365
- OGDEN, J. D. (1983) Tetanus in a dog. *Veterinary Medicine and Small Animal Clinician* **78**, 1549-1550
- RATCLIFFE, J. (1989) Tetanus in a dog. *Veterinary Record* **124**, 666
- RUBIN, S., FAULKNER, R. T. & WARD, G. E. (1983) Tetanus following ovariohysterectomy in a dog: a case report and review. *Journal of the American Animal Hospital Association* **19**, 293-298
- SANFORD, J. (1995) Tetanus – forgotten but not gone. *New England Journal of Medicine* **332**, 812-813
- SCHWEITZER, G. & NAIDOO, K. S. (1972) Fracture of femoral neck in a patient suffering from tetanus. *South African Medical Journal* **46**, 503-504
- SMARICK, S. D., HASKINS, S. C., ALDRICH, J., FOLEY, J. E., KASS, P. H., FUDGE, M. & LING, G. V. (2004) Incidence of catheter-associated urinary tract infection among dogs in a small animal intensive care unit. *Journal of the American Veterinary Medical Association* **224**, 1936-1940
- SOUBASIS, N., KOUTINAS, A. F., SARIDOMICHELAKIS, M. N. & POLIZOPOULOU, Z. S. (2002) Tetanus in the dog: a study of 6 cases. *European Journal of Companion Animal Practice* **12**, 19-23
- STEIHM, E. R. (1979) Standard and special human immune serum globulin as therapeutic agents. *Pediatrics* **63**, 301-319
- STEINMAN, A., HAIK, R., ELAD, D. & SUTTON, G. A. (2000) Intrathecal administration of tetanus antitoxin to three cases of tetanus in horses. *Equine Veterinary Education* **12**, 237-240
- TOOLAN, D. P. (1989) A case of tetanus in the dog. *Irish Veterinary Journal* **42**, 83
- VAKIL, B. J., MEHTA, A. J. & TULPUL, T. H. (1964) Recurrent tetanus. *Postgraduate Medical Journal* **40**, 601-603
- VAN HAM, L. & VAN BREE, H. (1992) Conservative treatment of tetanus associated with hiatal hernia and gastro-oesophageal reflux. *Journal of Small Animal Practice* **33**, 289-294
- WURST, Z. & KOMAREK, J. (1987) Tetanus in dogs: occurrence and results of treatment in the period 1966-1985. *Veterinarstvi* **37**, 548-549