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The Whelping Bitch and Paediatrics Mini Series

Session 3: Post-op Care of the Bitch and Neonates

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Puppies and kittens are considered to be neonates during their first two weeks of life, and paediatrics from two weeks to six months of age. Some add two subdivisions to the paediatric stage: infant (from two to six weeks), and juvenile (from six to 12 weeks). Although a kitten continues to grow beyond six months of age, and some breeds of dogs grow beyond 18 months, from a physiological point of view, a dog or kitten is developed by 12 weeks of age, as at this age the main systems are fully mature and, therefore, can behave like adult ones. This article deals mainly with the infant and juvenile periods, although some attention will be given to neonatal care.

From birth to 12 weeks of age, puppies and kittens show several differences in physical examination, imaging, haematology and biochemistry results when compared to adult dogs and cats. It is necessary to be familiar with the changes associated with growth and maturation to differentiate pathological changes from normal developmental variations. Furthermore, as individuals mature at varying speeds, reference values are not as useful as baselines and trends, which form the cornerstones of management and monitoring.

Major body system assessment – cardiovascular, respiratory, neurological, abdominal palpation and temperature – has to be performed in any sick puppy or kitten. This can be followed by a more thorough physical examination and diagnostic tests once any potential life threatening conditions have been identified and addressed.

Normal physiological parameters

	Weight	Temp	Heart rate	Resp rate	SAP	MAP	CVP
Puppies	60-500g at birth, doubled by 2 weeks	35.5 – 36,1°C at birth, 37.8°C at 4 weeks	180-200 bpm at birth	15-35 breaths per min	60-64mmHg at 4 weeks 135-145mmHg at 6 months	49mHg at 4 weeks 100mmHg at 6 months	8cmH2O at 4 weeks 2cmH2O at 6 months
Kittens	60 – 100g at birth, doubled by 2 weeks	36.7°C at birth, 37.8°C at 4 weeks	200-220 bpm at birth	15-35 breaths per min	No data available	No data available	No data available

Physical examination of the paediatric patient

Cardiovascular

The autonomic nervous system in neonates, infants and juvenile dogs and cats is immature, and they have a dominant sympathetic tone, resulting in pink mucous membranes and high heart rates, usually above 200 beats per minute. Due to this immaturity of the autonomic nervous system, until eight to 10 weeks of age, there is minimal to no control of contractility and vascular tone. Heart rate is the only way to control cardiac output, and this is particularly important in situations such as anaesthesia in which many drugs can reduce heart rate. Bradycardia in these patients is usually secondary to hypothermia or hypoxaemia and does not respond to atropine. Although heart murmurs are frequent and often lack clinical significance, congenital conditions do occur and arrhythmias are always abnormal. Mean arterial blood pressure is 49 mmHg at two months of age, gradually increasing to reach the adult value of 94 mmHg at nine months. However, the pulse pressure (the difference between systolic and diastolic pressure) is similar to that in adults, so pulses can be palpated relatively easily. Conversely, central venous pressure is 8 cmH2O at one month, and 2 cmH2O at nine months, which is the adult value. The circulatory tree, therefore, changes from a high volume-low resistance circuit at birth to a low volume-high resistance one when mature.

Respiratory

Respiratory rate is increased in paediatric patients, with a normal rate of approximately 25 to 45 breaths per minute. Lung sounds tend to be louder due to increased turbulence, and it is not uncommon to hear crackles and wheezes. These are more prominent in the youngest patients and thought to be caused by fluid in the small airways, collapsing alveoli due to insufficient surfactant, and immature smaller airways.

Abdomen

Abdominal palpation usually allows most abdominal organs to be identified. The circulatory characteristics and lower oncotic pressure often result in a very small volume of ascites being present. While not easy to detect even with ultrasound, this contributes to poor radiological detail and gives a ‘slippery’ feel to abdominal palpation. Intestinal contents tend to be soft or liquid, particularly before weaning. Due to the immaturity of the kidneys during the first 12 weeks, urine output is much higher than in adults and therefore the bladder tends to contain urine, even in dehydrated puppies or kittens.

Neurological

Attitude is one of the main aspects of the neurological examination. Puppies and kittens should be inquisitive, active and playful. Eyes open at 12 to 14 days, with normal vision at 21 to 28 days. The menace reflex is not present before eight to 12 weeks of age. It is important to evaluate the head shape and the presence and size of the fontanelle. Many puppies are born with a closed fontanelle, and in most cases it is closed by three months of age (6 months in miniature breeds). Neonates crawl when they are seven to 14 days old, can walk at 16 days and gait should be normal by day 21. The withdrawal reflex is present at seven to 19 days, and the pain reflex is present at birth. Maturation and personality vary between individuals, so, when assessing mentation, it is important to interact frequently with the puppy and to ask the owner to do so as well. Any change in mentation should be taken very seriously and trigger a search for its cause, the most common being hypothermia, hypoglycaemia and hypoxia.

Temperature

Core temperature will change during the first four weeks, increasing from 35.5 to 36.1.C at birth to 37.8.C by four weeks of age in puppies, and from 36.7.C at birth to 37.8.C by four weeks of age in kittens. It is important to note that, due to the immaturity of the autonomic nervous systems, stress or anxiety are not associated with hyperthermia in the first eight to 10 weeks. Additionally, paediatric patients have immature thermoregulation and are therefore more prone to hypothermia and its effects.

Diagnostic tests

Frequent sampling and manipulation often result in stress, discomfort and iatrogenic problems such as infections and anaemia from repeated blood samples. Using small syringes and needles as well as point-of-care testing devices, and carefully selecting the most discriminating tests, will help to prevent these. Nevertheless, clinical pathology and imaging are often necessary for diagnosis and monitoring response to therapy.

Haematology

Packed cell volume (PCV) tends to be high at birth, decreases over the first weeks of life, and then increases again to reach the adult values. Due to changes from fetal to adult haemoglobin and from a transplacental to a pulmonary respiration, in puppies PCV drops from 47.5 per cent to 29.9 per cent over the first four weeks of life, the increases to reach adult values at 12 weeks of age. In kittens, PCV drops from 35 per cent at birth to 27 per cent at four weeks and has reached the normal 35 per cent by 16 weeks of age.

In both puppies and kittens, haemoglobin concentration is roughly one third of the PCV value, as in adults. Because of the small blood volume (due to the patient's small size) and the fact that anaemia is often present, it might be advisable to administer a blood transfusion before the haematocrit reaches critical levels (at or below 15 per cent), particularly if frequent sampling is anticipated. Given the small size of these patients, the volumes needed are usually small, and one should avoid raising the PCV well over the expected normal value. Platelets are fully functional from birth, and their numbers are similar to those seen in adults. In puppies, leukocyte count stays stable at around 12,000 x 109 cells/l for the first eight weeks; with lymphocytes peaking on day 21, believed to be related to increased antibody formation. In kittens, leukocyte count increases from 9600 x 109 cells/l at birth to 23,000 x 109 cells/l at eight to nine weeks of age and then decreases to 19,700 x 109 cells/l at 16 weeks of age.

Blood reference values	PCV/ TS	Leukocyte count	Band count	Lymphocyte count	Eosinophil count	Prothrombin time	Partial thromboplas time
Puppies	47% at birth, 29% at 28 days	12,000 x 10^9/l at birth, stable for first 8 wks	500 x 10^9/l, peaks on day 7	5000 x 10^9/l, peaks on day 7	800 x 10^9/l, peaks on day 7	1.3 times the adult value on day 1, normal levels on day 7	1.8 times the adult value on day 1, normal levels of clotting factors on day 7
Kittens	35% at birth, 27% at 28 days	9600 x 10^9/l at birth, 23,000 x 10^9/l, at 8 weeks	No data	10,170 x 10^9/l, at 8 weeks, 8700 x 10^9/l at 16 weeks	2280 x 10^9/l at 8 weeks, 1000 x 10^9/l, at 16 weeks	1.3 times the adult value on day 1, normal levels on day 7	1.8 times the adult value on day 1, normal levels of clotting factors on day 7

Blood reference ranges in puppies and kittens.

Serum biochemistry

There are several differences in biochemistry values between paediatric and adult patients. The most relevant values for puppies and kittens are listed in Table 4. Albumin concentration is low at birth and gradually increases to reach normal levels by eight weeks of age. During this gradual increase in concentration, its effects in colloid oncotic pressure and drug binding will also change. Globulins are low at birth, and during the neonatal period are mostly maternal in origin (ingested as colostrum). By the end of the infant period (eight weeks) they have reached adult levels.

Alanine aminotransferase, γ-glutamyl transferase and bilirubin are elevated during the neonate and infant period and should be within adult reference levels by the end of the juvenile period. Cholesterol, on the other hand, will be low and gradually increase to reach adult levels before 12 weeks of age. While liver function an therefore bile acids are expected to be normal at eight weeks of age, the presence of increased bilirubin may preclude their use as a diagnostic tool. Blood urea nitrogen and creatinine are low at birth and gradually increase as the kidneys mature, reaching adult levels at the time of full development by eight to 12 weeks of age. Hypokalaemia is a common finding due to increased potassium losses by the immature kidneys.

During skeletal development, puppies and kittens have increased calcium and an increased calcium/potassium ratio. The increases will be more significant in ionized calcium than in total calcium. Alkaline phosphatase also tends to be elevated during skeletal growth, increases that can last until full body growth has been achieved (over one year in large breed dogs). In dogs, lactate is higher in neonates and infants, and concentration falls to reach the adult levels at approximately two months of age. There are no published data for kittens, although personal communications suggest the same phenomenon may occur. Nevertheless, the magnitude of the increases is small, so, from the clinical perspective, adult reference values can be used in most instances.

Clotting

Prothrombin time and activated partial thromboplastin time are nearly double the adult values at birth, and become normal at seven days of age, suggesting maturation of the coagulation system. This can be achieved faster if parenteral vitamin K is used. Colloids affect clotting in a similar manner to in adults.

Urinalysis

Immature kidneys have a low glomerular filtration rate, low renal plasma flow, exaggerated proximal tubule natriuresis and low concentrating ability, which makes them less able to counter changes in hydration. Blood flow in the immature kidney does not respond to angiotensin. Therefore, until six weeks of age, renal blood flow is predominantly dependent on mean arterial pressure. Glycosuria and proteinuria are normal findings and both exacerbate fluid losses. The low specific gravity, coupled with the usually low urea and creatinine, make assessment of urinary function difficult in these patients. Changes in urine output, urine dipstick and particularly the sediment are the most useful tools to evaluate kidney function and can be used to monitor hydration status.

Diagnostic imaging

The small size of puppies and kittens requires that fine screen radiographs be used at all times. In neonates, infants and less commonly in juveniles, the thymus sail sign can be seen in the cranial mediastinum, and the lung parenchyma has a higher water content, resulting in a more opaque appearance or even areas of collapse.

Abdominal x-rays can be frustrating as there is usually a lack of detail due to the poor body fat content and potentially the small volume of ascites present. There will be a lack of costochondral mineralisation and the growth plates will be open. Radiographs should be assessed for congenital defects such as peritoneo pericardio diaphragmatic hernia, mega oesophagus, orthopaedic deformities or lung pathology, as well as other signs such as cardiomegaly. Echocardiography can be easily performed if the patient is cooperative, although diagnosing most congenital cardiac diseases requires both expertise and sophisticated equipment. Abdominal ultrasound is very similar to that in adults except for changes such as very small volumes of free fluid, liquid contents in the intestines and less fat in the abdomen. The gall bladder is usually large as there is a relative cholestasis. The bladder is usually filled with fluid, and mild renal pelvic dilatation due to the high flows of urine is common.

Treatment principles for the critically ill paediatric patient

Some aspects of patient care present in virtually all sick patients are the need for pharmacological treatment and fluid therapy. Equally important, although not as frequently needed, are nutrition, oxygen therapy, analgesia, sedation or anaesthesia. Although clinicians are very familiar with these procedures in adult populations, it is important to be aware of the unique differences when applying them to paediatric patients.

Pharmacology

Drug absorption, distribution, metabolism and elimination are unique in neonates and paediatric patients for several reasons – they have a greater surface area: bodyweight ratio, a lower amount of body fat and a higher amount of body water, resulting in different volumes of distribution. They also have a lower amount of total protein and albumin (the protein to which most drugs bind), resulting in increased bioavailability of drugs that are highly protein bound. There is also reduced renal clearance of water soluble drugs, because of low glomerular filtration rate and renal blood flow in neonates, and later by a reduced renal excretion caused by immature renal tubules. The half-life of drugs excreted by the kidneys is, therefore, increased.

Similarly, as both phase I (oxidation) and phase II (glucoronidation) hepatic enzyme systems are not fully functional, most drugs requiring hepatic metabolism for excretion will achieve higher plasma levels, and drugs that require hepatic metabolism for activation will have lower concentrations in plasma. Furthermore, oral drugs that undergo first-pass metabolism are at risk of accumulating to toxic levels in plasma if the adult dose is given. The haematoencephalic barrier has greater permeability in paediatric patients. This allows substrates such as lactate to permeate into the brain for use as energy sources, but may also allow drugs (e.g., most antibiotics) to cross more readily than in adults. In general, during the paediatric period, it is safe to assume that most drugs will reach higher concentrations in the central nervous system than in adults. Due to the immature cardiovascular and autonomic nervous system, and the variation in individual maturation rates, the response to drugs targeting these systems can be variable and hard to predict. The route of administration must also be taken into account as it may influence bioavailability more than it does in adults. Oral drug absorption is much higher in the first 24 to 72 hours of life because of the greater permeability of the gastrointestinal tract. Neonates have a high gastric pH, and therefore should not receive antacids. Also, neonates and infants are unlikely to respond to pro-kinetics such as metoclopramide in the treatment of ileus or gastric atony. Some drugs have poor oral absorption due to the drug binding to milk components (e.g., calcium) and, in general, the intestinal flora is still developing and is very susceptible to disruption by oral antibiotics. The adjustments required for each particular drug will depend on the drug's characteristics. However, as a rule of thumb, when decreasing the dose it should be reduced by approximately 30 to 50 per cent, and, when widening the interval, it should be increased by 25 to 50 per cent (e.g., going from every eight hours to every 12 hours).

The first few weeks of life are of paramount importance in transitioning from a completely sterile environment in utero to a contaminated environment with constant interaction with microorganisms. Minimal interference with this process will reduce the chances of colonization by pathogenic microorganisms with the resulting local infections and sepsis.

Fluid therapy

Puppies and kittens have higher fluid requirements and are at a high risk of dehydration. The higher percentage of body water (caused mostly by extracellular water), the greater surface area:bodyweight ratio, the lower percentage of body fat and the higher urine production are the main contributing factors. Maintenance rates in paediatrics usually range from 100 to 180 ml/kg/day. Evaluating hydration status can be difficult using physical examination parameters (normally mucous membranes are wet even when the puppy or kitten may be severely dehydrated, and skin tent is present in well hydrated animals due to lack of fat) or clinical pathology (paediatric patients have low PCV, low albumin and low proteins). Due to the same developmental limitations, paediatric patients are more susceptible to over hydration. Puppies or kittens should be weighed at least twice a day using an accurate paediatric gram scale.

The route of fluid administration can also pose a challenge. The intravenous (IV) route is ideal, but sometimes may not be possible in dehydrated neonates or very small patients (Fig 1). In these cases, intraosseous access in the proximal femur, proximal humerus or tibial crest (in larger animals) can be used. This can be accomplished using either an 18- or 22-gauge spinal needle or an 18- to 25-gauge hypodermic needle. The area is prepared in a sterile manner, and the needle is inserted following the long axis of the bone. Gentle aspiration is applied to check the correct positioning and the catheter or needle is secured using a sterile bandage. Any drug or fluid that can be administered through IV can be administered intraosseously (IO) and samples can also be drawn from IO lines. IV access must be established as soon as possible after initial volume resuscitation, even if it requires a cut-down procedure. Intraperitoneal infusion can be used when IV or IO access is unavailable, but subcutaneous fluid administration should be avoided because absorption during hypovolaemia is minimal.

Nutrition

Due to their immature metabolism and high metabolic rate, puppies and kittens rely on frequent nutrient provision. The use of orogastric or nasogastric tubes can be a great help and minimise the stress associated with forced feeding (Fig 2).

Oxygen therapy

Oxygen therapy should be provided whenever there is difficulty breathing, either detected on physical examination (tachypnea, abnormal lung sounds, abnormal pattern), or via monitors (e.g., pulse oximetry). Lung sounds must be assessed in the context of clinical signs and breathing pattern, and, if the sounds are considered to be abnormal, it is better to err on the side of caution and administer oxygen. Care should be taken to minimise stress during oxygen administration (Fig 3), and inspired concentrations over 60 per cent should be avoided, particularly in neonates, given the potential for lung damage and retrolental fibroplasia associated with high inspired oxygen concentrations in these patients.

Analgesia, sedation and anaesthesia

Puppies and kittens are very sensitive to pain, and painful experiences can condition their physiology and behaviour for life (Fig 4). Every effort should be made to provide analgesia and to be as pre-emptive as possible. While non-steroidal anti-inflammatory agents should not be used until the animals are at least 12 weeks of age, opioids can be safely used in paediatric patients. Doses should be titrated to effect, usually starting at 25 to 50 per cent of the adult dose. With regard to anaesthetic and sedative choice, maintaining a high heart rate is a priority due to the young animals' dependency on heart rate to maintain cardiac output. Rather than adding anticholinergic agents such as atropine or glycopirrolate to increase heart rate, anaesthetic protocols and dosages should be chosen to minimise depressor effects on the heart rate.

Reproductive Emergencies

Uterine Prolapse

Uterine prolapse occurs during parturition when the cervix is open. It is more common in cats than dogs. External reduction should be attempted as soon as possible, because the longer the tissue is exposed, the more likely that severe edema and tissue necrosis will occur. The animal should be anesthetized and sterile lubricant applied liberally to the exposed tissue. The uterine horn is flushed with sterile saline under pressure. Mannitol or hypertonic saline can be used to reduce edema if necessary before attempting reduction. Once the uterus is replaced, the animal should be given 5-10 units of oxytocin IM to cause uterine involution. If the uterus stays in for 24 hours, further risk of prolapse is unlikely because the cervix should be closed. If the tissue is damaged or necrotic, ovariohysterectomy is recommended. Internal reduction of the prolapse can usually be achieved through a ventral abdominal incision. In some cases, reduction is impossible due to extreme engorgement of the prolapsed tissue. In these cases, the external segment can be amputated followed by ovariohysterectomy.

Eclampsia

Puerperal tetany or eclampsia occurs most commonly during the first 4 weeks postpartum, but can occur in the last few weeks of gestation. The condition occurs in bitches more frequently than queens. Puerperal tetany can be life threatening, caused by a depletion of ionized calcium in the extracellular compartment. Predisposing factors include improper perinatal nutrition, inappropriate calcium supplementation and heavy lactational demands. Small dams with large litters are at increased risk. Excessive prenatal calcium supplementation can lead to the development of puerperal tetany by promoting parathyroid gland atrophy and inhibiting parathyroid hormone release, thus interfering with the normal physiologic mechanisms to mobilize adequate calcium stores and utilize dietary calcium sources. Thyrocalcitonin secretion is stimulated. The use of a balanced growth (puppy / kitten) formula commercial feed without additional vitamin or mineral supplementation is optimal during the second half of gestation and throughout lactation. Supplementation with cottage cheese should also be avoided as it disrupts normal calcium-phosphorus-magnesium balance in the diet.

Metabolic conditions favoring protein binding of serum calcium can promote or exacerbate hypocalcemia, such as alkalosis resulting from prolonged hyperpnea during labor or dystocia. Hypoglycemia and hyperthermia can occur concurrently. Therapeutic intervention should be initiated immediately upon recognition of the clinical signs of tetany, without waiting for biochemical confirmation. The signs preceding the development of tonic clonic muscle contractions (progressing to seizures) include behavioral changes, salivation, facial pruritus, stiffness / limb pain, ataxia, hyperthermia and tachycardia. Immediate therapeutic intervention should be instituted with a slow intravenous infusion of 10% calcium gluconate (1–20 ml) given to effect. Cardiac monitoring for bradycardia and arrhythmias should accompany administration, their occurrence warrants temporary discontinuation of the infusion and a slower subsequent rate. Because cerebral edema can occur from uncontrolled seizures, diazepam (1–5 mg intravenously) or barbiturates can be used to control persistent seizures once eucalcemia is attained. Mannitol may be indicated for cerebral inflammation and swelling. Corticosteroids are undesirable because they promote calciuria, decrease intestinal calcium absorption and impair osteoclasia. Hypoglycemia should be corrected if present, and exogenous treatment for hyperthermia given if necessary. Once the immediate neurologic signs are controlled, a subcutaneous infusion of an equal volume of calcium gluconate, diluted 50% with saline, is given, repeated q 6–8 h until the dam is stable and able to take oral supplementation. Calcium gluconate or carbonate (10–30 mg/kg q 8 h) should be instituted. Each 500 mg calcium carbonate tablet (TUMS) supplies 200 mg calcium. Efforts to diminish lactational demands on the dam and improve her plane of nutrition are indicated. If response to therapy has been prompt, nursing can be gradually reinstituted until the neonates can be safely weaned, usually at a slightly early age (3 weeks) and concurrent supplementation with commercial bitch / queen milk replacement is encouraged. The administration of calcium throughout lactation, but not gestation, may be attempted in dams with a history of recurrent eclampsia (calcium carbonate 500–4000 mg/dam/day divided).

Uterine Rupture

Rupture of the uterus occurs most commonly with very large litters causing marked stretching and thinning of the uterine wall, especially in multiparous dams with dystocia. Immediate laparotomy for retrieval of fetuses and repair or removal of the uterus, as well as culture and lavage of the abdominal cavity, is indicated. The uterus should be carefully examined at any cesarean section for any areas with or prone to rupture. Peritonitis can result from an undetected uterine tear. A unilateral hysterectomy can be considered if the damaged area is limited and the dam valuable to a breeding program.

Metritis

Acute infection of the postpartum endometrium should be suspected if lethargy, anorexia, decreased lactation and poor mothering occur accompanied by fever and malodorous vaginal discharge. Metritis is serious and sometimes preceded by dystocia, contaminated obstetrical manipulations, or retained fetuses and/or placentae. Hematologic and biochemical changes often suggest septicemia, systemic inflammation reaction and endotoxemia.

Vaginal cytology shows a hemorrhagic to purulent septic discharge. Ultrasound of the abdomen allows evaluation of intrauterine contents and the uterine wall. Retained fetuses and placentae can also be identified with ultrasound. A guarded cranial vaginal culture is likely representative of intrauterine flora and should be submitted for both aerobic and anaerobic culture and sensitivities, and permits retrospective assessment of empirically selected antibiotic therapy. Bacterial ascension from the lower genitourinary tract is more common than hematogenous spread, and *Escherichia coli* the most common causative organism in both bitches and queens. Therapy consists of intravenous fluid and electrolyte support, appropriate bactericidal antibiotic administration and pharmacologic uterine evacuation, usually with prostaglandin f 2 alpha (in the United States) at a dose of 0.10–0.20 mg/kg q 12–24 h for 3–5 days. An ovariohysterectomy may be indicated it the bitch's condition permits, and she is poorly responsive to medical management. Ergonovine (0.2 mg/15 kg given once IM) is also an effective ecbolic agent, but may cause rupture of a friable uterine wall. Synthetic prostaglandins offer more uterine specific therapy where available. Oxytocin is unlikely to promote effective uterine evacuation when administered > 24–48 hours postpartum. Nurslings should be hand reared if the dam is seriously sick. Metritis can become chronic and cause infertility.

Critical Care of the Neonate

Hypothermia

In neonates, careful temperature regulation and awareness of normal homeostatic temperatures are imperative. Normal rectal temperature is 35.6C ± 0.7C (96F + 1.5F) in the first week of life and 37C-37.8C (98.6-100F in the second and third weeks of life; temperature should approximate that of a normal adult by 7 weeks of age.

Whilst neonates are normally hypothermic compared with adults, they are also likely to decompensate quickly and become severely hypothermic. Hypothermia can lead to bradycardia and intestinal ileus. Human neonatal incubators, heat lamps, circulating hot water blankets, hot water bottles and warm towels can all be used to increase the environmental temperature, with the ideal being 32°C (90°F) with 55-65% humidity.

To prevent overheating and possible thermal injury, neonates should be given room to crawl away from the heat source. In addition, as is recommended for hypovolaemic, hypothermic adults, hypothermic neonates should be warmed slowly over 1-3 hours to prevent heat stress and dehydration. Rapid warming of a patient may cause peripheral vasodilation, resulting in core body temperature shock as a result of the decreased circulating volume to the core.

Hypoglycaemia

Neonates are prone to hypoglycaemia from inefficient hepatic gluconeogenesis, decreased glycogen stores, and an immature glucose feedback mechanism. Anorexia, vomiting, diarrhoea, dehydration, and infection may all result in neonatal hypoglycaemia, Whereas the adult depends on long chain fatty acids as a substrate for the myocardium, the neonate depends on glucose and carbohydrate metabolism for energy to the brain and heart respectively. Persistent or recurrent hypoglycaemia in the neonate can potentially result in permanent brain injury. Because neonates have inefficient counter-regulatory hormonal regulation (e.g. adrenaline, glucagon, growth hormone, and cortisol) these hormones will not be released in response to hypoglycaemia.

Clinical signs

A variety of clinical signs may occur in the hypoglycaemic neonatal patient including tremors, crying, irritability, increased appetite, dullness, lethargy, coma, stupor and seizures. Besides starvation, and hypoxia, other common causes for hypoglycaemia in the neonatal patient are sepsis, poor environmental conditions, congenital metabolic defects such as glycogen storage disease, portosystemic shunts, ‘toy breed hypoglycaemia’ and hypopituitary dwarfism. Placental insufficiency and prematurity have been cited as causes of hypoglycaemia in the neonate immediately after birth.

Treatment

Early signs of hypoglycaemia which may include lethargy, decreased suckling, crying and a limp body should be treated immediately. The use of glucose syrup can be useful in neonates – and it provides an emergency treatment option at home for owners. IV glucose or dextrose boluses (0.5 – 1.5m/kg IV of 50% concentration diluted 1:1 – 1:2; or 2-4ml/kg of a 10% solution are preferred over PO glucose. Isotonic fluids supplemented with 2.5-5% glucose as a CRI can also be used. However caution should be used to prevent over-supplementation as prolonged hyperglycaemia can result in worsening dehydration via osmotic diuresis. . After treatment the blood glucose concentration should be monitored because of the hyperglycaemia attributable to the poor regulatory mechanisms in the neonate,

Hypovolaemia

In adult animals, compensatory mechanisms in response to hypovolaemia include activation of the renin-angiotensin-aldosterone system and the sympathetic nervous system. In a healthy adult, tachycardia, increased antidiuretic hormone release, vasoconstriction and decreased urine output are observed in response to hypovolaemia, in an attempt to maintain cardiac output.

In contrast, in the neonate, cardiac output cannot be increased by increasing cardiac contractility, as only 30% of foetal cardiac muscle is made up of contracting elements. Puppies also appear to have fewer sympathetic nerve fibres supplying the myocardium than adults. As a result, tachycardia in response to hypovolaemia may not occur.

Because these compensatory mechanisms are not fully developed, clinical evaluation of the neonate may be more difficult. For example, autoregulation of renal blood flow is decreased in young puppies in response to changes in arterial blood pressure or hypovolaemia. Concentration and dilution of urine in response to changes in extracellular fluid are limited in neonates but do increase with age. Neonates have immature kidneys, a lower glomerular filtration rate, lower blood urea nitrogen concentrations, inefficient countercurrent mechanisms, and short loops of Henle, and inefficient sodium reabsorption in the thick ascending limb of the loop of Henle, all of which contribute to the inefficiency of urine production.

In hypovolaemia patients, initial shock doses of a balanced crystalloid should be used (i.e. 30 – 45 ml/kg for dogs, 20-30ml/kg for cats). Serial examinations should be done after the bolus to reassess the response and evaluate the need for further fluid resuscitation. Maintenance fluid rates of 80 (paediatric) to 180 (neonatal) ml/kg/24 hours should be implemented quickly in hypoglycaemic neonates.

Hypoxia

In newborns, lung expansion is essential for the release of both surfactant and prostacyclin, which increases pulmonary blood flow and pulmonary vasodilation. In addition, nitric oxide synthesis is probably induced by foetal oxygenation and may also contribute to pulmonary vasodilation .As a result, there is less pulmonary vascular resistance at birth, resulting in closure of the ductus arteriosus.

Neonates exhibiting clinical signs of hypoxaemia (e.g. cyanosis, orthopnoea, tachypnoea, dyspnoea, abnormal auscultation) should be treated immediately with oxygen therapy. Because neonates are normally anaemic, it may be clinically more difficult to see cyanosis, as detection of cyanosis is dependent on haemoglobin concentration. Respiratory distress may be a result of decreased surfactant levels, congenital defects resulting in pulmonary hypertension, meconium aspiration, bacterial or vital pneumonia, and other conditions.

Hypoxia is not easily recognised clinically because puppies tend not to hyperventilate until they are several days old. During the first 1-2 hours after birth, respiratory and metabolic acidosis has been shown to be normal. In fact, most neonates recover within <45 minutes after birth without intervention. However treatment should be considered for those patients

with acidosis that lasts longer than 3 hours. Hypoxia causes serious stress that can lead to complications such as respiratory depression, translocation of intestinal bacteria and chilling, resulting in decreased resistance against bacterial infections. Studies have also shown that neonates that lack colostrum may develop necrotizing enterocolitis when they become hypoxic. Aspiration or infections, coughing and nasal discharge are considered to be causes of hypoxia.

Summary

In summary, it is important to remember that kittens and puppies are not just little cats and dogs, and must not be treated as such. Neonates and paediatric patients have unique anatomical and physiological characteristics, and we lack published physiological values for these populations. Meticulous attention to detail, the use of trends and variations for any given individual, and an awareness of the unique homeostasis and response to disease is the key to diagnosis and successful treatment.