Obstetrical and Gynaecological Emergencies in the Cat and Dog

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Care of the Neonate

A neonate is described as a puppy or kitten from birth to 6 months of age. During this time there are specific diseases that kittens and puppies may obtain.

The paediatric/neonatal patient can be divided into several groups based on age
Neonatal period: Birth to 2-weeks old
Transition period: 2- to 4-weeks old
Socialisation period: 4- to 12-weeks
Juvenile period: 12 weeks to 6 months old

The physical examination

Most errors in diagnosis of neonatal disorders occur during the physical examination. Important factors to consider are the history (information on the dam, littermates pedigree and patient), thorough observation and ancillary diagnostic aids.

Kittens and puppies less than 4 weeks old

A healthy neonate should be fat and sleek and sleep contentedly. Body weight is very important as it is usually the first indicator of illness. Body weights should be collected and recorded at birth, 12 hours later and daily for the first 2 weeks. Rules of thumb for weight gain is that puppies should double their birth weight by 10-12 days and kittens should double their weight by 2 weeks of age.

Species	Birth weights	TPR Data
Canine Toy breeds Medium Breeds Large breeds	 100-400g 200-300g 400-500g	HR > 200 bpm RR = 15-35 bpm Lower body temperature
Feline	100±10 g	HR > 200 bpm RR = 15-35 bpm Lower body temperature

Newborn puppies and kittens should not be left unattended or warmed on heating pads as they lack neuromuscular reflexes until they are 7 days old.

The normal neonatal nervous system: Eyes open at 12-14 days with the iris being blue-gray but it changes to their adult colour by 4-6 weeks of age. Vision is not normal until 3-4 weeks old. PLR's are sluggish until 4-weeks of age. Flexor tone is predominant in the first 4 days of life (comma shape) then extensor tone is dominant (lie with head extended). Pain perception is present at birth but withdrawal reflexes are poorly developed until approximately day 7. Neonates can crawl by 7-14 days of age, and they start walking by day 16 and by day 21 they are walking normally. The ears open at 2 weeks of age and the auditory system is functional by 3-4 weeks.

Examination of the neonate: The clinical examination should never be done on an unheated examination table. A neonate should be plump and round with no gross abnormalities of size or shape. Examine thoroughly for congenital abnormalities. Neonates should exhibit a rooting reflex. Inspect the skull for evidence of cleft palate, open fontanelle and harelip. The nose should be free of fluid. The oral cavity should be inspected for evidence of cyanosis, dehydration, cleft palate, and a sucking reflex. Mucous membranes should be pink and moist.

The respiratory system should be examined for symmetry, wounds, rib fractures, and spinal abnormalities. Breathing should be regular and unlaboured. The chest may be difficult to auscultate for heart murmurs and abnormal heart sounds. However the rate and depth of respiration, heart rate, muscle tone and activity are indicators of cardiopulmonary condition.

Abdominal examination: after feeding the abdomen will be enlarged but the neonate should be content. Abdominal enlargement with restlessness, weakness, vocalisation or complete silence may indicate illness or aerophagia. Inspect the umbilicus carefully for infection or abnormalities. Both micturition and defecation can be stimulated by rubbing the perineum with a warm moist cloth, and urine may be examined for haematuria, abnormal micturition, diarrhoea and constipation. Check the anus for patency.

Puppies and kittens 5 weeks to 6 months of age

Again observation is crucial. Start with examining the animal's response to its environment. At this stage try to make the examination experience positive.

The head: Examine the eyes, ears and oral cavity (especially the tonsillar crypts and under the tongue for burns). Cranial nerve function tests may include PLR's, jaw tone, facial sensation and swallowing reflex. The menace is not fully developed until the animal is 2-3 months old.

Cardiovascular system: Start with a visual inspection looking at respiratory rate, pattern and for evidence of respiratory distress. Then palpate the thorax for conformational abnormalities. Auscultate the thorax in a systematic pattern. Listen to respiratory sounds first then the cardiac sounds. Asymmetry in sounds is very important. Inspect the peripheral veins and assess CRT. Assess the pulse character and deficits, a "water-hammer" pulse (strong pulse fading quickly) may suggest PDA, while slow rising pulse suggests aortic stenosis. Weak thready pulses may indicate shock, hypovolaemia, or CHF.

Heart murmurs may be pathologic or physiologic. Follow up cardiac examinations are advised for any murmur persisting over 4 months of age. Continuous murmurs are almost always associated with PDA. Other murmurs may be systolic or diastolic.

Abdominal examination: Start with the skin and musculature. Genitalia should be palpated for openings. Look for umbilical hernias and assess contents (fat, bowel). Then palpate the abdomen in a systematic approach. Feel for changes in size, shape and texture. Intestinal palpation is very important. Normal intestines feel soft, slightly fluid or gas filled which are freely movable and non-painful.

Testicles should be descended by 4-6 weeks of age, and cryptorchidism should be suspected if the testicles have not descended by 16 weeks of age.

When taking the temperature observe the faeces and use this sample for faecal analysis if required. Examine the rectum and anus for inflammation or congenital effects.

Musculosketal system: Examine all limbs carefully, for symmetry. Firm pressure may be applied to the centre of the bone to detect pain from panosteitis. Palpate growth plates.

Sample Collection and Analysis in the Neonate

Urinalysis
Collect urine by perineal stimulation.
Glycosuria is normal in pups up to 8 weeks old.
Urine concentrating ability is impaired until the neonate is 8 weeks old

Blood Pressure
Lower in the neonate and difficult to record
Mean systolic BP at birth is 61±5 mmHg
Mean systolic BP in week 1-4 of life is 82±6 mmHg

Pulse Oximetry
Can use the hairless skin on the abdomen.
Normal SpO_2 should be greater than 90%

Liver
Can perform pre- and post – prandial serum bile acids at any age

Nervous System
Can collect 0.25 ml CSF for analysis.
Remember in the neonatal and transitional periods the neurological reflexes and responses are not fully developed.

Blood Sample Collection
Jugular venipuncture in lateral recumbency is the best method of blood collection. Gentle aspiration is needed to avoid collapse of the vein.

Normal Haematologic Parameters in the Neonate				
	Canine		**Feline**	
Parameter	**0-2 weeks**	**2-4 weeks**	**0-2 weeks**	**2-4 weeks**
RBC (x 10^6/µl)	3.6-5.9	3.4-4.9	5.0-5.5	4.6-4.8
Hb (gm/dl)	14.0-17.5	8.5-11.6	11.5-12.7	8.5-8.9
PCV (%)	33-52	27-37	33-37	25-27
MCV (fl)	89-93	73-83	65.5-69.3	52.7-55.1
MCH (pg)	28-30	23-25	22.4-23.6	18-19.6
HCHC (%)	32	32	33.7-35.3	32.5-33.5
WBC (x 10^6/µl)	6.8-23	23-25.5	9.1-10.2	14.1-16.5
Bands	0-4.8	0.3-1.2	0.04-0.08	0.07-0.15
Neutrophils	3.8-15.8	1.4-12.8	5.3-6.7	6.2-7.7
Lymphocytes	0.5-9.4	1.0-10.1	3.2-4.2	6.0-7.1
Monocytes	0.2-2.5	0.1-1.5	0-0.02	0-0.04
Eosinophils	0-2.8	0-1.8	0.5-1.4	1.2-1.6
Basophils	0-0.2	0-0.2	0.01-0.03	0

Normal Serum Biochemical Parameters in the Neonate				
	Canine		**Feline**	
Parameter	**0-2 weeks**	**4 weeks**	**0-2 weeks**	**4 weeks**
Bilirubin (µmol/l)	1.71-17.10	0-1.71	1.71-17.10	1.71-3.42
ALT (U/l)	10-337	9-24	11-24	14-26
AST (U/l)	10-194	14-23	8-48	12-24
ALP (U/l)	176-8760	135-201	68-269	90-135
TP (g/l)	34-52	39-42	40-52	46-52
Alb (g/l)	15-28	10-20	20-24	22-24
Cholesterol (mmol/l)	2.9-8.68	6.9-9.1	4.26-11.5	2.9-11.28
Glucose (mmol/l)	2.9-8.1	4.8-6.4	4.3-7.2	5.5-6.3

Normal Serum Biochemical Parameters in the Neonate		
Parameter	**Canine 5 weeks**	**Feline 4-6 weeks**
BUN (mmol/l)	4.2-6.7	NA
Creatinine (µmol/l)	31.8-43.3	44.2-61.9
Na (mEq/l)	143.9-149.9	147-158
Cl (mEq/l)	104.4-112.2	118-127
K (mEq/l)	4.9-6.4	3.7-5.6
Ca (mmol/l)	2.7-2.9	2.1-2.75
Phosphorus (nmol/l)	2.65-3.00	1.6-3.2

Tables from Miller, Current Vet Therapy XIII, 1995

Fluid Therapy in the Neonate

Neonates less than six weeks old are predisposed to dehydration because the extracellular fluid volume is higher, the renal capacity to conserve water is reduced, they have a large body surface area, and fluid loss through immature skin is higher. Secondly, the brain volume decreases during the first two days of life. Hypovolaemia or hypotension predisposes the neonate to brain haemorrhage especially if this is followed by rapid volume replacement with hyperosmolar fluids.

Estimation of dehydration by assessing skin turgor is ineffective in the neonate as they have higher water content in the skin. You must assess mucous membranes and urine colour (should be clear).

Fluid types

Lactated ringers solution, Ringers solution, 0.9% NaCl, and 5% dextrose solutions are most useful. Neonates may be unable to metabolise lactate to bicarbonate so Ringers solution may be better. Fluids should be warmed to body temperature before use.

Routes of administration

- Intravenous: Access via jugular*/or cephalic veins.
- Subcutaneous: Can use for maintenance fluid therapy only. It can be used in replacement of acute or chronic fluid losses provided the neonate is not dehydrated or hypotensive.
- Oral: Maintenance fluids provided they are not vomiting.
- Intraosseous: Use where vascular access is difficult. Sites include the tibial tuberosity, trochanteric fossa of the femur*, wing of ileum, and greater tubercle of the humerus. Fluid rates of up to 11 ml/min can be given by gravity. An 18-20g spinal needle is best. Place aseptically and it can be left in-situ for 72 hours. Manage as an IV catheter. Potential complications are osteomyelitis and pain.
- Intraperitoneal: This route is not recommended, as there is a high risk of peritonitis.

Administration of fluids

- Shock rates are 40-45 ml/kg/hr
- Maintenance rates are 40-50 ml/kg/day
- Monitor response to fluid therapy by frequent reassessment of clinical signs, measuring body weight, and assess cardiopulmonary and urinary function.
- Failure to correct dehydration may be due to underestimation of extent of dehydration or fluid losses or fluids are being administered at an excessive rate resulting in urinary and electrolyte losses. You can increase the daily fluid volume by an amount equivalent to 5% body weight to restore losses.

Complications of fluid therapy

- Overhydration with rapid infusion of fluid may result in vomiting, polyuria, exopthalamus diarrhoea, serous nasal discharge, chemosis, restlessness, tachycardia, cough, altered breathing pattern, pulmonary crackles and ascites.
- Catheter placement must be placed aseptically and checked at least daily for cleanliness, local pain and swelling.

Discontinuation of fluid therapy

- Stop when hydration is restored and the puppy or kitten can maintain fluid balance by eating and drinking
- Decrease fluid volume by 25-50% a day until weaned from fluids. At this stage you may change the route of fluid administration as well (IV to SQ).

Paediatric Intensive Care

Birth to 2week old kittens and puppies

A healthy neonate is firm, plump and vigorous. Crying occurs in response to pain, coldness, failure to nurse or loss of contact with the mother. MM's are usually hyperaemic until days 4-7 post partum.

A sick neonate is limp, relaxed with poor muscle tone. MM's are pale, gray, or cyanotic. They have reduced bowel sounds and approximately 60% of sick neonates have diarrhoea.

Care of the sick neonate

- ***External warming must be provided***
  Ideally a neonatal incubator can be used to maintain environmental temperature between 29-32°C and a humidity of 55-65%
  Hot water bottles, rice bags and heating pads can be used but the neonate must be able to move away from the heat source and the heat source covered with a towel to avoid burns

- ***Hypothermia (24-35°C) is common***
  Associated with respiratory depression, bradycardia, gastrointestinal paresis and coma.
  Must rewarm slowly
  Cannot commence oral nutrition until the neonate is warmed, as digestion is impaired
  Warm inspired air via an incubator or oxygen cage is the best method; alternatively you can give warmed fluids or a warm water enema

- ***Hypoglycaemia***
  Results from depletion of glycogen stores and immature hepatic function
  Test for in all sick neonates
  If not dehydrated or hypothermic give oral glucose (1-2 ml 5-15% dextrose solution)
  If mildly dehydrated give 2.5% dextrose and 0.45% NaCl SQ
  If severely depressed and/or dehydrated give parenteral glucose (0.25 ml/25g BWt 20% dextrose)

- ***Hydration***
  Dehydration may occur rapidly in neonates due to their rapid total body water turnover
  Hydration status can be difficult to assess. Skin turgor is unreliable as a result of increased water and decreased fat content of the skin
  Pale MM's, slow CRT in the absence of anaemia indicate circulatory collapse and 12-15% dehydration
  Treat with warmed IV fluids @ 1ml/30g BWt over 5-10 minutes. Continue fluid loading until colour and CRT improve with reassessment at 30 minute intervals until the patient is stable, then continue at maintenance rates

- ***Nutrition***
  Commence when dehydration and hypoglycaemia are corrected, and a suckling reflex is present
  Use a puppy or kitten milk replacer
  Stomach capacity is approximately 50 ml/kg
  Puppies should be fed 10 ml q 4-6 hours, gradually increasing by 1 ml/feed
  Kittens are fed 5 ml q 4-6 hours gradually increasing by 1 ml/day
  If diarrhoea occurs dilute the formula 1:2 with a balanced electrolyte solution
  Feed using a bottle, syringe or feeding tube. Care when using feeding tubes as there is an increased risk of improper placement, as the gag reflex does not develop until 10 days of age

- ***Vitamin K***
  Administer to any sick neonate less than 48 hours old or exhibiting signs of haemorrhage
  Give @ 0.5-2.5 mg/kg SQ or IM
  Puppies have decreased thrombin levels at birth making them more prone to haemorrhage

- ***Respiratory support***
  Respiratory distress may result from aspiration of amniotic fluid, atelectic lungs, or anoxia from premature placental separation
  Often present as failure to suckle following birth
  Puppies may have pulmonary contusions due to trauma from the dam
  Use supplemental oxygen

Specific Disease Disorders

1. ***Poor sanitation***
   May lead to infections of skin, eyes and umbilicus.
   Neonatal dermatitis results in crusting lesions of the head and neck at 4-10 days of age. Treat with a bactericidal shampoo and systemic antibiotics.
   Neonatal conjunctivitis occurs when a purulent exudate accumulates behind the eyelids before they completely open. Treat by gently separating the eyelids, cleaning and topical antibiotics.

2. ***Umbilical infections***
   Occur within the first 4 days of life.
   Due to faecally derived bacteria or *Streptococcus spp*.
   Treat by draining and flushing the abscess, providing IV fluids, antibiotics and any necessary supportive care.
   Prevent by applying antiseptic solution to the newborns umbilicus, administering peripartum antibiotics to dams with known genital infections. Ensure the whelping quarters are kept clean and dry.

3. ***Neonatal septicaemia***
   Results from a systemic infection.
   Can be secondary to lack of colostrum intake or to maternal infections such as mastitis or metritis.
   Organisms involved include *Eschericia coli, Staphylococcus spp, Streptococcus spp, Klebsiella spp, Enterobacter spp, Salmonella spp, Pasturella spp,* and *Pseudomonas spp*. Gram negative bacilli are most common.
   Bacteria enter the blood stream from the GIT, peritoneal cavity, respiratory tract, skin and associated wounds and urinary tract.

4. Viral infections

Canine herpesvirus and canine parvovirus type 1 may cause a rapid and fatal syndrome in puppies starting with crying, lethargy and anorexia followed by death.

CHV infections occur during late pregnancy or within the first 3 weeks of life. Severity of infection depends on maternal herpesvirus antibodies, stress and the presence of concurrent bacterial infections. CHV infection in-utero may result in foetal death, mummification, abortion, or neonatal death. Most puppy deaths are between days 9-14 of life. Clinically puppies present with sudden onset illness with crying, depression, anorexia, abdominal discomfort, bloating, shallow respiration, hypothermia and profound weakness (a sick puppy). Commonly infected pups die within 18-24 hours. CHV replicates best at low core body temperatures so warming is vital as well as supportive care.

5. Toxic milk syndrome

May result in bloating, green diarrhoea, crying and a red and oedematous rectum.

Remove puppies from the bitch and feed a milk replacer.

Look for underlying disease such as metritis and mastitis.

6. Fading Puppy or Fading Kitten Syndrome

Defined as neonates that die at birth or fail to survive the first 2 weeks of life. May be healthy at birth. Can include losses up to 12 weeks of age.

Losses of 15-40% can be expected in the first 12 weeks of life.

Generally occur as a result of congenital abnormalities, teratogenic effects, nutritional diseases resulting from the dam/neonates being fed an improper diet, abnormally low birth weights, traumatic insults during/after the birthing process, neonatal isoerythrolysis, infectious disease and environmental factors.

Lung abnormalities: May result in death at birth or during the first 2 weeks of life. Often identified at the first examination of after failing to thrive or with limited exercise tolerance.

Anatomic abnormalities: Include cleft palate, cranial deformities, agenesis of small or large intestine, cardiac abnormalities, extensive umbilical or diaphragmatic hernias, anomalies of the kidneys and lower urinary tract and musculoskeletal problems.

Teratogenic defects: Avoid the administration or application of any drug during pregnancy, especially those known to be teratogenic.

Nutrition: Dams fed poor quality diets during pregnancy may produce diseased or weak puppies or kittens. Taurine deficiency in cats may result in foetal resorbtion, abortion, still births, poor growing young. Malnutrition may also be due to a poor maternal blood supply (foetal competition).

Low birth weight: Associated with higher puppy or kitten losses. The birth weight is not affected by sex, litter size or weight of the dam. Small stature most likely due to lung anomalies or poor nutrition. Low birth weight is associated with a greater likelihood of still births or deaths during the first 6 weeks of life. A proportion of underweight kittens and puppies tend to be chronic poor doers. Many faders are normal size at birth but their growth rate is slow and they have below normal weights at the time of death.

Traumatic insults: Occur during the first 5 days of life. Associated with cannibalism, maternal neglect and dystocia. Often sickly kittens/puppies are cannibalised.

Infectious disease: Bacterial infections common post-weaning (5-12 weeks). Most deaths are attributable to primary infections of the respiratory tract, gastrointestinal tract or peritoneal cavity. Environmental factors are important, if unfavourable, illness is more severe and can result in high losses. Viral infections.

Miscellaneous factors: Intestinal parasitism, fatty liver syndrome.

Cat factors: Losses are lowest in the 5th litter; the 1st litter and greater than 5 litters have higher losses. Medium sized queens have lower litter losses, and losses are lowest if they have 5 kittens. Losses are highest in single kitten litters.
Investigation of fading puppies or kittens is warranted if pre-weaning losses are > 20% or post-weaning losses are >10%. You should also investigate litter losses if there are disproportionate losses due to a single cause. Investigation is based on a thorough clinical examination including history, physical examination, routine laboratory tests and a complete and accurate necropsy (culture, toxicology, and histopathology)

7. Neonatal isoerythrolysis

Immunogenetic disorder resulting from damage to the neonate's erythrocytes by maternal colostral acquired antibodies.
Incidence is low in DSH cats but can be responsible for up to 50% of neonatal deaths in certain pure-bred catteries that have not blood-typed parents.
Occurs only in the first few days of life as it is the only time exogenous Ab can gain access in the natural setting.
Colostral Ab's are directed against surface Ag's on neonatal erythrocytes resulting in accelerated removal or destruction of circulating RBC's, leading to anaemia.
Cats have three antigenic epitopes, A, B, and AB. All type B cats have strong haemagglutin and haemolysin against type A cells. Type A cats have low anti-B Ab's. These Ab's are naturally occurring.
Feline blood types A and B are erythrocyte phenotypes due to the action of two different alleles at the same autosomal gene locus. A is completely dominant over B, therefore cats with type A have AA or AB genotype. The AB blood type could be explained by the action of a third allele at the same gene locus that is recessive to A but dominant over B. Only homozygous BB genotype express blood type B.
All B type cats have strong naturally occurring alloantibodies, thus fatal neonatal isoerythrolysis (NI) can occur in type A offspring of type B cats bred to type A males. Matings of two type B cats can only result in type B offspring. If the father is genotype AA, all offspring are AB and are at risk of developing NI. If father is genotype AB half the offspring are AB and at risk of developing NI.
Neonates acquire maternal IgG Ab's via colostrum during the first 2 days of life. Between 6-8 weeks of age, kittens produce their own alloantibodies.
No previous transfusion or pregnancy is required for the production of alloantibodies in cats.
If the kitten is type A or AB and the queen type B, colostral Ab's bind and lyse erythrocytes in the kitten resulting in haemolysis (intra- and extravascular) and anaemia, chromoprotienuric nephropathy, organ failure and DIC.
Kittens are born healthy and nurse vigorously. Following colostrum intake the kittens show clinical signs within hours to days. May die peracutely or fail to thrive.
Key clinical sign is dark red-brown urine = haemoglobinuria. Can also develop anaemia and icterus, hypoglycaemia.
Surviving kittens may develop tail tip necrosis with sloughing of the tail tip between 3 days and two weeks of age. This may be due to cold reacting IgM Ab's leading to haemagglutination in capillaries.
Some kittens may only have failure to thrive if subclinical NI.

Management of NI

Kittens showing signs should be removed from the queen immediately to prevent further absorption of maternal Ab's. Duration of Ab transfer is approx. 12 hours, therefore you only

need to remove the kitten for the first 16-24 hours. Foster feed affected kittens with commercial milk replacer or place on a type A queen.
If severely anaemic, must replace erythrocytes in order to maintain tissue oxygen delivery. Must type the donor to ensure that it is compatible with queens' type B colostral Ab's. The queen is the ideal donor. Collect 2-3 ml of blood into EDTA or heparin and spin at 1000 rpm for 1 minute, remove supernatant and wash cells with an equal volume of saline, spin and resuspend in saline and transfuse. The trochanteric fossa is good. If greater than 3 days transfuse with type A blood.
Can avoid by preventing matings between type B queens and type A males
Can cross match blood with queen and kitten if suspicious.

Blood Cross Match Procedure

1. Use placental blood to do cross match with queens serum
2. Have a donor and control tube
3. Fill both tubes to 75-80% full with 0.9% saline
4. Add a drop of donor blood to each tube
5. Spin for 15 seconds @ 3500 rpm and discard supernatant
6. Add 2 drops of queen serum/plasma to donor test tube and 2 drops saline to control tube
7. Centrifuge and discard supernatant
8. Check tubes for agglutination

Feline Blood Types Based on Breed

Breed	% Type A	% Type B	AB
Siamese	100	0	
Burmese	100	0	
Tonkinese	100	0	
Russian blue	100	0	
DSH/DLH	90-99	1-10	Y
Maine coon	90-99	1-10	
Norwegian forest	90-99	1-10	
Abyssinian	80-89	11-20	Y
Birman	80-89	11-20	Y
Himalyan	80-89	11-20	
Persian	80-89	11-20	
Scottish fold	80-89	11-20	Y
Exotic and British shorthair	55-80	20-45	Y
Cornish and Devon rex Bengal	55-80	20-45	Y

2 to 6 Week Old Puppies and Kittens

During this period of life the most life threatening conditions are internal and external parasites, juvenile hypoglycaemia, dehydration from diarrhoea and trauma.
Renal function does not mature until 8 weeks of age so neonates are prone to dehydration and drug toxicities due to poor renal elimination.

1. ***Internal parasites***
   Can have a significant burden by 2-4 weeks old.
   Toxocara spp can be transmitted transplacentally and *Ancylostoma spp* can be transmitted in the dam's milk.
   Prevent by good parasite control in the dam prior to parturition.
   Toxocara spp cause weight loss, unthriftiness, abdominal distension and diarrhoea.
   Ancylostoma spp can cause a severe anaemia, which may require a whole blood transfusion. (Give intra-osseous or IV, can give via the intra-peritoneal route but very slow absorption of RBC's with 75% of RBC's absorbed over 72hr compared with 95% absorption in 5 minutes with IV/IO routes).
   Iron supplementation advised post transfusion.
   Treat worm infestation with pyrantel pamoate from 2-3 weeks old, every 2-3 weeks until 12 weeks old.

2. ***Protozoal parasites***
   Cause diarrhoea.
   Giardia and *Coccidia* common.
   Treatment for *Giardia* includes metronidazole 30 mg/kg PO for 7-10 days (care with toxicity) or febendazole 50 mg/kg SID for 7 days.
   Treat coccidiosis with sulfadimethoxine 50 mg/kg on day one, then 25 mg/kg daily until signs regress.

3. ***External parasites***
   Can be quite severe infestation resulting in severe anaemia
   Transfusion if anaemia very severe
   Ectoparasite treatment licensed for young kittens and puppies

4. ***Juvenile hypoglycaemia***
   May result from immature hepatic enzyme systems, lack of glycogen stores, increased metabolic requirements for glucose.
   Present with weakness, tremors, seizures, stupor and coma.
   Treat with intravenous or intraosseous glucose (0.5-1.0g/kg diluted to 5-10%).

5. ***Fatty liver syndrome***
   Causes ill-thrift in toy breed puppies at 4-16 weeks of age.
   Present with a sudden onset of severe illness with depression, anorexia, persistent crying, diarrhoea, rapid and shallow respiration, hypothermia, seizures and profound weakness.
   The disease usually ends in death 1-6 days later.
   Treat with glucose and general supportive care.

6. ***Dehydration***
   Potential problem in any neonate with diarrhoea.
   Give fluids IV or IO or SQ at 1-2 times maintenance (60-120 ml/kg/day).

Causes include overfeeding, lactose intolerance, excess solids, excess saturated fatty acids, parasites, infection, toxaemia, or improper handling of a milk replacement diet.

6 to 12 Week Old Puppies and Kittens

- Similar to adults in terms of renal function, body temperature, and vital signs
- They still have an increased maintenance water requirement (120-180 ml/kg/d) and caloric requirement (220 kcal/Kg/d)
- Maternal antibody is lost during this period and they are susceptible to infectious disease
- Life threatening diseases include FeLV, FIP, panleukopenia virus for kittens and canine distemper and viral enteritis in puppies.
- Other common emergencies include foreign body ingestion and electrical cord injury

1. ***Electrical Cord Injury***
   May have burns on lips, tongue, or oral mucosa.
   Consequence of electric shock is fulminant pulmonary oedema, that progressively worsens over the first 24 hours.
   Treat with supplemental oxygen therapy, furosemide (2mg/kg IV q 6-12 hours), ventilation, bronchodilator therapy.
   Most recover if survive the first 48 hours.

2. ***Juvenile Cellulitis***
   May be called puppy strangles or juvenile pyoderma.
   Idiopathic skin disease in pups 3-16 weeks old.
   Have facial swelling, lymphadenopathy, deep pyoderma of the head and face, fever, anorexia, and depression.
   No causative agent has been identified.
   Treat with topical antibacterial shampoos, cephalexin 20 mg/kg TID and immune-suppressive doses of prednisolone 2.2 mg/kg SID and tapering when symptoms resolve.
   Without treatment the disease can be fatal.

3. ***Canine Parvovirus Enteritis***
   Can be a rapidly fatal disease in pups aged 6 weeks to 6 months if not aggressively treated.

   Aetiology:
   Undeveloped DNA virus.
   May have acute myocarditis, haemorrhagic enteritis, or neonatal mortality.
   Can survive months to years in the faeces.
   Faecal infected fomites main cause of spread.
   Rotweillers and Dobermans predisposed.
   Normal bacterial flora is important in the course of the disease. They increase the rate of epithelial turnover exacerbating the disease and invade mucosa to produce sepsis.

   Pathogenesis:
   Initial replication in the lymphoid tissue of the oropharynx.
   During the subsequent viremia, virus enters rapidly dividing cells in bone marrow, lymphopoeitic tissue and crypt epithelium of the jejunum and ileum. Viremia terminated by development of serum neutralising antibody at 5-6 days PI.
   Virus replication in lymphoid tissue and bone marrow results in lymphopenia and neutropenia. Replication in crypt epithelial cells leads to collapse of intestinal villi, epithelial

necrosis and haemorrhagic diarrhoea. This allows normal bacterial flora to enter the mucosa and in combination with the immune suppression sepsis results.
If the puppy has partial immunity the consequences of viremia, immune suppression, and intestinal necrosis are less severe.

Clinical Features:
Incubation period is 5-10 days.
Clinical signs most common in dogs < 1 year (>75% between 6-18 weeks).
Depression, anorexia, pyrexia and vomiting are early signs.
Diarrhoea varies from soft and watery to haemorrhagic and fetid.
Leukopenia present in 80% of dogs at some stage. Lymphopenia and neutrophilia predominant.

Diagnosis:
Diagnosis made on appropriate clinical signs.
Faecal agglutination or ELISA test positive. This should be done on all suspected puppies. Care with recently vaccinated puppies as they may have a false positive result (2 weeks post vaccination).
Detection of virus particles in the faeces.

Treatment:
Affected puppies should be isolated from other hospitalised animals. Disinfect contaminated area with a household bleach diluted 1:30.
Supportive treatment required.

(a) Fluid therapy is the cornerstone of treatment. Restore fluid deficits as determined on Dextrans 70 @ 20 ml/kg. Avoid hypertonic saline as the puppy is already severely dehydrated.
Rehydrate the pup over 4 hours with a replacement electrolyte solution @ 3-10 ml/kg/hr. Remember to include estimated fluid losses into your fluid therapy plan. If Dextrans 70 or pentaspan is used less fluid is lost into the GIT and the total volume of fluid required can be reduced by up to 50%.
Once rehydrated maintain the pup on IV fluids with potassium chloride added. Hydration can be assessed by monitoring MM colour and refill, pulse quality, PCV and TP and urine output (1-2 ml/kg/hr).
Septic or ill puppies may become hypoglyceamic. Add dextrose to make a 2.5-5% solution in maintenance fluids.
Plasma or blood as required. If anaemic; whole blood is best. The best donor is a recovered parvovirus patient as it will have high titres of parvovirus antibody. Give 10-20 ml/kg over a 4 hour period.
If the pup is hypoproteinaemic, give plasma @ 10-20 ml/kg over 2-4 hours. Plasma also contains antibodies and serum protease inhibitors that may neutralise circulating virus and inflammatory mediators. Give the plasma early if indicated.
Continue colloid therapy if hypoproteinaemic @ 10 ml/kg BID or CRI 1 ml/kg/hr.

(b) Antibiotics
Trimethoprim sulpha unless sepsis present. If the patient severely depressed, has a profound neutrophilia (< 1000/µl) or evidence of sepsis a combination of ampicillin, amoxicillin, cephalosporin and gentamycin should be used.
Gentamycin is nephrotoxic. However once daily dosing in adult dogs has been shown to be effective and safe with no evidence of nephrotoxicity. I have used this in severely affected parvovirus puppies with no ill effects. In puppies receiving gentamycin ensure

they are adequately rehydrated prior to use and ensure they are on adequate IV fluid rates.

(c) Antiemetics/prokinetics

Metaclopramide CRI should be used as pups have an adynamic ileus which predisposes to bacterial overgrowth, and it is an antiemetic.

Ensure the pup does not become hypokalemic as this contributes to weakness and ileus.

If vomiting persistent add a phenothiazine antiemetic or droperidole to the therapy.

Intractable vomiting may respond to onandasteron (0.1-0.5 ml/kg q 6-12 hours). This works well in very ill puppies.

Naso-gastric tube for suctioning and nutrition.

(d) Analgesia with butorphanol or buprenorphine.

(e) Gut protectants

Puppies with intractable vomiting may develop an oesophagitis complicating recovery. Use famotidine 0.5 mg/kg IV or ranitidine 2 mg/kg IV twice daily.

(f) Immunotherapy

Use fresh frozen plasma.

In America you can purchase antiendotoxin serum to help neutralise bacterial endotoxin.

(g) Eliminate Intestinal Parasites.

The presence of intestinal parasites can exacerbate parvovirus enteritis by enhancing intestinal cell turnover and subsequent viral replication. Worm when vomiting ceases or give ivermectin 250 µg/kg SQ in non-collie breed dogs.

(h) Nutritional support

Very important!

Early enteral nutrition promotes intestinal regeneration. Start with lectade @ 0.1-0.4 ml/kg PO q 4 hours. Can also use Vital HN to provide further nutrition. Glutamine can be added @ 0.5 g/kg to promote intestinal healing as it is preferentially an energy source for gut cells. Early enteral nutrition has been shown to improve the puppy's recovery from parvovirus enteritis. Start feeding as soon as the pup comes into the hospital, even if it is vomiting.

Once vomiting has ceased start on an easily digestible, low-fat diet such as Hill's I/D or Royal Canin Intestinal diet in small frequent feeds. Once recovered gradually reintroduce the regular diet.

Complications:

Sepsis

Intussuception – palpate abdomen every 4 hours for early detection.

Adynamic ileus.

Intractable diarrhoea as a result of irreparable mucosal damage or persistent inflammation.

Prevention:

Vaccination.

Vaccination failure is commonly due to inadequate quarantine or hygiene practices. Have an immunity gap that cannot be avoided.

In an outbreak, bleach (1:30) is a good disinfectant. Can vaccinate with a modified live vaccine to offer protection as may get protective immunity in 3-4 days if seronegative.

Prognosis:
Most dogs that survive the first 2-3 days of treatment will recover.

4. ***Feline Panleukopenia***
   Similar to CPV but has a propensity to produce cerebellar disease if infected in-utero or in the early neonatal period.
   Anaemia more severe and usually require blood transfusions.
   Include vitamin B supplements in the nutrition protocol.

Pyometra

Cystic endometrial hyperplasia-pyometra complex is a potentially life threatening uterine disease that can affect both cats and dogs.

Pathogenesis

Cystic endometrial hyperplasia (CEH) is a progesterone mediated uterine disorder and it is usually the first lesion in the development of the disease. Normally progesterone stimulates the growth and secretory activities of the endometrial glands, which in turn promote bacterial growth. In the luteal phase of the oestrus cycle progesterone suppresses intrauterine leucocyte function in response to foreign stimuli, and it decreases uterine contractility. These functions are a prerequisite for normal pregnancy.

The non-gravid dioestral uterus is flaccid and contains secretions previously elaborated in response to oestrogen, however these secretions are also a potential medium for bacteria. Therefore, oestrogen may augment the effects of progesterone enhancing the development of CEH. Oestrogen may dilate the feline cervix allowing contamination of the uterus. Oestrogens from any source (endogenous or exogenous) enhance the stimulatory effect of progesterone by sensitising receptors to and enhancing the binding of progesterone. These effects can last for 9-12 weeks in normal bitches. Pyometra can develop in the absence of CEH.

Bacteria may reach the uterus by ascension from the genitourinary tract or haematogenous routes. Bacterial colonisation is normal in proestral or oestral bitches, however this overgrowth is cleared before it becomes a clinical problem. Bacterial infection results in the morbidity and mortality associated with pyometra. *Eschericia coli* is the most common isolate. Chen et al (2003) demonstrated that the virulence factors were identical to uropathogenic strains of *E. coli* that cause urinary tract infections in dogs and humans. These virulence factors appeared to enhance the pathogenicity of the organism in pyometra. Hagman (2002) identified identical isolates from both the urinary tract and the uterus of bitches with pyometra. These species of *E. coli* may adhere to specific antigenic receptors in the progesterone stimulated uterus. *E. coli* is a gram negative organism with a biologically active lipopolysaccharide endotoxin in the cell membrane. Endotoxin is released when these organisms die and will result in clinical endotoxaemia when serum levels exceed 0.05 ng/ml. Endotoxin release may be enhanced by antibiotics.

Other bacteria identified in pyometra include staphyloccoci, streptococci, *Klebsiella, Pseudomonas, Proteus, Haemophilus, Pasteurella* and *Serratia*. Intrauterine bacteria alone do not account for the pathogenesis of pyometra. Significant uterine disease or exposure to progesterone or oestrogen predisposes the bitch to pyometra. Contributing factors to the

development of pyometra are CEH, bacteria, dioestrus and elevated serum progesterone concentrations, exogenous progesterone or oestrogen admission.

There is a strong correlation between the onset clinical signs and recent dioestrus. The incidence is higher in bitches as they are spontaneous ovulators.

The incidence is increased when exogenous oestrogens are administered to bitches potentiating the effects of progesterone (see above), and if exogenous progesterone is given to queens.

Pathogenesis of polydipsia and polyuria: (1) Bacterial endotoxin affects sodium and chloride resorption in the loop of Henle, altering renal medullary hypertonicity and water resorption making urine dilute. (2) Renal tubular insensitivity to vasopressin results in a secondary nephrogenic diabetes insipidus (Vasopressin increases the reabsorption of water in the collecting ducts and increases the permeability of the medullary collecting duct to urea). (3) Renal tubular immune complex injury may also play a role. (4) Renal medullary washout may further impair urine concentrating ability.

Signalment and History

Pyometra can occur in bitches and queens of any age. In young bitches it is associated with exogenous oestrogens used for mismating injections. It is often diagnosed as early as standing heat and as late as 12-14 weeks after standing heat in bitches. The severity of the clinical signs depends on how quickly the client recognises the problem.

Any breed can be affected. There may be an increased risk in rough coated Collies, Rottweilers, Cavalier King Charles, Golden Retrievers, and English Cocker Spaniels. Breeds with a low risk include German Shepherds! and Dachshunds.

There appears to be little correlation in queens having previous litters being more susceptible to pyometra.

Open-cervix pyometra cases often have a vaginal discharge varying from serosanguinous to mucopurulent. In bitches this discharge is usually first noted between 4-8 weeks after standing heat. Other common signs are lethargy, depression, inappetance/anorexia, polyuria, polydipsia, vomiting and diarrhoea.

Closed-cervix pyometra bitches and queens are often severely ill at presentation, as there is no history of obvious vaginal discharge. Affected animals often have depression, lethargy, inappetance, polydipsia and/or polyuria, and weight loss. Cats may have an unkempt coat. They may also have vomiting and/or diarrhoea, progressively worsening dehydration, shock, coma and death.

Physical Examination

Abnormalities detected include depression, dehydration, fever, palpable uterine enlargement, and vaginal discharge. If the patient is septic or toxic they may have tachycardia, prolonged CRT, weak femoral pulses and hypothermia. Abdominal enlargement may be seen in the queen.

Avoid overzealous uterine palpation as it may result in uterine rupture.

Clinical Pathology

Haematology: The white cell count is variable. They may be neutropenic, neutrophilic with a varying degree of left shift, or the WCC may be normal. White cell counts are more likely to be elevated if the animal has a closed-cervix pyometra. There may be a normocytic, normochromic non-regenerative anaemia due to chronic disease. Septicaemia and toxaemia can suppress the bone marrow.

Biochemistry: Affected bitches may have hypoproteinaemia and hyperglobulinaemia. There may be a mild azotemia, and mild heptaocellular damage may be present secondary to dehydration and septicaemia. Cats may show similar biochemical changes.

Urinalysis and Urine Culture

The USG is very variable. Concentrated urine is secondary to dehydration. Dilute urine is secondary to PU/PD. Isothenuria or hyposthenuria is common in bitches with pyometra.

Urinary tract infections should be suspected if pyuria, haematuria and/or proteinuria are present. 30% of dogs with pyometra have a urinary tract infection. The proteinuria may be renal in origin due to immune complex deposition in the glomeruli. Midstream urine collection may result in false cultures due to contamination with vaginal flora. Blind cystocentesis should be avoided as there is a risk of puncturing the infected uterus. Ultrasound guided cystocentesis is best.

Radiology and Ultrasonography

Apart from pregnancy, the uterus should not be able to be identified. Radiographic visualisation of the uterus is abnormal. In pyometra the fluid filled uterus is larger in diameter than small intestinal loops and the loops of intestine are displaced cranially and dorsally. Look for evidence of peritonitis. Remember inability to visualise the uterus does not rule out pyometra.

Ultrasonography can determine uterine size, uterine wall thickness and the presence of fluid in the uterine lumen. Foetal remnants or placental tissue can be seen which can affect the success of medical therapy. We can also differentiate a gravid from non-gravid uterus. It is also useful for detecting stump pyometra.

Differential Diagnosis of Pyometra

Pyometra is a potential differential diagnosis in any intact female with polyuria and polydipsia and diabetes insipidus and pre-renal azotemia.

Stump Pyometra

This is an uncommon and difficult problem to diagnose as it involves inflammation and infection of the post-ovariohysterectomy remnant of the uterine body. If ovarian remnants are left at surgery the uterine remnant is under the normal influences of progesterone and oestrogen. It can be difficult to diagnose if the cervix is closed however, the clinical signs are similar to a normal pyometra. Ultrasonography is the best non-invasive method to diagnose stump pyometra. Many require an exploratory laparotomy to diagnose.

Treatment of Pyometra

Severely ill animals should be treated with IV fluids and IV antibiotics. Hypertonic saline and dextrans 70 is useful in treating septic shock. Bitches have a 10-15% incidence of bacteraemia and it is believed to be similar in queens.

Antibiotics effective against *E. coli* include clavulox, trimethoprim-sulpha, and cephalosporins. These should be continued for 7-10 days.

Correct acid-base and electrolyte disturbances and commence surgery as soon as the patient is adequately rehydrated. Severely septic animals are not candidates for medical management. Surgical removal of the uterus removes the septic focus enabling treatment of the secondary effects of sepsis. Therefore, surgery should not be delayed more than a few hours in a severely sick animal.

Emergency Medical Treatment of Pyometra

1. Assess severity of shock based on physical findings and clinical pathology
2. Collect ACT, PCV/TP, glucose and possibly haemogram
3. Place IV catheter and start isotonic crystalloid (0.9% NaCl or Lactated Ringers)
   (a) Replace estimated fluid deficit in 1-2 hours
   (b) If showing signs of shock - 90 ml/kg in the first hour for dogs
   40 ml/kg in the first hour for cats
   (c) If stable haemodynamically start at 5-10 ml/kg/hour
4. If septic shock or severe hypovolaemia
   Use hypertonic saline-dextrans to rapidly restore blood volume and improve perfusion.
   7% NaCl @ 4 ml/kg over 5-10 minutes
   70% Dextrans 70 @ 5 ml/kg over 5-10 minutes
   Continue on crystalloids at 5-10 ml/kg/hour
5. Start parenteral antibiotic therapy
   (a) If showing signs of septicaemia or hypovolaemia start IV
   (b) If stable haemodynamically give IM or SQ
   (c) As a rule start IV then continue orally once eating and drinking
   (d) Clavulox, trimethoprim sulpha, and cephalosporins are good choices.
   (e) Continue for 7-10 days
6. If a closed-cervix pyometra, restore normal haemodynamic parameters over 1-2 hours then take to surgery to remove septic focus
7. If pyometra surgery can be delayed until the following day or treated medically
8. Post surgery monitor for signs of septicaemia. In addition to IV fluids and IV antibiotics, treat septicaemia with colloids (10-20 ml/kg/day as CRI or twice daily boluses), fresh frozen plasma, packed red cells, gut protectants and antiemetics as required based on clinical pathology and patient monitoring

Surgical treatment

Ovariohysterectomy is the treatment of choice for pyometra.

The surgical procedure for ovariohysterectomy is described in any surgery text. However, the following precautions should be taken.

1. Handle the uterus carefully as it is very friable and may contribute to septicaemia.
2. Isolate the uterus from the abdomen with laparotomy sponges to avoid contamination.
3. Use triple clamping technique.
4. Tie off the uterine body at the cranial cervix to avoid leaving any uterine body. The remaining stump can be lavaged with saline and suctioned prior to release.
5. Oversewing the cervix is unnecessary unless the cervix is greatly distended. Disadvantages of oversewing include potential for abscess formation in the cavity between the stump and cervix; the remaining stump tissue and suture material can develop into a stump granuloma; and oversewing increases surgery time.
6. If uterine rupture has occurred lavage the abdomen with copious quantities of warm saline and treat aggressively for septic peritonitis (plasma, colloids, triple antibiotics, analgesia, and fluid therapy).

Surgical drainage has been described in dogs and cats to treat pyometra but it is not recommended.

Medical treatment of pyometra

Medical treatment is only recommended if the client has a very valuable bitch or queen who is less than 6 years old. It is contraindicated in severely ill patients or closed-cervix pyometra. Prostaglandin therapy is also contraindicated if there is a planned pregnancy, the presence of sepsis or septic peritonitis, significant organic disease, or the presence of mummified foetal remains.

Prostaglandin $F_{2\alpha}$ can be used in open-cervix pyometra where it exerts effects on the uterine myometrium, cervix and corpus luteum.

1. Prostaglandin $F_{2\alpha}$ stimulates uterine motility causing fluid to move towards the cervix. Women have reduced receptor numbers closer to the cervix and less smooth muscle.
2. Luteolytic effects of prostaglandin $F_{2\alpha}$ result in reduction of progesterone levels.
3. Relaxation of the cervix. This is variable.
4. Pregnancy must be ruled out before use, as prostaglandin $F_{2\alpha}$ is abortifacient. Use will cause abortion secondary to luteolysis in bitches after day 30, and queens day 40 of pregnancy. Placental progesterone is insufficient to maintain pregnancy until at least day 45 of pregnancy.

Hospitalisation during treatment is recommended and the patient checked three times a day. The patient should also be on bactericidal systemic antibiotics for 14 days. It has been suggested that you treat the patients in the morning then they can go home each evening.

Give Prostaglandin $F_{2\alpha}$ @ 0.1-0.25 mg/kg SQ q 24 hours for 3-7 days.

Following administration of the drug there are predictable physical reactions to the drug. These signs include panting, restlessness, salivation, emesis, tenesmus, diarrhoea, urination, mydriasis,

grooming, lordosis and kneeding (queen). Rarely bitches may develop an acute shock-like syndrome. These reactions diminish in severity and duration with subsequent treatments. These adverse reactions are due to the normal physiologic effects of endogenous prostaglandins.

Clinical response is not usually observed for at least 48 hours following commencement of therapy.

Patients should be rechecked at 7 days to ensure the bitch/queen is not clinically worse and consider ovariohysterectomy if she is deteriorating, and at 14 days post treatment for a normal sized uterus. Many bitches still have a vaginal discharge at this stage despite treatment with prostaglandins. Re-treatment should be based on physical examination, CBC and abdominal ultrasonongraphy or a sanguinous to mucopurulent discharge is present. Resolution of clinical signs varies from 82-100% in open-cervix pyometra following prostaglandin $F_{2\alpha}$ treatment. Reinstitution of treatment involves prostaglandin $F_{2\alpha}$ at 0.25 mg/kg for 7 days.

Successful short-term response to therapy is resolution of the clinical signs of pyometra. 64% in one study required one course of injections, while 36% required two series. Successful long-term response to therapy is the return of a normal oestrus cycle and if bred, conception and a live litter.

It is recommended that the dam be bred on her next oestrus cycle. This is because (a) they may have an abnormal uterus and recurrence of pyometra is possible, (b) pregnant animals may be less susceptible to infection and (c) there is no benefit skipping an oestrus cycle. Prostaglandins do not remove the underlying cystic endometrial hyperplasia. Conception rates in bitches are 40-82% and 85% in queens.

Synthetic prostaglandin $F_{2\alpha}$ (cloprostrenol and fluprosenol) are more potent than natural prostaglandin $F_{2\alpha}$ (dinoprost/lutalyse). Care must be taken, as there is no safe dose for synthetic prostaglandin $F_{2\alpha}$ determined.

Protocol for prostaglandin $F_{2\alpha}$ therapy in canine open-cervix pyometra (From Feldman and Nelson 2004)

1. Establish positive diagnosis
   (a) History and physical examination
   (b) CBC and other blood tests
   (c) Abdominal ultrasonography (radiography not as reliable)
2. Use natural prostaglandin (Lutalyse)
   (a) Day 1: 0.1 mg/kg, SQ, once
   (b) Day 2: 0.2 mg/kg, SQ, Once
   (c) Days 3-7: 0.25 mg/kg SQ, once daily
3. Antibiotics used during and 14 days following prostaglandin treatment
4. Re-evaluate
   (a) 7 days after completion of prostaglandin therapy
   (b) 14 days after completion of prostaglandin therapy
5. Re-treat at 14 days if
   (a) Purulent vaginal discharge persists
   (b) Fever, increased WBC, and fluid filled uterus persists

The antiprogesterone aglepristone has been used in dogs. Trasch et al (2003) treated 52 bitches with aglepristone. They had a 92% successful treatment rate at three weeks post-therapy. A 9.8% recurrence rate in pyometra at 3 months post treatment was observed. 37 animals had subsequent data on recurrence of cycles, with 8 bitches having a prolonged anoetrus period. The recurrence rate was minimised by the selection of bitches without ovarian cysts and cystic endometrial hyperplasia.

Gobello et al (2003) compared 2 protocols using aglepristone administered at 10 mg/kg, SQ, on days 1, 3, 8, and 15 (if not cured), combined with cloprostenol at the dose of 1 μg/kg, SQ on days 3 and 8, and 11. The second group received the same treatment with aglepristone as above but cloprostenol was given on days 3, 5, 8 10, 12, and 15 (if not cured). They found both treatments safe in the treatment of open-cervix pyometra. However, 20% of the patients developed pyometra before the next oestrus cycle. These findings were based on a small number of patients.

These treatment protocols may become useful in the near future, watch this space!

Protocol for prostaglandin $F_{2\alpha}$ therapy in feline open-cervix pyometra (From Feldman and Nelson 2004)

1. Establish positive diagnosis
   (a) History and physical examination
   (b) CBC and other blood tests
   (c) Abdominal ultrasonography (radiography not as reliable)
2. Use natural prostaglandin (Lutalyse)
   (a) Days 1-5: 0.1 mg/kg, SQ, twice daily
3. Antibiotics used during and 14 days following prostaglandin treatment
4. Re-evaluate
   (a) 7 days after completion of prostaglandin therapy
   (b) 14 days after completion of prostaglandin therapy
5. Re-treat at 14 days if
   (a) Purulent vaginal discharge persists
   (b) Fever, increased WBC, and fluid filled uterus persists

Medical Management of Dystocia and Caesarean Section

Dystocia is described as a difficult birth or the inability to expel the foetus from the uterus through the birth canal. Caesarean section is one method of treatment for dystocia.

Dystocia is an emergency condition where prompt treatment can result in live puppies or kittens being delivered and systemic consequences on the dam can be avoided.

A study in Rio de Janeiro (Chaves et al 2001) over 2 years found 2.5% of bitches presented for dystocia. The incidence was 66% in small breed of dogs, 23% in medium sized breeds and 10% in large breed dogs. 3.3% of cases were managed by obstetric manipulation, 40% required caesarean section and 57% received drug therapy.

Normal parturition

Pregnancy is maintained by progesterone secreted from the corpus luteum (CL). In the last 2 weeks of gestation in the queen placental progesterone maintains pregnancy. Parturition is due to foetal factors resulting in an increase in placental oestrogen secondary to stimulation of the adrenal cortex. The secretion of oestrogens and glucocorticoids by the foetal placental unit lead to luteolytic concentrations of prostaglandin $F_{2\alpha}$ in the placenta and myometrium. Oxytocin also increases the release of prostaglandin $F_{2\alpha}$ from the uterus. Oestradiol has been shown to directly promote prostaglandin synthesis by increasing the number of oxytocin receptors in the endometrium. Prostaglandin $F_{2\alpha}$ causes the CL to regress, reducing progesterone mediated blockade on myometrial contractions and prostaglandin $F_{2\alpha}$ synthesis is enhanced. The change in oestrogen:progesterone ratio also allows oxytocin to be released from the dams posterior pituitary dilating the cervix and enhancing uterine contractions. Relaxin is produced by both the ovary and placenta and it serves to bring structural changes to collagen in the interpubic ligament of the pubic bones which enables delivery of the foetuses and increasing distensibility of the cervix.

Stages of Labour

Stage I labour: Begins with the onset of uterine contractions and ends with dilation of the cervix. This is often not obvious in the bitch or queen. It can last between 6-24 hours in the bitch and is seen as restlessness, nervousness, anorexia, shivering, panting, vomiting pacing and nesting behaviour. In the queen this stage lasts 2-24 hours. In addition to the behaviours listed above the queen may also groom and vocalise.

Stage II labour: This begins with full dilation of the cervix and ends with complete expulsion of the foetus.

Stage III labour: Begins after expulsion of the foetus and ends with expulsion of the placenta. Bitches with more than one foetus vary between stage II and III labour.

Both stage II and III labour vary and many bitches deliver pups over 2-3 hours up to 24-36 hours. Contractions are usually visible during these stages. The time between the commencement of stage II labour and the birth of the first pup varies between 10-30 minutes. The placenta is usually expelled within 5-15 minutes of the birth of the pup. Breech presentation is normal.

In the queen the entire birthing process is over within 2-6 hours following the delivery of the first kitten, but it can take up to 24 hours. A fall in rectal temperature precedes delivery of kittens by at least 12 hours. Parturition can take from one to several days, especially if there is a disturbing environmental factor (owner, another cat). Placentas are expelled shortly after the delivery of the kitten.

After whelping the uterus undergoes involution where the uterus repairs itself. This usually occurs during the first 4-6 weeks post-partum. Commonly an odourless green, dark red/brown or bloody vaginal discharge (lochia) is observed. At the end of involution the uterus enters a period of anoestrus.

Evaluation of the dam and foetuses

1. History

Dystocia can present in several ways to the veterinarian. Often history is gathered over the phone prior to presentation. Common causes are;

(a) The animal has shown no signs of labour and is determined overdue by the owner
(b) Significant decrease in rectal temperature in past 24 hours but no signs of first or second stage labour
(c) Straining but failure to deliver a foetus after 20-30 minutes
(d) One or more newborn were produced but labour has ceased even though there are more foetuses in utero.

Breeding dates, vaginal cytology information, and progesterone peak at mating are important dates in determining if the pregnancy is full term. Parturition will occur at 57 to 72 days from breeding date, or 57±3 days from first day of dioestrus or 65±1 day from LH peak. Also enquire as to the size of the stud.

The clinician should ask whether the owner has tried vaginal manipulations or given oxytocin. Also look at the colour of the vaginal discharge as a dark green/black discharge suggest placental separation.

A fall in rectal temperature below 37.5°C usually indicates parturition will likely occur within the next 24-48 hours.

Guidelines for seeking veterinary attention during parturition

1. The dam has reached her due date without any signs of labour or a temperature drop
2. No signs of stage I labour 12-18 hours after temperature drop
3. Failure to progress to stage II labour after 6-8 hours of stage I labour
4. Active straining for 20-30 minutes or there are weak intermittent abdominal contractions for 1 hour and no foetus has been produced or only foetal membranes are presented
5. Been greater than 1 hour between the delivery of foetuses with no further signs of active labour

2. Physical examination of the dam

Perform a thorough physical examination on the patient. Look for evidence of hypoglycaemia, hyopcalcaemia, and sepsis. Palpate the uterus looking for pain/discomfort, the presence of foetuses and movement, and uterine contractions. Assess the mammary glands for development and milk.

Perform a digital vaginal examination. Ensure you do not get bitten during this procedure. Sterile gloves and aseptic preparation of the vulva is recommended. The cervix is too cranial to be palpated in the majority of bitches. However, the size and shape of the birth canal can be assessed, and the presence of soft tissue strictures, tissue bands, or septa detected. Feel for the presence of foetal membranes and foetuses within the birth canal. Feathering (stimulation of the dorsal vagina) indicates if uterine inertia is present.

3. Laboratory data

PCV/TP and blood glucose should be performed on all bitches. Serum calcium has been suggested although most dry chemistry tests are unreliable in detecting hypocalcaemia.

Serum progesterone may be useful in healthy overdue bitches. Normal concentrations are >2ng/ml, but this falls below 2 ng/ml 24-48 hours prior to parturition.

4. Foetal monitoring

Ultrasound examination can provide direct information on foetal viability. The measurement of foetal heart rate is useful and can indicate whether caesarean section is required as bradycardia (<150 bpm in bitches) indicates anoxia/placental insufficiency. Without ultrasound it is difficult to assess foetal viability.

5. Radiographs

Chemical sedation should be avoided due to effects on foetuses. I usually do a lateral radiograph including the pelvis. From this we may determine the approximate number of foetuses, whether the foetus will pass through the dams pelvis, possible foetal death, and the relative position of foetuses at the pelvic inlet.

Causes of dystocia

Uterine inertia is due to failure of uterine contractions to expel the foetus. Complete uterine inertia occurs when no foetuses are expelled, partial uterine inertia results when part of a litter is delivered but the uterus fails before complete delivery of the litter. Primary uterine inertia may be due to small litter size, hypocalcaemia, infection, very large litters, uterine trauma or torsion, and malnutrition. Secondary uterine inertia is a result of foetal obstruction, usually the uterus tires secondary to foetal-maternal oversize, or a small birth canal.

Causes of Dystocia in the Bitch or Queen
(From Feldman and Neslon, 2004)

Cause	Dog (%)	Cat (%)
Maternal:	**75.3**	67.1
Primary complete inertia	48.9	36.8
Primary partial inertia	23.1	22.6
Birth canal too narrow	1.1	5.2
Uterine torsion	1.1	-
Uterine prolapse	-	0.6
Uterine strangulation	-	0.6
Hydroallantois	0.5	-
Vaginal septum formation	0.5	-
Foetal:	**24.7**	**29.7**
Malpresentations	15.4	15.5
Malformations	1.6	7.7
Foetal oversize	6.6	1.9
Foetal death	1.1	1.1

Medical Treatment and Management of Dystocia

(a) Manual Therapy

Manual delivery is restricted to removing a sole foetus from the vaginal vault. Fingers are the safest and most reliable tools. Use plenty of lubricant and gentile traction may be sufficient. Due to the narrow diameter of the birth canal instruments such as spay hooks, sponge forceps and placental instruments must be used. Care must be exercised as these can cause injury to the foetus and dam. Any traction on the foetus should be in a posterior and ventral direction (follow the birth canal). Gentle shifting from side to side may relieve the obstruction.

Following delivery of the foetus the vagina should be examined for injury, which may have occurred.

The health of the bitch is paramount and if no progress is being made with manual delivery (after 10 minutes of gentle traction) a caesarean should be considered. Also if there is a vaginal obstruction, foetal malposition or a large oversized pup palpable, proceeding to caesarean section may be the best option.

(b) Medical Therapy

Oxytocin

This is useful with uterine inertia, ensuring foetal or maternal obstructions are not present. It should be used judiciously. Before using use radiography, vaginal examination, and abdominal palpation to rule out contraindications for use.

IV oxytocin may result in tetanic contractions of the uterus, which are poorly coordinated contractions that fail to expel the foetus. This may be avoided by diluting oxytocin (10 U/l in 5% dextrose) and titrating to achieve effect contractions. However using low doses of IV oxytocin alone appears to work well.

In order for oxytocin to cause effective uterine contractions, normal blood calcium levels are required. A serum calcium of < 2.25 mmol/l in association with dystocia may indicate calcium supplementation is required.

If the dam is dehydrated or hypovolaemic IV fluid therapy is indicated. It will not only compromise the dams' health but it will affect uterine perfusion and placental exchange of oxygen and nutrients.

Oxytocin Therapy in the Bitch

(a) Initial arbitrary dose is 5-20 U/kg IM/IV. 2 U/kg is good starting dose up to 20 U/kg and place in a quiet area.
(b) If no response in 30-40 minutes give a second dose of oxytocin. This can be preceded by 10% calcium gluconate 2-10 ml slow IV.
(c) If no response in 30-40 minutes give a third dose of oxytocin. This can be preceded by 50% dextrose 5-10 ml slow IV
(d) If no response after third injection of oxytocin caesarean is advised
(e) If pup expelled can give oxytocin at hourly intervals
(f) Ensure all pups are delivered before discharge

Oxytocin Therapy in the Queen

(a) In cats give 2-4 U oxytocin IV.
(b) If no response in 20 minutes give a second dose of oxytocin. This can be preceded by 10% calcium gluconate 1-2 ml slow IV.
(c) If no response in 20 minutes give a third dose of oxytocin. This can be preceded by 50% dextrose 2 ml slow IV
(d) If no response after third injection of oxytocin caesarean is advised
(a) Care with oxytocin as it can cause placental separation and foetal death.
(b) Ensure all kittens are delivered before discharge

(c) The Agitated or Nervous Bitch

An anxious, frightened or excited dam or owner may result in inhibition of labour. Light sedation with conservative doses of acepromazine may be beneficial. Handling of the dam should take place in a gentle and quiet manner at the veterinary clinic.

Surgical management of Dystocia

This is known as a caesarean section.

(i) Indications for Caesarean Section

The aim of caesarean is to maximise the number of live puppies or kittens delivered. It is indicated in every case where there are multiple foetuses retained and foetal heart rates are depressed (<150 bpm). Other indications include primary or secondary uterine inertia unresponsive to oxytocin and calcium supplementation, foetal oversize, soft tissue or bony obstructions of the birth canal, incorrigible malposition precluding vaginal delivery, foetal death in-utero and uterine torsion or rupture.

Caesareans may be planned in breeds such as Bulldog, Labrador and Golden retriever, Mastiff, and Yorkshire Terriers. In these cases the dam should have an accurate mating date in order to time the surgery. Depending on litter size a due date should be days 55-57 of dioestrus and surgery should be performed 48 hours prior to scheduled delivery date.

Neonatal survival can be affected if the due date is unknown and the dam has no signs of dystocia. In this situation performing a caesarean too early may result in foetal death. The bitch should be at least 57 days from her last breeding.

Guidelines for Caesarean Section

1. Multiple foetuses retained and foetal heart rates are depressed (< 150 bpm).
2. Primary or secondary uterine inertia unresponsive to oxytocin and calcium supplementation.
3. Foetal oversize
4. Soft tissue or bony obstructions of the birth canal
5. Incorrigible malposition precluding vaginal delivery
6. Foetal death in-utero
7. Uterine torsion or rupture.
8. Previous caesarean section?

Anaesthesia and Fluid Therapy

The dam should be placed on IV fluids prior to surgery because the dam has increased cardiac work and decreased reserve due to an increased cardiac output and blood volume and decreased RBC's. Isotonic crystalloids are usually sufficient. Colloids and hypertonic saline may be beneficial if the dam is severely hypovolaemic or septic.

Pregnant dams have a decreased pulmonary reserve and safety from hypoxia and an increased anaesthetic gas exchange due to decreased lung volume and functional reserve, and an increased oxygen consumption and increased minute volume and alveolar ventilation.

Foetuses have an inefficient drug metabolism, minimal cardiopulmonary reserve and a risk of hyperthermia. These result from immature renal and hepatic pathways, an immature cardiovascular-pulmonary system, and an immature thermoregulatory system.

Anaesthesia will affect both the dam and foetuses and there is no one safe protocol. Use a protocol you are familiar with. Prepare the dam as much as possible prior to anaesthetic (clip, prep abdomen) and pre-oxygenate via mask prior to induction.

Induction of Anaesthesia for Caesarean Section

(a) Acepromazine premed and mask induction with isoflurane and isoflurane-oxygen maintenance. Disadvantage is that animals may fight the anaesthetic during induction.

(b) Propofol very slow IV (avoids hypotension) and intubation and isoflurane-oxygen maintenance. Very safe and can intubate very light with practice provided given very slow.

(c) Ketamine 5-10 mg/kg and diazepam 0.25 mg IV and intubation and isoflurane-oxygen maintenance. Can get prolonged sedation in newborn.

Sevoflurane can also be used in place of isoflurane.

Regional anaesthesia has also been suggested in order to reduce general anaesthetic doses.

Analgesia with opioids in humans has been shown to not affect foetal oxygenation or neonatal pH when given parenterally. However, if used parenterally neonates more often

received naloxone compared to when epidural opiates were used in one study (Leighton et al, 2002). Opiod analgesia is good pre-surgery to prevent wind-up and lower doses of analgesia are required if used before surgery. Another large study in humans also found epidural analgesia had a better outcome for the mother and the neonate when compared with parenteral opiod analgesia (Halpern et al, 1998).

(ii) The Surgery

To minimise anaesthetic depression of the foetuses in-utero, removal from the uterus should be undertaken in an expedient manner.

Key factors to consider during surgery are;

1. Hysterotomy incision can be made in the body of the uterus and should be large enough to allow easy removal of the foetus. In breeding bitches it has been suggested that an incision in each horn may be better to reduce scarring of the uterine body.
2. If the placenta can be removed easily, remove at the time of surgery otherwise leave it to pass naturally.
3. Clamp the umbilicus 2-3 cm from the neonate
4. Hand newborn to assistants for resuscitation.
5. Close the hysterotomy incision with 3-0 or 4-0 absorbable suture with a two-layer closure. The first layer is a simple continuous pattern avoiding entering the uterine lumen and the second layer is a continuous inverting pattern (Cushing pattern).
6. Lavage the abdomen with warmed saline.

(iii) Resuscitation of the Neonate

1. Undertaken by assistants.
2. Remove the amniotic sac from the mouth and head.
3. Vigorous rubbing for up to 10-15 minutes often revives the newborn.
4. Fluid should be cleared from the mouth and nares if present by suction, cotton swabs or gentle swinging in a downward motion. Care with postural drainage as injury may occur from pressure on the internal organs or dropping.
5. Doxapram 1-2 drops sublingullay may stimulate respiration if not breathing. Mouth to mouth or catheter intubation can be used.
6. Inspect for gross birth defects.
7. Place in warmed area (33°C).

To Spay or Not to Spay

If the dam has evidence of reduced uterine viability or putrefaction of foetuses has occurred, ovariohysterectomy is recommended.

Many animal welfare clinics will spay the dam at time of caesarean as it prevents further breeding, which is one of their major goals. Ovariohysterectomy at the time of caesarean may slow the patients post-surgical recovery and potentially predispose them to sepsis or DIC. I personally do not spay at the time of surgery unless they have life threatening complications.

Robbins and Mullen (1994) found ovariohysterectomy and removal of the foetuses to be a safe and alternative method for the treatment of dystocia. They found one cat to have a clotting disorder, 3 cats had significant anaemia, one dog had uroperitoneum and one cat died, with an overall complication rate of 9%. Another study in bitches (Mojzisova et al, 2003) undergoing routine ovariohysterectomy looked at the phagocytic activity of blood leukocytes and mitogen-

induced blastogenesis of lymphocytes. The ingestion capacity of leukocytes was decreased significantly immediately after surgery. Mitogen-induced blastogenesis of lymphocytes was depressed significantly in the first 48 hours and this did not return to normal for 7 days post surgery. If this occurs in healthy bitches then it may be more pronounced in a bitch with dystocia. Food for thought!

Post Operative Complications

Potential complications of caesarean section include hypovolaemia and hypotension, uterine laceration, haemorrhage, gastrointestinal tract or urinary tract trauma. Endometritis and peritonitis can also occur.

In humans repeat caesarean sections are common in mothers who have had a previous caesarean section. A study by Trujillo-Hernandez et al (2002) found the three most frequent indications for first caesarean in women were dystocia and cephalopelvic disproportion (45%), foetal distress (12.8%), and pelvic presentation (9.9%) and the main indicators for repeat caesarean section were previous caesarean section (51%), dystocia (20%) and pelvic presentation (6.2%). A change in surgical technique from an anterior uterine body midline hysterotomy (similar to technique used in companion animals) to a low transverse hysterotomy has reduced the requirement for repeat caesarean section (Slatter, 2003).

In animals that have had a caesarean section the jury is out as to whether they will require a repeat caesarean section. Animals that have had a caesarean should be observed carefully during parturition to ensure that dystocia does not recur necessitating a repeat caesarean.

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