Jan. - Feb. 2026 Vol 4 Issue2

THE CAMPUS

we could be heroes!

The first and only vet. med. students' journal in Romania and probably the best in Europe

love is in the air!

THE CAMPUS

we could be heroes!

PUBLISHER

Ex Terra Aurum - USAMV Bucharest

SCIENTIFIC COMMITTEE

Dr. Emilia Ciobotaru
Dr. Iuliana Ionascu
Dr. Dan Cringanu
Dr. Gheorghe V. Goran

GENERAL COORDINATOR

Dr. Seralp Uzun

CHIEF EDITORS

Vet.Med.Stu. Giovanni Marciano
Vet.Med.Stu. Rădoi Ana-Maria

EDITORS

Vet.Med.Stu. Giovanni Marciano
Vet. Med. Stu. Irene Rita Romanelli
Vet. Med. Stu. Dinica Maria
Vet. Med. Stu. Sally Mustafa
Vet.Med.Stu. Assunta Piccolo
Vet.Med.Stu. Alessia delle Cave
Vet.Med.Stu. Rădoi Ana-Maria
Vet. Med. Stu. Blanche Nicolle
Vet. Med. Stu. Anne-Kristoffy Bluteau
Vet. Med. Stu. Daria-Ioana Marghidanu
Vet. Med. Stu. Rama Halabi
Vet. Med. Stu. Esther Cheron

THE CAMPUS
we could be heroes!

THE CAMPUS
we could be heroes... ABROAD!

BACK TO CAMPUS, BACK TO CARING

Dear students, veterinarians, professors, and animal lovers,

As we step into this new academic year, we return from the break with fresh energy, renewed curiosity, and a stronger commitment to our shared passion: veterinary medicine.
Whether you are a student, clinician, professor, or a dedicated pet owner visiting our hospital, we are excited to have you with us on this journey. Each new year brings its own challenges and opportunities, but it also offers a chance to grow, to question, and to rediscover why we chose to care for animals in the first place.

In the issues to come, you can look forward to topics that are just as spectacular and thought-provoking as ever: intriguing clinical cases, emerging research, ethical debates, stories from practice, and reflections from students finding their way in this demanding, yet deeply rewarding profession.

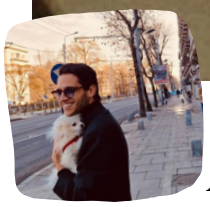
We wish you a year filled with inspiration, resilience, and meaningful learning experiences—both in the classroom and in the clinic. May this new beginning bring you confidence in your skills, compassion in your practice, and joy in every small step forward.
Welcome back, and let's make this year unforgettable—for us, for our patients, and for the profession we are building together.

The Editorial Team of The Campus.

The Campus

thecampus.vet

AFRICAN SWINE FEVER IN EUROPE: EPIDEMIOLOGICAL EVOLUTION AND EMERGING CHALLENGES



Article by Giovanni Marciano

Abstract

African Swine Fever (ASF) is a highly contagious viral disease affecting domestic pigs and wild boar, characterized by high mortality rates and severe socio-economic consequences. Although ASF is not zoonotic, its impact on animal health, food security, and international trade has made it one of the most significant transboundary animal diseases worldwide. Over the last two decades, the epidemiological scenario of ASF in Europe has changed dramatically, shifting from a limited and geographically confined disease to a widespread and persistent threat. This article provides an overview of the African Swine Fever Virus (ASFV), its biological characteristics and pathogenesis, and compares the epidemiological situation in Europe approximately twenty years ago with the current context, highlighting the emerging challenges for disease control and prevention.

Introduction

African Swine Fever (ASF) is a viral disease of suids characterized by high mortality rates and the absence of effective vaccines or treatments (Dixon et al., 2005). Originally described in Africa in the early 20th century, ASF remained confined to specific geographic regions for decades (Penrith et al., 2013).
In Europe, ASF was considered a largely controllable disease until the early 2000s. However, since the introduction of ASFV into Eastern Europe in 2007, the disease has progressively expanded, becoming endemic in several regions and involving both domestic pigs and wild boar populations (Sánchez-Vizcaíno et al., 2015).

This shift has transformed ASF into a major challenge for European animal health systems.

African Swine Fever Virus: Virology and Biological Characteristics

African Swine Fever Virus (ASFV) is a large, enveloped, double-stranded DNA virus belonging to the family Asfarviridae and the genus Asfivirus (Dixon et al., 2005). It is the only known DNA arbovirus, capable of infecting both vertebrate hosts and soft ticks of the genus Ornithodoros.
The virus exhibits remarkable environmental resistance, surviving for extended periods in blood, tissues, carcasses, and pork products (EFSA, 2023). More than 20 genotypes of ASFV have been described, with genotype II being responsible for the current European outbreaks (Gallardo et al., 2014).
ASFV primarily targets monocytes and macrophages, which play a central role in the host immune response (Sánchez-Cordón et al., 2018).

Pathogenesis of African Swine Fever

The pathogenesis of ASF depends on viral virulence, infectious dose, and host susceptibility (Tulman et al., 2009). Following infection, usually through the oral or nasal route, ASFV initially replicates in the tonsils and regional lymph nodes before spreading systemically via the bloodstream.

Key pathogenic mechanisms include:

- Replication in macrophages, leading to immune dysfunction
- Excessive pro-inflammatory cytokine release, contributing to systemic inflammation
- Vascular damage and hemorrhages, associated with disseminated intravascular coagulation

Highly virulent strains cause acute or peracute disease, characterized by high fever, anorexia, hemorrhages, and mortality rates close to 100% (Sánchez-Cordón et al., 2018).

Transmission and Epidemiology

ASFV transmission occurs through:

- Direct contact between infected and susceptible animals
- Indirect contact via contaminated fomites
- Ingestion of contaminated pork products
- Contact with infected wild boar or carcasses

Wild boar play a crucial role in the maintenance and spread of ASF in Europe, acting as a reservoir and significantly complicating eradication efforts (EFSA et al., 2018).

ASF in Europe Twenty Years Ago

Approximately twenty years ago, ASF in Europe was largely limited and geographically contained. The disease was mainly restricted to:

- Sardinia, where ASF had been endemic for decades due to traditional pig-farming practices (Mur et al., 2014)
- Historical outbreaks in Western Europe, including Spain and Portugal, which had been successfully eradicated by the mid-1990s (Arias et al., 2002)

At that time, ASF was considered a regional problem with limited involvement of wild boar and minimal transboundary spread (Sánchez-Vizcaíno et al., 2013).

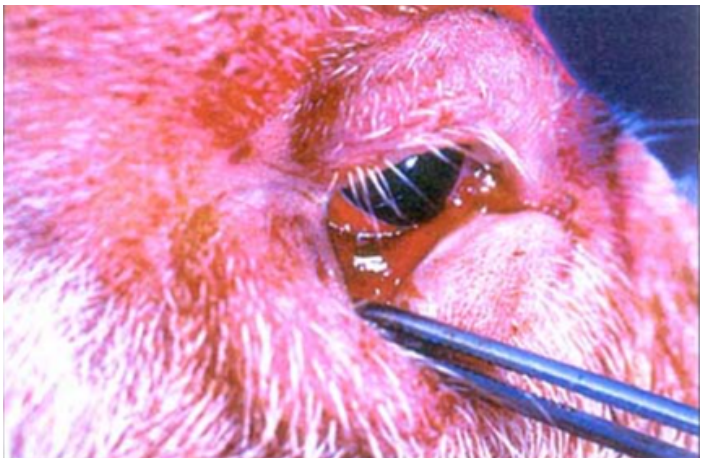
Current Epidemiological Situation in Europe

The current epidemiological scenario of ASF in Europe is markedly different. Since the introduction of ASFV genotype II into Georgia in 2007, the virus has spread progressively across Eastern, Central, and parts of Western Europe (Rowlands et al., 2008).

Key features of the present situation include:

- Endemic circulation in wild boar populations
- Recurrent outbreaks in domestic pig farms
- Persistent environmental contamination
- Significant economic losses due to culling and trade restrictions

Despite a recent decrease in outbreaks in domestic pigs in some EU countries, ASF remains firmly established in the European ecosystem (EFSA, 2024).



Congested ocular mucosa
The mucous membranes of the eyes of this pig are deeply congested, and some of the redness may be due to haemorrhage.

Source: www.fao.org

Comparison Between Past and Present Scenarios

Compared with the situation twenty years ago, ASF in Europe has evolved from:

- A localized and controllable disease
- to
- A complex transboundary animal disease involving wildlife reservoirs and long-term persistence

This evolution highlights the increasing difficulty of controlling ASF using traditional eradication measures alone (Sánchez-Vizcaíno et al., 2015).

Current Challenges and Future Perspectives

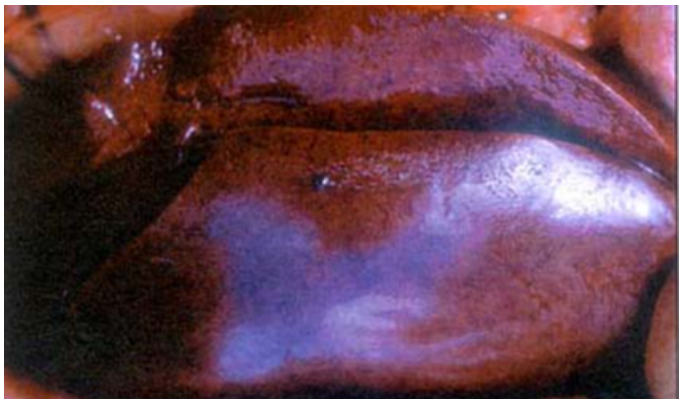
The control of African Swine Fever in Europe remains a complex challenge that goes beyond traditional disease management. A major limiting factor is the role of wild boar populations, which act as a persistent reservoir of ASFV and contribute to long-term environmental contamination (EFSA et al., 2018). The survival of the virus in carcasses and the difficulty of applying biosecurity measures in wildlife significantly hinder eradication efforts.

Another critical issue is the heterogeneity of pig production systems across Europe. While commercial farms often maintain high biosecurity standards, small-scale and backyard farms remain particularly vulnerable to ASF introduction and may facilitate transmission between wildlife and domestic pigs (Mur et al., 2016).

Human-mediated spread continues to play a central role in ASF epidemiology. The movement of contaminated vehicles, equipment, feed, and pork products represents a significant risk factor for long-distance dissemination of the virus (EFSA, 2024). In this context, compliance with biosecurity measures and effective risk communication are essential.

Early detection through passive surveillance, especially the testing of wild boar found dead, remains one of the most effective tools for ASF control (EFSA et al., 2018). However, surveillance systems require sustained investment and international coordination.

Finally, the absence of an effective vaccine continues to limit control strategies. Despite ongoing research, ASFV immune evasion and genetic complexity hinder vaccine development, making prevention and long-term management the primary control options at present (Rock, 2017).



Haemorrhages in kidneys
Pinpoint to larger haemorrhages on the kidney capsule.

Source: www.fao.org

Close-up of flushed/cyanotic skin
In white-skinned pigs, the ears, tail, legs and underside appear deeply flushed and may develop a bluish tinge (cyanosis).

Source: www.fao.org

Peracute ASF
In the peracute form of the disease, death may occur before any clinical signs appear.

Source: www.fao.org

Enlarged spleen
The spleen is often markedly enlarged and dark in colour.

Source: www.fao.org

Conclusions

Over the last two decades, African Swine Fever has evolved from a geographically limited and largely controllable disease into one of the most significant transboundary animal health threats facing Europe. The progressive spread of African Swine Fever Virus across the continent has profoundly altered the epidemiological landscape, revealing the vulnerability of European pig production systems to highly resilient and complex pathogens.

This review emphasizes the importance of understanding the biological characteristics and pathogenesis of ASFV in order to fully interpret its epidemiological behavior. The virus's capacity to infect and disrupt key immune cells, induce severe systemic inflammation, and persist in the environment provides a biological basis for its high transmissibility and persistence. These features help explain why ASF has proven exceptionally difficult to eradicate once established, particularly in regions with dense wild boar populations.

The comparison between the European ASF situation twenty years ago and the current scenario highlights a clear transition from sporadic and localized outbreaks to a state of long-term endemicity in several areas. The involvement of wildlife reservoirs, combined with human-mediated transmission and heterogeneous farming systems, has transformed ASF into a disease that cannot be addressed through emergency responses alone. Instead, it requires sustained and adaptive management strategies.

From a control perspective, the absence of an effective vaccine continues to represent a major limitation. While vaccine development remains a critical long-term objective, current efforts must prioritize prevention, early detection, and strict biosecurity measures. Passive surveillance, particularly in wild boar populations, remains a cornerstone of early warning systems and should be strengthened through coordinated European initiatives.

Ultimately, African Swine Fever should be regarded not only as a veterinary health issue, but also as a broader challenge affecting food security, rural economies, and international trade. Addressing ASF in Europe will require long-term political commitment, harmonized surveillance and control policies, and close collaboration between scientists, veterinary authorities, farmers, and wildlife managers. Only through an integrated, science-based approach can Europe hope to mitigate the impact of ASF and enhance the resilience of its pig production sector in the years to come.

References

1. *Arias, M., Sánchez-Vizcaíno, J. M., & Morilla, A. (2002). African swine fever eradication: The Spanish model. Trends in Emerging Viral Infections of Swine, 133–148.*
2. *Dixon, L. K., Escribano, J. M., Martins, C., Rock, D. L., Salas, M. L., & Wilkinson, P. J. (2005). Asfarviridae. In Virus Taxonomy (pp. 135–143). Elsevier.*
3. *EFSA, AHAW Panel. (2018). African swine fever in wild boar. EFSA Journal, 16(7), 5344.*
4. *EFSA. (2023). Epidemiological analyses of African swine fever in the European Union. EFSA Journal, 21(5), e07818.*
5. *EFSA. (2024). African swine fever overview and epidemiological update. EFSA Journal, 22(2), e08321.*
6. *Gallardo, C., et al. (2014). African swine fever virus genotype II in Europe. Transboundary and Emerging Diseases, 61(4), 344–353.*
7. *Mur, L., et al. (2014). Thirty-five-year presence of African swine fever in Sardinia. Transboundary and Emerging Diseases, 61(4), 314–321.*
8. *Penrith, M. L., Vosloo, W., & Jori, F. (2013). African swine fever virus eradication in Africa. Virus Research, 173(1), 228–246.*
9. *Rock, D. L. (2017). Challenges for African swine fever vaccine development. Journal of General Virology, 98(12), 2859–2870.*
10. *Rowlands, R. J., et al. (2008). African swine fever virus isolate Georgia 2007. Emerging Infectious Diseases, 14(12), 1870–1874.*
11. *Sánchez-Cordón, P. J., Montoya, M., Reis, A. L., & Dixon, L. K. (2018). African swine fever: A re-emerging viral disease. Veterinary Research, 49, 71.*
12. *Sánchez-Vizcaíno, J. M., Mur, L., & Martínez-López, B. (2013). African swine fever: An epidemiological update. Transboundary and Emerging Diseases, 59(s1), 27–35.*
13. *Sánchez-Vizcaíno, J. M., Mur, L., & Martínez-López, B. (2015). African swine fever control strategies in Europe. Veterinary Microbiology, 165(1–2), 1–6.*
14. *Tulman, E. R., et al. (2009). ASFV pathogenesis and immune evasion. Virus Research, 173(1), 14–22.*

SILENT BLEEDERS: UNDERSTANDING VON WILLEBRAND DISEASE IN DOGS

Article by Irene Romanelli

Introduction

Von Willebrand disease (vWD) may sound like a complicated medical term, but for many dog owners, it's a very real part of their pet's health journey. It is considered the most common inherited bleeding disorder in dogs, and this condition silently affects a wide range of breeds. Some dogs live with it their whole lives without any issues, while others experience unexpected bleeding episodes that can be frightening for both pet and owner. Understanding how the disease works, how to recognize it, and how to manage it it's very important, in order to give an affected dog a safe, happy, and comfortable life.

What Exactly Is von Willebrand Disease?

At its core, von Willebrand disease is a disorder of blood clotting. When a blood vessel is injured (even by something small, like trimming a nail too short), the body relies on a protein called von Willebrand factor (vWF) to help platelets stick to the damaged area and form an initial clot. If vWF is missing, too low, or not working properly, a dog may bleed longer than normal.
There are three main types of vWD, each with its own level of severity:

- Type I – The dog produces vWF, but not enough of it. This is the most common and usually mild form. Many affected dogs live normal lives without obvious symptoms. DOBERMAN PINSCHERS are especially well known for this type, but many other breeds can be affected.
- Type II – The dog produces vWF, but the protein is abnormal and doesn't function as it should. This type tends to be more severe.
- Type III – The dog produces almost no vWF at all. This is the rarest and most serious form, and dogs can experience significant, spontaneous bleeding episodes.

Although any dog can theoretically inherit vWD, certain breeds have a much higher likelihood of carrying or expressing the gene. Breeds commonly associated with vWD include:

- Doberman Pinscher
- Standard and Miniature Poodle
- Shetland Sheepdog
- Scottish Terrier
- Chesapeake Bay Retriever
- Corgi
- German Shorthaired and Wirehaired Pointer
- Manchester Terrier
- Golden Retriever
- Bernese Mountain Dog

Mixed-breed dogs can also inherit the condition if both parents carry the affected gene.

How to Recognize the Symptoms

Some dogs with vWD never show noticeable signs, while others experience regular or severe bleeding events. Common signs include:

- Prolonged bleeding from even tiny cuts
- Longer than normal bleeding after surgery or dental cleanings
- Nosebleeds without clear cause
- Bleeding gums, especially when chewing toys
- Bruising under the skin (hematomas)
- Blood in urine or stool
- Excessive bleeding during heat cycles or after whelping in females

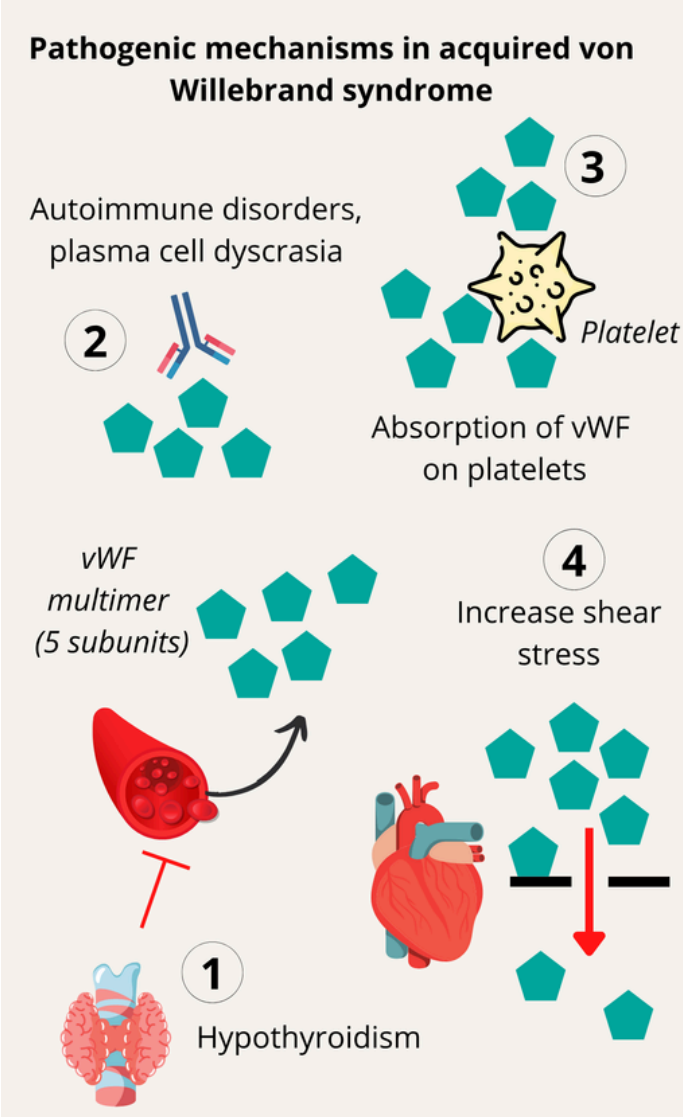
Symptoms can also appear suddenly during times of stress, illness, or heat cycles, which temporarily lower vWF levels.

Diagnosis vWD

Diagnosis of von Willebrand disease relies on several complementary tests. Buccal Mucosal Bleeding Time evaluates how long a small oral cut takes to clot. The vWF antigen test measures the amount of von Willebrand factor circulating in the blood. Genetic testing, performed through a cheek swab or blood sample, identifies whether a dog is clear, a carrier, or affected. Standard coagulation panels help exclude other causes of abnormal bleeding. Genetic screening is particularly useful for breeders and for dogs undergoing procedures such as spaying, neutering, or dental surgery.

Managing and Treating von Willebrand Disease

Most dogs with von Willebrand disease can live normal, active lives when the condition is managed correctly. Treatment focuses on reducing bleeding risks and supporting clotting when needed. Desmopressin can temporarily boost von Willebrand factor, especially in Type I cases, and is commonly used before surgery. In emergencies or major procedures, plasma or cryoprecipitate transfusions supply essential clotting factors. Certain drugs—such as aspirin and some NSAIDs—should be avoided because they can worsen bleeding. In more severe cases, lifestyle adjustments like limiting rough play and monitoring for unexpected bleeding help keep dogs safe. Careful planning before any surgical or dental procedure remains essential to prevent complications.



Genetics and Breeding Considerations

Because von Willebrand disease is inherited, thoughtful breeding choices play a key role in reducing its impact. DNA testing distinguishes clear dogs, carriers that show no signs but can transmit the gene, and affected dogs. Using these results, breeders can prevent high-risk pairings and steadily decrease the frequency of the mutation in their breeding lines.

Life of a Dog with vWD

Even though von Willebrand disease can sound alarming, many dogs with mild to moderate forms lead completely normal lives. With awareness, preparation, and a good relationship with your veterinarian, you can manage the condition effectively.

CYSTOTOMY IN DOGS AND CATS: INDICATIONS, SURGICAL TECHNIQUE, AND POSTOPERATIVE MANAGEMENT

Article by Esther Cheron

Cystotomy is a routine soft-tissue surgery in dogs and cats, mainly used to remove uroliths when medical therapy fails. It is also indicated for bladder trauma, tumors, foreign bodies, and chronic lower urinary tract disease. The procedure and its key steps, risks, and postoperative care are reviewed in this article.

Introduction

Soft tissue surgery plays a crucial role in veterinary medicine, particularly in the management of abdominal and urinary tract disorders. Among these procedures, cystotomy is one of the most frequently performed surgeries in small animal practice. It is considered a fundamental surgical technique for both veterinary students and practitioners.

In dogs and cats, cystotomy is most commonly indicated for the removal of urinary bladder uroliths. Although medical dissolution and dietary management are preferred whenever possible, surgical intervention becomes necessary when conservative treatments fail or when complications such as urinary obstruction, recurrent infections, or bladder trauma occur. When performed correctly, cystotomy is associated with a high success rate and a favorable prognosis.

Indications for Cystotomy

Cystotomy is indicated in a variety of urinary bladder conditions in dogs and cats. The most common indications include:

- Removal of uroliths that cannot be medically dissolved
- Chronic or recurrent lower urinary tract disease
- Urinary bladder rupture or penetrating trauma
- Removal of blood clots or foreign material
- Collection of full-thickness bladder biopsies
- Suspected bladder neoplasia

In cats, cystotomy is often associated with obstructive lower urinary tract disease, while in dogs it is frequently performed due to urolithiasis. A thorough diagnostic workup is essential to determine whether surgical intervention is necessary and to rule out alternative treatment options.

Surgical Anatomy and Preoperative Consideration

The urinary bladder is a hollow muscular organ with four layers. The neck and trigone are very sensitive and must be avoided surgically to preserve continence.

Preoperative Evaluation

Before performing a cystotomy, a complete clinical evaluation is required, including:

- Physical examination
- Urinalysis and urine culture
- Abdominal radiography and/or ultrasonography
- Complete blood count and serum biochemistry

Patients with urinary obstruction or electrolyte imbalances should be stabilized prior to surgery. Appropriate antimicrobial therapy should be selected based on culture and sensitivity results whenever possible.

Surgical Technique

1. Surgical Approach

Cystotomy is performed under general anesthesia using a ventral midline celiotomy approach.

The urinary bladder is gently exteriorized and isolated from the abdominal cavity using moistened laparotomy sponges to prevent contamination.

2. Bladder Incision

A stab incision is made on the ventral surface of the bladder body, away from the trigone. This location minimizes hemorrhage and reduces the risk of postoperative complications. The incision is extended as needed to allow adequate visualization of the bladder lumen.

3. Removal of Bladder Contents

Uroliths or foreign material are carefully removed using atraumatic forceps. The bladder and urethra should be flushed retrograde with sterile saline to ensure complete removal of residual stones or debris.

4. Bladder Closure

The bladder is closed using a single-layer appositional or inverting suture pattern with an absorbable monofilament suture material. A leak test is performed by injecting sterile saline into the bladder before abdominal closure. The abdominal wall is closed routinely.

Postoperative Management

Postoperative care is essential to ensure proper healing and prevent complications.
Key aspects include:
• Analgesia using opioids and non-steroidal anti-inflammatory drugs
• Antibiotic therapy based on culture and sensitivity results
• Monitoring urine output and signs of dysuria or hematuria
• Temporary urinary catheterization if indicated
Most dogs and cats regain normal urinary function within a few days following surgery.

Complications

Although cystotomy is generally safe, potential complications may occur, including:
• Urine leakage and uroabdomen
• Postoperative hemorrhage
• Surgical site infection
• Residual or recurrent urolithiasis
• Urethral obstruction

Careful surgical technique, thorough bladder flushing, and appropriate postoperative monitoring significantly reduce the risk of complications.

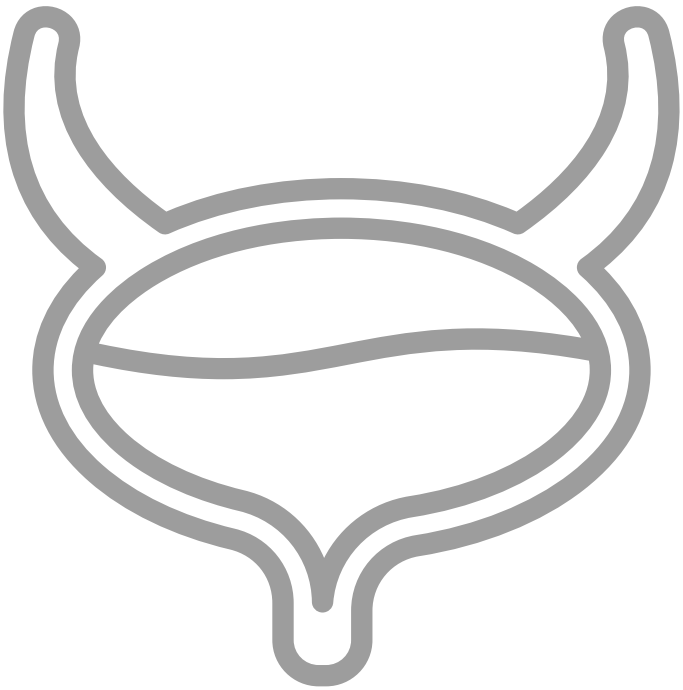
Prognosis

The prognosis following cystotomy in dogs and cats is excellent in most cases. Long-term success largely depends on identifying and addressing the underlying cause of the urinary disorder, particularly in patients with recurrent urolithiasis. Dietary management and regular follow-up are essential to prevent recurrence.

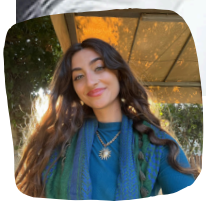
Conclusion

Cystotomy remains a cornerstone procedure in small animal soft tissue surgery. When properly indicated and performed, it is a safe and effective method for managing urinary bladder disorders in dogs and cats. A solid understanding of bladder anatomy, meticulous surgical technique, and comprehensive postoperative care are essential for optimal outcomes.

References :
1. Fossum, T. W. (2019). Small Animal Surgery (5th ed.). Elsevier.
2. Birchard, S. J., & Sherding, R. G. (2016). Saunders Manual of Small Animal Practice. Elsevier.
3. Osborne, C. A., Lulich, J. P., & Kruger, J. M. (2009). Medical and surgical management of urolithiasis. Veterinary Clinics of North America: Small Animal Practice, 39(3), 509–528.
4. Tobias, K. M., & Johnston, S. A. (2018). Veterinary Surgery: Small Animal. Elsevier.



FROM VILLAGE PRIDE TO GLOBAL RECOGNITION



Article by Rama Halabi

The Straight Egyptian Royal World Cup 2025 is an international event dedicated to Straight Egyptian Arabian horses.
While it is sometimes describedas beauty focused or status driven, the event also reflects long-standing relationships between humans and horses shaped by care, responsibility, and shared history.
This article connectsthe Royal World Cup through the story of Al Waheeb AA, whose victory in 2025 carried both professional significance and deep meaning for his community.
By focusing on his upbringing, pedigree and cultural context, the article is meant to show why such competitions remain relevant for veterinary practice and for understanding the historical role of horses in West Asia and the wider MENA region.

Introduction

Horse competitions oftenprovoke mixed responses. To some observers, they represent dedication, tradition, and excellence. To others, they appear as beauty pageants shaped by prestige, external validation and possibly exploitation. These critiques are valid, particularly when competitions are viewed only through their public presentation.
Yet such events also reveal how horses are raised,trained and cared for over long periodsof time. What is rewarded in the arena is directly influenced by everyday practices in training and veterinary care far beyond a single competition. For this reason, events such as the Straight Egyptian Royal WorldCup matter not only culturally, but also in ways directly related to animal health and welfare.
Born in 2019 at Al Halabi Studs,Al Waheeb AA's success did not emerge suddenly. It was the outcome of years of care and decisions extending from his home to an international stage.

Before Al WaheebAA's victory was announced officially, it was already known in Daliyatel Karmel. I first heard about it through my father, as the news moved through every day conversations within the community. The way people spoke about it made clear that this was not experienced as an individual achievement, but as shared pride.

In Palestine, horses are part of lived experience. Knowledge about care, patience and responsibility is passed on through observation and daily practice rather than formal instruction. Horses are understood as beings that require time, consistency and respect. This approach shaped Al Waheeb AA long before he entered an international arena.

Horses have long been part of Palestine's history. In Daliyat el Karmel, my great-grandfather kept horses, and I grew up on stories about them—stories of community, resilience, and care that shaped my father's and uncles' childhoods.
For many in the community, his victory felt like recognition of years of quiet work that usually remains unseen, these competitions are by no means the only measure of greatness yet moments like these offer acknowledgment for care, commitment and values.

Lineage and memory

Al Waheeb AA's pedigree is often mentioned in professional contexts, particularly his connection to Al Ayal AA. Within the community and among breeders, lineage is not only about records or rankings. It functions as a form of memory, connecting horses across time through shared qualities and stories.
Names such as Algmara Al-Sheikh, Birwaz Al-Bustan and Rawaf Al-Bidayer are remembered in similar ways and at more advanced levels for some.

These horses are valued not only for titles but for balance, presence and reliability. Mentioning them situates Al Waheeb AA within a broader narrative shapedby many hands and many years. These horses are not just for show but rather apart of a larger family. He is not the first horse from the country to be internationally recognised but his story is what reminded me of our shared love of horses that connects people from different backgrounds of the country who would've otherwise never met.

Time, care, and veterinary relevance

Al Waheeb AA has been under structured care since 2019 by Al Halabi stud.From a veterinary perspective, this time frame is central. Horses expected to perform at elite levels cannot be rushed without cost. Physical development, mental stabilityand resilience depend on gradual progress and consistent care.

His upbringing involved attention to feeding, regular health monitoring and training that respected his pace. These choices reduce injury risk, support long-term soundness and allow the horse to remain calm and responsive in demanding situations.

For veterinary practice, this is wheret he relevance of such competitions becomes clear. They reflect the consequences of everyday decisions about care long before a horse enters the arena.

It is easy to reduce events like the Royal World Cup to appearance and prestige. Yet what is valued publicly often shapes what is practiced privately. When calmness, harmony and balance are rewarded, they encourage approaches that support animal wellbeing.

Horses, History,and West Asia

Horses have held a central place in the societies of West Asia and the wider MENA region for centuries. They were bred within communities that relied on them for movement, work and survival. Endurance, adaptability and trust mattered long before modern competitions existed.

Remembering this history places contemporary events within a longer story. Al Waheeb AA's journey connects a village, shared cultural memory and a global stage without separating one from the other.

Conclusion

His journey offers something deeper than a competitive title. His path reflects the slow and steady work that happens far from arenas.

For me, his story is less about the outcome of a single event and more about what it reveals: how veterinary care, cultural memory and commitment can intersect in the life of one horse. It invites reflection on the long histories and everyday practices that sustain horses in West Asia. Competitions may offer visibility but the real meaning lies in the human and animal relationships that exist regardless of recognition. Al Waheeb AA's presence in the arena simply made visible what his community already knew.

A DROP OF DANGER: PERMETHRIN TOXICITY IN CATS

Article by Blanche Nicolle

This article addresses the often overlooked risk of permethrin-based spot-on flea treatments for dogs in households with cats. While safe for dogs, permethrin is highly toxic to cats due to their hepatic metabolism. We review exposure scenarios, clinical signs from tremors to seizures, emergency management, and stress the importance of rapid treatment and owner education to prevent fatal outcomes.

Figure 1- source : original

A Hidden Hazardin Our Homes

Many pet owners, striving to protect their beloved canine companions from fleas and ticks, unwittingly introduce a grave danger to their feline household members. Permethrin, a synthetic pyrethroid commonly found in dog-specific flea and tick spot-on treatments, is a potent neurotoxin for cats.

While dogs possess the enzymatic pathways to safely metabolize permethrin, cats lack sufficient glucuronosyltransferase enzymes, leading to prolonged exposure of the nervous system to the toxin. This critical metabolic difference is often misunderstood by the public, making permethrin toxicosis a tragically common and preventable veterinary emergency. This article aims to raise awareness among pet owners, veterinary students, and practitioners about the critical threat of permethrin toxicosis in cats, focusing on prevention, recognition, and emergency management.

The Permethrin Paradox:Why Cats Are Uniquely Vulnerable

Permethrin belongs to the pyrethroid class of insecticides, which work by disrupting sodium channel function in insect neurons, leading to hyperexcitability and paralysis. In mammals, these compounds are typically rapidly metabolized and excreted.

The vulnerability of cats stems from a metabolic limitation known as glucuronidation deficiency, a crucial detoxification pathway in the liver. This deficiency means that permethrin remains in its toxic, active form within the feline system for an extended period, accumulating to harmful levels and causing severe neurotoxic effects.

Common Exposure Scenarios:

- Direct Application: Accidental application of dog permethrin products directly onto a cat.
- Secondary Exposure: Cats grooming or cuddling with a recently treated dog, absorbing permethrin through skin contact or ingestion of residues. This is the most common route of exposure.

- Environmental Contamination: Cats coming into contact with bedding, furniture, or carpets

where a recently treated dog has rested.

Clinical Signs: Recognizing the Emergency

The onset of clinicalsigns can range from minutesto several hours post-exposure, depending on the dose and route of exposure. The signs are primarily neurological, reflecting permethrin's direct neurotoxic action.

Other signs may include hyperesthesia (increased sensitivity to touchor sound), vomiting, diarrhea, dyspnea (difficulty breathing), and ultimately, coma and death if left untreated.

Emergency Management: Every Minute Counts

Permethrin toxicosis is a veterinary emergency. Immediate, aggressive action is crucial for a positive outcome.

Clinical Sign	Description	Severity
Muscle Tremors	Fasciculations, especially around the face and head, progressing to whole-body tremors.	Moderate to Severe
Hypersalivation	Excessive drooling.	Mild to Moderate
Ataxia	Incoordination, wobbly gait.	Moderate
Seizures	Generalized tonic-clonic seizures, a severe and life-threatening manifestation.	Severe
Hyperthermia	Elevated body temperature (>40.5 °C) from sustained muscle activity.	Severe
Depression/Weakness	Lethargy, inability to stand or move normally.	Variable

Initial Steps for Pet Owners (Before reaching the vet)

Owners should seek immediate veterinary care if exposure is suspected; before transport, these steps are crucial:

1. Decontaminate: Immediately and thoroughly wash the cat with a mild dish soap (e.g., Dawn) in lukewarm water. Multiple washes may be necessary. Wear gloves to protect yourself.
2. Rinse and Warm: Rinse the cat thoroughly to remove all soap and residue. Keep the cat warm after washing to prevent hypothermia, which can worsen the prognosis.
3. Transport: Transport the cat to the nearest veterinary clinic as quickly as possible, bringing the suspected product packaging for identification.
4. Do NOT: Induce vomiting or apply any "home remedies."

Veterinary Protocols: Triage and Treatment

On arrival, rapid assessment and treatment are essential, focusing on decontamination, neurological control, and supportive care.

Stabilization and Decontamination

- Triage (ABC): Ensure Airway, Breathing, Circulation are maintained. Administer oxygen if the cat is dyspneic.
- IV Catheter: Establish intravenous access for fluid administration and medication.

Thorough bathing: Immediately wash the cat with mild dish soap to reduce absorption; repeat if needed, and carefully clip matted fur if necessary.

Control of Neurological Signs

- Muscle Tremors/Seizures:

○Methocarbamol: The first-line muscle relaxant for tremors (50-150mg/ kg IV slowly, to effect)
○ Benzodiazepines (Diazepam or Midazolam): Effective for controlling seizures(0.5-1 mg/kg IV)
○ Refractory Seizures: May require more potent agents like Propofol (Constant Rate Infusion, CRI) or Phenobarbital.

- Hyperthermia: Activelycool the cat if the body temperature is excessively high (e.g., cool IV fluids, fan, cool water enemas)

Supportive Care

- Fluid Therapy: Administer intravenous fluid therapy to maintain hydration, support renal function, and help with toxin elimination.
- Thermoregulation: Maintain normothermia post-bathing.
- Monitoring: Continuously monitorvital signs (heart rate, respiratory rate, temperature), neurological status, blood pressure, and bloodwork (electrolytes, kidney/liver values).
- Nursing Care: Provide nutritional support (if recumbent), manage the bladder (expression or catheterization), and lubricate the eyes to prevent corneal drying.

Prognosis and Prevention: A Call to Action

Prognosis varies with dose, speed of decontamination, and supportive care. Prompt, aggressive treatment can lead to full recovery, though severe cases with uncontrolled seizures may be fatal. Prevention relies primarily on owner education.

Key Prevention Messages:

- "Read the Label!": Emphasize that dog productsare NOT for cats. Highlight the "FOR DOGS ONLY" warning.
- Separate Animals: Ifa dog is treated with a permethrin-containing product, keep the dog and cat separated for a minimum of 72 hours (or as per product instructions).
- Avoid Contact: Prevent cats from sleeping with or grooming recently treated dogs.
- Consult Your Vet: Always consult a veterinarian for appropriate, cat-safeflea and tick control products for ALL household pets.

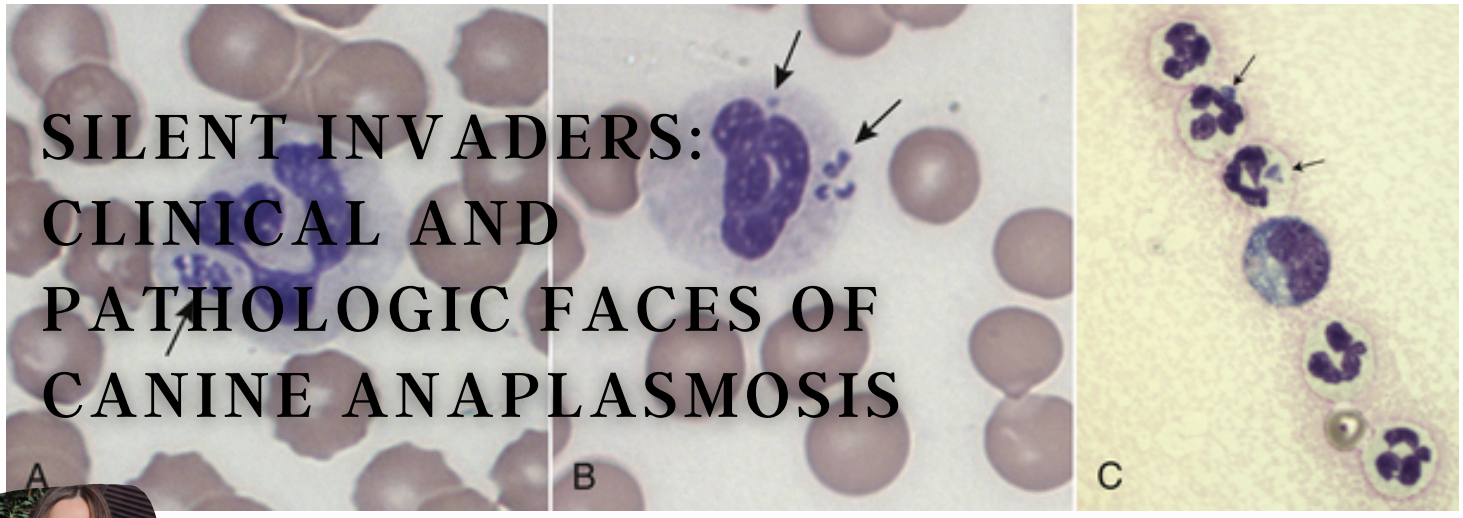
Conclusion: Protecting Our Feline Friends

Permethrin toxicosis is a serious yet preventable threat to cats. Understanding their metabolic vulnerability, recognizing clinical signs, and acting promptly are key to protection.

Immediate, aggressive veterinary care can greatly improve outcomes, but prevention through public and professional education remains essential, ensuring pet owners recognize “DOGS ONLY” warnings and choose safe parasite control for all animals.

References :

1.Di Pietro S. et al. (2022) Treatment of Permethrin Toxicosis in Cats by Intravenous Lipid Emulsion.
2.Linett P-J. (2008)Permethrin toxicosis in cats.
3.Volmer P. et al. (1998)Warning against use of some permethrin products in cats.
4.Malik R. et al. (2017)Permethrin Spot-On Intoxication of Cats.
5.Daza M.A. (2004).Most common poisonings in small animals.



SILENT INVADERS: CLINICAL AND PATHOLOGIC FACES OF CANINE ANAPLASMOSIS

Article by Rădoi Ana-Maria

Canine anaplasmosis is a tick-borne disease caused mainly by Anaplasma phagocytophilum and A. platys, obligate intracellular bacteria that infect neutrophils and platelets, respectively, explaining the associated clinical and hematologic findings.

Figure 1- source : todaysveterinarypractice.com/parasitology/diagnosing-and-managing-anaplasma-infection-in-dogs/

Following inoculation via tick bite, the organisms invade target cells and form intracytoplasmic morulae, sometimes visible on blood smears.

A. phagocytophilum infection of neutrophils leads to immune dysfunction, systemic inflammation, and mild-to-moderate vasculitis.

A. platys induces cyclic thrombocytopenia with recurrent episodes of decreased platelet counts. Systemic inflammation and vascular involvement may result in arthritis, polyarthritis, and, less commonly, neurologic or ocular signs.

Clinical signs

Many infections are subclinical. When disease is apparent, clinical signs are typically acute and nonspecific:

- fever,
- lethargy,
- inappetence,
- weight loss,
- lymphadenomegaly,
- splenomegaly,
- stiffness,
- shifting leg lameness,
- joint pain,
- occasionally vomiting and diarrhea.

Thrombocytopenia can cause bruising, petechiae, nosebleeds, or other hemorrhages. Severe cases or co-infections may lead to neurologic signs, uveitis, edema, or respiratory distress.

A. platys infections more often present with mild-to-moderate recurrent hemorrhagic tendencies.

Diagnosis (clinical and laboratory)

Diagnosis is based on:

- history (tick exposure, season, endemic area);
- physical exam (fever, joint pain, lymphadenomegaly, bleeding);
- CBC: thrombocytopenia is common; mild anemia, leukocytosis or leukopenia may be present;
- blood smear: morulae in neutrophils (A. phagocytophilum) or platelets (A. platys) – limited sensitivity;
- rapid serologic assays (ELISA SNAP-type) – detect antibodies;

- PCR – reference method for confirmation and speciation .

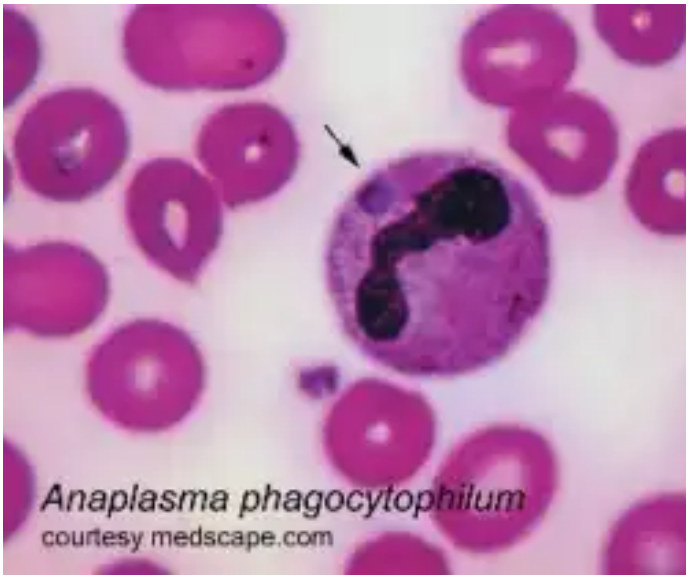


Figure 2- source : www.vetmed.auburn.edu/academic-departments/dept-of-pathobiology/diagnostic-services/molecular-diagnostics/anaplasmosis-canine-feline/

Treatment

Doxycycline is the treatment of choice, usually (once daily or divided twice daily) for 14–28 days . Most dogs show marked clinical improvement within 24–48 hours. In severe cases, additional supportive care is indicated:

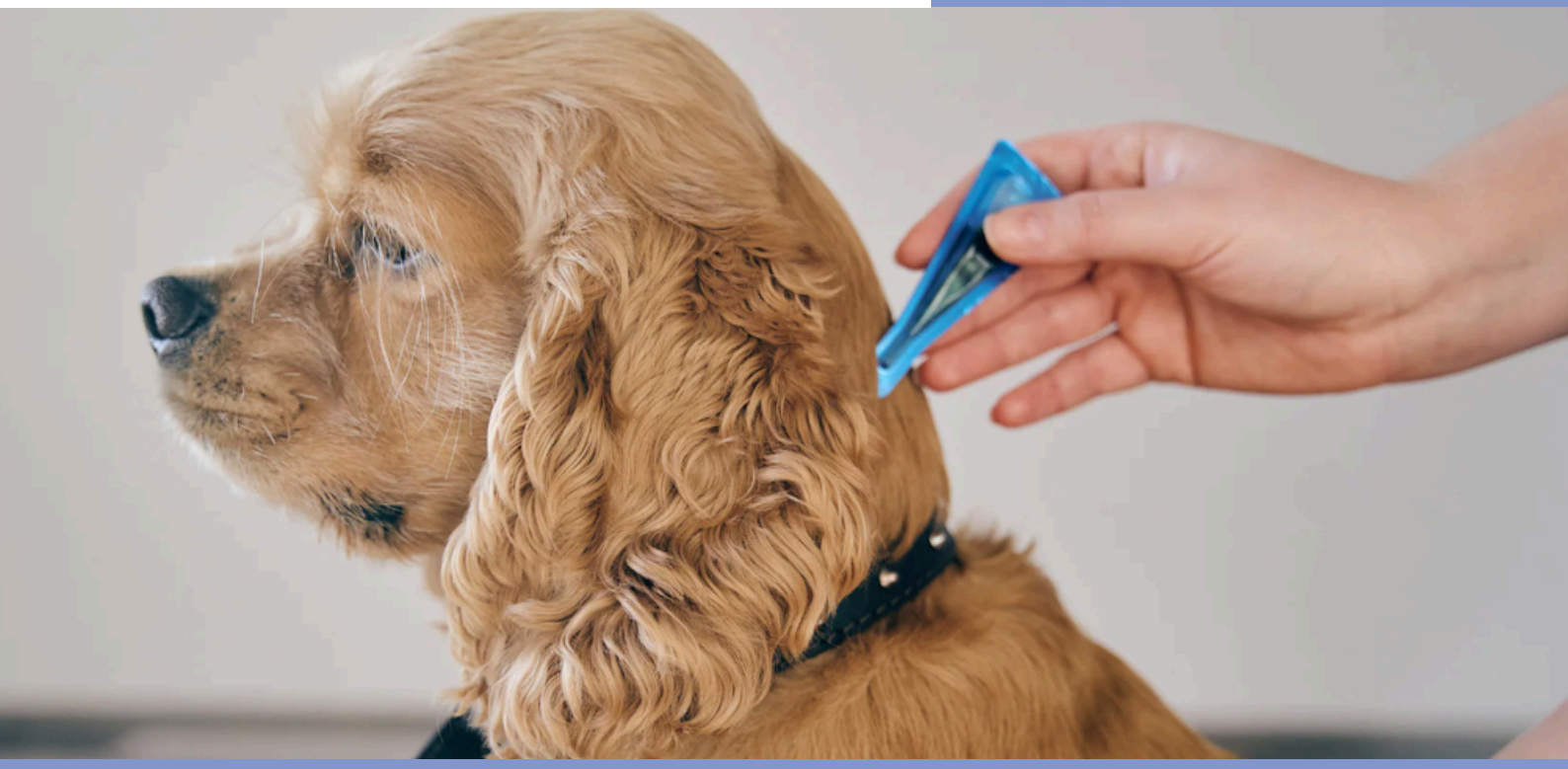
- fluid therapy, anti-inflammatory drugs (with caution), and analgesics;
- blood or platelet transfusions for severe hemorrhage;
- monitoring coagulation status and organ function.
- Complete elimination of the organism is not always guaranteed; subclinical carrier states can persist.

References :

1. *https://vcahospitals.com/know-your-pet/anaplasmosis-in-dogs*
2. *https://www.msdvetmanual.com/infectious-diseases/rickettsial-diseases-in-dogs/anaplasmosis-in-dogs*
3. *https://pmc.ncbi.nlm.nih.gov/articles/PMC8705095/*
4. *https://todaysveterinarypractice.com/wp-*
5. *content/uploads/sites/4/2024/04/TVP-2024-0506_Anaplasmosis.pdf*
6. *https://www.idexx.com/files/anaplasmosis-next-steps.pdf*
https://gsvs.org/blog/anaplasmosis-dogs-emergency/

Universitatea de Ştiinţe Agronomice şi Medicină Veterinară din Bucureşti
Facultatea de Medicină Veterinară

THE CAMPUS
we could be heroes...

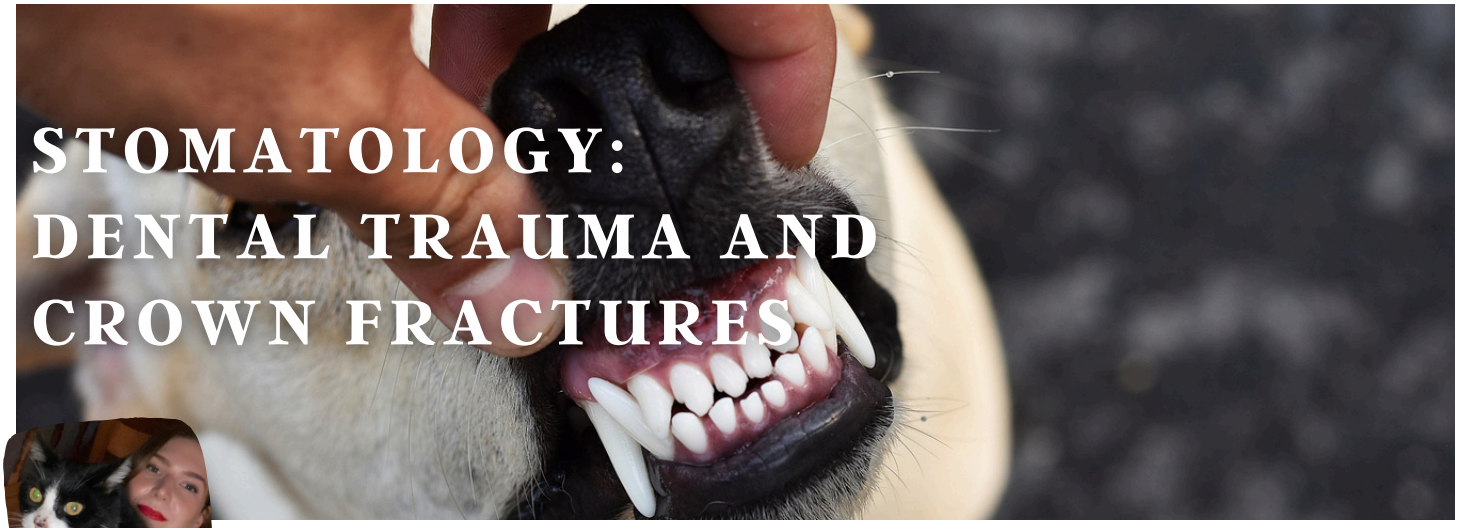


STOP PARASITES BEFORE THEY START

Fleas, ticks, and mosquitoes don't wait. These tiny pests can carry dangerous diseases like Lyme, anaplasmosis, or heartworm, threatening your dog's health year-round. Regular preventive treatment keeps your pets safe and your home protected.

PROTECT YOUR DOG, PROTECT YOUR FAMILY

Parasites aren't just itchy nuisances—they can transmit serious infections to both pets and humans. Trusted veterinary care and consistent prevention are the fastest ways to keep your dog healthy and your family safe.



STOMATOLOGY: DENTAL TRAUMA AND CROWN FRACTURES

Article by Anne-Kristoffy Bluteau

What is stomatology in veterinary medicine?

Veterinary stomatology is the study and clinical management of oral and dental disorders across species, covering anatomy, eruption pattern, occlusions, periodontal disease, tooth fractures, endodontics and oral masses. It integrates clinical examination, dental radiography, anesthesia and species-specific treatments (from prophylaxis to extractions and maxillofacial surgery) to preserve oral function and patient welfare.

Normal dentition in dogs and cats

Normal dentition in domestic species is typically heterodont and diphyodont, with a deciduous set replaced by permanent teeth according to predictable eruption schedules. Tooth structure (enamel, dentin, cementum, pulp; Fig. 1, 2) and the supporting periodontium determine function and common disease presentations. Familiarity with species-specific dental formulas, eruption timelines and radiographic appearance is essential for accurate diagnosis, age estimation, and treatment planning in clinical practice.

Key definitions:

- Heterodont: different tooth types (incisors, canines, premolars, molars)
- Diphyodont: two successive sets of teeth (deciduous then permanent)

Dental trauma and crown fractures: emergency management

Coagulopathies such as platelet disorders, clotting factor deficiencies, DIC (disseminative intravascular coagulation) or even Angiostrongyliasis (A. vasorum) can lead to secondary hemothorax in pets, so it is important also to keep them in mind when the traumatic cause is ruled out.

Figure 1: Feline tooth cross-section anatomy

Clinical context and priorities

Dental trauma is common in small animal emergency practice and can range from minor enamel chipping to complicated crown-root fractures, luxation and avulsions. In the acute setting priorities are:

- Rapid assessment of life-threatening injuries • Pain control
- Preservation of tooth vitality when possible
- Prevention of infection
- Clear communication with the owner about prognosis and follow up

Structured decision making reduces the risk of chronic pain, pulp necrosis, or alveolar bone complications.

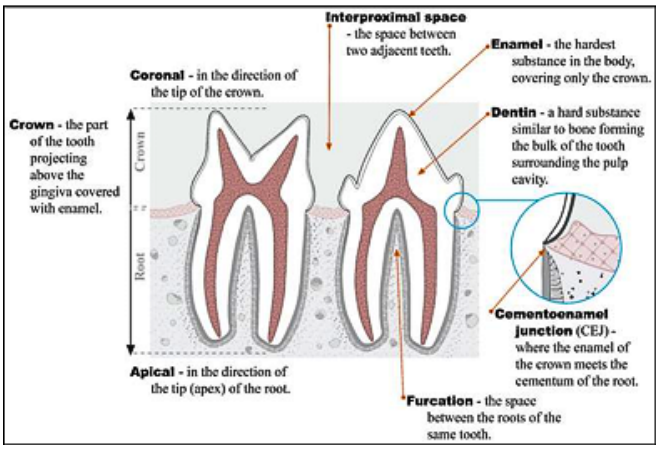


Figure 2: Sagittal section through canine tooth

Initial triage

- Primary survey: ensure respiration, circulation and neurological status are stable before oral assessment.
- Focused history: time and mechanism of injury (high energy impact, bite, fall…), appetite and behavior changs, previous dental diseases, and any first-aid already performed. Note the time elapsed since the injury and whether fragments or a tooth was retrieved.
- Analgesia: provide immediate multimodal analgesia appropriate for the patient (systemic opioids, NSAIDs if not contraindicated, local nerve blocks when indicated) before detailed oral manipulation

Clinical examination

Extra-oral examination	Intra-oral (initial) examination	Definitive examination under general anesthesia
• Facial swelling • Bruising • Lacerations • Occlusion changes • Mandibular/maxillary symmetry • Possible jaw fractures • Palpate regional lymph nodes	• Inspect for bleeding, visible fractures, displaced teeth, foreign bodies and soft tissue laceration • Avoid prolonged restraint or aggressive manipulation causing pain	• Full intra-oral charting, periodontal probing, pulp exposure assessment • Percussion testing • Complete intra-oral dental radiographic survey to assess root integrity, pulp chamber, alveolar bone and luxation extent

Table 2: Steps in the clinical examination

Classification and implication for management:

- Uncomplicated crown fracture (enamel +/- dentin, no pulp exposure): pain can be mild, risk to pulp depends on the depth of dentin exposure and time since trauma. Radiographs are done to rule out subgingival extension
- Complicated crown fracture (pulp exposure): higher risk of pulp necrosis and infections, options include direct pulp capping (rarely done), partial pulpotomy (done in selected cases), or root canal therapy/extraction depending on tooth value, feasibility and patient factors.
- Crown-root and root fractures: require radiographic evaluation, coronal fragment mobility, periodontal involvement and root displacement will influence the prognosis. Many root fractures ultimately require extraction of coronal fragments and monitoring or extraction of the root depending on healing.
- Luxation and avulsion: repositioning and splinting (luxation) or reimplantation (avulsion) protocols depend on extra-alveolar time, storage medium and degree of contamination. Avulsed permanent teeth have guarded prognosis if extraoral time is prolonged. Decisions differ for deciduous teeth (usually do not reimplant).

Species	Type of dentition	Deciduous teeth	Permanent teeth
Dog	Heterodont, diphyodont, carnivore-adapted	42 = 2(I3/3 C1/1 P4/4 M2/3)	28 = 2(I3/3 C1/1 P3/3)
Cat	Heterodont, diphyodont, obligate carnivore	30 = 2(I3/3 C1/1 P3/2 M1/1)	26 = 2(I3/3 C1/1 P3/2)

Table 1: Dentition type and dental formulas in dogs and cats

Immediate management steps

- 1. Stabilize and analgesia: systemic pain control and local anesthesia for wound management.
- 2. Control bleeding and soft tissue injuries: irrigate wounds with saline, suture mucosal lacerations when indicated after debridement. Avoid primary closure over infected tooth fragments.
- 3. Radiographic evaluation: essential before definitive treatment. Acquire intra-oral radiographs of injured are and contra-lateral teeth for comparison.
- 4. Decision tree for fractured teeth: o Uncomplicated crown fracture: smooth sharp edges, apply temporary protective dressing, plan definitive restoration or monitoring, provide analgesia and recheck. o Complicated fracture: if tooth is restorable and client accepts restoration, consider endodontic therapy (root canal) as definitive. If the extraction is more appropriate (multi-rooted, poor periodontal support, or patient factors) then do it. In immature teeth, vital pulp therapy or partial pulpotomy may be considered if within an appropriate time window and facilities exist. o Crown-root or root fractures: if coronal fragment is mobile or infected → extract the coronal fragment and assess root fragment. If root fragment is stable and apical fragment intact, monitor radiographically for healing.
- 5.Luxation/avulsion: attempt repositioning/reimplantation for permanent teeth when feasible and within a favorable time frame. Splint for 2-4 weeks depending on injury, consider systemic antibiotics and tetanus prophylaxis where relevant. In deciduous teeth → typically extract rather than reimplant.
- 6. Post-operative care: o Analgesia plan o Short course antibiotics only when indicated (e.g. severe contamination, systemic signs, alveolar bone involvement) o Soft food and oral hygiene instructions o Scheduled rechecks with radiographs at 2-4 weeks and 3-6 months to monitor pulp and periapical health.

Special considerations and pitfalls

- *Radiographs are indispensable:* many root fractures or subgingival extensions are occult on visual inspection.
- *Timing matters:* prompt management of pulp exposures and reimplantation attempts improves outcomes, record times precisely.
- *Toothe value and patient factors:* When deciding between restoration, endodontics, or extraction, weigh occlusal function, root anatomy, patient temperament, owner finances, and expected follow-up compliance.
- *Documentation and client communication*: describe findings, uncertainties, treatment options, expected complications and follow-up schedule in writing. Obtain informed consent for possible additional procedures if radiographs reveal more extensive damage under anesthesia.
- *Referral thresholds:* Complex crown-root fractures needing advanced restoration, unclear radiographs, or cases where function must be preserved should be referred to a stomatologist.

Conclusion

Dental trauma and crown fractures need rapid assessment of stability, pain, and tooth vitality. Radiographs under anesthesia guide restoration, endodontics, or extraction. Early referral to a veterinary dentistry specialist is advised for complex cases.

References:

- *[1] Andreasen, J. O., Andreasen, F. M., & Andersson, L. (2013). Textbook and color atlas of traumatic injuries to the teeth (4th ed.). Wiley Blackwell.*
- *[2] Bellows, J. (2010). Feline dentistry: Oral assessment, treatment, and preventative care. Wiley Blackwell.*
- *[3] Bourguignon, C., Cohenca, N., Lauridsen, E., Flores, M. T., Day, P. F., Hicks, L., Andreasen, J. O., Fouad, A. F., Tsilingaridis, G., Kahler, B., & Levin, L. (2020). International Association of Dental Traumatology guidelines for the management of traumatic dental injuries: Fractures and luxations. Dental Traumatology, 36(4), 314–330. https://doi.org/10.1111/edt.12578.*
- *[4] Feline tooth cross-section blog illustration. https://cdn.shopify.com/s/files/1/0931/5473/8517/files/feline-tooth-cross section-blog-illustration.jpg?v=1745914905.*
- *[5] Levin, L., Bourguignon, C., Cohenca, N., et al. (2020). International Association of Dental Traumatology guidelines: General introduction and overview. Dental Traumatology, 36(5), 355–363. https://doi.org/10.1111/edt.12574.*
- *[6] Merck Veterinary Manual. (n.d.). Table: Dental formulas of various animal species. Merck Veterinary Manual. https://www.merckvetmanual.com/multimedia/table/dental-formulas-of-various-animal-species.*
- *[7] MSD Veterinary Manual. (n.d.). Table: Dental formulas of various animal species. MSD Veterinary Manual. https://www.msdvetmanual.com/multimedia/table/dental-formulas-of-various-animal-species.*
- *[8] Soltero Rivera, M. (2021, August). Dentition and dental nomenclature of animals. MSD Veterinary Manual. https://www.msdvetmanual.com/digestive-system/dental-development-and-anatomy/dentition-and-dental-nomenclature-of animals.*
- *[9] World Small Animal Veterinary Association. (2019). WSAVA global dental guidelines. https://wsava.org/wp content/uploads/2020/05/WSAVA-Dental-Guidelines.pdf.*

UNITED VETERINARY MEDICINE

This initiative seeks to bring the veterinary community together, overcoming cultural, economic, and societal barriers. By combining perspectives and expertise from universities around the world, the project highlights the powerful benefits of international collaboration. Each institution contributes its own strengths and faces unique challenges, allowing participants from Europe to Asia to gain both personal growth and professional development.

Goals and Objectives

The project promotes global veterinary collaboration to enhance knowledge sharing, increase awareness, and improve animal health outcomes through international cooperation and academic exchange.

Jan-Feb 2026

Shape the future with us

This project stands as a unifying call for veterinarians and veterinary students worldwide, inspiring them to unite, share expertise, and collaborate across cultural and geographic divides. By cultivating a sense of global solidarity and common purpose, the initiative seeks to establish a dynamic and well-connected international veterinary community. Such synergy will not only elevate the standards of veterinary medicine but also improve the lives of the animals we care for and the people who love them. Through shared knowledge and joint action, we can shape a future where veterinary care transcends borders, delivering compassionate, high-quality treatment to animals everywhere.

Think beyond borders

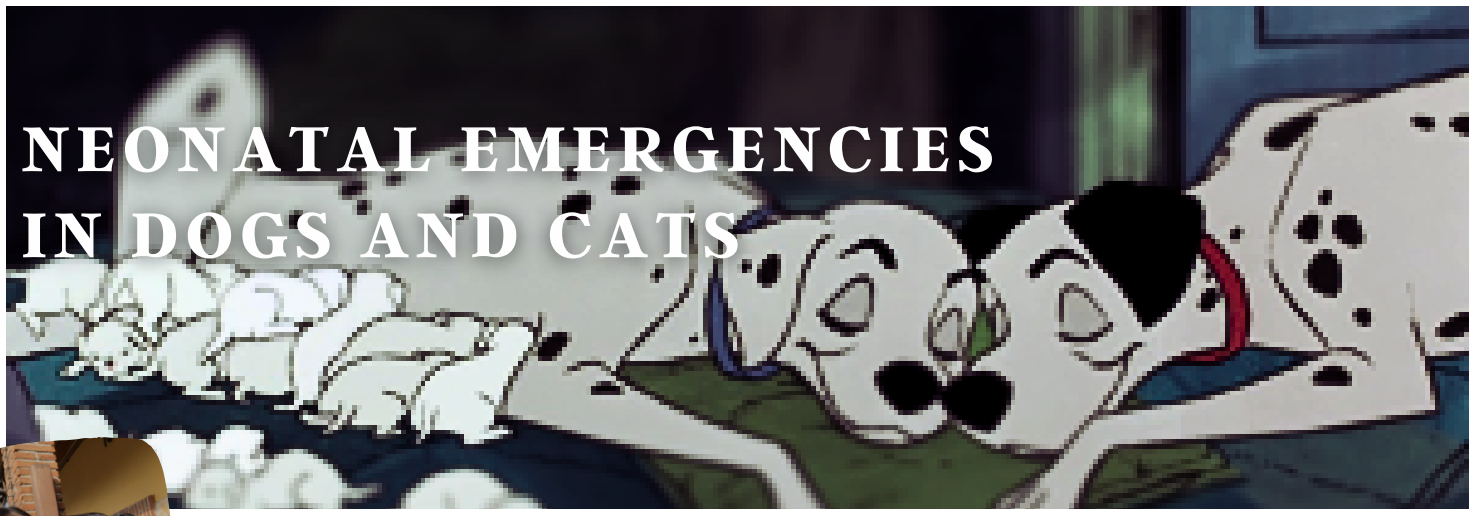
International collaboration fosters veterinarians' skills, knowledge, and relationships across borders, promoting global unity and cooperation in veterinary medicine.

Ideas beyond borders

The Campus

Join The *Campus...ABROAD* project to improve veterinary care through global collaboration. Curiosity has no borders!

thecampus.vet



NEONATAL EMERGENCIES IN DOGS AND CATS

Article by Assunta Piccolo

Introduction

The neonatal period, which in dogs and cats roughly corresponds to the first two weeks of life, represents a critical phase characterized by high vulnerability. Neonates have immature immune systems, limited thermoregulation capacity, and total dependence on the mother for nutrition, hygiene, and physiological stimulation. Neonatal mortality can reach significant levels: in dogs it ranges from 10% to 30%, while in cats it can reach 20–25% in domestic or non-optimized breeding environments. Prevention and early recognition of neonatal diseases are therefore essential to ensure the survival and health of puppies and kittens.

Physiology and Neonatal Characteristics

In canine and feline neonates, many physiological functions are still immature. Body temperature ranges from 35.5 to 37°C in the first hours of life, gradually increasing as they mature. Respiration and circulation show peculiar characteristics: the ductus arteriosus and foramen ovale close progressively, while respiratory and heart rates are higher than in adults. Maternal colostrum is the main source of antibodies and nutrients, with maximum immunoglobulin absorption occurring within the first 12 hours of life. Reflexes such as sucking, seeking the nipple, and responding to skin stimulation are essential for survival, as is maternal stimulation for urination and defecation.

Major Neonatal Diseases

Weak Puppy or Kitten Syndrome (Fading Syndrome) Weak neonate syndrome is a multifactorial clinical condition that can rapidly lead to death if untreated. Major causes include hypothermia, hypoglycemia, systemic infections, congenital malformations, and insufficient milk intake. Clinical signs: lethargy, difficulty suckling, continuous crying, hypothermia. Management: gradual warming using an incubator or lamp, administration of oral or intravenous glucose, nutritional support, and targeted antibiotic therapy if infection is present.

Neonatal Hypothermia

Neonates cannot maintain body temperature in cold environments. Hypothermia is one of the most frequent causes of early mortality. Signs: reduced activity, inability to suckle, slow breathing, cold extremities. Intervention: gradual warming, maintaining ambient temperature at 30–32°C, monitoring neonatal body temperature, and, if necessary, providing nutritional support.

Hypoglycemia

Hypoglycemia is common in debilitated or hypothermic neonates, particularly in toy breed puppies and low-birth-weight kittens. Clinical signs: tremors, weakness, seizures, loss of consciousness. Management: immediate glucose administration (oral or intravenous), blood glucose monitoring, regular nutrition, and prevention through frequent nursing or supplementation.

Neonatal Infections

Infections can be vertical (transmitted in utero or via infected colostrum) or horizontal (environmental, contact with other animals). Common agents: bacteria (E. coli, Streptococcus spp., Staphylococcus spp.), viruses (canine herpesvirus, feline calicivirus). Signs: red or purulent umbilicus, fever, lethargy, diarrhea, sepsis. Prevention: proper nest hygiene, environmental disinfection, umbilical care, monitoring of mother and neonates.

Dehydration and Malnutrition

Frequent causes include insufficient maternal milk or inability to suckle. Signs: weight loss, dry mucous membranes, decreased skin elasticity, reduced motor activity. Management: supplemental or artificial feeding, parenteral fluid therapy in severe cases.

Congenital Malformations

Congenital malformations can compromise survival. Examples: cleft palate, anal atresia, heart defects, hydrocephalus. Diagnosis: clinical examination in the first days of life, X-rays or ultrasound if necessary. Management: surgical intervention when possible, prognostic evaluation in cases incompatible with life.

Diagnosis and Monitoring

Careful and consistent monitoring of neonates is essential for their survival and healthy development. Daily checks are recommended, including body weight, aiming for regular gains of at least 5–10%, as well as close observation of body temperature, heart rate, and respiratory rate. Assessment of suckling reflexes and response to stimuli provides insight into neurological and overall health, while hydration status and mucous membrane color help detect early signs of illness. A thorough review of maternal history—including vaccination status, pregnancy complications, birthing events, and environmental conditions—is crucial for identifying potential risk factors. The use of neonatal incubators or heated boxes not only ensures optimal thermal support but also allows safer and more controlled monitoring of critical neonates, reducing stress and improving survival outcomes.

Copyright,Darwinbulldogs.com

Emergency Management

Timely and appropriate management of emergencies in neonatal puppies and kittens is crucial, as the neonatal period is characterized by high vulnerability. The most common emergencies include hypothermia, hypoglycemia, respiratory distress, and dehydration. Rapid intervention can make the difference between survival and death.

Neonatal Resuscitation

In neonates with respiratory arrest or severe respiratory distress, resuscitation should follow specific protocols: Respiratory stimulation: Dry the neonate with clean gauze and stimulate the respiratory reflex. Position the neonate with the head forward and body slightly inclined to facilitate the drainage of fluids from the nose and mouth. Chest compressions: If heart rate is <60 bpm and there is no spontaneous breathing, perform gentle chest compressions (approximately 1/3 of chest width). Recommended frequency: 120 compressions/min, alternating 2 compressions with 1 ventilation. Oxygen therapy: Administer via nasal cannula, mask, or oxygenated incubator, especially in neonates presenting cyanosis or respiratory distress.

Hypothermia Management

Hypothermia is often the primary condition leading to cardiac failure, hypoglycemia, and poor suckling ability: Gradual warming: Use incubators, heat lamps, or thermal blankets. Avoid abrupt warming, which can cause peripheral vasodilation and shock. Temperature monitoring: Ideal value: 36.5–37.5°C during the first 48 hours, gradually increasing to 38–38.5°C. Nutritional support: Provide small amounts of milk or glucose solution during warming if the neonate is not sucking spontaneously.

Hypoglycemia Management

Neonatal hypoglycemia is common, especially in small or premature puppies: Clinical signs: tremors, lethargy, seizures, coma. Immediate intervention: Administer 10–20% glucose solution orally or intravenously (carefully calculated, 0.5–1 ml/100 g body weight). Repeat blood glucose monitoring every 2–4 hours until stabilized. Prevention: frequent feeding, weight monitoring, and observation of activity.

Continuous Monitoring

In critical neonates, intensive monitoring is essential to prevent relapses or complications: Daily body weight: variations <5% are acceptable; decreases indicate problems. Heart and respiratory rate: reference values for puppies: HR 200–220 bpm, RR 15–35 breaths/min; for kittens: HR 180–220 bpm, RR 20–40 breaths/min. Monitor temperature and hydration status. Observe suckling behavior and reflexes.

Practical Approach

A practical algorithm can be summarized as follows: Rapid assessment: breathing, heart rate, temperature. Resuscitation if necessary: respiratory stimulation → chest compressions → oxygen. Concurrent treatment: warming + nutritional support + correction of hypoglycemia. Intensive monitoring during the first 24–48 hours, with prompt interventions if vital parameters deteriorate.

Prevention

Prevention of neonatal diseases requires an integrated approach: Maternal health management during pregnancy, including vaccination and screening for infectious diseases; Optimal nest management: temperature, hygiene, tranquility; Availability of colostrum and regular nursing; Education of breeders and owners on neonatal surveillance and first aid; Possible use of specialized artificial milk in the absence or insufficiency of maternal milk.

Copyright, Amoreaquattrozampe.it

Conclusion

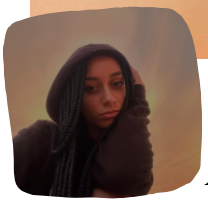
The neonatal period in dogs and cats is extremely delicate, but most diseases can be prevented or treated with timely intervention and proper management. Collaboration between the veterinarian and breeder or owner is essential to ensure survival and healthy development of neonates. Emerging technologies, such as advanced incubators and remote monitoring, are valuable tools to further improve standards of care.

Copyright, CANVA

References:

- *Pereira, K. H. N. P.; Fuchs, K. d. M.; Mendonça, J. C.; Xavier, G. M.; Câmara, D. R.; Salgueiro Cruz, R. K.; Lourenço, M. L. G. (2024). Neonatal Clinical Assessment of the Puppy and Kitten: How to Identify Newborns at Risk?*

THE KING COBRA (OPHIOPHAGUS HANNAH):



Article by Alessia Delle Cave

From feared predator to medical resource

beyond common perceptions and learning to understand their biological and ecological roles. Few animals embody this contrast better than the king cobra (Ophiophagus hannah), a species widely feared for its venom, yet of extraordinary scientific and medical importance. The king cobra is the longest venomous snake in the world and one of the most fascinating reptiles from both a zoological and a medical perspective. Its venom, behavior and relationship with humans make it an ideal example of how wildlife biology and medicine intersect.

General characteristics

The king cobra belongs to the family Elapidae, which includes cobras, mambas and coral snakes. Despite its common name, it does not belong to the genus Naja, but to its own genus, Ophiophagus, meaning “snake eater”. This name already reveals one of its most distinctive ecological traits. Adult individuals can reach 4–5.5 meters in length, making them the longest venomous snakes known. The body is slender but muscular, with olive, brown or black coloration, often with pale crossbands. When threatened, the king cobra raises the anterior part of its body and spreads a hood, a defensive display that serves primarily as a warning rather than an invitation to attack. From a veterinary and biological standpoint, this behavior highlights an important concept: venomous snakes are generally defensive, not aggressive animals.

Geographic distribution and habitat

The king cobra is native to South and Southeast Asia, including:
• India and Sri Lanka
• Thailand, Vietnam and Malaysia
• Indonesia and the Philippines
• Southern China

Its preferred habitats are tropical forests, bamboo thickets, mangroves and forest edges, often near water sources. It can also be found in agricultural areas, which increases the risk of human–snake encounters. Habitat destruction and deforestation are major threats to the species, contributing to its current classification as Vulnerable by the IUCN.

Lifestyle and feeding behavior

Unlike many snakes, the king cobra is primarily diurnal, meaning it is active during the day. It relies heavily on vision and chemoreception to locate prey.

Diet

The species is highly specialized:
• it feeds mainly on other snakes, including venomous species
• occasionally consumes large lizards

Due to the size and nutritional value of its prey, the king cobra does not feed frequently. After a large meal, it may go weeks or even months without eating, depending on metabolic demands and environmental conditions.

Water intake

Like most reptiles, the king cobra:

• does not drink water frequently
• obtains water from prey and environmental sources
• may drink directly from puddles or streams when available

Reproduction

A distinctive and often overlooked feature of king cobra biology is its unique reproductive behavior: it is the only snake known to build a nest. The female gathers leaves and vegetation into a mound, lays her eggs inside, and then guards the nest until they hatch. This unusually high level of parental care is rare among reptiles and highlights the species' remarkable behavioral adaptation.

Venom composition and effects

From a medical and veterinary standpoint, king cobra venom is notable for its potent mix of neurotoxins, cardiotoxins, and enzymatic components such as phospholipases. These substances disrupt neuromuscular function, impair cardiac muscle, and contribute to rapid systemic deterioration. Clinically, envenomation leads to progressive paralysis, respiratory failure, and potentially cardiovascular collapse. Without rapid intervention, death typically results from respiratory paralysis.

Antivenom and clinical relevance

Antivenom production is a striking example of how veterinary science directly supports human medicine. It begins with administering controlled, non-lethal doses of venom to animals—most often horses—so they can develop targeted antibodies. These immunoglobulins are then collected and purified to create the final therapeutic product. The success of antivenom relies on rapid administration, correct species-specific formulation, and proper dosing. Because adverse reactions can occur, ongoing research and refinement of production methods remain essential to improving safety and effectiveness.

Copyright, wikibigino.miraheze.org

Medical and scientific importance

King cobra venom has significance far beyond emergency medicine, serving as a valuable tool in biomedical research. Its components help scientists investigate neuromuscular transmission, develop new analgesic and neuroactive drugs, and deepen understanding of cardiovascular physiology. Many modern therapeutics trace their origins to animal toxins, underscoring that conserving venomous species is not only vital for ecosystems but also essential for medical progress.

Conservation and final considerations

The king cobra, despite its fearsome image, is not naturally aggressive toward humans; most bites happen through accidental or defensive encounters. Scientifically, it stands out as an important top predator, a valuable model for toxin research, and a reminder that fear often comes from limited understanding. Learning more about this species supports coexistence, conservation, and scientific progress.

References:

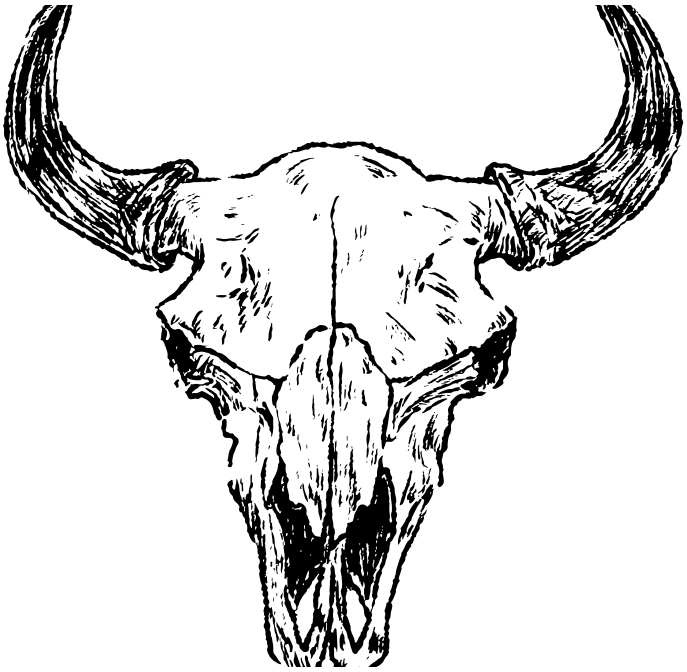
- *1. Gutiérrez JM et al. Snakebite envenoming. Nature Reviews Disease Primers, 2017.*
- *2. World Health Organization (WHO). Guidelines for the management of snakebites., 2016.*
- *3. Kardong KV. Venomous reptiles and their toxins. University of California Press.*
- *4. Fry BG et al. The evolution of venom systems in reptiles. Annual Review of Ecology, Evolution, and Systematics.*
- *5. Whitaker R., Captain A. Snakes of India: The Field Guide.*

REVIVING THE EUROPEAN BISON: FROM EXTINCTION TO REWILDING

Article by Daria-Ioana Marghidanu

The European bison (Bison bonasus) is the largest herbivore in Europe, surpassing species such as the aurochs, moose, and reindeer. It typically measures up to 2.9 meters in length and stands about 1.85 to 1.95 meters tall, weighing anywhere between 800 and 1,000 kilograms. In captivity, they can live up to 30 years, their lifespan in the wild generally ranging from 18 to 24 years, with females tending to live longer than males. Regarding physical characteristics, the wisent is slightly lighter than the buffalo (North American bison) and stands taller at the shoulder. However, it has a shorter mane on the head, neck, and forequarters, with longer tails and horns than its American counterpart.

Historically, King Sigismund I of Poland introduced the death penalty for poaching European bison as early as the 1500s, reflecting an early recognition of the species' importance. Despite these early protection efforts, centuries of hunting and habitat loss took a heavy toll. The last wild bison in Poland was killed in 1919, and the final wild individual worldwide was shot in the Caucasus in 1927. By that time, fewer than 50 European bison survived globally, all confined to zoos and captive breeding programs. In other regions, the decline occurred even earlier: the last bison in Moldova was killed in 1762, while in Transylvania the species disappeared by 1790. These events highlight the severe pressures that human activity placed on the European bison, underscoring the critical importance of conservation efforts in the modern era.



First rewilding efforts started as early as 1951, in Poland. In Romania they were reintroduced in 1958, when the first two animals were brought from Poland and kept in a reserve in Hațeg. The idea of having free-roaming bison in Romania only emerged in 1999, through a program supported by the World Bank and the European Union.

Today, they inhabit protected forest areas in Poland, Belarus, and Moldova, with additional herds living in Lithuania, Ukraine, Russia, and Kyrgyzstan and in zoos in 30 countries. Presently, our country houses over 350 wild wisents which inhabit Vânători-Neamț Natural Park, Țarcu Mountains and Făgăraș Mountains.

fig 1 - Map of European bison populations, 2020 source: Wikipedia

Rewilding European bison has emerged as a powerful tool for ecological restoration, biodiversity conservation, and socio-economic development. Ecologically, their grazing, trampling, and movement create open spaces and maintain dynamic ecosystems, supporting a variety of plants, insects, birds, and other mammals, as well as contributing to carbon sequestration. For instance, the 2014 reintroduction of bison to a 52-square-kilometer grassland area in Romania's Țarcu Mountains resulted in an estimated 54,000 tons of carbon being drawn down annually. Socially and economically, bison attract ecotourism and raise awareness about the importance of preserving Europe's natural heritage, their presence reconnecting communities with wildlife and enhancing cultural identity. However, despite the succes that the reintroduction of this species has had as of now, there are also a series of challenges that need to be adressed. The biggest problem derives from the fact that the entire existing population descends from only 12 individuals, as a result the current population exhibiting very low genetic diversity. Therefore, the comeback of the wisent isn't just about saving a species, it's about breathing life back into Europe's wild landscapes and reconnecting people with nature in ways long forgotten. The wisent's return is a bold reminder that with dedication and vision, even the most endangered creatures can reclaim their place in the wild.

References:

- *oneearth.org wwf.ro*
- *Wikipedia.org*

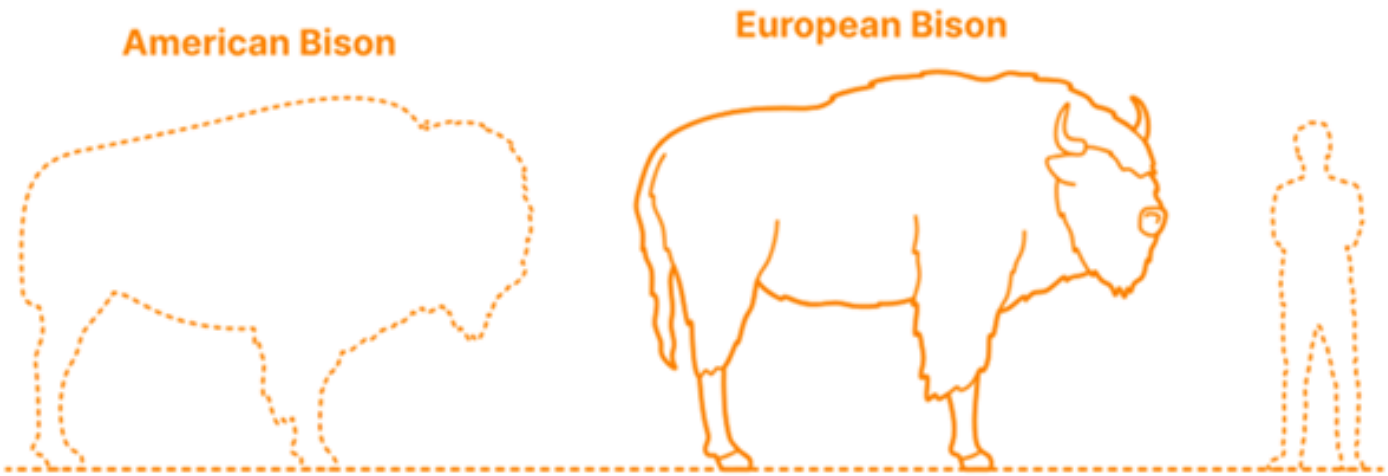


fig 2 - source: Dimensions.com

THE NEW STRIDES IN VETERINARY MEDICINE: FIP, A CURABLE DISEASE NOW?

Article by Sally Mustafa

If youre a fellow cat owner or cat enthusiast, you have definitely heard of FIP, Feline Infectious Peritonitis.

One of the most devastating diseases that happens to our feline friends, for a long time, a cat getting diagnosed with this meant, unfortunately, an immediate death sentence.

The disease itself is rare, and its symptoms are varied so it's also tricky to diagnose. Combined with all its effects, it's a huge pain to the cat, as well as it's always a big challenge to clinicians and also owners who have to deal with the devastating outcomes.

As veterinarians, seeing FIP is never easy, even personally, the first euthanasia I ever witnessed was due to FIP. However due to the constant strides in veterinary medical science and doctors all over the world working hard, we have a new breakthrough.

A ray of hope for treating FIP, New drugs that have improved survival chances greatly for cats diagnosed with FIP, so what are these treatments, and how do they work?

The breakthrough antiviral: GS- 441524:

The main active metabolite of the human antiviral remdesivir, which was developed by a pharma company that was doing research into antivirals for RNA viruses like Ebola and Coronaviruses, in 2018 Veterinary researchers published the first demonstration showing how it strongly blocks the replication of FIP, a year later follow up studies started showing more promising results and dramatic improvements, and with many group efforts, now around 5 years later.

FIP has been able to transform from a virtually uniformly fatal disease to one that is treatable and often curable and the majority of cases.

How does this drug work?:

1- GS-441524 enters infected cells where it is phosphorylated (activated) by the cell's enzymes into a nucleotide triphosphate form.

2- This activated form mimics the normal RNA building blocks that the coronavirus uses to copy its genetic material.

3- When the viral RNA polymerase incorporates GS-441524 instead of the natural building block, it disrupts the copying process, essentially causing premature termination of viral RNA replication.

4- Without effective replication, the virus can't spread to new cells, and the cat's immune system can begin clearing the infection .

How the drug is used:

This drug is available in two forms: injectable (s.c. or i.v.), the most commonly used for precise dosing, and oral (capsules or liquid), recently developed to allow easier at-home administration. Dosage depends on the cat's weight and type of FIP—wet, dry, ocular, or neurological. The typical dose is 4–6 mg/kg once daily, though more advanced cases may require higher doses. Treatment usually lasts around 12 weeks or longer, depending on severity. Many cats show improvement within days, including reduced fever and increased appetite, but completing the full course is essential to ensure recovery and prevent relapse. Studies show that most cats who finish treatment achieve full recovery and can live normal lives.

The reality, where are we going with this treatment?:

As of 2026, access to this treatment is getting better and better.

A couple of years ago, it was pretty much impossible to get access of this drug without some vets resorting to risky means of acquisition due to it still not being widely legal to use in veterinary medicine and available. However, now it is becoming widely legal in many countries such as Australia, the UK, Canada, the USA, and many nations in the EU and Asia.
In Romania, it is still not formally approved; however, some pharmacists make tailored similar formulations that fit veterinary legislation. And some vets use remdesivir as an alternative legally under the cascade rules.

Another challenge due to the limited availability is the cost, also with it being a long treatment course.

The cost can be hefty- it can reach around 800-3000 euros, not including all the diagnostics and vet visits. and not many owners can afford that.

In general, treating FIP is not just about giving a drug, it's about navigating diagnostic uncertainty, legal gray zones, high costs, daily hands-on care, emotional strain, and inconsistent veterinary support, all while racing against time.

However, with all that, it is a big leap and a breakthrough for us as pet owners and vets to be able to live in an era where medical science is so advanced.

Simply the discovery and use of this represents a turning point in veterinary medicine, showing that even long fatal diseases can eventually become treatable eventually.

While also us needing to highlight the urgent need for better regulatory pathways, equitable access to life-saving drugs, and a more proactive, evidence-based approach to advancing care and welfare for animals worldwide.

Copyright, goodhouret.click

References:

International Cat Care (iCatCare). (2025). An update on the treatment of feline infectious peritonitis. International Cat Care. https://icatcare.org/resources/icatcare_fip_update_july25.pdf Kleszynski, B., DVM. (2025). Feline infectious peritonitis (FIP): Signs, diagnosis, and treatment. PetMD. https://www.petmd.com/cat/conditions/infectious-parasitic/feline-infectious-periton itis Stokes Pharmacy. (2024). Feline infectious peritonitis (FIP) treatment with compounded GS-441524. Stokes Healthcare. https://www.stokespharmacy.com/fip/

UNDERSTANDING REPRODUCTIVE DISORDERS AND BEHAVIORAL REGULATION IN PET BIRDS

Article by Dinică Maria

Reproductive issues represent some of the most common and potentially life-threatening medical conditions in avian medicine. Unlike their wild counterparts, who are governed by seasonal environmental cues, pet birds often live in "artificial spring" conditions. Constant food availability, stable temperatures, and extended photoperiods (artificial lighting) can keep a bird in a state of chronic reproductive stimulation.

I. The Avian Reproductive Cycle: Cues and Triggers

Birds are oviparous, meaning they lay and incubate eggs. In the wild, the reproductive cycle is a strictly timed biological event triggered by increasing day length (photoperiod), temperature, and food availability.

Physiological Foundation

Most female pet birds possess only a left ovary and oviduct. The process of egg formation is metabolically demanding, taking approximately 24 hours. It begins in the infundibulum (fertilization), moves to the magnum (albumen deposition), the isthmus (shell membranes), and finally the uterus or shell gland, where calcification occurs. To support shell production, females store calcium in their bones (hyperostosis), a process driven by estrogen.

Captivity and Reproductive Stimulation

In household environments, several factors inadvertently signal to a bird that it is time to breed: photoperiod(>12 hours of light a day), nutrition(diets high in fats and calories, like seed-based diets), perceived mates(the human owner is often perceived as one, stimulating courtship).

II. Behavioral Challenges and Misplaced Bonds

Behavioral problems in pet birds are frequently rooted in sexual frustration or overstimulation. Hand-raised parrots, in particular, may develop "maladaptive bonding" with their owners.

Manifestations of Reproductive Behavior

When a bird reaches sexual maturity and identifies its owner as a mate, it may exhibit: excessive screaming when leaving the room, over-grooming or plucking driven by hormonal frustration or the lack of a suitable nesting site, territoriality over a perceived nest site or "mate-guarding" the owner from other family members.

Management through Behavior Modification

Correcting these issues requires a multi-pronged approach: reducing day length to 8–10 hours, limiting petting to the head and neck only. Avoid "cuddling" or feeding warm foods by hand/mouth, as these are viewed as courtship rituals, converting the bird to a portion-controlled diet and encouraging foraging to redirect energy toward "working" for food rather than breeding.

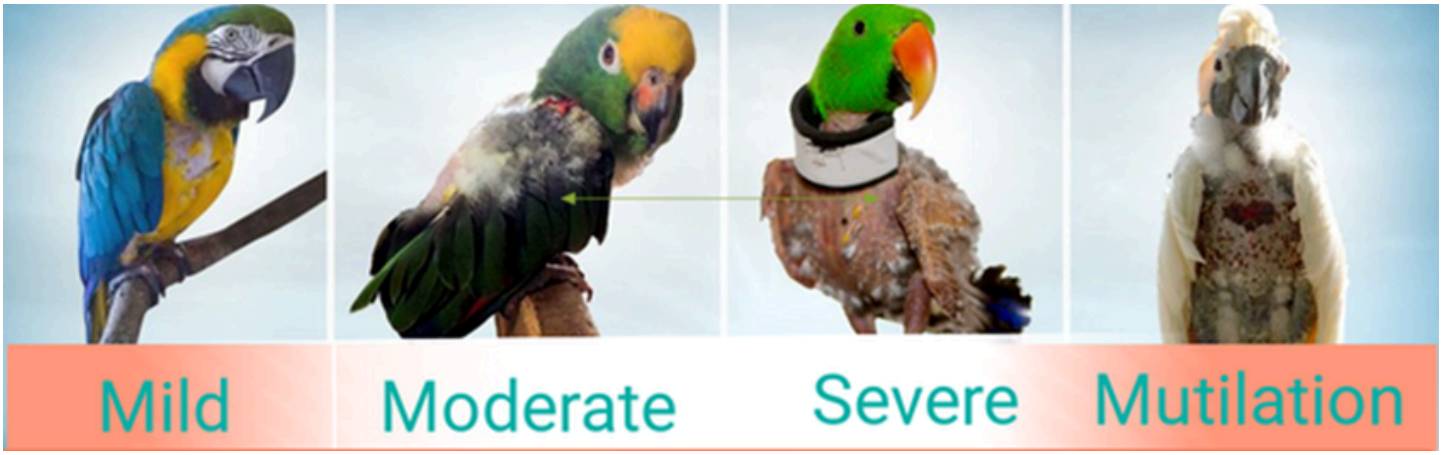


Figure 1- source : birdsupplies.com/pages/feather-plucking-what-products-do-i-need?

III. Cloacal Prolapse (Vent Prolapse)

Cloacal prolapse involves the protrusion of the cloacal lining or oviduct through the vent. This condition is a medical emergency and is particularly prevalent in adult Umbrella and Moluccan cockatoos.

Causes and Contributing Factors

While the exact etiology remains under study, several commonalities exist among affected birds: delayed weaning: many were hand-raised and showed prolonged begging behaviors, stool retention: some birds are taught to "hold it" until they are taken out of the cage or given a command, leading to flaccidity and eventual prolapse, sexual straining: misplaced sexual attraction to a human can lead to frequent straining and masturbatory behaviors, weakening the cloacal sphincter.

Treatment and Prevention

Early intervention involves cleaning and replacing the tissue, often using stay sutures to hold it in place. However, surgery (such as cloacopexy) is rarely successful long-term without significant behavior modification. If the bird continues to perceive the owner as a mate and strains to "offer" its cloaca or hold stool, the prolapse is likely to recur.

IV. Critical Reproductive Pathologies

Egg Binding (Dystocia):

Egg binding occurs when a hen is unable to expel an egg. This is common in smaller birds like cockatiels, budgerigars, and lovebirds, often as a result of chronic egg laying.

Risk Factors:

Obesity, lack of exercise, and nutritional deficiencies —specifically Calcium and Vitamin A. Calcium deficiency results in soft-shelled eggs that the oviduct cannot effectively grip and push.

Clinical Signs:

A bird sitting on the cage floor, depression, wide-based stance, and "tail bobbing" (indicating difficulty breathing due to the egg's pressure).

Figure 2- source : careforbirds.com/2020/12/14/%E3%80%90how-to-discourage-birds-from-laying-eggs%E3%80%91/

Intervention:

Treatment involves stabilization in a warm, humid incubator, fluid therapy, and parenteral calcium. If medical management fails, manual extraction or surgery may be required.

Figure 3- source : www.reddit.com/r/cockatiel/comments/1c7085h/egg_bound_no_idea_what_to_do/

Excessive or Chronic Egg Laying

This is a syndrome where a bird lays repeat clutches or more eggs than is physiologically sustainable. It leads to the depletion of calcium stores, potentially causing pathologic fractures and metabolic collapse. Management centers on removing nest boxes, toys the bird is bonded to, and utilizing hormonal therapy.

EIgg Yolk Coelomitis

This occurs when yolk material enters the body cavity (coelom) instead of the oviduct. It causes severe inflammation and can lead to secondary bacterial infections (e.g., E. coli). Signs include abdominal distention and respiratory distress.

V. Advanced Regulation: Hormonal Implants

When environmental and behavioral changes are insufficient, veterinarians turn to pharmacological regulation.

GnRH Agonists (Deslorelin)

The Companion Zoological Animal Research (CZAR) laboratory at UC Davis has pioneered research into the use of Deslorelin implants. Deslorelin is a sustained-release hormonal contraceptive that suppresses the production of reproductive hormones (LH and FSH).

Figure 4- source : cluckin.net/egg-yolk-peritonitis-the-causes-symptoms-and-treatment-of-eyp

Efficacy: The duration of effect varies significantly between species. Research is ongoing to determine the precise dosages needed to suppress life-threatening egg production in parrots.

Limitations: Hormonal implants should not be viewed as a "quick fix." They are most effective when used as a bridge while the owner implements permanent environmental and behavioral changes.

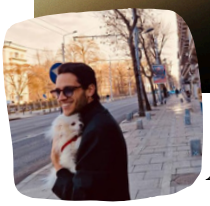
Conclusion

Reproductive health in pet birds is a delicate balance of biology and husbandry. The "humanization" of parrots, while fostering a deep bond, often leads to physiological states that the avian body cannot sustain. By understanding the triggers of reproductive activity—photoperiod, diet, and social bonding—owners can prevent common tragedies like egg binding and cloacal prolapse. Ultimately, the successful management of the pet bird requires viewing it not as a "little human," but as a highly sensitive biological organism governed by natural cycles that must be respected in the home.

References and bibliography :

·https://www.msdvetmanual.com/exotic-and-laboratory-animals/pet-birds/reproductive-diseases-of-pet-birds#Behavioral-Problems_v23359079
·https://www.merckvetmanual.com/bird-owners/disorders-and-diseases-of-birds/reproductive-disorders-of-pet-birds#Cloacal-Prolapse-(Vent-Prolapse)_v3224145
·https://doi.org/10.1002/9781119650522.ch7
·https://czar.vetmed.ucdavis.edu/research/reproductive-regulation-birds

CANINE PARVOVIROSIS IN PUPPIES: AN EMERGENCY NOT TO BE UNDERESTIMATED



Article by Giovanni Marciano

Abstract

Canine parvovirosis is one of the most serious viral diseases affecting puppies worldwide. Caused by canine parvovirus type 2 (CPV-2) and its variants, this disease is characterized by severe vomiting, diarrhea, dehydration, leukopenia, and high mortality if untreated. Although highly effective vaccines are available, outbreaks are still reported, especially in unvaccinated or incompletely vaccinated puppies. One of the reasons parvovirosis remains such a formidable challenge is the extraordinary environmental resistance of the virus. CPV is a small, non-enveloped DNA virus that withstands extreme conditions, including heat, cold, humidity, and many common disinfectants. The virus can survive for months to years in contaminated environments, making control and eradication difficult without proper hygiene measures. Early recognition, rapid diagnosis, and aggressive supportive care are fundamental for survival. Fluid therapy remains the cornerstone of treatment, but pharmacological support, nutritional supplementation, and careful monitoring of biochemical and hematological parameters are equally essential.

Etiology and Pathogenesis

Canine parvovirus belongs to the family Parvoviridae. After fecal–oral transmission, the virus targets rapidly dividing cells, especially intestinal crypt cells and bone marrow precursors. This leads to intestinal villus collapse, malabsorption, hemorrhagic diarrhea, and profound immunosuppression.

Puppies are most at risk between 6 weeks and 6 months of age. In neonatal pups, CPV may also infect myocardial cells, leading to myocarditis, though this form is now rare due to vaccination.

Clinical Presentation

The incubation period is 3–7 days. Clinical signs include:

• Profound lethargy and anorexia
• Repeated vomiting
• Severe, often bloody diarrhea
• Rapid dehydration
• Fever or hypothermia in advanced cases
• Marked leukopenia If untreated, death occurs due to hypovolemic shock, electrolyte derangements, sepsis, or disseminated intravascular coagulation (DIC)

Diagnosis

The diagnosis of parvovirosis is usually straightforward based on the clinical picture, but laboratory confirmation is always important.

- Rapid fecal tests (ELISA) can detect viral antigens within minutes; however, false negatives are possible, especially in very early or late stages of infection.
- PCR testing is more accurate, as it directly detects viral DNA.

Routine bloodwork provides essential information:

- Leukopenia and neutropenia are typical and correlate with disease severity.
- Biochemistry abnormalities often include hypoalbuminemia, hypoglycemia, electrolyte imbalances, and metabolic acidosis.

The Importance of Fluid Therapy

If there is one rule in treating parvovirosis, it is this: start fluid therapy immediately. Dehydration and shock are the primary killers in parvoviral puppies, and restoring circulation is the first and most urgent step.

- Shock resuscitation: Dogs in hypovolemic shock may require up to 80–90 mL/kg of crystalloids (such as Lactated Ringer's or 0.9% NaCl) given intravenously in divided boluses of 20–30 mL/kg, with constant reassessment.
- Maintenance fluids: Once stabilized, puppies need 40–60 mL/kg/day, plus additional fluids to replace ongoing losses from vomiting and diarrhea.
- Glucose supplementation: Many puppies develop hypoglycemia; therefore, 2.5–5% dextrose is often added to IV fluids. In case of acute hypoglycemia, a bolus of 0.3–1 mL/kg of 50% dextrose (diluted 1:2) can be administered, followed by a continuous glucose infusion.
- Potassium supplementation: 20–40 mEq/L of potassium chloride can be added to fluids and infused at no more than 0.5 mEq/kg/h to correct life-threatening hypokalemia.
- Plasma or colloids: In cases of severe protein loss, plasma transfusion can help restore oncotic pressure.

Fluid therapy is dynamic-it must be constantly adapted to the puppy's condition, guided by repeated checks of hydration, blood glucose, and electrolytes.

Monitoring Glucose in Puppies

Puppies are not just small dogs; their metabolism is unique. They have smaller glycogen reserves in the liver and an immature ability to regulate blood sugar. In parvovirosis, continuous vomiting and diarrhea, combined with sepsis, rapidly deplete these limited reserves.

- Normal blood glucose for puppies: 80–120 mg/dL
- Hypoglycemia: blood glucose < 60 mg/dL, which can cause weakness, seizures, coma, and death

This is why blood glucose monitoring every few hours is crucial. Intravenous glucose supplementation is not optional—it is lifesaving.

Therapeutic Protocol:

- Maintenance supplementation: Add 2.5–5% dextrose to IV fluids for at-risk puppies.
- Acute hypoglycemia:
  - Bolus: 0.3–1 mL/kg of 50% dextrose, diluted 1:2 with saline, IV
  - Followed by continuous infusion of 2.5–5% dextrose

Frequent monitoring (every 2–4 hours) is essential to prevent both hypoglycemia and rebound hyperglycemia.

Antiemetic Therapy

Persistent vomiting is not only distressing but also accelerates dehydration and prevents oral refeeding. For this reason, antiemetic coverage is a cornerstone of parvovirosis management.

- Maropitant citrate (1 mg/kg IV once daily) is highly effective as it blocks NK-1 receptors, acting on both central and peripheral pathways.
- Metoclopramide (0.2–0.5 mg/kg IV every 6–8 hours, or as a continuous rate infusion [CRI] at 1–2 mg/kg/day) can be used alongside maropitant to promote gastric emptying and reduce nausea.
- Ondansetron (0.1–0.2 mg/kg IV every 8–12 hours) is a serotonin (5-HT3) antagonist that is effective when vomiting persists despite other therapies.

Combination therapy is often required, and its importance cannot be overstated: by controlling vomiting, we protect the puppy from further losses, allow earlier reintroduction of food, and significantly improve comfort and survival.

Antibiotic Therapy

Although parvovirus itself is not affected by antibiotics, the destruction of the intestinal barrier allows dangerous bacteria to enter the bloodstream.

Without antibiotics, sepsis is almost inevitable.

- Metronidazole (10–15 mg/kg IV or PO every 12 hours) provides coverage against anaerobic bacteria.
- Ceftriaxone (25–50 mg/kg IV or IM every 12–24 hours) adds broad-spectrum coverage against both Gram-positive and Gram-negative bacteria.

Rationale: Antibiotic therapy in parvovirosis is primarily preventive, protecting the patient from bacteremia and sepsis secondary to intestinal mucosal damage.

Pain Control

Parvovirosis is painful. The intestinal inflammation and repeated vomiting cause significant distress. Effective pain management not only improves welfare but also contributes to recovery.

- Buprenorphine (0.01–0.02 mg/kg IV, IM, or SC every 8 hours)
- Butorphanol (0.2–0.4 mg/kg IV or IM every 4–6 hours)

These are the most commonly used analgesics in parvoviral puppies. NSAIDs are generally avoided, as they can worsen intestinal damage and increase renal stress in dehydrated patients.

Advanced Pain Management: The Role of Methadone and Low-Dose Ketamine

While buprenorphine and butorphanol are often sufficient to control mild to moderate discomfort in parvoviral puppies, some patients experience severe visceral pain that requires a stronger approach. In such cases, methadone and low-dose ketamine can play an important role.

- Methadone, a full μ-opioid receptor agonist, provides more potent analgesia than butorphanol and is particularly effective for visceral pain. It can be administered at 0.2–0.4 mg/kg IV, IM, or SC every 4–6 hours, offering consistent pain relief without excessive sedation when carefully titrated.
- Ketamine at sub-dissociative doses acts as an NMDA receptor antagonist, preventing central sensitization ("wind-up" pain) and enhancing the effects of opioids. In parvovirosis, it can be particularly useful for puppies experiencing persistent abdominal pain. Ketamine is not used for anesthesia in this context, but rather as a continuous low-dose infusion (10–20 μg/kg/min), sometimes preceded by a small bolus of 0.25–0.5 mg/kg IV.

This multimodal approach allows for effective pain control while minimizing sedation and supporting recovery in severely affected puppies.(McKune 2011).
When combined, methadone and ketamine provide potent analgesia, though careful monitoring is required. This protocol can significantly improve welfare in the most painful cases of parvovirosis.

According to the literature, multimodal analgesia is considered the standard of care in canine intensive care settings (Carregaro et al., 2018; Grubb et al., 2020). The synergistic effect of methadone and ketamine improves comfort and reduces the need for higher doses of opioids, minimizing sedation while effectively controlling severe visceral pain.

Nutritional Support and Probiotics

For many years, veterinarians withheld food from parvoviral puppies. Today, we know this was a mistake. The intestine needs nutrients to heal, and early enteral feeding improves survival. Small, frequent meals of highly digestible food are recommended as soon as vomiting is controlled.
Adding probiotics can also be beneficial. Certain strains of Lactobacillus, Bifidobacterium, and Enterococcus can restore healthy gut flora and even support the immune system.

Assisted Feeding: Nasogastric vs. Esophagogastric Tubes

One of the most delicate moments in managing parvovirosis is the reintroduction of nutrition. Puppies are often too weak, nauseated, or unwilling to eat, yet the intestine desperately needs nutrients to begin healing. When oral feeding is not possible, veterinarians may use assisted enteral feeding with either a nasogastric (NGT) tube or an esophagogastric (EGT) tube.

- Nasogastric tube (NGT):
- The tube is inserted through the nostrils and advanced into the stomach. Its main advantages:
  - Can be used in unstable patients without general anesthesia
  - Allows continuous infusion of liquid diets, providing small amounts of nutrients to the intestine
  - Limitations:
  - Small diameter restricts diet to liquid formulas only
  - May irritate nasal passages, causing sneezing
  - Puppies may try to remove it by scratching

- Esophagogastric tube (EGT):

- Inserted through the mouth into the esophagus and stomach, it has different characteristics:
  - Wider diameter allows feeding of more calorically dense diets, useful for patients with high energy needs
  - Better tolerated for medium-term feeding, as it does not irritate the nose

Both methods ensure that nutrients reach the intestine, supporting healing and recovery in severely affected puppies.
On the downside, placing an EGT usually requires short anesthesia, which can be risky in critically ill puppies. Additionally, because the tube bypasses the mouth, patients lose the normal stimulation of chewing and salivation, which can slightly affect digestion.
Both methods, when properly chosen and monitored, can make the difference between life and death by maintaining energy supply, supporting gut recovery, and preventing further catabolism.
The decision on which tube to use depends on the individual patient's condition, tolerance, and expected duration of nutritional support.

Immunostimulation with Granulokines

One of the most dangerous aspects of parvovirosis is the drop in
white blood cells, which leaves the
happy defenseles recent diars recombinant
granulokines-recombinant
proteins that stimulate the bone marrow to produce new white cells.
* Filgrastim (G-CSF) at 5 pg/kg SC once daily can significantly reduce the duration of neutropenia.
* Sargramostim (GM-CSF) at 5-10 pg/kg SC once daily is another option, though less commonly used.
These drugs, originally developed for human
medicine,
can make a crucial difference in the
sickest puppies, helping them survive until their immune system recovers.

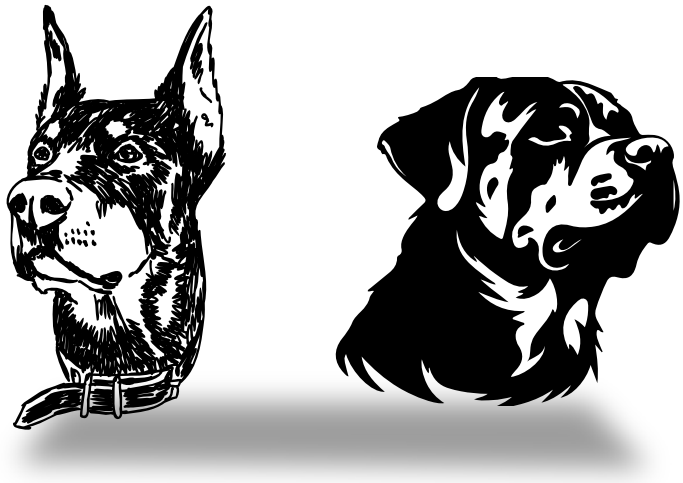
Hematological Considerations

Leukopenia is a classic finding in parvovirosis and strongly correlates with prognosis. Severe neutropenia (<1,000/µL) is associated with increased mortality.(Decaro et al., 2005; Greene, 2012)

Particular attention must be paid when leukopenia shifts to leukocytosis, which often indicates secondary bacterial infection and systemic inflammation, raising the risk of sepsis (Smith-Carr et al., 2019).
Monitoring hematology guides antibiotic therapy, granulokine use, and overall prognosis. Platelet counts and packed cell volume (PCV) should also be followed, as they help detect complications such as disseminated intravascular coagulation (DIC) or severe anemia.

Breed Susceptibility

One of the most intriguing and clinically important aspects of canine parvovirosis is the observation that not all breeds are affected equally. While any unvaccinated puppy is at risk, veterinarians around the world have noted that Dobermans, Rottweilers, and Miniature Pinschers are particularly vulnerable (Houston et al., 1996; Breed-related risk factors, PubMed 3003015).



The reasons for this increased susceptibility are not yet fully understood, but several theories have been proposed:

- Immunogenetic differences: Studies suggest that certain breeds may have variations in their major histocompatibility complex (MHC) genes, which play a central role in how the immune system recognizes and responds to pathogens. In Dobermans and Rottweilers, specific alleles may reduce the efficiency of antigen presentation, leading to a weaker or delayed immune response against parvovirus (Houston et al., 1996; Kennedy et al., 2000).
- Maternal antibody interference: Puppies are initially protected by antibodies acquired from their mother's colostrum, but these antibodies gradually decline in the following weeks.

If maternal antibodies remain high at the time of early vaccination, they can neutralize the vaccine virus before the puppy's immune system has a chance to respond. This "window of susceptibility" may be particularly problematic in breeds like Rottweilers and Dobermans, where maternal antibodies sometimes persist longer, leaving them unprotected during a critical period (Pollock & Carmichael, 1982; Greene & Decaro, 2012).

- Differences in intestinal immunity: Another hypothesis is that breed-related differences in gut-associated lymphoid tissue (GALT) may influence how effectively the immune system controls viral replication in the intestine. A less efficient mucosal immune barrier could contribute to more severe intestinal damage and bacterial translocation (Decaro & Buonavoglia, 2012).
- Clinical observations: Veterinarians often report that even with early and aggressive therapy, Dobermans and Rottweilers show higher rates of complications such as sepsis, persistent leukopenia, and hypoalbuminemia. This clinical evidence reinforces the idea that these breeds are not just "unlucky" but truly biologically predisposed (Houston et al., 1996; Goddard & Leisewitz, 2010).

For practitioners, this knowledge is not just theoretical: it has practical implications. Puppies of these susceptible breeds should be considered high-risk patients, requiring strict vaccination protocols, meticulous hygiene, and especially aggressive early treatment if they show signs of infection.
For breeders, awareness of this predisposition is critical, as prevention through timely vaccination becomes even more vital.

Prognosis

Without treatment, parvovirosis kills more than 90% (Houston et al., 1996; Goddard & Leisewitz, 2010) of infected puppies. With intensive supportive care, survival rates rise to 70–90%(Greene & Decaro, 2012; Decaro & Buonavoglia, 2012). Factors such as severe leukopenia, hypoglycemia, and hypoalbuminemia lower chances of recovery. Early intervention can change the outcome dramatically.

Prevention: The Power of Vaccination

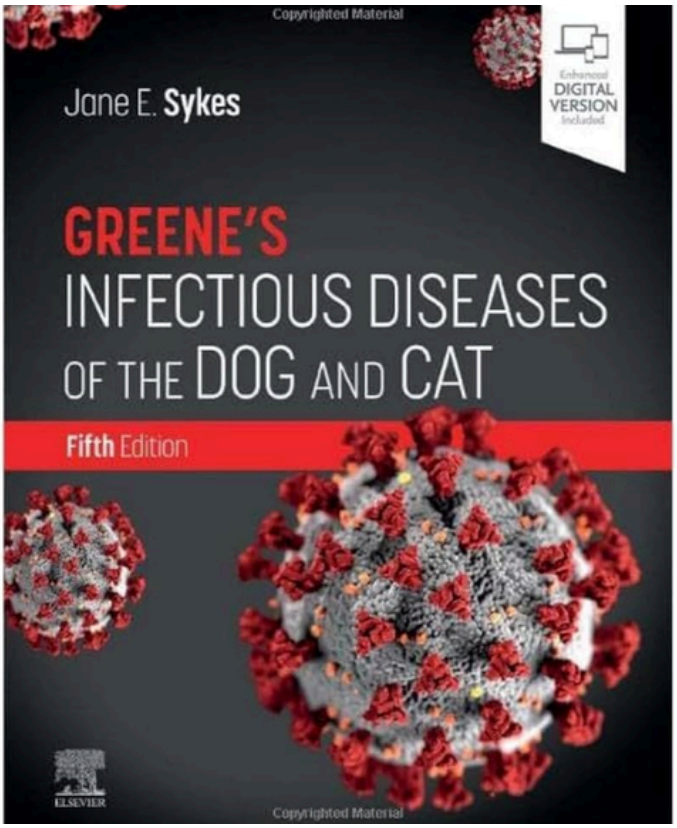
The best way to fight parvovirosis is to prevent it altogether. Vaccination is highly effective, but only if performed correctly. Puppies should start their vaccine series at 8 weeks of age, with boosters every 3–4 weeks until at least 16 weeks to ensure protection once maternal antibodies wane (AAHA/WSAVA, 2016). Adult dogs require regular booster shots to maintain protection, typically with a booster one year after the initial series and then every 3 years thereafter (Greene & Decaro, 2012).
Owners must understand that one missed dose can leave a puppy vulnerable, especially because maternal antibodies can interfere with early vaccines if given too soon or not repeated at proper intervals (Pollock & Carmichael, 1982; Greene & Decaro, 2012). Combined with strict hygiene and effective disinfection (e.g., with bleach) and avoiding contaminated areas, vaccination is our most powerful weapon against parvoviral disease.
For veterinarians, the message is clear: treating parvovirosis is possible, but prevention through vaccination is always better, safer, and far less costly (Goddard & Leisewitz, 2010; Greene & Decaro, 2012).

Conclusion

Canine parvovirosis is not just another disease—it is a true veterinary emergency that combines virology, intensive care, and client education. The virus is extraordinarily resistant, the clinical signs are dramatic, and the risks are high.
But with rapid intervention—including fluid therapy, glucose monitoring, antiemetics, nutritional support, probiotics, immunostimulation, and constant monitoring—many puppies can be saved.
At the same time, we must never forget the bigger picture: vaccination remains the key to defeating parvovirosis. It protects not only individual dogs but the entire canine community, sparing countless puppies from suffering and giving them the healthy start in life they deserve.

References:

- *1. Decaro N. Buonavoglia C. Canine parvovirus-A revicto of epidemiological and diagnostic aspects, with emphasis on type 2c. Vet Microbial 2012;155(1):1-12*
- *2. Greene CE, Addie DD. Canine Parvovirus Infections, In: Greene CE, ed. Infectious Diseases of the Dog and Cat. 4th ed. St. Louis: Elsevier; 2012. p. 67-75.*
- *3.Otto CM, Drobat: KJ, Soter C. Endotoremia and tumor necrosts pector acrote tn dogs sour naturaly occurring parcoviral enteritis J Vet Intern Med. 1997:11(2):63-70.*
- *4. Prittie J. Canine parvoviral enteritis: a reviete of diagnosis, management, and prevention. J Vet Emerg Crit Care. 2004:14(3):167-176*
- *5.Nelson RW, Couto CG. Small Animal Internal Medicine 6th ed. St. Louis: Elsevier: 2019.*
- *6. Papich MG. Saunders Handbook of Veterinary Drugs oin ed. a. Louis: Biscoter: 2020.*
- *7.Savigny MR, Macintine DK. Use of rocombinant human granulote colony-stimulating factor in dogs with parvoviral enteritis. J Vet Emerg Crit 2000:10(3):161-167.*
- *8.Gómez DE. Weese JS. The potential role of probiotics in the management of canine enteric discase. Vet Med Res Rep. 2018;9:71-82*
- *9. Gow AG. Papasouliotis K. et al. Hypoalbuminemia in dogs: canses, diagnosis and treatment. J Small Anim Pract. 2011:59(7):377-384.*
- *10.Houston DM, Ribble CS, Head LL, et al. Epidemiologic and clinical features of canine parvovirus infection in dogs: 100 cases (1983–1988). J Am Vet Med Assoc. 1996;208(12):2012-2018.*
- *11.Goddard A, Leisewitz AL. Canine parvovirus. Vet Clin North Am Small Anim Pract. 2010;40(6):1041-1053.*
- *12.Pollock RV, Carmichael LE. Maternal antibody and neonatal immunization to canine parvovirus. Vet Immunol Immunopathol. 1982;3:343-356.*
- *13.Truyen U, Parrish CR. Canine parvovirus: virus evolution and host adaptation. Vet Microbiol. 2013;165(1–2):1-4.*
- *14.Kennedy MA, Kitchener A, et al. Breed-related differences in immune response: implications for canine parvovirus. Vet Immunol Immunopathol. 2000;77(3–4):203-212.*
- *15AAHA/WSAVA. 2016 Canine Vaccination Guidelines. J Am Anim Hosp Assoc. 2016;52(2):95-108.*



For anyone interested in a deep and practical understanding of infectious diseases in pets, **Green's Infectious Diseases of Dogs and Cats** *is an outstanding resource. The book provides comprehensive coverage of viral, bacterial, fungal, and parasitic infections, detailing their pathogenesis, clinical signs, diagnostic approaches, treatment options, and prevention strategies. Beyond the basics, it explores epidemiology, zoonotic risks, and the complexities of co-infections, helping readers understand how multiple factors interact in real-world cases. The text is supported by current research and case examples, making it both scientifically rigorous and clinically applicable. Its clear explanations and structured format make it particularly useful for veterinary students, practitioners, and anyone seeking a thorough, reliable guide to infectious diseases in dogs and cats. Whether used for study, clinical reference, or general enrichment, this book offers a complete and engaging overview of the challenges and considerations involved in keeping pets healthy in the face of infectious threats.*

Universitatea de Științe Agronomice și Medicină Veterinară din București

Facultatea de Medicină Veterinară

WSAVA Global Veterinary Community

PARVOVIROSIS

Why does Parvovirus is still a threat ?

Parvovirus remains a threat because it is extremely contagious, survives for months in the environment, and causes severe disease in unvaccinated animals—especially puppies. Incomplete vaccination, poor hygiene, and rapid viral spread continue to fuel new outbreaks. Prevention depends on strict sanitation and consistent vaccination.

WSAVA Global Veterinary Community

2024 guidelines for the vaccination of dogs and cats – compiled by the Vaccination Guidelines Group (VGG) of the World Small Animal Veterinary Association (WSAVA)

AUTHORS:

R. A. Squires *,1, C. Crawford†, M. Marcondes‡ and N. Whitley§

*Formerly, Discipline of Veterinary Science, James Cook University, Townsville, QLD 4814, Australia
†College of Veterinary Medicine, University of Florida, 2015 SW 16th Avenue, Gainesville, FL 32608, USA
‡Department of Clinical Medicine, Surgery and Animal Reproduction, São Paulo State University, Rua Sergipe 575, ap. 3
São Paulo, 01243-001, SP, Brazil
§Internal Medicine, Davies Veterinary Specialists, Manor Farm Business Park, Higham Gobion, Hertfordshire, SG5 3HR, U
1Corresponding author email: ra.squires@hotmail.com

[Correction added on 23 August 2024, after first online publication: The copyright line was changed.]

Journal of Small Animal Practice • Vol 65 • May 2024 • © 2024 The Authors. Journal of Small Animal Practice © 2024 British Small Animal Veterinary Association

WSAVA Global Vaccination Guidelines

The World Small Animal Veterinary Association (WSAVA) promotes a unified global approach to preventing infectious diseases in companion animals. Through its evidence-based vaccination guidelines, WSAVA aims to improve protection against major pathogens such as canine parvovirus, distemper, and adenovirus.

The initiative highlights the importance of core vaccines, appropriate booster timing, and consistent education for veterinarians and pet owners. By strengthening preventive medicine and harmonizing vaccination practices worldwide, WSAVA supports a coordinated effort to reduce the impact of preventable diseases and improve animal health globally.

SAVE THE DATE

23RD EVECC CONGRESS

4-6 JUNE 2026

EDINBURGH-SCOTLAND

WWW.EVECC-CONGRESS.ORG

3RD JUNE 2026
VECCUS
PRE CONGRESS DAY
POINT OF CARE ULTRASOUND

EVECC Congress 2026

EVECC
EUROPEAN VETERINARY EMERGENCY AND CRITICAL CARE CONGRESS

ECVECC · ROYAL CANIN · B BRAUN SHARING EXPERTISE · BSA · IVC EVIDENSIA