PREFACE

In recent years there is a spectacular growth of pet animal population in our country with changing social structure. Changing lifestyle in the form of the rise in nuclear families and double income households have encouraged the growth of pet ownership in urban areas of Orissa.

The health management of pet animals, due to several reasons has not received much attention. In urban areas the pet practice is an important challenge Veterinarians have to face. Pet practice is essentially different than large animal practice, especially when the cost of treatment is considered. The pet owners demand quality treatment irrespective of the cost. For this reason, Veterinarians are required to keep them abreast of the latest knowledge and skill. The owners are well aware of the advancement in medical diagnostic and therapeutic facilities and they expect medical breakthrough that have occurred for human to be available for their pets with the same diseases.

Pet practice has undergone a sea change and continues to develop commensurate with varied demands and expectation of the clients. The challenges have become arduous because secrets of creation of life and death are continuously being unfolded with advancement of biological science and its related applied part in the field of medicine. Therefore, today's practitioner is burdened with much greater responsibility to satisfy the health management of pet animals.

Pet animal practice must remain relevant to changing needs of society. Practitioners aim should be to further, to improve the quality of services to meet the global challenges and also bring awareness among our clients of the challanges ahead.

The CVE programme is meant for updating the knowledge and skill of pet practitioners so that they can remain on the "Cutting Edge" of their profession and continue to provide a quality service to their clients.

Prof. S. K. Ray

CONTENTS

INFECTIOUS DISEASES IN DOGS AND PREVENTIVE VACCINATION PROTOCOL

N. Sahoo
Department of Medicine, College of Veterinary Science, Bhubaneswar

Dogs, like other animals, are susceptible to many infectious diseases caused by either micro-parasites or macro-parasites. It is worth mentioning that majority of those diseases are curable after advent of suitable chemotherapeutic agents. However, the incidence of certain infectious is raising their heads where a large number of factors are thought to be playing a role e.g., microbial adoption and change, susceptibility to infection, changing ecosystem, human activities etc. Here are brief descriptions of some common infectious diseases among dogs along with the vaccination protocol wherever available.

Distemper: It is caused by Morbillivirus of the family Paramyxoviridae which affects a wide range of organs including the skin, brain, eyes, intestinal and respiratory tracts. The virus is transmitted through the air being contaminated by the cough and urine of infected animals. Dogs of any age can be affected; however, majority is less than 6 months of age. The common signs are nasal and eye discharge, coughing, pneumonia, diarrhea, vomiting, biphasic fever and seizures. Puppies that recover may have severe tooth enamel damage. The nose and foot pads of the young dog may become thickened, hence the nickname "hard pad disease." A polymerase chain reaction test can be performed. Blood examined microscopically may show canine distemper inclusion bodies. There is no specific treatment for canine distemper. Therapy is largely supportive which mainly include antibiotics, intravenous fluids and anti-seizure medications. Supportive care of patients with neurological signs is less rewarding. Disease can be prevented through timely vaccination.

Hepatitis: The disease caused by canine adenovirus type 1 (CAV-1) is a disease of the liver and other body organs. The primary mode of transmission is by direct contact with an infected dog. Initially, the virus affects the tonsils and larynx causing a sore throat and coughing. The cornea may appear cloudy or bluish due to edema within the cell layers forming the cornea; hence the name 'hepatitis blue eye'. As the liver and kidneys fail, one may notice seizures, increased thirst, vomiting, and/or diarrhea. Occasionally dog suffers from pneumonia. As there is no specific treatment supportive care are indicated. Vaccines containing either CAV-1 or CAV-2 are used to immunize pet.

Coronavirus infection: Canine coronavirus is a single stranded RNA type of virus with a fatty protective coating. It is spread by virus shedding in the feces of infected dogs. The primary symptom associated with canine coronavirus infection is diarrhea. Canine coronavirus (CCV) is the second leading viral cause of diarrhea in puppies with canine Parvovirus being the leader. Most puppies, however, will recover after several days of mild to severe diarrhea.

Therapeutic management aims against dehydration. Vaccines are available for immunization. Sanitation with commercial disinfectants is highly effective.

Parvovirus infection: Parvovirus infection, a highly contagious disease, is caused by Type CPV-2, 2a, 2b, 2c.The disease is spread through contact with feces containing the virus. Virus can be found in the feces several days before clinical signs of disease appear. The most common form of the disease is the intestinal form having the predominant clinical signs of diarrhea and vomiting that is often bloody. The course of the disease is often one to two weeks. In absence of ELISA and PCR, CBC is performed to determine the severity as well as prognosis of the disease. The treatment of parvovirus is directed at supportive therapy consisting of parenteral antibiotics, antipyretics, antiemetics, hemostats, antiulcers and restricting the food during periods of vomiting. Intravenous administration of a balanced el electrolyte solution is preferred to replace fluids lost through vomiting and diarrhea. Through vaccination protocol, the window of susceptibility can be made as small as possible.

Rabies: Rabies, a multi-species disease, is caused by Rhabdoviridae and transmitted by exposure to rabid animal. The virus exists in nature through Sylvian cycle or Urban cycle. Pathogenesis is dependant on location and severity of bite, species of animals involved. The incubation period varied from 14-90 days. Virus Invades peripheral nerve tissues- sensory and motor nerve - neuromuscular junction-binds with acetyle choline –axon – spinal cord – limbic system of brain (Furious form) – neocortex (Paralytic form). The virus spreads to the saliva, tears, milk, and urine. Clinical features are similar in most cases with variations between individuals. Furious form is easily recognized clinically. Dumb (Paralytic) form of rabies is difficult to diagnose and requires laboratory tests. Death occurs in 2-14 days. In absence of suitable treatment, wound toilet, ethnoveterinary practices and vaccination with RIG should be instituted without delay. Pre exposure vaccination and bio-security measures are considered ideal to prevent this fatal disease.

Parainfluenza: Canine parainfluenza is a highly contagious respiratory disease. The disease can progress to pneumonia in puppies or chronic bronchitis in older dogs. The canine parainfluenza virus is transmitted through contact with the nasal secretions of dogs that are infected with the disease. High humidity and exposure to drafts can enhance a dog's susceptibility to the disease. An unproductive, but persistent cough is one of the common symptoms of canine parainfluenza. The cough generally lasts 10 to 21 days. Dogs with canine parainfluenza have runny nasal discharge.The labored breathing becomes more acute with physical activity and excitement. In some cases, a low-grade fever may be present. Alone, the canine parainfluenza virus is usually not a serious problem. However, when it teams up with other pathogens, it can turn into a serious case of kennel cough. Chest x-rays coupled blood test can diagnose the infection. It is a vaccine-preventable disease.

Leptospirosis: It is a bacterial disease of zoonotic importance. Organism can penetrate mucous membranes or abraded skin and multiply rapidly upon entering the blood system. From there they spread to other tissues including the kidneys, liver, spleen, nervous system, eyes, and genital tract. In acute infections fever, shivering, and muscle tenderness are the first signs. Then

vomiting and rapid dehydration may develop. Severely infected dogs may develop hypothermia followed by death. In subacute infections, the animal usually develops a fever, anorexia, vomiting, dehydration, and increased thirst. The dog may be reluctant to move due to muscle or kidney pain. Animals with liver involvement may develop icterus. The possibility of chronic or sub-clinical form can not be ruled out. A positive diagnosis can be made through a blood test. Treatment consists of antibiotics, fluid replacement, and controlling the vomiting and the problems associated with the corresponding kidney or liver infections. Prevention involves keeping animals out of contact with potential sources of infection including contaminated water sources, wildlife reservoirs, or domestic animals that are infected or chronic carriersThere are currently many different vaccines available on the market for a wide variety of species and serovars i.e., L icterohaemorrhagiae L. grippotyphosa and L. pomona. Unfortunately, vaccination against one strain does not protect against the other strains.

Ehrlichiosis: Canine ehrlichiosis, a tick-borne riskettsial disease, is caused by multiple strains of Ehrlichia spp. The clinical signs of ehrlichiosis are exhibited in three phases. Phase I is characterized by fever, depression, lethargy, loss of appetite, shortness of breath, joint pain, stiffness and bruises. The animal may appear normal or show slight anemic in phase II which can last for months or years in subclinical form. The last chronic phase can be mild or severe where weight loss, anemia, neurological signs, bleeding, inflammation of the eye, edema in the hind legs and fever may be seen. German Shepherds and Doberman Pinschers tend to have a more severe chronic form of the disease. Recovered dogs are not immune to infection. A decrease in the number of platelets in the blood is the most common laboratory finding in all phases of the disease. The diagnosis is based on the typical clinical signs, and blood tests i.e., IFA, ELISA and PCR tests. Examination of blood smear may of help during acute phase. Tetracycline or doxycycline for 3-4 weeks, blood transfusions and intravenous fluids depending on the severity of the disease are included in the line of treatment.The drug, imidocarb dipropionate, is sometimes used in conjunction with the antibiotics. If immune-mediated arthritis or decrease in platelets occurs, corticosteroids (e.g., prednisolone) may be given. There is no vaccine for ehrlichiosis. Tick control is the main way to prevent ehrlichiosis.

Malassezia infection: Malassezia pachydermatis, potential opportunistic yeast, is a normal resident of the skin, commonly found in external ear canal, on mucosal surfaces (oral and anal), in the anal sacs, interdigital areas, lips, rectum and vagina of dogs. Environmental factors such as high temperature and humidity influence the incidence of infection, either dermatitis or otitis. The disease though not fatal, is not uncommon among dogs and the clinical manifestations are often aesthetically disagreeable to the owners. Certain predisposing and perpetuating factors either prolong the course of disease or create hindrance in recovery process. The disease is characterized by variable degrees of head shaking, pain, pruritus, foul odor and exudation in otiotis and black pigmentation, lichenification, pruritus, fowl odour and alopecia in dermatitis. Cytological study preferably through New Methylene Blue stains is extremely helpful as well as reliable to identify the pathogens of dermatological importance i.e., bacteria, yeasts, mites and fungi. Antifungal

agents i.e., clotrimazole, ketoconazole, fluconazole and itraconazole can be used both topically and orally to combat the infection. Proper managemental practices can prevent this infection.

Pyoderma: Any condition, infectious, inflammatory, and/or neoplastic etiologies, of the skin that results in the accumulation of neutrophilic exudate can be termed a pyoderma. However, pyoderma refers to bacterial infections of the skin where Staphyloccocal intermedius is the predominant etiologic agent. Bacterial pyodermas are popularly classifed by depth of infection i.e., (1) pyodermas limited to the epidermis and hair follicles are referred to as superficial pyoderma and (2) those involve the dermis or cause furunculosis are referred to as deep pyoderma. Warm, moist areas on the skin, such as lip folds, facial folds, neck folds, axillary areas, dorsal or plantar interdigital areas, vulvar folds, and tail folds, often have higher bacterial counts than other areas of skin and are at an increased risk for infection. Pressure points, such as elbows and hocks, are prone to infections, possibly due to follicular irritation and rupture due to chronic repeated pressure. The most common clinical sign of bacterial pyoderma in dogs is scaling; pruritus, alopecia, papules or pustules, pain, crusting, odor, and exudation of blood and pus. The diagnosis of pyoderma is usually based on clinical signs and examination of skin scraping. The primary treatment of superficial pyoderma is with appropriate antibiotics for 1-4 weeks. All clinical lesions should be resolved for at least 7 days before antibiotics are discontinued. Chronic, recurrent, or deep pyodermas typically require 8-12 wk or longer to resolve completely. The choice of antibiotic is lincomycin, clindamycin, erythromycin, ceftriaxone+ tazobactum, chloramphenicol, cephalosporins, amoxicillin trihydrate-clavulanic acid. Pyodermas that do not respond to therapy should be treated based on culture and sensitivity. Topical antibiotics may be helpful in focal superficial pyoderma. Use of medicated shampoos containing benzoyl peroxide, chlorhexidine or chlorhexidine-ketoconazole at daily/weekly/ fornightly interval may accelerate the recovery process.

Vaccination protocol: It is known very well that prevention preferably through vaccination is worth more than the best cure. In this connection information about maternal derived antibodies, windows of susceptibility, breed susceptibilities, the possibility of unidentified strains, the effectiveness of different vaccines, vaccination protocols and more over the health status of the pup are of much value. The generally recommended protocol is to vaccinate puppies beginning at 6-8 weeks of age, and revaccinating every 2-3 weeks until the puppy is 16-20 weeks of age. A booster is given at one year of age and every 1-3 years. The vaccination can be started at an early age of 4weeks where the dam is unimmunized during pregnancy. The vaccine-preventable disease in dogs are rabies, parvo, distemper, hepatitis, parainfluenza, corona and leptospirosis.

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USE OF HORMONES IN CANINE REPRODUCTION

S. K. Harichandan Ray, Ex- Prof. & Head,
Deptt. of Animal Reproduction , Gynaecology & Obstetrics.
College of Veterinary Science and Animal Husbandry,
O.U.A.T., Bhubaneswar-3

The reproductive processes of animals are primarily under endocrine control. Need based application of hormonal therapies to circumvent reproductive problems of animals require thorough knowledge about various endogenous reproductive hormones and their specific physiological role in performing various reproductive events pertinent to the species.

Reproductive activity in the bitch differs from polycyclic pattern of other animal species both clinically and physiologically. Initiation of estrus cycle is suggestive of attainment of puberty while it is basically regulated by complex interaction of hypothalamus-pituitary -ovarian axis. Unlike other species, the endogenous endocrine profile and their specific role in canine differs in many aspects for controlling estrus cycle and pregnancy. Generally bitches are considered monocyclic although many of them exhibit more than one estrus cycle in a year with marginal seasonal effect. The age of attainment of puberty varies between 6 to 30 month with late onset of puberty in larger breeds. The reproductive or estrous cycle of domestic dog (Canis familiaris) is classified into four phases i.e. proestrus, estrus, diestrus and anestrus. The first two phases are conspicuous and appreciable due to their clinical and physiological significance and express average duration of 9 days each. The diestrus phase roughly starts from ceassation of behavioral estrus in concurrence with rising level of progesterone and last about 75 days (60-90). Anestrus is the quiescent and functionless phase of estrus cycle with no reproductive activities with an average duratio of 125 days (15-265). In bitch the initiation of proestrus and late proestrus is under the influence of estrogen (17-â estradiol). Contrary to other species the bitch becomes receptive to male with rising level of progesterone along with receding estradiol. In bitch , prolactin maintains corpus luteum activity (luteotrophic) in late diestrus both in pregnant and non pregnant bitches and physiologically uterine PGF_2á may not be responsible for maintenance or regression of the corpora luteum in bitch. Moreover the pregnancy is basically maintained by luteal progesterone with tropic support from pituitary LH and prolactin in 2nd half of pregnancy.

With this preamble, exogenous administration of hormone in the dogs and bitches in the context of canine reproductive has been highlighted. Generally some steroid hormones or their analogues were routinely used in the control of canine reproduction and to resolve some pathological disorders. However, recently prostaglandin and hormones and anabolic agents have given a new dimension as a novel therapy to treat various reproductive disorders, pregnancy termination and

estrus control. While treating canine patients many factors such as age, breed, breeding history, possible side effects and attitude of owner should be considered rationally before instituting hormonal therapy.

Induction of ovulatory estrus and its anomalies.

Estrus Induction:

Natural estrus expression requires synchronized action of hypothalamic pituitary –gonadal hormones. On the basis of this analogy various hormonal products or their analogs were attempted to mimic natural process to induce estrus and pregnancy. The most effective drug protocols to induce fertile estrus are suggested.

PMSG(eCG) and HCG (nCG) protocol

i. 500-1000 iu PMSG i/m repeated after 6 days.

ii. PMSG 20 IU i/m daily for 5 days and hCG 500 IU i/m on 6th day. Pretreatment with estrogen (estradiol 100 mg daily i/m for 5 days) showed better conception rate.

iii. Estrogen DES (Diethyl Stilbosterol) 5mg orally for 6 to 9 days (not recommended)

iv. GnRH analogues 1.25 µg s/c every 190 minutes for 11 days

v. HCG + GnRH

HCG9(500 – 1000) IU i/m for two days followed by GnRH 50µg i/m daily for two days

vi. Dopamine agonist

a. Bromocriptine 20µg/kg/bid/orally X 21 days

b. Cabergoline 5µg/kg/once daily/orally X 7-10 days (pregnancy induced in 93%.)

Persistent estrus:

Defined as combined duration of proestrus and estrus is more than 6 weeks. This condition may arised due to prolonged estrogenic activity, functional ovarian follicular cysts and granulosa cell tumour . It may lead to bone marrow depression, anemia, leucopenia thrombocytopenia and cystic endometrial hyperplasia. LH or Progesterone treatment may resolve prolonged estrogenic activity.

Irregular estrus:

Endometrial involution and repair takes 130-150 days. Inter estrus interval less than 4 months usually infertile.

Causes - Hereditary, uterine disease and ovarian cyst.

Treatment: (The aim of this therapy is to induce temporary anestrum.)

Mibolerone- 30µg/10kg/orally or

Testosterone (0.5µg/kg/i/m at 5 days interval.

Anestrum:

Primary anestrum denotes lack of estrus exhibition by 24 months of age. Hormonal assay of bitches show elevation of both FSH (>290 ng/ml) and LH (>200 ng/ml) indicating lack of negative feed back from ovarian steroid.

Incase of normal /intact bitches the same drug protocol may be used to induce estrus.

Silent heat:

Iin bitches it may be defined as no manifestation estrus symptoms including proestrus and male attraction, even though the animal possesses cyclic ovaries. In this condition progesterone level is more than 2ng/ml indicating functional ovary.

Estrogen therapy may induce overt heat symptoms.

Primary anestrum may precipitate due to hypothyroidism, which is a common cause of infertility. Anestrum, prolonged inter estrus interval, abortion, mummification and stillbirth are common gynecological complications.

Diagnosis - Assay of T_4 (T_4D) and TSH in blood.

Treatment - L thyroxine (0.01 to 0.02 mg/kg) orally twice daily with regular hormonal assay.

Drug induced anestrus :

Bitches receiving either androgen , progesterone or glucocorticoids for a long duration may not exhibit estrus symptoms. Withdrawal of inciting agents may cause recovery.

Secondary anestrus :

Prolongation of inter estrus interval or fails to cycle by 10 to 18 months of previous cycle.

Estrus may be induced by hormonal applications such as FSH & LH.

Estrus suppression:

Progestin :

Megestrol acetate – 0.55 mg/kg /once daily orally 7 days prior to expected proestrus for 32 days. Or 2.2 mg/kg/once daily orally after onset early proestrus for 8 days.

Megestrol acetate for 3 days (2.2 mg/kg/orally) prior to mating will prevent conception.

MPA(Medroxy progesterone) 2mg/kg/i/m every three month in anestrus period.

Proligestrone- the drug has antigonado-tropic properties.

Dose 30mg/kg/s/c to be repeated after 3 months and later every five months.

Androgens :

Mibolerone- Dose- 30 to 80 µg orally for 30 days or more could suppress estrus. On discontinuation the bitch returns to estrus.

Testosterone :

Dose- 100 mg testosterone propionate i/m once weekly and 25 to 50 mg (methyltestosterone) orally twice in a week could prevent estrus expression.

GnRH agonist :

Implants could prevent attainment puberty and suppress estrus expression in cyclic bitches.

Mismating:

Avoiding an unwanted pregnancy is best accomplished by preventing the access of fertile male to bitch. In bitch fertilization is nearly 100 percent and it is difficult to terminate pregnancy once the embryo is implanted. If accidental mating occurs the animal should be presented within 72 hrs post coitus for prevention of pregnancy.

Treatment:

Estrogens:

i. DES (diethyl stilbosterol) 0.1 mg/kg orally for 3to 5 days.

ii. Estradiol benzoate – Dose – 0.01 mg/kg/i/m or s/c on day 3,5, & 7 after mating .

iii. Estradiol valerate 0.1mg/kg/i/m (should not exceed 3.0 mg)once

iv. Tamoxyfen citrate (Estrogenic activity in bitch)

Dose- 1mg/kg/twice daily /,,,,,,,,,,,,, / 10 days at late proestrus or estrus. It prevents conception or transportation of ovum to the uterus.

Termination of pregnancy:

Treatment:

PGF_2á (lutalyse)

Dose- 20 µg/kg bodywt / s/c thrice daily . or

150-200 µg s/c twice daily till the pregnancy terminates.

(5 to 21 days of pregnancy could be terminated by above therapy)

Cloprostenol- (for 14 to 28 days of pregnancy)

Dose- 10µg/kg /intravaginally

or

2.5 µg/kg/s/c three times a day till the pregnancy is terminated. How ever PGF_2á not effective in vey early pregnancy (1-10 days).

Glucocorticoids (Late gestation)

Dexamethasone- 5mg i/m twice daily for 10 days.

Or

Dexamethosone- 0.1 to 0.2 mg/kg body wt. orally /twice daily X 5 days followed by decreasing dose up to 10 days.

The oral therapy may be given along with PGF_2á (250 µg/kg/s/c twice daily)

Prolactin inhibitors (25 to 40 days termination)

Bromocriptine- 20 to 100 µg/kg body wt/orally for 4 to 7 days.

Cabergoline – 1.65 to 5.0µg/kg/once daily orally or s/c for 5 days.

Pre treatment with PGF_2á (coloprostenol or lutalyse) for 3 days followed by Cabergoline could induce abortion in 100 percent of cases.

Mifepristone – (Ru 486)- it is generally used as abortificant drug in human female

Dose- 2.5 mg/kg/orally twice daily for 4-5 days and could terminate canine pregnancy at 32 days and abortion occurs 3-5 days following treatment . Higher doses (10-20mg/kg body wt.) could terminate early pregnancy (10-26 days)

Hormonal infertility:

Anovulation : Administration of hcg (25 IU/kg/i/m) will induce ovulation in bitches at mid estrus, consistent with the rising of progesterone (>1µg/ml)

Hypoluteoidism (Lutal deficiency) - Pregnancy is maintained by luteal progesterone and should be more than 2ng/ml. Less than this level will terminate pregnancy indicating luteal insufficiency.

Progesterone in oil 8 mg/kg body wt/i/m at 48 hrs interval and should be discontinued 1 week before expected date of parturition.

Ovarian follicular cyst (anovulatory):

Treatment:

Surgical removal of ovary or ovario hysterectomy (OHE) most effective.

Hormonal treatment

GnRH (50µg/i/m) as a single dose or two does LH (500 IU) through intra-muscular route at 48 hrs. interval may be effective in some cases.

Cystic endometrial hyperplasia -pyometra complex (CEH- Pyometra)

It is a progesterone mediated uterine disease associated with bacterial colonization and retained corpus luteum. Treatment of such cases involves surgical (ovario hysterectomy) and medicinal approach. The PGF_2á is most preferred drug of choice which causes luteolysis, myo-metrial contraction, cervical relaxation and expulsion of pyomic content.

Treatment- serum progesterone should be measured prior to treatment

Lutalyse- 100 µg/kg body wt/s/c TID for two days followed by 200 µg/kg/s/c once for 2-7 days or until the uterus size returns to normal (Progesterone should be >1ng/ml).

If the serum progesterone is less than 1ng/ml lutalyse 200 µg/kg/s/c once or twice daily for 2 to 7 days.

In both types, antibiotics and other supportive treatment is indicated until cleared clinically.

Other anomalies:

Expulsion of foetus, uterine inertia, post partum haemorrhage ,post partum endommetritis, retention of fetal membranes and milk obstruction may be treated with Oxytocin , which induces contraction of smooth muscle of uterus and mammary gland. Oxytocin is contraindicated in obstructive dystokia

Oxytocin 5-10 IU given as intravenous drip or repeated small doses in case of uterine inertia since it may result in uterine rupture.

Vaginal prolapse:

Prolonged estrogenic stimulation during late proestrus and estrus lead to vaginal prolapse with higher incidence in large breeds.

Spontaneous regression occurs following ovulation with concurrent rise in LH

Treatment-

GnRH (2.2 µg/kg/im) or hCG (500-1000 IU/im) will induce ovulation and suppression of prolapse by 7 days.

Other reproductive anomalies

Pseudopregnancy/ pseudocyesis

Urinary inconsistency-

Following ovario hystectomy (OHE) many bitches suffer from micturition difficulty. Estrogen treatment for a short duration may relieve micturition difficulty.

Mammary neoplassia:

Testosterone or Tamoxifen(estrogen receptor blocker) may reduce its progression.

Tamoxifen at the dose rate of 0.5 to 1 mg/kg body wt. /orally once daily proved to be more effective.

Adverse Effect of Hormonal Therapy:

Estrogen:-

Bone marrow depression, Applastic anemia , leucopenia, thrombocytopenia, bleeding into body cavities, endometritis , cystic endometrial hyperplasia, pyometra and dibetes.

Progesterone:-

Increased appetite , weight gain , lethargy ,mammary development , masculinization of fetus,

adrenal suppression, pyometra, acromegaly and mammary tumors.

Androgen:-

Clitorial hypertrophy, thick vaginal discharge, increased mounting, aggressive behavior and masculinization of female fetus.

PGF_2á :-

Vomition, hypersalivation, anorexia, defecation, polyurea, polydipsia, dehydration and lactation. Side effect is minimized by giving atropine sulphate (0.025 mg/kg i/m) 15 minutes prior to PG treatment.

Bromo criptine:

Vomition & anorexia

Conclusion :

There is no area of veterinary therapeutics in which there is a greater uncertainty than the use of drug specifically hormones in breeding canine species and risk is far greater while treating pregnant animals. Hormonal treatment behaves as two edged weapon and individual idiosyncrasies are not ruled out. Hence hormonal treatment may be undertaken with utmost caution following rational evaluation of requirement and post treatment side effect.

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PSEUDOPREGNANCY

S. Das, Professor and Director of Clinics,
Teaching Veterinary Clinical Complex,
Faculty of Veterinary Science and Animal Husbandry,
O.U.A.T., Bhubaneswar

Pseudopregnancy is also known as false pregnancy, a term as pseudocyesis in the human being and pseudopregnancy in other mammals, is the appearance of clinical and/or subclinical signs and symptoms associated with pregnancy when the person or animal is not pregnant. Clinically, false pregnancy is most common in veterinary medicine (particularly in dogs and mice). False pregnancy in humans is less common, and may sometimes be purely psychological. It is generally estimated that false pregnancy is caused due to changes in the endocrine system of the body, leading to the secretion of hormones which translate into physical changes similar to those during pregnancy. A related appropriate term is galactorrhea, defined as spontaneous develop of the mammary gland with secretion of clear liquid to true milk.

Pseudo pregnancy is a normal phenomenon in the intact bitch. It is reported that 64.3% of intact bitches exhibit pseudo pregnancy regularly and 7.1 % were reported to exhibit clinical sign intermittently. 87 percent of intact dogs are reported to exhibit signs of false pregnancy 2 or more times in their live. However many bitches exhibit mild lactation and galactorrhea with no other noticeable sign. Prolactin is higher in bitches that have overt psuedopregnancies as compared to those that have covert psuedopregnancies. Diestrus can be considered a covert pseudopregnancy. Overt pseudopregnancy, or pseudocyesis or pseudogenetra occur in some bitches.

Pathophysiology

Pseudo pregnancy is a diestrual endocrine disorder, commonly occurs at the end of diestrus after serum progesterone concentration abruptly decline. Normal intact bitch exhibits lacteal phase of 2 months regardless of breeding and pregnancy status, mammary gland undergoes glandular development after every estrus cycle. Serum P4 concentration rise until 13 to 16 days and then decline within last 7 days of diestrus. No significant difference was found in serum p$ concentration during diestrus between pregnant and nonpregnant bitches or with covert and overt false pregnancy or between bred or unbred bitches. Most bitches show evidence of pseudo pregnancy being very variable with little or no signs. While overt pregnancy will range from mammary development and lactogenesis and will undergo mock parturition with nesting, loss of appetite, straining, emotional attachment to inanimate objects.

Other events that exacerbate the signs are self nursing by the bitch, massage of the mammary glands by the owner, hot packs of the mammary glands, and milking the glands out.

Iatrogenic pseudogenetra can be caused by ovariohysterectomy in diestrus. This results in a similar progesterone decline as seen in the naturally occurring condition.

Progesterone supplementation during a pseudogenetra will temporarily stop the signs, but the removal of the progesterone will result in a recrudescence of the signs.

Endocrinology

The bitch has a prolonged luteal phase with persistence of corpus luteum for 70-80 days in nonpregnant animals. The peripheral P_4 concentration are similar to those of pregnant bitches. Concannon *et al* (1975) obtained mean maximum value of 29 ng/ml for pregnant and 27 ng/ml for nonpregnant bitches. However, there was a lot of individual variation with peak values after LH peak in both pregnant and nonpregnant bitches between 8-28 days. Decline in progesterone concentration in late diestrus concentration is accompanied by increase in serum estrogen and prolactin concentration plasma prolactin concentrations are elevated 2 to 5 times in late diestrus in all intact bitches than early diestrus. Mammary gland development is stimulated by the prolonged exposure to serum progesterone and production of milk is stimulated by presence of plasma prolactin.

Clinical Sign

Pseudogenetra or overt pseudopregnancy occurs at the normal expected whelping time. It include physical signs like abdominal enlargement, mammary development and development of maternal instinct such as nesting, mothering and adopting inanimate objects.

Diagnosis:

1. Physical examination by exclusion of pregnancy is carried out and is achieved by
    a. Lateral abdominal radiograph 50 days after breeding.
    b. Abdominal Ultrasonography taken more than 24 to 28 days after breeding .
2. Analysis of single mammary fluid sample to assess the protein content..
3. Prolactin assay.

Treatment :

No treatment is best.

Remove all physical causes such as self nursing by the bitch, massage of the mammary glands by the owner, hot packs of the mammary glands, and milking the glands out.

Short term oral therapy for a period of 4 to 5 days with Dizepam, a mild tranquliser. But, Phenothiazine tranquilizers can cause more milk release because they are dopamine antagonists and dopamine is a prolactin antagonist, so it will result in more lactation.

Administration of progestin like megesterol acetate @ 2.5 mg/kg body wt. orally daily for 8 days.

Administration of androgens commonly testosterone propionate at @ 0.6 to 37 mg/ kg body wt. for 2 to 10 days. It is reported that a dose of 16 mg/kg/day orally for 5 days is optimal to show clinical improvement. Mibolerone is an androgen and has been shown to improve 100% of

the physical and 90% psychological signs when administered @ 0.016mg/kg for five days. It cured 47% of the physical and 77% of the psychological signs.

Use of serotonin antagonists like cabergoline @ 1.5 to 5 mg/kg/day orally for 2-8 days cause clinical response within 3 to 4 days. Metergoline is also a serotonin inhibitor and also inhibits prolactin. The dose is 2 mg BID for 10 days. But it may cause whining and aggression.

Dopamine agonists such as Bromocriptine @ 30 mg/kg/day orally for 10 to 15 days is reported to cause decreased lactation and behavioral signs. But emesis is reported to occur, may be controlled with treatment with Metoclopromide @ 0.5 mg/kg.

Estrogens are described as a therapy but have limited clinical use due to side effects like uterine and vaginal hypertrophy, blood dyscariasis, prolonged sexual receptivity and pyometra.

Discomfort secondarily occur due to mammary gland engorgement may be reduced by cold and warm compress on engorged mammae alternately or by wrapping ace bandage on mammary gland.

Sequelae to pseudogenetra

Mastitis

Mammary hypertrophy

Owner irritation.

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GYNAECOLOGICAL EMERGENCIES IN PET ANIMALS

D. N. Mohanty, Proffesor and head, dept. of Gynaecology
Faculty of veterinary science & animal husbandry
Bhubaneswar, Orissa

The classical stages of the canine estrus cycle has been earlier documented[1]. This was prior to the understanding of the hormones. So his classifications of various stages were based on sexual behaviour and physiologic changes in the reproductive tract. At present the definitions are based on; behavioural, clinical, physiologic, cytologic and endocrinological changes in the bitch.

Prebreeding examination of the bitch prior to breeding should be encouraged with respect to :

1. General physical examination
2. Immunization record
3. Testing for heart worm and other parameters.
4. Screening for genetic diseases (281 nos.-1988; 400 nos.-1994)
5. Radiography – for hip dysplasia (hereditary) –pg.41
6. Thyroid testing

Understanding the physiology and endocrinology of pregnancy and eutocia in bitch is necessary for preventing, diagnosing and treating the anomalies. Although the exact mechanisms for initiation of parturition has not been completely known, but studies in bitch and other species have provided data that allow veterinarian to understand and identify the problem.

Basing on the endocrinology of canine parturition the following factors can be the contributory factors.

1.	Corticosteroids	-	Fetal ACTH increased, release of fetal cortisol
2.	PGFM (13,14-dihydro 15-keto-prostaglandin F_2C)	-	Increased synthesis and release of PG
3.	Progesterone	-	Prepartum decrease to < 1-2 µg/ml
4.	Prolactin		- Increase in PRL conc. from 40± 7 µg/ml to 117 µg ± 24 µg/ml within 32 hrs to parturition.

Predicting Onset of Parturition:

Predicting day of whelping is difficult as the canine gestation length can range from 57 to 72 days [2].

When canine gestation length is timed from ovulation, the duration is more precise, ranging from 62 to 64 days.

The predictions of delivery date are:

1.	Breeding date	- 57-72 d from a single breeding
2.	Ovulation date	- 64 -66 d after serum LH surge (determined by serum LH)
3.	Serum P_4	- 1-1.9 µg/ml-63-65 d 2-3.9 µg/ml – 63-65 d 4-10 µg/ml- 62-64d Ø Parturition within 12 -24 hrs after P_4 conc. drops< 1-2 µg/ml
4.	Diestrus vaginal serum	- 57d (approx) after onset of cytologic diestrus (decreased superficial cells and increased small intermediate cells)
5.	Rectal temperature	– Drop to 98.8°F, between 8-24 hr. prior to parturition
6.	Radiographic appearance of fetuses (prepartum)	- 20-22d , spine, skull , ribs 2-9 d, caudal vertebrae, fibula, paws 3-8d, teeth
7.	Ultrasonographic appearance	- fetal biparietal diamt. and fetal trunk diamt.
8.	Onset of lactation	- 2 wks before parturition
9.	Nesting behaviour	- 5-7 d prior to parturition to first stage of labour.
10.	Cervical , vaginovestibular and vulvar relaxation	- Near parturition
11.	Lochia (greenish black discharge)	- following placental sepration, with whelping occurring 1-2hr of its presence.

The most commonly encountered gynaecological emergencies in pet animals are:

1. Uterine inertia
2. Dystocia
3. Uterine torsion
4. Inguinal hernia
5. Uterine rupture

6. Septic metritis and toxaemia
7. Metritis
8. Hypocalcaemia
9. Pregnancy toxaemia
10. Medical induction of parturition

1. Uterine Inertia:

PRIMARY – Characterized by a failure to expel normal sized fetuses through a birth canal that is normal except for an incompletely dilated cervix.

It is complete if no signs of second stage labour occurs.

Etiology— Mechanical , hormonal , physical and genetic components [3].

This occurs when parturition begins normally, but uterine contraction stops before expulsion of the puppy. Mostly occurring with :

1. Inherited breed predisposition (Terrier) with overstretched uterus containing large litter.
2. Inadequate uterine stimulation in one or two-pup litters.
3. Systemic disease- hypocalcaemia
4. Obesity
5. Uterine infection in septicemia
6. Inadequate nutrition
7. Uterine torsion
8. Trauma

Ø Serum calcium measurement in affected bitches

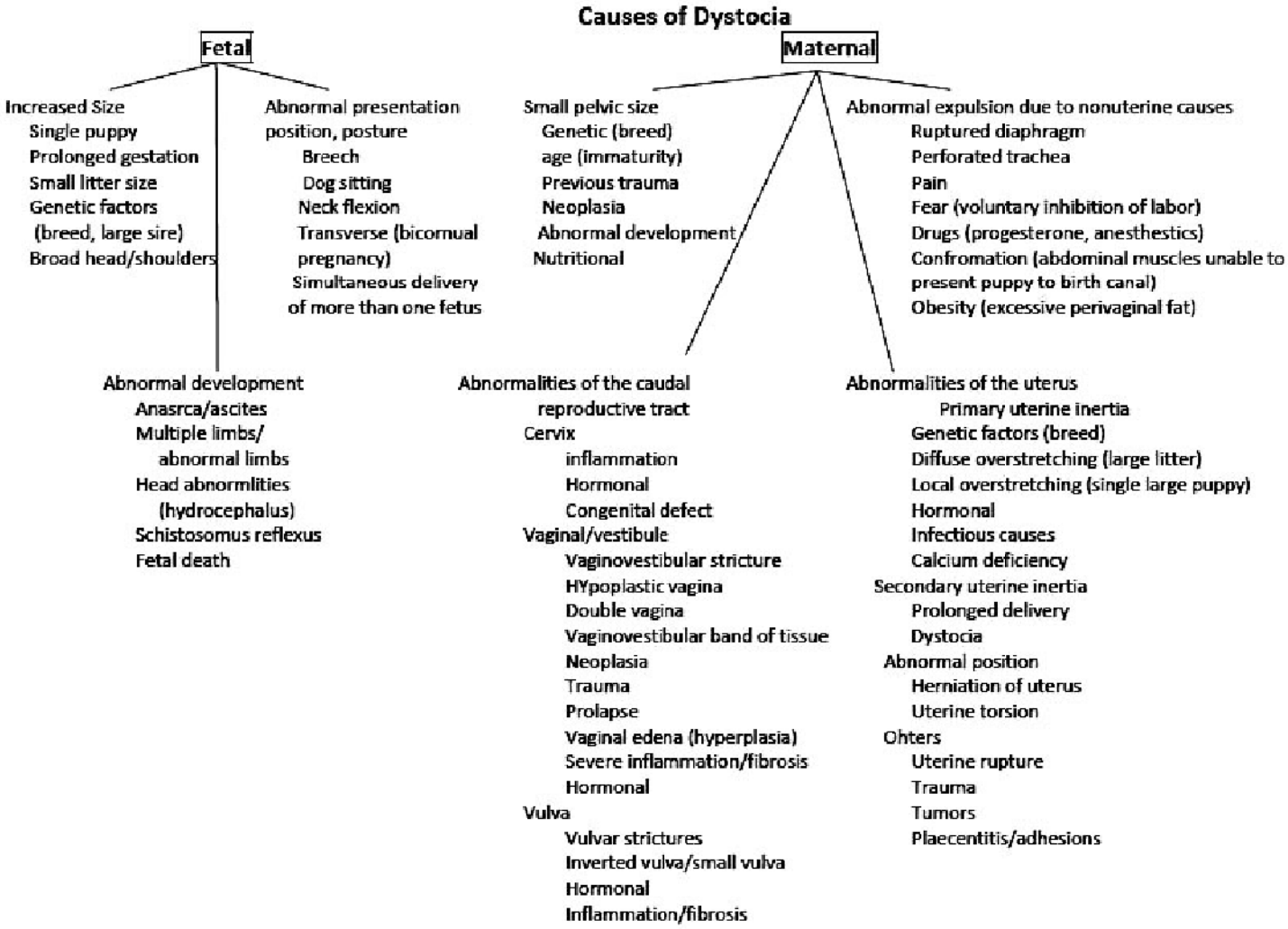
Prognosis- Guarded to good

SECONDARY - Prolonged uterine contractions failing to expel a fetus.

A. Strong and frequent stage II '!Abdominal straining that fails to produce a pup within 30 minutes.

1. Perceived failure to start parturition on time.

2. Dystocia:


Causes of Dystocia
Fetal
Maternal
Increased Size
Single puppy
Prolonged gestation
Small litter size
Genetic factors
(breed, large sire)
Broad head/shoulders
Abnormal presentation
position, posture
Breech
Dog sitting
Neck flexion
Transverse (bicornual
pregnancy)
Simultaneous delivery
of more than one fetus
Abnormal development
Anasrca/ascites
Multiple limbs/
abnormal limbs
Head abnormlities
(hydrocephalus)
Schistosomus reflexus
Fetal death
Small pelvic size
Genetic (breed)
age (immaturity)
Previous trauma
Neoplasia
Abnormal development
Nutritional
Abnormal expulsion due to nonuterine causes
Ruptured diaphragm
Perforated trachea
Pain
Fear (voluntary inhibition of labor)
Drugs (progesterone, anesthestics)
Confromation (abdominal muscles unable to
present puppy to birth canal)
Obesity (excessive perivaginal fat)
Abnormalities of the caudal
reproductive tract
Cervix
inflammation
Hormonal
Congenital defect
Vaginal/vestibule
Vaginovestibular stricture
HYpoplastic vagina
Double vagina
Vaginovestibular band of tissue
Neoplasia
Trauma
Prolapse
Vaginal edena (hyperplasia)
Severe inflammation/fibrosis
Hormonal
Vulva
Vulvar strictures
Inverted vulva/small vulva
Hormonal
Inflammation/fibrosis
Abnormalities of the uterus
Primary uterine inertia
Genetic factors (breed)
Diffuse overstretching (large litter)
Local overstretching (single large puppy)
Hormonal
Infectious causes
Calcium deficiency
Secondary uterine inertia
Prolonged delivery
Dystocia
Abnormal position
Herniation of uterus
Uterine torsion
Ohters
Uterine rupture
Trauma
Tumors
Plaecentitis/adhesions

2. Perceived failure to progress normally with delivery of puppies once labour has begun.

Predisposing Factors :

1. Fetal and maternal factors and many of these occurring together [4].

   e.g:- Foetal oversize a fetal factor leads to secondary uterine inertia, a maternal factor.

SO WHILE PREDICTING OR DIAGNOSING DYSTOCIA IT IS ESSENTIAL TO CHARACTERIZE MATERNAL AND FETAL FACTORS.

During second stage of labour strong abdominal straining or tenesmus is suggestive of pup presence in birth canal.

Assistance may be necessary if the bitch does not deliver the pup within 30 minutes. Pup lodged in the birth canal can die if complete placental separation is not followed by delivery.

Obstruction compromises other pups remaining in the uterus.

Eventually active straining and labour will subside as secondary uterine inertia develops.

B. Weak or Intermittent stage II'! Abdominal straining that fails to produce a pup within 4 hrs (1st pup) or 2 hrs(between pups).

After 4 hrs of onset of the stage II labour ¯ Pup is not delivered or 2 hrs between pups is suggestive of infective uterine contraction to advance the foetus through the birth canal.

These bitches frequently respond to medical management with oxytocin or calcium.

Incidence of stillbirth rises as the time interval between delivered pups increases.

Reliable sign of beginning of whelping ->

Presence of lochia or uteroverdin (greenish –blackish vulvar discharge) indicates placental separation has begun.

It has reported that sometimes after the death of the caudal foetus in the uterus before term, lochia will pass and the remaining pups are born normally at term[5].

IMPORTANT NOTE:- Passage of lochia from a term bitch signifies whelping to commence within 1-2 hrs . Failure signifies a potential dystocia and if not relieved within 24 hrs the entire litter of pups may die.

Common observation to be noted by the owner:

i. Copious amount of clear, water like vulvar discharge - Allantoic or amniotic fluid

ii. Confusion with the placental fluids and urination.

iii. Observing a sac or bubble protruding from the vulva lips - caudal portion of allantochorion.

All these signifies the presence of a pup in the birth canal.

Diagnostic Evaluation[5] :

To confirm - i. Pregnancy is present

ii. Parturition is not proceeding normally

iii. The cause of dystocia

iv. Detect maternal anal/or fetal compromise

The necessary evaluation should be based on –

a. Complete history – general health , prior illness, previous reproductive performance (breeding dates), past whelping & dystocia, rectal temp. measurements during pregnancy.

b. Physical exam.- temp.,pulse, respiration rate, capillary refill, hydration status.

c. Thorasic auscultation – cardio pulmonary function

d. Abdominal palpation – fetal presence

e. Palpation of fetal movement and auscultation of fetal heart beats (absence does not confirm fetal death)

f. Inspection of mammary gland- normal or abnormal secretion of milk

g. Examination of vulva – lochia or blood

h. Digital examination of the vestibule and vagina – ascertain the relaxation of the birth canal, presence or absence of foetus, soft or bony tissue impingement on the birth canal.

i. Abdominal radiography – fetal number , size, position, signs of fetal death.

Radiographic signs of fetal death –

i) Presence of gas within foetal body cavities or blood vesels.

ii) Alteration in the spatial relationship between bones of the axial skeleton.

iii) Overlap of foetal cranial bones

iv) Failure of the skeleton to calcify or continue to grow

j. Ultrasonographic examination – Presence or absence of intrauterine fetal viability. it has been suggested that fetal distress can be predicted using ultrasonographic observation of brady cardia when fetal tissue pH reaches 7.05 and fetal heat rate is 40 to 130 beats/min[6].

k. External monitoring devices and hand held Doppler units (Whelp Wise) - Assessing uterine contractile activity and fetal heart rate.

imp. note:- Fetal heart rate - < 130 beats/min; poor viability of pups if not delivered within next 2-3 hrs.

< 100 beats /min; immediate veterinary intervention

l. Laboratory evaluation - Complete blood count, serum chemistry (glucose, calcium) urine analysis

Treatment:

I. Manipulative treatment -

Extracting malpositioned or slightly oversized foetus (snook ovariohook, sponge forceps, clamshell forceps) only these instruments are to be used when fetus is dead or there is adequate room space.

Protruding puppy from vulva - Lubricating the birth canal and grasping with a gauze sponge. Grasping tail or limb are not to be encouraged .

Puppies obstructing the birth canal and barely protruding are to be recovered by C.S.

II. Medical treatment-

Thumb rule for indicating medical treatment

(i) Bitch is in good health

(ii) Labour has not been unduly protracted

(iii) Cervix is dilated

(iv) Fetal size is consistent for vaginal delivery.

When medical treatment produces a slow response and many pups remain in utero then indicate C.S.

Administration of ecbolic drugs is not indicated in obstructive dystocia The various forms of medical treatment to be considered are;

a. Oxytocin:

Action- uterine contractions and milk ejection

Synthetic oxytocin .Half life is 1-2 mins

Safe for both bitch and pups.

But high and repeated doses results uterine hyper stimulation or (uterine tetany) with fetal distress[5].

Recommended dose - 5-20 units/dog, i/m at 30-40 min interval, poor response may be seen when the extracellular calcium conc. are low

b. Calcium- Added to oxytocin administration

Oxytocin increase frequency where as calcium increases strength of uterine contractions

(myometrial contraction). Combination results a direct action on the rate of Ca ion influx into the myometrial cell [7].

Dose - Slow i/v over 3-5 min, 10% calcium gluconate @ 0.2 ml/kg or 1-5 ml/dog s/c., discontinued if the animal is restless or change in the heat rate and rhythm occur.

c. Ergonovine - Ergot alkaloid

Not to be used for treating canine dystocia rather to be used for uterine contraction and vasocerotictism in post partum haemorrhage.

Dose - 10-30 µg/kg per os or i/m

Combination with oxytocin in women results in less blood loss post partum but it causes nausea, B.P. & vomiting.

d. Glucose for clinical management

Oral glucose or 5 to 10% glucose solution i/v

e. Tranquilizers- indicated for

i. overcoming voluntary inhibition of parturition

ii. facilitate clinical & vaginal examination.

Uses is to be discouraged

Barbiturates and promazine derivatives are poorly metabolized by fetal liver.

Occasionally, dopamine antagonists (acepromazine) and phenothiazine block dopamine receptors can be given post partum to increase pituitary release of prolactin.

MEDICAL APPROACHES FOR TREATING CANINE DYSTOCIA

History	Approach
1 or 2- pup litter or fetal oversize	C.S.
5 or more pups remaining in uterus	C.S.
4 or less pups remaining in uterus with a non obstructed birth canal	1.Oxytocin 0.1 – 2.0IU/kg i/m not exceeding 20IU 2. If pup is born with 30 mins repeat oxytocin at 30 min interval if delivery slows add calcium 3. If put is not born with 30min of oxytocin admn., give 10% (calcium gluconate 0.2ml/kg) i/v not exceeding 5 ml. Repeat oxytocin after calcium . If no pup is born after 30 min. C.S.

III Uterine Torsion:

Relatively uncommon due to long and freely movable uterine horns. One or both horn can twist along the long axis or arround the opposite horn, or the entire body can rotate[8].

Symptoms - Severe pain with abdominal distension

Haemorrhagic vulva discharge

Tachycrdia

Signs of shock

Dystocia.

Severe torsions can cause obstruction of the blood supply to the uterus resulting in thrombosis or rupture of the uterine vessel, congestion, shock, fetal and /or maternal death. Rupture may occur at parturition.

Diagnosis - From clinical signs

Ultrasonographic exam.

Exploratory laparotomy

Treatment - Immediate surgical correction (hysterotomy to remove foetus or hysterectomy if thrombosis and gangrene are present)

IV Inguinal Hernia:

Occasional occurrence when pregnant uterine horns enter through inguinal ring and can result in dystocia.

Congenital defects - Basset hound, Cairn terrier, Basenji, Pekingese, West Highland White terrier.

Treatment - 1. Surgical repair of the hernia for preventing ischemic compromise.

2, C.S. for delivery of term pups.

V Uterine rupture:

Rare in bitch

Follows uterine trauma or trauma

Condition may go undiagnosed until dystocia results when puppies fail to enter the birth canal.

Death of foetus immediately when fetus expelled to abdominal cavity and be resorbed (if fetal calcification has not occurred) or retained as mummified foetus.

Possible sequel – Peritonitis.

VI Septic Metritis and Toxaemia:

It is evident by 48-72 hr after intra uterine fetal death.

When dystocia gets undiagnosed or untreated, the entire litter dies within 24 hrs.

The foetus then serves as the substrate for infection with ascending vaginal bacteria.

Diagnosis - History or ultrasonography

Symptoms - Elevated rectal temperature, degenerative left shift of WBC.

Treatment - Aggressive fluid and antibiotic therapy

Hysterotomy.

VII Metritis:

Acute puerperal metritis a disease of immediate post partum period (0 to 7d post whelping) is severe inflammation or the endometrium and myometrium that causes systemic illness in the bitch.

Etiology- Retained placenta

Retained pups

Macerated and decomposed pups

Prolonged delivery

Bacteria thrive in retained or devitalized tissues leading to inflammation of endo- and myometrium and if untreated leads to septicemia and toxaemia.

Symptom- Depression

High rectal temp.(103°-105°F)

Putrid, reddish brown uterine discharge

Hypovolumic shock from dehydration

Septicemia or endotoxaemia

Diagnosis- Cytologic evaluation of uterine discharge

i. Neutrophils – degenerative

ii. Bacteria

iii. Erythrocytes ,endometrial cells and muscle fibers from decomposing fetuses

iv. Leukopenia with immature neutrophils.

Treatment- 1. Treating shock- replacing fluid deficits

2. Initiating broad spectrum antibiotics

3. Dextrose i/v (if hypoglycemia)

Once the bitch is stabilized, surgical intervention is required to remove remaining placenta and/or devitalized fetal or uterine tissues.

Then culturing the contents.

Merits of infusing the uterus with antibiotics or draining uterine contents are unknown[5]. Antibiotic infusions may be contraindicated[9] as it impede phagocytic function of uterine neutrophils. Antiseptic solutions infusion may damage uterine neutrophils .

Role of various ecbolic agents in treatment is uncertain . Using ergonovine is not recommended because of uterine rupture.

Efficacy and safety of using PGF_2á have not been studied carefully. Hormones as in bovine cases it may be judiciously used depending on the integrity of the myometrium and uterine wall.

VIII. Hypocalcemia: Eclampsia, Puerperal tetany

Depletion of calcium in the extracellular compartment characterized by nervousness elevated body temp., dry mouth ,sclera, panting , restlessness, whining, tremors, staggering. It may occur prior to parturition and is far more common during the first few weeks post partum. Observed generally in smaller breeds.

Symptoms - Restlessness ,pacing, panting, reluctance to care for the pups, stiffness before the onset of muscle tremors, tetany and convulsions.

Diagnosis 1. Hyperthermia- 105°F (due to increased muscle activity)

2. Electrocardiogram – Deep, wide T waves, prolonged Q-T interval

Taller R waves

3. Blood calcium- < 7 mg/100ml (normal 9-11mg/100ml) Confirmative diagnosis.

4. Blood glucose- Normal (differentiating from pregnancy toxemia)

Differential diagnosis of seizures - epilepsy, meningoencephalitis, poisoning.

5. Magnesium conc. – Normal

Treatment 1. Slow i/v administration of calcium gradual cooling the bitch.

2. 10% solution of calcium gluconate (0.22 to 0.44 ml/kg)i/v

3. Glucocorticoids not to be used (decreases intestinal absorption, enhance renal excretion of calcium)

4. Oral administration of calcium or Vit.D may be of benefit after initial i/v treatment.

5. Removing pups to reduce lactational drainage.

Prevention - Balanced diet with Ca. P ratio (1:1 to 1:2:1)

Dietary cation–anion difference may be more crucial than calcium intake during pregnancy in preventing hypocalcaemia . Feeding highly anionic (acidic) diet were more responsive to parathyroid hormone , enabling quick mobilization of calcium from bone .

Current recommendation for preventing eclampsia in bitch –

Feeding diets during pregnancy that are not excessive in calcium. So during lactation calcium supplementation to bitches is imperative for preventing Eclampsia.

Dry dog feed diets – high percentage of soybean meal or bran

IX Pregnancy Toxaemia:

Can occur days to weeks before parturition and is also associated with prolonged gestation and dystocia.

Etiology- Inadequate nutrition

Bitches with large litters

Ø Ketonuria without glucosuria is a hallmark of prepartum pregnancy toxemia in bitch.

Symptoms - Weakness, inability to stand, seizures and coma.

Hepatic lipidosis

Diagnosis - Urine ketones in the absence of urine glucose hypoglycemia

Treatment - Supplemental nutrition

I/v dextrose administration

Medical induction of parturition with glucocorticoids.

X. Medical Induction of Parturition:

Indication - Pregnancy compromising with maternal health (Preg. Toxaemia)

Preterm induction of parturition in the healthy bitch has not been well studied. The common drugs that can be tried are:

i) Glucocortricoids;

Dexamethasone is effective in terminating with 2 to 16 days after initiation of treatment.

Dose - 0.2 mg/kg/os TID for 5 days followed by

0.16 to 0.02/kg/is TID for next 5 days

Termination of canine pregnancy of 57 -58 day at a dose rate of 0.4 mg/kg parenterally one time[10].

Long term glucocorticoids enhance viability of pups by enhancing maturation of fetal lungs [11].

(ii) Prostaglndings;

Terminate pregnancy after 3 to 5 day at the dose rate given S/C or i/m

a. 20/µg/kg every 8 hr

b. 30µg/kg every 12 hr for 72 hr

c. 250µg/kg every 8 hrs for 4 days.

Majority of the fetus expelled are live with placenta intact.

(iii) Oxytocin; Not known whether it can induce parturition in term bitch or with elevated P_4conc.

(iv) Mifipristone(RU-486)

Progesterone antagonists can induce premature delivery in bitches[12]. Bitches on 32d pregnancy can be induced with mifepristone, expelling dead foetus or dark mucoid vulvar discharge. But it is unknown whether it can induce near term.

XI Retained Fetal Membrane:

The persistence of green genial discharge after 12 hrs. of birth of last puppy is indicative of a retained after birth.

Vaginal exploration with finger & by fiddling to bring out the umbilical cord & gentle traction is applied to withdrawal the placenta.

The uterus is palpated through the abdominal wall& detect it's presence (in small bitch) as an egg like distension. Fine parts of the bitch raised & firm pressure is applied to the distended part. By this separation occurs & the placenta is expelled immediately.

Treatment is to be repeated after a few hours.

Oxytocin is administered half an hour after the last fetuses have been delivered for expulsion of the terminal placenta if present.

Radiographic examination of the abdomen may be undertaken.

In no response by Oxytocin administration or abdominal manipulation , lapratomy is indicated for milking the fetal membrane along the uterus towards the cervix. If this fails hysterotomy can be performed to relieve them[13].

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EPILEPSY IN DOGS AND ITS MANAGEMENT

R. C. Patra and S. K. Parida
Department of Medicine, College of Veterinary Science and Animal Husbandry,
Orissa University of Agriculture and Technology, Bhubaneswar – 751 003

Epilepsy refers to series of frequent seizures or convulsion and does not identify a disease, but rather describes a clinical sign. It is associated with paroxysmal self-limiting functional cerebral disturbance characterized by abnormal electrical activity of the brain leading to overt clinical signs of random muscle movement and sudden loss of consciousness. Each attack of epilepsy in dogs, like in human beings, can have seizures and seizure disorders. Basically, brain cells use electrical and chemical signals to communicate, which can either activate or shut off another neuron. Seizures are thought to be caused when there is an imbalance of excitatory and inhibitory signals in the brain. The malfunctioning of brain results in firing a barrage of electrical signals through the nervous system and is manifested by convulsion either in a specific body part, or more commonly, many areas at once. Many a times, the word epilepsy is used interchangeably with seizures, convulsions, attacks or fits. It is one of the top three concerns among more than 80 health issues in dogs, according to recent survey conducted by American Kennel Club. It affects anywhere from 0.5 to 5.7 percent of canine population.

A seizure presents a terrifying spectacle- especially because the dog seems to have no awareness of the surrounding. Most seizures are not life threatening and will end in a matter of seconds, although it may seem like hours. However, the dog with seizures may requires immediate and critical veterinary intervention if the seizures continues to persist for more than 30 minutes without relapse. The dog may paddle his feet or become rigid and violently thrash about.

There are many possible reasons why a dog might have a seizure. Anything capable of changing the nerve function within the brain may produce a seizure. Known causes of seizures include infectious diseases (distemper), metabolic diseases (hypoglycemia), toxic substances, neoplasia (tumors), and certain bacterial and fungal organisms. A metabolic disorder, such as hypoglycemia (low blood sugar), is often the cause in toy breeds. Hypocalcemia (low calcium) can cause seizures in bitches, nursing puppies. Puppies with distemper are also prone to seizures. Sometimes, a dog can develop seizures after an accident or a blow to the head. A post traumatic scar may develop and result in seizures. Encephalitis, a brain infection that causes inflammation, can lead to convulsions. Brain tumors, more often found in older dogs, can cause malfunctioning in the nervous system inducing seizures. Other causes of seizures include heat stroke, congenital malformation of the brain, and ingestion of toxic chemicals.

The epilepsy in dog is broadly classified into two types.

1. Idiopathic or Primary Epilepsy- No known cause for the condition and it is assumed to be an inherited condition

2. Secondary Epilepsy - This diagnosis is used when a specific cause for the seizures can be established. A veterinarian will normally run a variety of tests to rule out possible physiological or toxic causes before diagnosing the dog as having the idiopathic epilepsy.

In a retrospective study, Varshney and Ali (2000) observed that the problem was more common in adults and males than younger dogs and females. Extra-cranial causes noted in such problem in dogs included strychnine toxicosis, hypocalcemia, and hypoglycemia by *Babesia gibsoni*, drug intoxication, and intracranial causes were viral encephalitis, canine distemper etc. Besides, there is a long list of potential causes. A diagnosis of idiopathic epilepsy is generally made when all other seizure-inducing factors have been ruled out.

Idiopathic epilepsy is the most common cause of recurrent seizures in dogs and the age of onset is usually between 1 and 3 years. Some practitioners use a broader range, from 6 months to 5 years. The published prevalence ranges from between 0.5 to 4.1 percent and males are more affected than females (Jaggy et al., 1998). In 125 cases of confirmed idiopathic epilepsy, seizures were recorded in dogs of all the age groups and the peak age for the onset of first seizure was between one and five years. On the other hand epilepsy is called acquired or secondary when it occurs in previously healthy dogs whose brain has become seizure-prone for some specific reason.

Breed predisposition and inheritance:

The genetic basis of idiopathic epilepsy in dogs has received considerable attention in recent years. Certain breeds seem particularly prone to developing it, suggesting the role of a genetic factor. Among the breeds most commonly affected are Beagles, Dachshunds, Dalmatians, German shepherd dogs, Keeshonds, Boxers, Cocker and Springer spaniels, Collies, Golden and Labrador retrievers, miniature Schnauzers, Poodles, St.Bernards, Siberian Huskies, and Wire-haired terriers. Following recognition by the Swiss Labrador retriever club that seizures in this breed were becoming a matter of increasing concern, a study was conducted involving 799 pedigree certificates from a population of healthy and epileptic dogs (55) from 11 generations (Jaggy et al., 1998). The study revealed that males were no more affected than females. The increased manifestation of seizures in some subpopulation and the repeated occurrence in different families of the same sire suggested that there was genetic basis for the condition in the breed. The results also supported the hypothesis of a polygenic, recessive mode of inheritance that requires to be defined through objective test-mating programme.

Possible causes of seizures in dogs of different age group

- **Under 8 months-** Developmental Disorders, Encephalitis or Meningitis, Trauma, Portacaval shunt, Hypoglycemia, Toxins, Intestinal parasites, Idiopathic Epilepsy (rare)
- **8 months to 5 years-** Idiopathic Epilepsy (most common), Developmental disorders, Trauma, Encephalitis or meningitis, Acquired hydrocephalus, Neoplasia (tumor), Portacaval shunt, Hypoglycemia, Electrolyte disturbances, Hypothyroidism, Toxins
- **Over 5 years-** Neoplasia (tumor), Degenerative disorders, Vascular disorders, Hypoxia (lack of oxygen in body tissues), Hypoglycemia, Idiopathic Epilepsy, Trauma, Encephalitis or meningitis, Acquired hydrocephalus, Serious Liver disease, Hypocalcemia, Electrolyte disturbances, Hypothyroidism

Pathophysiology

A seizure is a transitory disturbance of brain function. It has a sudden onset, ceases spontaneously, and tends to recur. A seizure results from a sudden and uncontrolled electric discharge of neurons in the cerebral cortex of the brain due to paroxysmal depolarizing shift of neurons. Neurons can spontaneously discharge for several reasons, including decreased inhibitory neurotransmitter activity, increased excitatory neurotransmitter activity or combination of both. Neurotransmitter activity can be altered by a change in the cell membrane or the internal cell metabolism. At the beginning of a seizure, only a few highly unstable neurons may spontaneously discharge. This initial discharge can cause surrounding neurons or neurons in the opposite brain hemisphere to discharge as well and spread the seizure activity by a processes referred to as kindling and mirroring, respectively. Spontaneous discharge can be triggered by almost any alteration in a neuron's environment. Neuronal changes influence the threshold for depolarization that causes seizure activity. High frequency and low amplitude paroxysmal discharge with either a focal or generalized distribution have been observed in majority of cases of idiopathic epilepsy, and electroencephalographic features during interictal period reamin consistent, despite anaesthesia (Jaggy and Bernardini, 1998)

There are many evidences justifying the role of immediate early gene c-fos in physiological and pharmacological properties in the central nervous system. To elucidate the role of c-fos in seizure induction, male wistar rats were pretreated with antisense c-fos and nonsense c-fos oligodeoxynucleotides 12h prior to intraperitoneal administration kainic acid that induces neuronal discharges. Antisense c-fos unlike nonsense oligodeoxynucleotides inhibited the number of wet dog shakes and the appearance of limbic motor seizures. The anticonvulsant effects were associated with reduction of both Fos and NGFI-A immunoreactivity and neuroprotection in the hippocampus, thalamus and primary olfactory cortex-amygdaloid region suggesting that c-fos plays a role in the generation of kainic acid-induced limbic seizures and neuronal death (Panegyres and Hughes, 1997). Astrocytes support the neuronal function and helps in altering the seizure susceptibility through ATP production and aconitase activity. Inhibition of aconitase activity in astrocytes lowers the doses of both kainic acid and pilocarpine that is required to induce behavioural seizure (Lian and Stringer, 2004)

Clinical manifestation

Most seizures occur in three stages, each characterized by specific clinical signs. The first part of a seizure, called the *aura*. This often goes unnoticed but the dog shows changes in the behaviour signaling an impending seizure, and is clinically characterized by apprehension, restlessness, nervousness, and salivation. The period of aura lasts from a few seconds to few days. The aura is followed by the actual seizure, called the *ictus*. Although it seldom lasts for more than one minute, it can be a very disturbing event to the owner. During the seizure, the animal usually collapses onto its side and experiences a series of violent muscle contractions associated with paddling of the feet and rigidity of the body. Loss of consciousness, excessive salivation, and involuntary urination and defecation may also occur in more severe seizures. The period immediately following the seizure is known as the *postictal* phase. It usually lasts less than one hour but may last as long as one or two days. The animal may show signs of confusion, disorientation, restlessness, and temporary blindness. In cases of repeated seizures, the inter-ictal period denotes the interval between seizures.

Some animals are normal during this period and others are not, depending on the cause of the seizures.

Behavioural changes associated with seizures include loss of memory, lack of consciousness, altered muscle tone or movement, alteration in visual, auditory, or olfactory hallucinations and salivation, urination, defecation, or other autonomic nervous system disruptions.

Thus, depending on severity of the seizure and the duration of different stages, it is termed as Grand mal, when the seizure is severe. The term petit mal refers to a generalized seizure with a specific EEG pattern. However, the above two terminologies are no more used. A series of seizures within a short period of time, with the dog regaining consciousness between seizures is termed as Cluster seizures. Status epilepticus refers to rapidly repeating seizures with no period of consciousness between them. Seizures are classified as either partial or generalized. In the former case, neurons discharge in a specific area. In case of generalized seizures (also referred to as tonic-clonic seizures) involve an animal's entire body. During the first (i.e. tonic) phase, which usually lasts between 10 and 30 seconds, an animal falls to the ground, loses consciousness, and rigidly extends to legs; it may also stop breathing or shake. In the second (i.e. clonic) phase, the animal's legs make running or paddling, its mouth makes chewing motions, and it may continue to shake. In addition, an animal may urinate, defecate, have dilated pupils, salivate excessively, vocalize, or vomit during either phase. A seizure may alternate between tonic and clonic phases once or repeatedly for the duration of the seizure. The entire seizure usually lasts between 1 and 2 minutes.

Thus, depending on the clinical manifestations, seizures are classified as follows.

Generalized Seizure: Tonic-colonic (Grand Mal or Mild)

In the grand mal seizure, the tonic phase occurs as the animal falls, loss consciousness, and extends its limbs rigidly. Respiration also stops (apnea). This phase usually lasts 10-30 seconds before the clonic phase begins. Clonic movements include paddling of the limbs and/or chewing. Other signs that appear during the tonic or clonic phase are dilation of the pupils, salivation, urination, and defecation. The mild seizure involves little or no paddling or extension of limbs, and usually no loss of consciousness. Generalized seizures are usually associated with primary epilepsy.

Petit Mal Seizure (aka Absence Seizure)

Depending on the authority quoted, petit mals are described as either very rare or usually unrecognized in animals. Signs are brief (seconds) duration of unconsciousness, loss of muscle tone, blank stare, and possibly upward rotation of eyes. According to one authority (Kay), the term petit mal is misused by veterinarians and should only be accorded to cases manifesting very specific clinical signs and EEG abnormalities.

Partial Seizures

Movements are restricted to one area of the body, such as muscle jerking, movement of one limb, turning the head or bending the trunk to one side, or facial twitches. A partial seizure can progress to (and be mistaken for) a generalized tonic-clonic seizure, but the difference can be established by noting whether or not a seizure starts with one specific area of the body. Partial seizures are usually associated with secondary epilepsy.

Complex Partial Seizure (aka Psychomotor or Behavioral)

It is associated with bizarre or complex behaviors that are repeated during each seizure. People with complex partial seizures may experience distortions of thought, perception or emotion (usually fear), sometimes with unusual visual, olfactory, auditory and gustatory sensations. If dogs experience the same things, it may manifest lip-smacking, chewing, fly-biting, aggression, vocalization, hysterical running, cowering or hiding. Vomiting, diarrhea, abdominal distress, salivation, blindness, unusual thirst or appetite, and flank biting are other signs. There is an obvious lack of awareness though usually not lack of consciousness. Abnormal behaviors may last minutes or hours and can be followed by a generalized seizure. Complex partial seizures are usually associated with secondary epilepsy.

Cluster Seizures

Cluster seizures refer to multiple seizures within a short period of time with only brief periods of consciousness.

Status epilepticus

Status epilepticus can occur as one continuous seizure lasting 30 minutes or more, or a series of multiple seizures in a short time with no periods of normal consciousness. It can be difficult to tell - emergencies. Most status patients usually suffer from generalized tonic-colonic seizures. Though status epilepticus can occur with either primary or secondary epilepsy, it may also suddenly arise in dogs with no previous history of seizures (traumatic brain injury, toxins, or disease).

Seizure threshold

It has been reported that each animal inherits a "genetically determined predisposition to seizures", and seizures occur when this threshold is exceeded. In other words, a physical condition, which may cause seizures in a low-threshold animal, may not cause seizures in a "normal" animal. The seizure threshold is apparently exceptionally low in animals that suffer from idiopathic (primary) epilepsy. An animal's threshold can also be altered by other means. Certain types of tranquilizers (e.g. acepromazine) may induce seizures in animal with a low threshold. The medical condition of alkalosis is reported to decrease the threshold.

Management of seizures

There is no cure or standard protocol for treating idiopathic epilepsy. However, seizures can be controlled with anticonvulsant drugs. The purpose of the treatment is to decrease the frequency, duration and severity of the seizure. Treatment is individualized for each animal based on its history and physical examination. No single drug is always effective; several drugs or a combination of drugs may have to be tried before a successful treatment is found. Also, it could take several weeks to establish a therapeutic dosage that works for an individual dog. This may indicate a need to change medication or alter the dose. It might be necessary to medicate the dog several times daily for the rest of its life and the medication schedule must be closely followed. Variance from the schedule may potentiate a seizure or series of seizures.

The etiological factors in cases of secondary epilepsy should be given due to attention and therapy should be instituted to minimize or eliminate the problem that gives rise to seizure. However, in such cases anticonvulsants are to be given to suppress the motor activities. Blood tests have to be performed to rule out/ identify metabolic disorders, toxic chemicals in the dog's system, infection

or inflammation. A fasting blood glucose test can reveal certain metabolic causes for seizures. A neurological examination can point towards an underlying disease of the central nervous system, which may be confirmed by tests done on the dog's cerebrospinal fluid. This fluid is collected from dogs under general anesthesia. More advanced tests, such as electroencephalograms (EEG) and brain scans are rarely carried out due to lack of facility and well trained personnel with sound knowledge on interpreting the findings. They are performed only when a dog's epilepsy proves to be difficult to treat, and they are generally available only at well-equipped animal hospitals.

The two most popular medications for long term seizure control are phenobarbital and potassium bromide. Each is an effective anticonvulsant, which can be used alone or in combination. However, 10 to 50% of the dogs having seizures are found to refractory to Phenobarbital sodium (Podell, 1995). Bromide is a halide anticonvulsant that offers an effective alternative to Phenobarbital and other barbiturates for treatment of epilepsy in dogs (Schwartz et al, 1991). The use of potassium bromide (KBr) as an anticonvulsant in dogs with severe signs (seizures) has been very helpful, particularly in cluster seizures. The drug is not metabolized by the liver and has fewer drug interactions than Phenobarbital. Primidone, once more frequently prescribed, is no longer recommended, as it seems to cause greater liver toxicity than Phenobarbital. Its administration needs to be carefully monitored by veterinarians to insure that a therapeutic dose has been achieved. Trepanier et al (1998) recorded 50% reduction in seizure frequency in 72% of epileptic dogs following initiation of treatment with potassium bromide. Long term control of psychomotor seizures can be achieved with carbamazepine despite its serum low to un-measurable concentration. This has been attributed to the effects of its metabolite on seizure control; thus serum concentration of the drug is not the useful guide to clinical efficacy in epileptic dog (Holland, 1988).

Failure of treatment

There are many reasons why medical treatments can fail. The biggest reason is the owner's lack of proper administration of the prescribed drug. The progression of an underlying disease (such as brain tumor) may resist treatment. Also, gastrointestinal disorders can affect drug absorption, and tranquilizers may stimulate seizures. Drug interactions can occur and adversely affect the level of anticonvulsant drug in the dog's system. And it just might be that a particular drug may not work for that animal.

Alternative therapies

These range from acupuncture to vitamin therapy. Traditional acupuncture therapy for epileptic dogs involves the placement of needles in up to 10 areas of the body. Needles can be left in place from 20 minutes to over a month. Acupuncture is not usually considered a substitute for drug therapy, but is used in conjunction with them. Of 5 dogs with intractable epilepsy, followed after gold bead implants in acupuncture points, 2 dogs relapsed after five months. Two reports of epileptic dogs given acupuncture in the ear (Shen-men point) are more positive. One dog enjoyed a six-fold increase in time between seizures; the other was seizure-free for 200 days after a previous history of monthly seizures.

Some forms of epilepsy respond to supplementation of vitamin B6, magnesium, and manganese. It has long been known that a deficiency of vitamin B6 or any interference with its function can cause seizures in any mammalian species including man and dog.

Control and Awareness of pet owners

While epilepsy can't be cured, it can be controlled. It is important to brief the dog owner that it is a chronic condition and needs lifelong management and the dog can have a good life even if it has epilepsy. Most seizures are not life threatening and will end in a matter of seconds, although it may seem like hours. The foremost important tips the dog owner should be advised is to remain calm and to remove objects that the dog might knock into and to place a towel or mat under the dog's head if it is banging against a hard floor.

There is little danger of the dog swallowing his tongue. Of greater concern is a dog aspirating vomit if it throws up during an attack, so owners should quickly move a dog away from anything it could inhale. The owner should be advised to sit with dog and soothe it during the period of recovery. It may sometimes take a dog several minutes or longer to recover from a seizure, and comforting presence of the dog owner really matters.

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DIAGNOSTIC RADIOGRAPHY IN SMALL ANIMAL PRACTICE

Indramani Nath
Professor, Department of Veterinary Surgery and Radiology,
College of Veterinary Science and Animal Husbandry,
O.U.A.T., Bhubaneswar - 751003.

Radiology is used in small animal practice

1. As a diagnostic aid
2. To select methods or techniques of treatment
3. To detect previously unrecognized lesions.
4. To monitor efficacy of a treatment schedule.
5. To screen normal animals for morphological evaluation in an attempt to eradicate inherited diseases by selective breeding.
6. To determine age of the animals.

Types of X-ray machines

1. Portable (KV: 70 – 110, mA: 15 – 35)
2. Mobile (KV: 90 – 125, mA: 40 – 300), suitable for radiography in small animals
3. Fixed (KV: 120 – 200, mA: 300 – 1000)

N.B.: Most human X-ray machines are suitable for radiography of pet animals.

Parts of X-ray machine

1. Control panel
    - On-off switch
    - Voltage and voltage compensator control
    - Kilovoltage selector
    - Milliammeter and milliamperage control
    - Timer and exposure botton
    - Fluoroscopy control
2. Transformer assembly

Types of cassettes (may be hinge type or clip type or tension type)

1. 6' X 8'
2. 10' X 12'
3. 12' X 16'

Types of X-ray films

1. Screened film: Need less exposure time, most suitable for pet animals.
2. Non-screened film: Need long exposure time. Suitable to detect hair line fracture, slight bony changes and in dental radiograph.

Storage of X-ray films

1. The films should be stored in an air tight film box inside a dark room, packets kept in a vertical position (not one over the other), away from moisture at a temperature of 10^0C to 20^0C.

Loading of X-ray films

1. Film loading should be done on a dry, clean surface inside a dark room under a safe light.
2. Film should be handled delicately and any accidental splashing of processing solution should be avoided.
3. Care should be taken not to keep the covering of the X-ray film inside the cassette.
4. The clips of the cassette should be tightly closed before opening the room.

Adjustment of factors on the control panel for obtaining a diagnostic radiograph

1. KV:
    - Higher is the KVP, better is the radiograph as when higher KVP is used then more mistake can be accommodated and we will still produce a diagnostic radiograph.
    - Centimeter thickness – KVP relationship: For each centimeter change in thickness, you must add or subtract 2 KVP up to 80 KVP; from 80KVP to 100KVP you must add or subtract 3KVP; and above 100KVP you must add or subtract 4 KVP for each centimeter of thickness. e.g. If the thickness of the part is 4cm, then KVP = 40 + 2 X 4cm = 48
2. mA:
    - Generally mA of 100 or 160 is sufficient for small animal radiograph. There exists a relationship between KVP and mA. When a change is required Ma is either halved or doubled with corresponding change in kv
3. Exposure time:
    - It should be as minimum as possible.

Radiographic positioning of the animal

1. Skull: ventrodorsal or dorsoventral, lateral and frontal
2. Cervical region: ventrodorsal or dorsoventral, lateral
3. Thorax: ventrodorsal or dorsoventral, lateral and standing lateral
4. Thoracic spine: ventrodorsal and lateral
5. Abdomen: ventrodorsal or dorsoventral, lateral and standing lateral
6. Lumbar spine: ventrodorsal and lateral
7. Pelvis: ventrodorsal and lateral
8. Femur: anteroposterior and lateral
9. Stifle joint: posteroanterior and lateral
10. Tibia and fibula: posteroanterior and lateral
11. Tarsal joint and foot: anteroposterior and lateral
12. Scapula and shoulder joint: posteroanterior and lateral
13. Humerus: anteroposterior or posteroanterior and lateral
14. Elbow joint: anteroposterior and lateral
15. Radius and ulna: anteroposterior and lateral
16. Carpal joint and foot: anteroposterior and lateral

Types of processing solutions

1. Powder form (most suitable and most commonly used)
2. Liquid form

Processing of X-ray film

The five steps of processing are

1. Developing
    - The films are generally put inside the developing solution for a period of 4 – 5 mins at 68 ^{0}F (20 ^{0}C).
    - This converts the latent image to a visible one.
    - Once developing solution is prepared it should not be kept for more than 3 months.
2. Rinsing
    - After developing it is put inside the rinsing solution to stop over development.
    - It is generally done in the running tap water.
    - Rinsing time is 10 – 30 secs.

3. Fixing
    - After rinsing the films are transferred in to the fixing solution.
    - It retains the silver image permanently and harden the emulsion.
    - Fixing time ig generally twice that of the developing time.
4. Film washing
    - After fixing films are washed in a tank with provision of running tap water
    - Generally 20mins of washing time is required.
5. Film drying
    - After washing the films is drained and hang up to dry.
    - During drying the films should not be coming in contact with each other.

Interpretation of a radiograph

A number of important steps must be fully completed before interpretation of a radiograph can be attempted

1. The case history
2. The physical examination
    - Generally, the purpose of radiography is to confirm a clinical diagnosis or impression, not to make the diagnosis.
    - It becomes evident that a very detailed and complete physical examination is necessary, first to establish the reason radiographs should be taken, and second to determine what part of the animal is to be radiographically examined.
3. The correct radiographic procedures
    - A radiograph changes a three dimensional subject in to a two dimensional flat plane. For this reason it becomes necessary to make at least two views 90^0 to each other and other angular views as the condition demands.
    - It may be easy to find out an abnormal object on a radiograph, however without a second view it is impossible to determine if the abnormal object is located inside the animals body or outside the animals body on the skin.

A radiographic diagnosis consists of two important steps

A. Location of the lesion (determining whether or not an abnormal structure exists)
    - The normal anatomy must be known for each area.
    - In order to help establish a mental picture of normal anatomical parts, always place the radiographs on the viewer in a standard manner. It is interesting to note that there are eight possible ways to position a radiograph.

- The basic radiographic signs of pathology have been classified in terms of changes in the following: 1. Size, 2. Architecture, 3. Contour, 4. Density, 5. Position and 6. Function. By noting changes in any of these normal anatomical structures, the pathological lesions are located.
- With the radiographs placed on the illuminator, a quick general survey should be made to locate obvious pathological lesions. A normal note should be made of any obvious pathological lesions. Next, and most important, a more detailed examination of the radiograph should be made. Only a systematic, methodological examination of each radiograph will prevent overlooking lesions beyond those expected.
- In a complete examination of the radiograph, one could begin with the osseous system. Examine each bone following the contour of the cortex throughout. Compare one side with the other side, the left leg with the right leg and so forth. It often pays to radiograph the opposite leg; this is essential in young animals. Examine each and every bone and process such as the individual ribs. Wherever possible compare one side with the other. This is particularly necessary in the study of spine, pelvis, limb and skull. After examination of the osseous system is completed, the other systems should be carefully studied.
- In the abdomen study the gastrointestinal system (the liver, the stomach, the small and large bowel) examine the contour, size and position of each part.
- Compare the systematic examination by studying the urinary system (the kidney, the bladder and the external structures).
- The genital system comes next. This study usually entails observation of the areas of the ovaries and the uterus in the female, and the areas of the prostrate and external genitalia in the male. Most of the internal genitalia structures are not normally visible in the dog and cat, and, if seen are abnormal.
- Then the spleen should be studied to determine position, size and contour.
- Finally observe all other areas of the radiograph, which include every location and part not previously covered such as the skin, and the subcutaneous areas and the areas within the abdomen with no evident viscera (e.g. those that are ventral to the liver, dorsal to the kidneys and so on).
- Other areas in the body, such as the thorax, the skull and the extremities, must be covered with equal care.
- It is nearly impossible for a veterinarian to remember details of all anatomical parts of each animal. If in doubt, do not hesitate to compare the radiograph in question with that of a known normal animal. If radiographs of normal animals are not available, refer to the normal radiographic anatomy.
- For veterinarians, preconceived ideas can be misleading in the examination of the radiographs.

- Do not get too close to the radiograph, particularly in the examination of the abdomen and thorax. Often an evident mass is missed by examining the radiograph with the eyes too close to the viewer.
- Move back and often the lesion will then become quite evident. Glance back and forth across the radiograph; a normal structure or an abnormal lesion might then become evident.
- For fine bone structures examine the parts with the eyes close to the radiograph. A magnifying glass often helps in close examination of a part.
- A spot light allows observation of normally overexposed areas of a radiograph such as soft tissue structures around the bone. Minimal new bone growth normally missed under the usual illuminator light can be shown
- It is helpful to restrict the light showing around the edges of a radiograph that does not fill the entire surface of an illuminator. This can be done with card board strips, a radiographic envelope or other radiographs.
- Locating the lesion is only half of the problem. The abnormal anatomy must next be named or classified (radiographic diagnosis). If a definitive diagnosis can not be made, a differential diagnosis must be made in order of probability.

B. Classification of the lesion (making a definitive or differential diagnosis)

- Once a pathological lesion has been located, it becomes necessary to attempt to determine what is wrong and what pathological syndrome is present.
- A scheme that might help in categorizing pathological lesions would be either to try: 1. To classify the observed condition under one of the categories (1. Developmental, 2. Metabolic, 3. Traumatic, 4. Infectious, 5. Neoplastic, 6. Degenerative) or 2. To attempt to rule out some of the categories. This will, in the end, provide a more logical conclusion about each lesion.

Radiographic science of pathology

1. Uterus (pregnancy and pyometra)
   - Normal non-gravid uterus cannot be visualized on a radiograph. If pathological lesions are of sufficient size they can be seen.
   - Examination of gravid uterus is necessary when dystocia or a retained foetus is there.
   - The examination should also include the pelvis also to determine any deformity either traumatic or developmental.
   - The time of ossification of foetal skeleton occurs between 40 – 50 days and sometimes it may be more.
   - A gravid uterus prior to foetal skeletal ossification and pyometra appear similar i.e. posterior ground glass appearance in posterior ventral abdomen.
   - Any ascites can be diagnosed from free fluidity of the part.

2. Fracture
    - It is the break in continuity of hard tissue.
    - In some fracture, fracture line may not be visible immediately.
    - Take radiograph after 24 – 48 hrs of stability.
    - While describing a fracture, the type of fracture, location, degree of angulation should be considered.
3. Hip dysplasia
    - It is the most commonly observed developmental deformity in dogs especially in large breed of dogs like German Shepherd.
    - For radiograph the animal should be positioned in ventro-dorsal positioning with hind limbs extended.
    - The radiograph should include both of the hip joints to detect any abnormality easily.
4. Osteosarcoma (Bone cancer)
    - It occurs at extremities of long bones.
    - There is distinct mottled or mosaic appearance of cortex and medulla.
    - They very rarely cross a joint.
    - Periosteum is raised, due to inflammatory condition giving a triangular appearance called as Codman's triangle.
    - There is also sunburst appearance.
5. Osteomyelitis
    - There is infection. Septic inflammation of the bone. A clear involuntrum will be observed on radiograph, which is the junction between live bone and dead bone. The dead bone i.e. sequestrum has to be removed.

Different contrast radiographic techniques practicable in the field condition are

1. Barium swallow technique (Oesophagography)
    - This technique is used to evaluate structural and functional status of esophagus.
    - This is indicated to diagnose obstruction, stenosis, diverticulum, perforation of esophagus.
    - Barium sulphate is prepared with light cream consistency.
    - First of all a plain radiograph should be taken.
    - Then barium swallow can be administered @ 1 – 2ml/ kg b wt.
    - As the last swallow is given, radiograph is taken.

- The mucosal folds of esophagus appear as linear streaks and barium is quickly cleared in to stomatch.
- If it is accumulated then there is obstruction.
- If there is outpouching, there may be a diverticulum.

2. Barium series
   - This technique is used to examine gastrointestinal tract.
   - Keeping the animal fasting for 24 hrs barium sulphate is given @ 6 – 12ml/kg b wt.
   - Take radiograph at different intervals till barium sulphate is completely excreted out in the feces.
3. Barium enema
   - It is indicated to outline colon and rectum.
   - Keeping the animal fasting for 24 hrs barium sulphate should be introduced through rectum with hindquarters raised after giving a soapy water enema.
4. Pneumocystography
   - It is the contrast study of the urinary bladder.
   - It can be done by catheterization and evacuating urine.
   - Then the contrast agent i.e. air can be administered by a disposable 50ml syringe.

N.B.: Barium Sulpahte is available in the form of powder, suspension and paste. But the powder form is most commonly used.

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CLINICAL INTESTINAL DISORDERS IN PET ANIMALS

Indramani Nath
Professor, Department of Veterinary Surgery and Radiology,
College of Veterinary Science and Animal Husbandry,
O.U.A.T., Bhubaneswar - 751 003.

The rule of thumb that carnivorous intestine is about 5 times the length of the trunk applies well to dog and cat. The small intestine (duodenum, jejunum and ileum) is about 4 times the length of the large intestine (cecum, colon, and rectum). The duodenum is best recognized by its relation to stomach. The jejunum is empty and ileum has additional ileocecal fold attached to antimesenteric border. The cecum is most distinctive of all being blind and short. The colon is identified by its relation to cecum and rectum. It is wider in diameter and contains fecal matter.

The common clinical disorders of intestines encountered in pet animals are as follows

Intestinal obstruction:

Types:

The obstruction may be mechanical or paralytic.

Causes:

Mechanical block to the passage of intestinal contents may be in the lumen (foreign bodies, intussusceptions, neoplasia, inspissated feces), in the wall (inflammatory) or outside the wall (hernia, adhesion). A wide variety of foreign objects may be ingested particularly by young animals. Once the object has passed through the pylorus, the next smallest lumen is the distal duodenum and proximal jejunum, the most common site of obstruction.

Signs:

The signs of intestinal foreign body obstruction are variable, depending on the location of the foreign body and propensity of the foreign body to cause vascular disruption and necrosis of intestinal wall. Anorexia, depression, abdominal tenderness and vomiting are commonly seen. An object completely lodged in the proximal small intestine stimulates profuse vomiting with loss of acid secretions from the stomach and alkaline secretions from the gall bladder, pancreas and duodenum. Animals with vomiting may have either normal pH or primary metabolic acidosis. Obstruction of the distal jejunum may not stimulate vomiting, but results in distension of the intestinal lumen by fluid and gas. Vomiting may cause dehydration and weakness. Defecation may decrease in frequency and the stool may be blood tinged.

Diagnosis

- Based on history: Intestinal obstruction should be considered in any animal that vomits. If symptomatic medicinal treatment does not improve the condition of the animal within 24 hrs further diagnostic evaluation is necessary.
- Abdominal palpation
- Abdominal radiography: Obstruction of distal small intestine produces greater dilation. A standing lateral view reveals foreign bodies. Contrast examination permits confirmation of diagnosis. Intraluminal obstruction appears as radiolucent area surrounded by contrast material outlining the foreign body. A radiographic examination six hours after administration of barium permits diagnosis of most proximal small intestinal obstruction. 24 hr study is best in diagnosing most distal small intestine obstruction.

Treatment

- The treatment for foreign body obstruction is exploratory celiotomy. Abdomen is exposed through ventral midline incision to permit adequate inspection of the entire gastrointestinal tract.
- If foreign body has not caused any vascular obstruction it is removed through enterotomy made distal to and slightly over the foreign body. Enterotomy incision is closed with 3/0 synthetic absorbable suture in Cushing and Connell pattern.
- If the foreign body has caused necrosis of the intestinal wall, then rejection and end to end anastomosis is indicated.
- Sharp foreign bodies like sewing needle are best left alone. The transit of the foreign body is monitored by periodic abdominal radiograph and animal is monitored for sign of peritonitis. In most cases the needle passes without complications.
- String foreign bodies (thread, nylon) can produce a unique form of intestinal obstruction. If the bowel is perforated, signs of peritonitis will be observed, treatmemt is exploratory celiotomy. The string is found along the mesenteric site of the intestine and pulled out.

Intestinal Intussusceptions:

It is produced by vigorous contraction that forces one segment of intestine into the adjacent segment (diarrhea, parasitism, canine distemper). Ileum is very much prone to getting telescoped in to the caecum. Most common in pups.

Types

Ileo-ileal/ Ileo-caecal/ Caeco-caecal/ Caeco-colic

Symptoms

Vomition/ colic/ straining/ passing small quantity of faeces/ semi solid/ blood stained / rectum empty.

Diagnosis

- Contrast radiography in puppies
- Exploratory laparotomy

Treatment

- Following laparotomy, invagination is corrected by gentle traction of the bowel that has become telescoped.
- If adhesion enterectomy and anastomosis is performed.

Principle of intestinal surgery

- Operative fluid therapy: A dog presented with intestinal disorder need to be operated within hours unless the fluid deficit is partially corrected before surgery the animal may not survive after operation. Ringer's lactate is the best suited to body pH. Ringer's lactate @ 10 ml per kg body weight per hour should be given. 3 ml of fluid should be administered for each ml of blood loss. If PCV fall below 20, then whole blood should be administered @ 20ml/ kg body wt. Pale mucous membrane, weak pulse, prolonged capillary refill time (more than 2 sec), requires accelerated rate of administration of ringer's lactate. The maximum volume given should not exceed 80ml/kg body weight.
- Antibiotic prophylaxis: Since the patient for operation carry a risk of infection, the administration of antibiotic should be started before contamination occurs.
- Timing of surgery: Surgery for mechanical obstruction of intestine should be performed with in 12hrs after diagnosis has been made.

Colonic impaction

Obstipation or intractable constipation is an acquired form of megacolon in both dogs and cats.

Causes:

It is due to impaction with foreign material or feces mixed with hair. This is more common in older animals.

Signs:

- History of repeated straining to defecate while passing little or no feces, anorexia, vomiting, straining to defecate with passage of small amounts of liquid feces containing blood or mucous and weight loss.
- There is abdominal pain, crying, stiff gait, arched back and reluctance to move.
- Plain abdominal radiograph reveal the colonic impaction. Contrast study with barium enema can confirm the diagnosis.

Treatment:

Fluid therapy, relief of obstruction, enema and manual decompression are often necessary to relieve a chronic colonic impaction. Decompression can generally be accomplished with

large volume soapy luke warm water administered through a douch cane with raised hind quarters. Colostomy may be indicated in extreme cases.

Rectal prolapse:

Reddish, hemispherical protrusion in the anal region with transverse fold.

Types:

- Protrusion of the anal mucous membrane – Prolapsus ani
- Prolapse of the posterior portion of the rectum – Prolapsus ani et recti
- Prolapse with invagination of colon/ rectum

Causes:

- Loss of tone of sphinter ani
- Loosening of rectal mucous membrane
- Loosening of attachment of rectum to peri-rectal tissue
- Very young and very old animals are susceptible
- Straining due to constipation/ diarrhea, parturition are the exciting causes

Treatment:

- Reduction and retention of prolapsed mass: Washed with mild antiseptic solution. Cleaning with normal saline/ sugar solution/ salt solution (hypertonic) with cold application helps in withdrawal of fluid. The mass should be lubricated with bland oil. Epidural anesthesia is helpful for easy reduction. Putting a purse string suture around anus leaving space for defecation (introduction of a cylindrical object/ glass bottle/ needle cover/ syringe barrel) helps. Snipping of the necrotic mucosa.
- Following laparotomy; colopexy and ventopexy may be tried.
- Amputation: May be advised as last resort. Passing two silk threads one horizontally, one vertically through center of the prolapse close to anus, cut away the portion of bowel behind the suture, suture is drawn from lumen of anus after cutting at centre and knot is tied (Moller's method). Application of series of mattress suture anterior to the prolapsed level before amputation
- Post-operatively laxative diet, keeping hind quarter at higher level than the fore quarter may be advised.

ANAL ADENITIS

Anal sacs are modified sebacious & apocrine glands located on either side of the anal opening. Secretion has lubricating effect, assist the passage of feces. At times get inflammed, swollen, bluish, fluctuating, and very painful. Contain pus, burst & may produce anal fistula.

Signs

- Licking, biting anal area, tail base or skin on either side of perineum.
- Rubbing anus on ground and tail chasing
- Anal sacculitis with impaction.

Treatment

- Contents discharged by pressure between a finger in rectum and an overlying thumb.
- Sac irrigated with saline, povidone-iodine.

ANAL TUMOURS, WART, CYSTS, LIPOMATA

Causes

Androgen dependent.

Treatment

- Surgery preferred, some reoccur.
- Bleeding controlled by proper ligation by holding stumps, prevention of sepsis is important due to proximity of anus.
- Castration reduce recurrence.
- Adenomas are ovoid, cryosurgical removal is often successful.

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DENTAL DISEASES AND DISORDERS OF DOGS AND CATS

S. K. Ray
Formerly Professor and Head & Emeritus Scientist (I.C.A.R.)
Department of Veterinary Medicine
Orissa University of Agriculture and Technology, Bhubaneswar - 751003

Dental disease is the main oral disorders of dogs and cats and is of major importance in pet practices.

Pet's dental health is just as important to his or her overall health. Dental disease does not affect just the mouth. It can lead to more serious health problems affecting intestinal tract, heart, lung, kidney and joints. Dental diseases of dogs and cats is one of the most commonly overlooked areas of pet care practice, with many of them suffering from undiagnosed, painful dental disorders.

Dog is born edentulous (without teeth). By 6-8 weeks of age the deciduous or primary teeth erupt.

Puppies and kitten with deciduous teeth

In this age group there are two types of dental problems occurring - traumatic damage to the baby teeth and oral cavity and improper eruption of the adult teeth. The baby or deciduous teeth are very thin and fragile. They are not firmly anchored in place by strong mineralized bone and can be easily broken or pulled out of position. Therefore, the most common problems encountered in this young age are traumatic injuries, sometimes self inflicted, sometimes inflicted by well meaning owners.

Puppies are very oral and enjoy having things in their mouths. Puppies are given hard objects to chew and playing "tug of war" with them. By pulling we can either fracture or luxate (pull out of the position) the primary `canine' or 'fang' teeth. Since the adult fang teeth are developing under the gums close by to where the baby fangs are, these developing adult teeth can also be damaged. This can cause them to either never erupt or to come up in an improper position. This improper tooth location can cause injury to the mouth's soft tissue.

Hard objects like "Indestructible Bones'", hooves, stick and rocks can break teeth. Catching a flying saucer-like play toy in mid-air can also lead to teeth breaking. These types of injuries are very painful and usually result in the tooth dying and many cause an infection or abscess of the bone. Signs of a possible oral problem include difficulty in eating or holding objects, bleeding or drooling. The bone and overlying gums will be sensitive to the touch, swollen and the infection can start a draining abscess - a condition called a "gum boil". Any broken baby teeth detected should be extracted.

Kittens, unlike puppies, usually break their baby fangs by running into doors, steps and walls. In addition to broken teeth, kittens and cats are notorious for chewing on electric cords and ornamental plants - which cause serious injury to the oral cavity.

Malocclusion

Malocclusion (abnormalities in the position of the teeth) is common in dogs, but also occurs in cats. It is the second most common dental problem seen in young dogs and cats where the permanent teeth erupt improperly. This condition is due to either trauma or the presence of persistent baby teeth. Normally as the permanent tooth erupts, it does so directly under the roof of the deciduous tooth causing it to break down, which then allows the adult tooth to push it out. Sometimes the bud of the permanent tooth is not directly positioned under the deciduous counterpart. This improper positioning causes the permanent tooth, during its formation, to glide off the baby tooth root and erupt abnormally. This malpositioned adult traumatizes the soft tissue in the mouth causing the pet pain and possible subsequent infection. In addition, food often becomes trapped between the baby tooth and adult tooth causing the development of gum infection. As a rule there should never be two of the same tooth type occupying the mouth at the same time. By frequently checking the pet's teeth between the ages of 14 to 24 weeks of age, any double presence of teeth will be detected and can be immediately corrected. Never wait for the baby tooth to fall out by itself if you see even the slightest protrusion of the adult crown next to it. The teeth that are most often affected by the presence of retained baby teeth are the small front incisors and the canine or fang teeth. The lower fang teeth usually come in towards the inside of their deciduous (baby) counterparts. That means they will erupt into the hard palate if the baby fangs do not fall out promptly and are not extracted in time. This condition, if uncorrected, will cause a permanent hole in pet's hard palate creating a direct connection between the mouth and nasal cavity. To correct this, the specialist will often construct an acrylic incline plane or "sliding ramp" to allow the inward directed lower fang to be forced out into a normal position. This is a very common occurrence in toy breeds but can occur in all animals.

Adult Dogs and Cats : Dental Fractures

In this mature age group, a variety of oral problems that can occur. Fractured teeth, as in young animals, if left untreated will cause abscesses and facial swellings. The fang teeth or canines, and the most important chewing teeth, the "carnassial" teeth, are often affected. The carnassial or shearing teeth are the upper 4^{th} premolars and the lower 1^{st} molars. They do 90 percent of the animal's chewing. Because of the tremendous chewing forces that an animal can exert, any indestructible chew toys can cause these teeth to fracture and expose their nerve centers. These important chewing teeth if injured should be saved by a Dental specialist rather than extracted. A veterinary dentist will perform a root canal treatment that prevents infection from going up the tooth and into the bone, and also allows the tooth to remain functional. If the tooth's crown is substantially damaged, the dentist will take impressions and have a dental laboratory cast a metal "Jacket Crown". The metal crowns are indestructible and will prevent further injury to the tooth. The metal crown's Dobermans, and Rottweilers, and to the sporting breeds like the Retrievers and Setters. Quite often dogs that have been kenneled or have exhibited separation anxiety damage their teeth by chewing on their cages. The damage that is done occurs on the distal or back surfaces of the teeth. The enamel and dentin are worn down and the tooth appears hook like. The normally white enamel at these worn areas becomes discolored to yellow or brown. These weakened teeth are more

prone to further wear, fracture and exposure of the root canal. A veterinary dentist can strengthen the tooth with a 3'4 crown, which covers the sides and back area of the tooth with metal and thereby prevent further damage to the tooth.

Periodontal Disease

Periodontal disease is an infection of the tissue surrounding the teeth that takes hold in progressive stages. It is the number one disease that affects the dog's mouth after the age of two. This is a very slow, insidious disease that affects all the supporting structures of the teeth.

It starts out as a bacterial film called **plaque**. The bacteria attach to the teeth. when the bacteria die they can be calcified by calcium in saliva. This forms a hard, rough substance called **tartar or calculus** which, allow more plaque to accumulate. Initially plaque is soft and brushing or chewing hard food and toys can dislodge it. If left to spread, plaque can lead to **gingivitis** an inflammation of the gum, causing them to become red and swollen. Bad breath and bleeding red gums are the most consistent signs noticed at the early stages of the disease. The animal might drop food and rub its mouth as well.

As plaque and tartar develop below the gum line, when not cleaned, the plaque and tartar build up continues unchecked; infection can form around the root of the tooth.

In the final stages of periodontal disease, the gums start to recede and supporting ligaments that hold the tooth to the jawbone and the bone itself becomes damaged. Deep pockets of infection cause pus, bleeding and pain. The affected animals are more reluctant to chew on hard food and quite often an abscess develops in the gum and jawbone. The animal starts to loose weight, avoids having its face or head touched, and seems to become all of a sudden "much older" overnight.

This stage of the disease requires the intervention of a dental specialist who is skilled in periodontal therapy and surgery. Dental radiographs to be taken to determine the degree of bone destruction and diseases; radiograph will be beneficial in establishing an appropriate treatment plan to save the animals teeth. Often by deep root therapy and splinting teeth, the dentist can encourage new supportive bone to form. If nothing is done, the pet will succumb to the final stages of periodontal disease.

Due to advanced destruction of the jawbone, the teeth are lost. In the case of toy breeds, often the lower jaw will fracture because the bone around the teeth is severely damaged. Advanced periodontal disease that affects the upper fang teeth can lead to permanent oro-nasal fistula where the nasal and oral cavities are actually connected. Often the dog has sneezing episodes that lead nose bleeds. When the tooth finally does fall out, there is a permanent non-healing hole between the mouth and nasal cavity that needs to be surgically repaired.

It should be obvious that bad breath, secondary to gum disease is very serious and should be acted upon immediately.

FORLS (Feline Odontoclastic Resorptive Lesions)

FORLS are very common oral disease and occurs in 60 per cent of cats. It is otherwise called as cat cavities. Normally the lesion starts after the age of two. The most common signs

of FORL are that the cats eagerly approach their food bowls but then walk away without eating. These FORLS are extremely painful what is happening, usually at the gum line or neck of the tooth, in the activated tooth eating cells, called odontoclasts, start attacking the teeth. This leads to the tooth developing a cavity or hole into the root canal that is extremely painful. If left untreated. the crown of the tooth snaps off leaving the roots of the tooth to cause irritation and drainage.

The affected cats show lack of appetite and weight loss. The gums in the affected area seem to be growing up and into the teeth. Bleeding may occur from the gums. In the end stage of the disease, a swollen bulging gum where the tooth used to be replaces the missing crown. At the present time, we do not know what causes the tooth-eating odontoclasts to be activated to start destroying the teeth. We do see a higher incidence of the disease in cats with moderate periodontal disease. Siamese and oriental cats have a higher incidence of the disease. Cats that have immune suppressive viruses such as Feline Leukemia "Felv" or Feline Immunesupressive Virus "FIV" have a greater tendency to develop the FORLS. At the present time a Veterinary dentist can slow the progression of the tooth destruction by using special fluoride leaching fillinas. If the teeth are severely damaged they need to be surgically removed in order to allow for proper healing.

Cancer

Animals 8 years and older (less frequently young animals) can develop oral cancer. The third most common site for caner is the oral cavity. In young animals, problem can involve tumors that affect the teeth directly. Odontomas are tumors that evolved from tooth bud and, fortunately, benign and properly excise will not return. Unfortunately, most oral cancers are malignant which needs they will not only grow locally but also can often spread or metastasize to other locations.

Conclusion

Out pet's teeth, like our own, can be damaged and thus require regular appropriate care. Depending on the breed, age, and upbringing, we can have different oral problems to deal with. Very important is the daily care and inspection of our special friend's mouth. The oral cavity is the gateway to either health or disease. A long, healthy, comfortable life for our friend can be assured by constant oral attention. It is up to us to guarantee this quality of life for our pets. Being proactive and taking care of problems immediately will help prevent further spreading of problems that can affect other organ systems in the body.

I like to quote "Hale - 2003" which reads as "In the domestic environment, dogs have no real need to defend territory. They have no need to prehend and kill live prey animals. In short, the domesticated pet dog does not need teeth at all. This may seem like an odd statement for a veterinary dentist to make, but I feel quite strongly that a dog is far better off having no teeth than having bad teeth. My preference is that a dog should have a full set of healthy, functional teeth, but preserving bad teeth in the face of a poor or questionable prognosis serves no positive purpose."

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ENDOPARASITIC DISEASES IN DOG AND THEIR CONTROL MEASURES

Mitra Ranjan Panda
Deptt. of Veterinary Parasitology
College of Veterinary Science & Animal Husbandry., O.U.A.T., Bhubaneswar

INTRODUCTION

The pets, particularly dog is a loyal companion of man since time immemorial. A dog is reliable, affectionate and also having a sense of consistent routine life. It is a great stimulus for laughter, play, exercise and keep their masters active. Presence of this animal or other pets offer opportunities for developing a new life style. A pet helps alleviate the suffering of people in distress, depression, loneliness, anxiety. Humans are able to form a strong emotional bond with the dog and vice-versa. A guide dog is the eyes of its blind owner and act as a trusted companion to bridge the gap with outside world. Animal assisted therapy or pet therapy can work wonderfully on the minds and act as occupational therapy. However, a dog, in order to render these useful service, must be in a good state of health. Like other animals dogs also suffer from many diseases including parasitic infections.

Endoparasites or internal parasites are those which occur in different internal organs, body cavities and blood vascular systems include helminthes and protozoa. Most of the endoparasites, though are not fatal, have long standing debilitating effects on pet dogs which affect their normal activities and performance. More important is, a good number of endoparasites those infect the dogs are zoonotic in nature and potentially harmful to the dog owners, particularly children. Therefore, it is important to know the endoparasites which infect the dogs, their harmful effects on dogs as well as on human beings, mode of infection, prompt diagnosis and effective treatment for successful control.

HELMINTHES INFECTING DOGS :

TREMATODES

*Hetero*phyes heterophyes
Metagonimus yokogawai
Apophallus donicum
Cryptocotyle lingua
Cryptocotyle concava
Cryptocotyle jejuna
Opisthorchis tenuicolli
Nanophyetus salminicola
Opisthorchis viverreni
Clonorchis sinensis
Echinochasmus perfoliatus
Artyfechinostomum sufrartyfex
Paragonimus westermani
Paragonimus kellicoti
Schistosoma spindale
Schistosoma incognitum

GENERAL MORPHOLOGY:

- Body is flat/leaf like (*Opisthorchis*),may be globular with thick fleshy or slender/cylindrical (Schistosome).
- These are usually hermaphrodites ,but in schistosome sexes are separate.

GENERAL LIFE CYCLE

These are heteroxenous and have indirect life cycle. Adults are endoparasites of dog.

Eggs are passed out with the faeces. If they land in water they develop into a swimming larva (the miracidium) that emerges to penetrate a snail.

A number of asexual reproductions occur in the snail producing a large number of cercariae.

The snail is eaten by 2nd IH (a crustacean : crayfish usually) in which develops the metacercariae that infect the dog when eaten. These then penetrate the gut into the body cavity, reach site of predilection.

There may be involvement of more than one intermediate hosts (IH).

1st IH : may be snails of various species.

2nd IH: may be snail, tadpole of frog, fish, larva of nematoceran flies, crustacean etc.

Infective stages (metacercaria) ingested by dog through contaminated feed/drinking water or ingestion of 2nd IH.

On reaching digestive tract of dog, metacercaria excyst and release immature flukes which may migrate to site of predilection.

In family Schistosomatidae, furcocercous cercariae penetrate the skin of dog.

Digestive Tract

Alaria americana is a common trematode in the intestine of dogs. It is also called ***Alaria canis*** .

Nanophyetus salmincola The trematode itself is not a major problem as far as dog parasites go but it transmits a rickettsial organism (*Neorickettsia helminthoeca*) that causes **"salmon poisoning**" disease.

Enteritis, bloody stool and lymph node enlargement occur.

There are a number trematodes which rarely occur in the GIT of dogs such as *Mesostephanus, Echinochasmus, Apophallus, Cryptocotyle, Phagicola, Plagiorchis* and *Sellacotyle.*

Liver:

Opisthorchis and ***Metorchis* spp.** live in the bile ducts and gall bladder.

Opisthorchis tenuicollis : Dogs are infected by eating the fish.

In large numbers they can cause thickening and blockage of the bile ducts .

Respiratory System

Paragonimus kellicotti and ***Paragonimus westermani*** are the important lung flukes. These flukes are fairly large, fleshy trematodes with a spinose skin and are found in cysts in the lungs.

Leucocytic infiltration and fibrous tissue formation around **hazel nut** shaped cysts. Cirrhosis, cystic dilation of bronchi ,pseudopneumonia, abscess formation, peritonitis and bronchitis are the common pathological lesions. Rusty coloured sputum is the main clinical sign.

Blood and Lymph

- Schistosomes: ***Schistosoma incongnitum***, lives in the veins draining the intestines of dogs.
- Cercariae are mobile and when they break out of the snail they swim about until they find a good host and penetrate the skin.
- These larval trematodes get into the circulatory system and move to the veins in which the adults develop.
- These worms are either male or female and spend most of their adult lives in permanent copula. The female is held in a long groove called the gynecophoric canal along the length of the male's body.
- *Schistosoma spindale:* occurs in the veins draining the intestines of dogs in India and Indonesia. A similar parasite called *Heterobilharzia americana* is found in the veins of the gut in dogs and a variety of wild carnivores in the southeastern United States.
- These cause phlebitis in the mesenteric veins, haemorrhage in intestinal mucosa, profuse diarohhea/dysentery, dehydration, anaemia, hypoalbuminaemia, oedema.

CLINICAL SIGNS

Clinical signs differs as per the site of predilection and severity of infection.

DIAGNOSIS

By finding eggs in faeces, secretion, excretion, blood etc.

TREATMENT

1.symptomatic treatment.

2 Anthelmintic: Praziquantel,Niclofolan, Niclosamide, Albendazole, Triclabendazole etc can be prescribed in appropriate dose.

CONTROL MEASURES

- Dogs should not be fed with raw/salted/improperly cooked fishes. Salted fish should not be fed within 10 days.
- Freezing and cooking of all fish used for food is essential .

- Eradicate snail IH by spraying CuSo4 @1:150 million parts in water. Other molluscicides are sodium pentachlorophenate, di-nitro-o-cyclohexyl-phenol@3-5:1 million parts of water, pentachlorophenol,2-4,dinitro-6-phenyl phenol.
- Treatment of human night *soil with ammonium sulphate to kill fluke eggs.*

Paragonimus westermani and Paragonimus kellicoti :

- Raw fresh-water crustacean should not be fed to animal host.

Schistosoma spindale *and* ***Schistosoma incognitum***

- Immunization against *S. incognitum* using 60Co irradiated cercariae**.**
- Treatment of infected persons and animals.
- Biological control measures by predation by larval stages of Echinostomain snail IH,
- Microsporidial protozoa :Nosema eurytremae cause damage to intra-molluscan stages.
- Competetion between molluscan species such as Helisoma spp.to.eliminate Bulinus glabrata.
- Application of molluscide such as frescon, Bayluscide to water bodies.
- Increase the speed of water in irrigation channels as snail IH prefer slow moving or stationary water.
- Night soil treatmentby formentation for 25-45 days before allowed to water bodies**.**

Nanophyetus salminicola

- Prevention of consumption of raw /uncooked fish.
- In accidental consumption apomorphine can be given within 3 hours of consumption**.**

CESTODES

Includes following species

Dipylidium caninum

Taenia hydatigena

Taenia ovis

Taenia pisiformis

Taenia multiceps

Taenia serialis

Echinococcus granulosus

Echinococcus vogeli

Mesocestoides tenuis

Diphyllobothrium latum

GENERAL MORPHOLOGY

Body is tape/ ribbon like without alimentary tract.

Measures few mm to several cm.

Body consists of head(scolex),followed by unsegmented neck,and strobila having proglottids separated by transverse constrictions.

Scolex may have suckers (acetabula) with hooks.(class Eucestoda) or may have long,narrow,weakly muscular grooves (bothria: in class Cotyloda).

These are hermaphrodite, metameric repetitionof reproductive organs.

Posterior mature proglottids,packed with eggs are known as gravid proglottid.

Self/cross fertilization occurs between proglottids.

GENERAL LIFE CYCLE

Life cycle is indirect,larval stage is known as metacestode. Development of larval stage require one or more IH. Metacestode may be classified as procercoid/plerocercoid/tetrathyridium/ cysticercoid/cysticercus/strobilocercus/coenurus/ hydatid.

1st IH: rodents,canines,herbivores,oribatid mite (*Mesocestoides tenuis)* ,crustacia (*Diphyllobothrium latum),*dog flea/dog louse *(Dipylidium caninum).*

2nd IH: dog,cat,mice,reptiles,birds,amphibia.

PATHOGENESIS AND CLINICAL SIGNS

1. **Digestive Tract:** The dog-sheep tapeworm, ***Taenia hydatigena*** , a common dog parasites in sheep raising areas where dogs are fed sheep viscera or where they find sheep carcasses. The intermediate host is an herbivore like sheep in which the bladder like cysticercus larva is found in the muscles and viscera. Sheep are infected by eating eggs on grass contaminated by the faeces of the dog in which tapeworm proglottids are shed. In dog prepatent period is 51days.in liver migration of young cysticerci cause haemmorrhagic and fibrotic tracts. Mature ones are not so harmful.
2. Two species of ***Echinococcus*** infect dogs as a result of their having eaten raw meat from sheep, caribou, cattle, kangaroo or rodents. They are ***Echinococcus granulosus*** and ***E. multilocularis***.

   Humans are accidental hosts to the larval stage that is contained within large cysts spread throughout the internal organs including the liver, lungs, bones and brains. This is hydatid disease or hydatidosis. Rupture of cysts leads to anaphylactic reaction in man.
3. Another dog-sheep tapeworm is ***Multiceps multiceps***. The adult tape worm looks like *Taenia hydatigena* but the larval form in the intermediate host such as sheep, cattle and goats is a bladder-like thing called **a coenurus**. Inside the coenurus are many small larvae, each of which will mature to an adult when eaten by the dog or any wild canids. The coenurus is located **in the brain** of the intermediate host.

4. Another species called ***Multiceps serialis*** infects dogs. The intermediate host for this one is a rabbit, hare or squirrel where the coenurus is in the brain or muscles.
5. ***Dipylidium caninum*** is a small tapeworm of dogs.
6. The broad fish tapeworm is a is a parasite of fish eating mammals including dogs. It is called *Diphyllobothrium latum* .Infections in dogs occurs by eating poorly cooked or raw fish.

DIAGNOSIS

By finding eggs/gravid segments in faeces of animal.

By finding metacestode stage in various tissues /muscles/organs of the I.H.

TREATMENT

Praziquantel :5-7.5 mg/kg body wt.

Niclosamide :75-100mg/kg bwt.

Mebendazole :22mg/kg bwt. For 3 days.

Fenbendazole,albendazole and other combination

CONTROL

Species:

- **Prevention of dogs from taking raw offals /meat and infected small wild animals like rabbit, rat, mouse**
- **Do not allow dogs to defecate in parks, vegetable gardens, and grazing fields.**
- **Immunization of dogs with irradiated *Echinococcus granulosus* protoscolices and secretory antigens from adults of Echinococcus granulosus grown in vitro.**
- *For Mesocestoides tenuis* **control of oribatid mites is essential.**
- **Avoid feeding of raw offals/meat to dog.**
- *For Diphyllobothrium latum*
- **Dogs should not be fed raw fish .Proper cooking of fish before feeding to animal.**

C.NEMATODE

- *Toxascaris leonine*
- *Toxocara canis*
- *Ancylostoma caninum*
- *Ancylostoma braziliense*
- *Filaroides osleri*
- *Thelazia callipaeda*

- *Thelazia lacrymalis*
- *Spirocerca lupi*
- *Gnathostoma spinigerum*
- *Dirofilaria immitis*
- *Trichuris vulpis*
- *Capillaria plica*
- *Capillaria aerophila*
- *Dictophyma renale*
- Oncicola canis

Morphology

- Body shape is elongated,cylindrical and tapering at both ends and not metamerically segmented.
- Cuticle may show variable structures.
- Filariform / rhabdity form/ trichurid oesophagus.
- Sexes are separate. In most species males bear cuticular expansion at posterior extremity known as bursa.

General life cycle

- In the life cycle of nematode ,it has 4 moults,successive stages are

  1) 1st stage larva(L1) 2) 2nd stage larva (L2) 3) 3rd stage larva (L3) 4) 4th stage larva (L4) and 5th stage i.e adult.
- First 3 stages are pre parasitic ,may be free living/ in intermediate hosts.
- Pre parasitic L3 stage is sheathed in old cuticle and called infective stage.(exception: in ascarid worms L2 stage present in the eggs are infective).
- In heteroxenous lifecycle the IH are: beetle,earth worm, snail, slug, gastropod, mollusc,house fly, stable fly, cockroach, grass hopper, isopod, cyclopes, mosquito, tabanid fly, oligochite, annelid etc.

PATHOGENESIS AND CLINICAL SIGN

Digestive Tract:

The tissue nematode ***Spirocerca lupi*** may be in nodules in the esophagus, stomach or aorta of dogs where it may be rather benign. Worms are red in colour and coiled in a circle, hence the name. It may cause difficulty in swallowing if in the esophagus, or cause development of aneurysms if in the aorta. Both can cause the dog to be very sick. It may even cause development of malignant tumors. It is world wide but usually in warmer regions. The life cycle involves a dung eating beetle as intermediate host in which larvae develop and the dog is infected by ingesting the

beetle. If beetles are eaten by an unsuitable host, the larvae encyst in the tissues and infect the dog when the transport host is eaten. In the dog, larvae penetrate the stomach wall, enter the arterial system and form the nodules in the aorta and adjacent tissues. The adults develop there.

The large ascarid nematode, ***Toxocara canis***, is dog round worm. It causes one of the most important parasitic diseases of dogs**, Toxocarosis**. The life cycle is direct but transport hosts can be involved. Typically eggs are shed with the faeces and dogs or other mammalian hosts are infected by eating contaminated food. Eggs hatch in the intestine and larvae penetrate the gut wall, get into the venous system and thence to the liver where they develop. They then migrate to the heart and then the lungs. They develop further in the lungs, break out into the bronchioles and eventually get to the trachea and esophagus and are swallowed. In the gut they develop to the adult worm. This is the case in young pups. In older pups many larvae in the lungs get back into the circulatory system and migrate to all areas of the body where they encyst. They can stay here for a long time. Then, if they are in a female dog that has puppies, they can resume migration and infect the puppies through the placenta or the mammary glands.

Toxascaris leonina is another of the dog round worms that is very similar to ***Toxocara*** and occurs in both dogs and cats.

Human can serve as transport host for ***T. canis***. Larvae migrating through our tissues (**visceral larva migrans)** are responsible for the disease condition called **Toxocarosis** in humans.

Hookworm infection in dogs is another true parasitosis that can result from moderate to heavy loads of ***Ancylostoma caninum***, ***A. brasiliense or Uncinaria stenocephala.*** The more serious pathogen is ***A. caninum.*** Death can quickly result from blood loss, especially in pups. Pups can be infected through the mother's milk (**transmammary transmission)** because the life cycle is similar to that described for ***Toxocara*** as it involves a tissue and pulmonary migration. One major difference is that larvae actively penetrate the skin of the definitive host and get directly into the circulation from there. Hookworms are very common dog parasites and are also dog round worms, but not so large as the **Ascarids.**

Humans are susceptible to the larvae of hookworms that infect dogs and cats. **Cutaneous larva migrans** is caused by the larvae wandering around under our skin, usually cat hookworms.

Dogs also have Canine **strongyloidosis** due to ***Strongyloides stercoralis***, and are infected with ***Trichinella spiralis and Trichuris***.

Respiratory System:

The most common lung and bronchiole dog parasites are the migrating larvae of hookworms and ***Strongyloides*** which are dog round worms that mature in the small intestine but go through a lung migration before they get to their permanent home in the gut. They will cause respiratory problems.

Filaroides and ***Crenosoma*** are dog lungworms found in the trachea and lungs of dogs and other carnivores worldwide**.** ***Filaroides osleri*** forms nodules in the bronchi and trachea in which the adult worms are located. They shed eggs into the trachea which are coughed up and swallowed. Eggs pass out with the faeces and the next definitive host gets infected by accidentally eating food contaminated with the eggs containg infective larvae. the life cycle is direct. ***Crenosoma vulpis*** is another lungworm in dogs in Europe, China and eastern North America. Adults are in the

bronchi and instead of eggs, they release mobile larvae which get swallowed and pass out with the faeces. These infect an intermediate host snail which is then eaten by the definitive host (the dog). Larvae then penetrate the gut wall into the blood vessels and are carried to the lungs where they breakout and mature in the bronchi.

Bronchial capillariasis due to ***Capillaria aerophila*** .

These dog roundworms are found in the trachea, bronchi and small bronchioles in dogs and other carnivores in Europe, Russia, Eurasia and North and South America. The thin worms similar in shape to the whipworms but are the same slender diameter for the full length. The life cycle is direct. There are other capillarid nematodes in the lungs and urinary bladder .

Blood and Lymph:

There are several species of filarid dog round worms that occur in dogs throughout the world. These are the heartworms and lymphatic worms since the adults live in the blood vessels of the heart or the lymph nodes and vessels. They all produce mobile larvae called microfilariae that are released into the blood stream. The life cycle requires an intermediate host which is usually a mosquitoe or other biting arthropod that picks up the microfilariae in a blood meal and then injects them into the next host it bites. Important heartworms are ***Dirofilaria*** and a couple of species of the genus ***Brugia*** that are residents of lymph nodes and vessels.

Dirofilaria immitis is the causative agent of heartworm disease in dogs in North America. It is transmitted from one host to another by mosquitoes and is a constant threat every year when mosquitoes are active. Permanent damage can be done to the heart and vessels as well as the lungs and liver. Acute cases may result in death.

Muscles and Tissues

Dipetalonema reconditum is found throughout the world and is very similar to the heartworm *Dirofilaria*. However, dog and cat fleas are the intermediate hosts. Adults of this dog parasite are found in connective tissue under the skin but the microfilariae are circulating in the blood. Another very similar dog round worm also found in subcutaneous connective tissue in dogs is ***Dracunculus insignis.*** It is found in North America and is similar to ***Dracunculus medinensis*** of humans (which also infects dogs).

Trichinella spiralis is an intestinal parasite, the larval forms are common in the flesh of dogs or other carnivores.

Urogenital System

Only two roundworms are in the urogenital system of the dog. The giant kidney worm ***Dioctophyma renale*** is found in the kidney and sometimes other organs. It is also found in many other mammals, including man.

A **capillarid** dog round worm similar to those in the bronchi and trachea, ***Capillaria plica*** is found wound through the mucosa of the urinary bladder.

DIAGNOSIS

- By finding characteristic egg/ larvae in body secretion,excretion ,fuids.
- From typical clinical symptoms.
- Using trichinoscope in case of *Trichinella* infection.

TREATMENT

Ascariosis :

Pyrantel pamoate @orally 5-10 mg/kg bwt.:

Pyrantel ebonate: @.14.4 mg/kg bwt

Doramectin :@ 200µg/ kg bwt s/c or i/m.

Piperazine :@orally 200-300 mg/kg bwt.

Fendazole, benzimidazole compounds.

Diethyl carbamazine @50mg/kg bwt.

Hook worm diseases

Albendazole @5-7.5 mg/kg bwt.

Fenbendazole@ 50 mg/kg bwt.

Levamizole @7.5 mg/kg bwt.

Spirocercosis

Tetramizole @15 mg/kg bwt.

Oxfenbendazole orally @5 mg/kg bwt.

Albendazole orally @5-7.5 mg/kg bwt.

Dirofilariosis

Diethylecarbamazine @6.6mg/ kg bwt for 3-4 days by parenteral route.

Milbemycin oxime@ 0.5 mg/kg bwt.orally.

Arsenicalthiacetarsamide @0.1ml/0.45kg bwt i/v bid for 2-3 days in case of adult worms.

Levamisole @ 10mg /kg bwt orally for 15-20 days.

Dithiazanine iodide @ 2 mg/ 0.45kg bwt for 7 days.

CONTROL:

- Control of IH are recommended such as spraying of insecticide, Molluscide rodenticide, acaricide etc.
- General hygiene measures and public education.

- Segregation and treatment of affected animal and persons.periodic deworming should be done.
- Hervivores that act as IH should not be allowed to graze marshy land that contain snails and flies.

Regular cleaning of animal sheds and proper disposal of carcass, excreta etc.

D. PROTOZOAN PARASITE

Trypanosoma rangeli	***Isospora neorivolta***
Trypanosoma cruzi	***Isospora ohioensis***
Trypanosoma congolense	***Sarcocystis cruzi***
Trypanosoma suis	***Sarcocystis levinei***
Trypanosoma evansi	***Toxoplasma gondii***
Giardia canis	***Neospora caninum***
Entamoeba histolytica	***Hepatozoon canis***
Entamoeba coli	***Babesia canis***
Entamoeba hartmanni	***Babesia gibsoni***
Entamoeba gingivalis	***Babesia vogeli***
Entamoeba canibuccalis	***Encephalitozoon cuniculi***
Isospora burrowsi	***Balantidium coli***
Isospora canis	***Ehrlichia canis***

LIFE CYCLE STAGES

During its life cycle, a protozoan generally passes through several stages that differ in structure and activity.

Trophozoite is a general term for the active, feeding, multiplying stage of most protozoa. In parasitic species this is the stage usually associated with pathogenesis.

In the hemoflagellates the terms amastigote, promastigote, epimastigote, and trypomastigote designate trophozoite stages that differ in the absence or presence of a flagellum and in the position of the kinetoplast associated with the flagellum.

A variety of terms are employed for stages in the Apicomplexa, such as tachyzoite and bradyzoite for ***Toxoplasma gondii***. Other stages in the complex asexual and sexual life cycles seen in this phylum are the merozoite (the form resulting from fission of a multinucleate schizont) and sexual stages such as gametocytes and gametes.

Some protozoa form cysts that contain one or more infective forms. Multiplication occurs in the cysts of some species so that excystation releases more than one organism. For example, when the trophozoite of ***Entamoeba histolytica*** first forms a cyst, it has a single nucleus. As the

cyst matures nuclear division produces four nuclei and during excystation four uninucleate metacystic amoebas appear.

Giardia lamblia has the same number of internal structures (organelles) as the trophozoite. However, as the cyst matures the organelles double and two trophozoites are formed. Cysts passed in stools have a protective wall, enabling the parasite to survive in the outside environment for a period ranging from days to a year, depending on the species and environmental conditions. Cysts formed in tissues do not usually have a heavy protective wall and rely upon carnivorism for transmission. Oocysts are stages resulting from sexual reproduction in the Apicomplexa. Some apicomplexan oocysts are passed in the feces of the host.

Babesia canis: If an infected dog is bitten by a tick, the parasites are taken in with the blood meal. These then first reproduce in the tick's intestinal cells and then in the eggs being developed by the tick. When the tick eggs hatch and the larval ticks develop and begin feeding on a dog, they transmit the Babesia canis to it.

Coccidia of dog:(*Isospora canis*) 3 generations of schizonts develop beneath the epithelium of distal portion of lower third intestinal villi. Gamonts are found in the epithelial cells, sub epithelial connective tissue of intestinal villi. And mucosa of large intestine. The prepatent period is 9-11 days and patent period is about 4 weeks.

PATHOGENESIS:

The Digestive Tract

Protozoa can be found in the mouth, small and large intestines. The most common are the coccidia. Dogs have three different species of the coccidian parasite ***Isospora*** in the cells of the small intestine.

Heavy infections cause diarrhoea, bloody stools and occasionally dysentery.

Giardiasis: diarrhoea and dysentery is seen.

Amoebosis : Chronic infection is common. Parasite penetrate deep into mucosa and cause inverted flask shaped ulcer opening in to bowel lumen. Occasional abdominal pain, nausea,flatulence, irregular bowels,headache,and fatigue,dysentery with blood and mucus.**Toxoplasmosis** : fatal case seen in young dogs with concurrent infection/immunosupression.Intermittent fever, tonsillitis,dyspnoea, diarrhoea,vomiting,lesions in brain and spinal cord with progressive paresis.

Blood and Lymph

There are a few protozoan dog parasites found in the blood. There are flagellates (***Trypanosoma***) and sporozoans related to the blood parasites (***Hepatozoon*** and ***Babesia***). These are transmitted by ticks and are particularly important. ***Babesia canis*** occurs in dogs world wide but,the intermediate hosts or vectors are tropical or subtropical. The parasite is limited by distribution of the intermediate hosts..

DIAGNOSIS : based on clinical signs and presence of oocyst in faeces.

In case of blood protozoan diseases blood parameter changes along with microscopic finding of developmental stage confirm this.

TREATMENT

Amoebosis :

Metranidazole is the drug of choice@ 750 mg orally thrice daily for 5-10 days.

Tinidazole is more effective.

Giardiasis:

Quinacrine:@50-100 mg/kg bwt bid for 2-3 days

Chloroquin,diodoquin,metronidazole@ 250mg/kg bwt bid for 10 days

Babesiosis :

Phenamidine isethionate@0.3mlof a 5% sol./kgbw (15 mg/kg bwt) s/c .repeat after 5 days

Diminazine aceturate @3.5 mg/kg bwt s/c or i/m as single dose.

Imidocarb dipropionate@6 mg/kg bwt s/c or i/m as single dose.

Quinoronium sulphate @2.5 mg/kg bwt as 0.5% sol. Through s/c route.

Toxoplasosis:

Sulphadiazine and pyrimethamine

Trypanosomosis:

Quinapyramine (antrycide): 3mg/kg bwt as 10% aq.sol.

Suramins :3-5 ml of 10% sol. Followed by the same dose at 3-4 week interval. prophylactic dose: 0.3-0.5g/kg bwt.

Diminazene aceturate 3.5 mg/kg bwt.

Phenathridine compound@1mg/kg bwt.

CONTROL:

- In case of coccidian infection, isolation of affected animal along with proper treatment is necessary.
- keeping away carnivores(dog) from animal houses, feed water and bedding.
- uncooked offal/meat should never be fed to carnivores(dogs). Freezing drastically or heating above 55•C for 20 min. can kill cysts in muscle/organs.
- Good hygiene and frequent check up help in control.
- Fly control measures should be taken.

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BIOCHEMICAL CONSTITUENTS AS AID OF DIAIGNOSIS OF DISEASES IN DOGS

P. C. Bisoi
Professor, Department of Biochemistry
College of Veterinary Science and Animal Husbandry, Bhubaneswar.

Diagnosis of a disease is an art of precisely knowing the cause of a disease. Success of treatment of a disease depends upon the accurate diagnosis. The diagnosis is based on the history, careful clinical examination of animal, laboratory examination and co-relation and interpretation of findings. Laboratory diagnosis helps the veterinarian to arrive on any conclusion. According to a well known, diagnostician 'A veterinarian who does not need a laboratory is uninformed and a veterinarian who is totally dependent upon the laboratory findings is inexperienced and in either case the animal is in danger.'

Serum biochemical constituents are normally the secretory or excretory products of the cell or the fraction of the dietary nutrients. These constituents always remains within a range at different physiological states of a healthy individual. An abnormality in the function of the body (or tissue) in most cases represents an abnormality of the functions of the constituting cell which are reflected in the fluids bathing them and in turn in plasma and urine. The analysis of blood and urine thus provides the veterinarian with information of great clinical importance for the diagnosis and follow-up of disease. The analysis is performed on a no. of body fluids and juices such as blood, urine, cerebrospinal fluid, bile, gastric juice, pancreatic juice etc. Amongst these because of the ease with which the samples may be obtained and the change that occur in disease, blood and urine analysis have continued to play a dominant role in clinical chemistry.

Analysis of Biochemical Constituents of blood

Proteins : Serum or plasma proteins is a very complex mixture of a variety of protein and their determination is clinically of much value in diagnosis. According to electrophoretic mobility

they are broadly grouped as Albumin, $a_1$globulin, a_2-globulin, B-globulin, y-globulin and fibrinogen. The total plasma protein of dog ranges between 5.4-7.1 gm/dl.

Albumin: It is a major protein of plasma. It has many function in blood. It ranges between 2.6-3.3 g/dl. The variation in the range is helpful for diagnosis of certain diseases.

Increase: Occurrence is rare. It is found during dehydration with diminution in the water content of blood.

Decrease:

(a) Nephritis and Nephrosis with excessive loss of albumin in urine.

(b) Inadequate supply of protein as a result of impaired protein digestion and absorption in peptic ulcer, advanced T.B., malignancy, pancreatic disease etc.

(c) Impaired synthesis in chronic hepatic diseases particularly cirrhosis, infection, severe anaemia.

(d) Excessive catabolism as in uncontrolled diabetes mellitus, thyrotoxicosis, prolonged febrile illness.

Globulin:

It is a mixture of more than 30 different proteins.

A_1 globulin : It includes a_1-acid glycoprotein :

Increase: Observed in acute and chronic inflammatory diseases, cirrhosis of liver, many malignant condition.

Decrease : Hepatic disease, malnutrition, nephritic syndrome.

a_1Fetoprotein : It is used as a marker of hepatic carcinoma. The porotein is not normally synthesized in adult liver. The concentration increases in hepatocellular carcinoma in adult age.

a_1 antitrypsin :

Increase : Acute or chronic inflammation, malignancy, liver disease, myocardial infarction.

Decrease: Nephrotic syndrome, emphysema of lungs, juvenile cirrhosis of liver.

a_2 globulin : It includes ceruloplasmin, haptoglobin, a_2 macroglobulin etc.

Ceruloplasmin: It is a copper containing a_2 globulin increase is observed during inflammatory process, pregnancy, malignancy and decrease is observed in wilson's diseasse.

Haptoglobin : It is a protein which prevents excretion of hemoglobin.It's concentration decreases in intravascular haemolysis and concentration increases during inflammatory condition.

B-globulin : It includes lipoprotein, transferrin,

c-reactive protein, hemopexin etc.

Transferrin: It is a iron transporting protein. The level increases in iron deficiency anaemia and during last month of pregnancy and decreases in cirrhosis of liver, nephrotic syndrome, myocardial infarction and malignancies.

Hemopexin: It is increased during diabetes mellitus, ducheme muscular dystrophy in some malignancies and decrease in hemolytic disorders.

Gamma-globulin: It includes the antibodies which are responsibkle to give protection to the animal. The level increases during infectious diseases and decreases in some genetic diseases associated with immunoglobulin synthesis.

Fibrinogens: It is a protein involved in blood coagulation. Higher level is found in multiple myeloma, Nephrosis, hepatitis, most acute infection and decrease is observed in genetic defect in synthesis, hepatic insufficiency etc.

Blood glucose: It is the major source of energy for monogastric animals. The levels of blood glucose varies between 70-118 mg/dl. Hyperglycemia is found in condition like diabetes mellitus, endocrine disorders like hyper pituitarism, hyper adrenalism and hyper thyroidsim, intracranial diseases, asphyxia, anaesthesia etc. Hypoglycemia is observed in insulin excess, endocrine disturbance like, hypoadrenalism, hypopituitarism, hypothyroidism, and in certain physiological condition such as pregnancy, starvation, lactation etc. A glycohemoglobin i.e. Hb1AC has been reported to reflect the average blood glucose level over several weeks and shows promise of being an effective means of monitoring insulin therapy both in human and dog.

Lipids: The plasma lipids are present as complex with protein molecules, the lipoproteins. Changes in the lipid components in disease result in changes in the type, of lipoproteins present in the plasma. According to density they are of 4 types i.e., chylomicron, VLDL, LDL and HDL. Among them LDL and HDL are important clinically. Lipemia in Dogs are of relatively frequent occurance. There are 5 types of hyperlipoproteinemia are seen in human beings. Type-I hyperlipopreteinemia are found in puppy and dogs.

LDL: It contain major amount of cholesterol. Increase in level of LDL indicates that the animal is more prone of artherosclerosis and myocardial infarction. Increased level is observed in xanthoma, myxodema, coronary heart diseases, nephrotic syndrome, diabetic acidosis etc.

HDL: It involves in removal of cholesterol from peripheral tissue to liver. It is a beneficial lipoprotein fraction – Increase level of HDL is found in diabetic acidosis, glycogen storage disease and low in xanthoma, nephrotic syndrome, chronic billiary obstruction.

Cholesterol : It is a important lipid fraction of the body which act as a precursor for several body constituents. The normal concentration ranges between 135-270 mg/dl. Variation in the level is associated with several disease conditions. Increased level is observed in diabetes mellitus, nephrotic syndrome, artherosclerosis, hypothyroidism, hepatic and billiary tract disease lower concentration is found in pernicious anaema, haemolytic jaundice, hepatocellular damage, hyperthyroidism etc. Diabetic dogs showed hypercholesterolemia in association with increased HDL 1 (2) and LDL (B) levels. In acute pancreatitis moderate increase in cholesterol and triglycerides were observed.

Non-protein nitrogenous substance: It includes urea, uric acid, creatinine, amino acid etc.

Urea: It is a metabolic product of protein metabolism, which is normally excreted through urine. Higher level is observed mainly in the diseases associated with kidney. Decrease is rarely observed and found in liver damage.

Creatinine: It isa excretory product of creatine. The level normally increase during kidney disease and muscular dystrophy.

Uric acid: It is a metabolic product of purine metabolisms. Any defect in synthesis and metabolism of purine causes over production of uric acid accumulation of which leads to gouty arthritis. Also increased in diseases like polycythemia, leukemia, pernicious anaemia etc.

Enzymes

Enzymes are biocatalysts which enhance the multiple reactions of cell metabolism and play a vital role in normal cellular function. Certain enzymes leak into the circulation or other extra

cellular fluids only when the cells are damaged. The conc. of these enzymes in serum, urine or other fluids to an extent is related to the degree of cell damage and the no. of cells involved. Some enzymes occur in different molecular forms which differ in chemical properties but catalyse the same reaction. Different forms are localize to specific organs or tissue in different conc. Depending in their functional needs . The property of relative organ specificity of enzymes and isoenzymes is being exploited extensively for arriving at a laboratory diagnosis of diseased organs.

Amylase: It splits starch in to glucose. Salivary gland and pancreatitis secret the enzyme. Damage to glandular cells or secretory pathway of these organs may cause amylase to enter blood stream.

Increase in amylase activity is observed in acute pancreatitis, perforated peptic ulcer, intestinal obstruction. Peritonitis

Lipase: It catalyzes the hydrolysis of esters in the alpha position of TG. The enzyme synthesized in secretory cells of pancreas and appears in circulation following damage to the tissues.

Increase in lipase activity is observed in acute pancreatitis, perforated peptic ulcer, carcinoma of pancreas.

LDH: It is enzyme involved in glycolysis. It is found in most tissue but found in higher concentration in muscle, heart, liver etc.

Increase: Increase in LDH activity is found in muscular dystrophy, pernicious anaemia, liver disorder, leukemia etc.

It has 5 isoenzyme types i.e. LDH_1 to LDH_5. LDH_1: Increase indicates heart disease. LHD_4 and LDH_5- Increase indicates liver diseases.

ALP: The enzyme found in many tissues but higher level is found in bone, liver, intestine and placenta etc. It has 3 isoenzyme from i.e. Hepatic form – Fast, Intestinal farm – Slowest, Osseous and placenta – medium.

Increase is observed in diseases like obstructive jaundice osteo blastic sarcoma, rickets, acute liver disease etc.

It is used as a marker of bone disease and obstructive jaundice.

ACP: It is used as a marker of metastatic prostatic carcinoma.

CPK: It is a organ specific serum enzyme. It has four isoenzyme CPK_1 CPK_2, CPK_3 and $CPK_{Mt.}$ Increase is observed in muscular dystrophy, myocardial infarction.

AST: Incases is observed in myocardial infarction, moderate increase during liver and muscle diseases; haemolytic anaemia.

ALT: Marked increase in viral hepatitis and moderate increase in cirohesis of sliver, obstructive jaundice, skeletal muscle disease and myocardial infarction.

Choline esterase: It is a important enzyme which involve in hydrolysis of acetyl choline. The level of activity increase during nephrioic syndrome, acute myocardial infarction and decrease during acute liver disease, malnutrition, acute infectious disease and organ phosphorus poisoning etc.

In addition to above enzymes there are several other enzymes which are estimated for diagnosis of some specific diseases.

These blood constituent are also helpful in ascertaining the function of various organs like liver, kidneys, pancreas etc. for example.

Liver Function: To know the function of liver the level of protein, albumin, cholesterol ester, and the enzymes like – ALP, LDH, transminase are measured.

Increase in activity of enzyme ALP, LDH and ALT are indicative of liver dysfunction.

Decrease in level of cholesterol ester and level of albumin are also indicate liver dysfunction.

Kidney function: Increase in the level of creatinine and urea beyond the normal range is indicative of kidney damage.

Pancreatic function: Increase in level of amylase and lipase activity indicates dysfunction of pancreas.

The level of blood constituent also helps to know the prognosis of treatment and for follow-up of disease condition.

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STERILIZATION IN PET ANIMAL PRACTICE

Indramani Nath and Jayakrushna Das
Department of Veterinary Surgery and Radiology,
College of Veterinary Science and Animal Husbandry,
O.U.A.T., Bhubaneswar - 751 003.

The term sterilization/ castration are used to mean removal of the testicles or removal of the ovaries. But by common use the term is only confined to removal of testicles. Removal of ovary is denoted by the term spaying or oophorectomy. The term ovariotomy is used for removal of diseased ovary rather than normal ovary.

Indications:

1. Prevention of breeding nuisance (population control of stray dogs) to reduce prevalence of rabies.
2. Neoplastic growths or crushing injuries affecting the tesicle.
3. In enlarged prostrate.
4. Perineal hernia.
5. To make the animal more docile and domesticated.
6. To avoid fighting tendencies in Tom cats.
7. The urine of male cat has a strong pungent smell and this smell is much less after castration.

Anesthetic protocols:

1. Xylazine- Atropine- Ketamine- Diazepam

Pre-medication

- Xylazine @ 1mg/ kg b wt (administered intramuscularly, maximum dose 1ml)
- Atropine @ 0.04mg/ kg b wt (however, there is increasing evidence that atropine should not be given as a premedicant and should only be administered following induction to maintain cardiac output).

Induction:

To be given 10 minutes after administration of Xylazine and Atropine.

- Ketamine @ 2.5mg/ kg b wt + Diazepam @ 0.25mg/ kg b wt.
- Mix equal volumes of ketamine (50mg/ ml) and diazepam (5mg/ ml) in the same syringe.
- Dose: 1ml of mixture per 10 kg bw, given slowly intravenously to effect, to premedicated dog.

Maintainance:

Increments to be given at half the induction dose.

Fluid therapy:

Ringer's lactate should be administered by I/V route throughout the surgical procedure.

Respiration:

- Open mouthed with gag and spontaneous respiration/ via endotracheal tube.
- Endotracheal tube inserted cuff inflated if necessary.

N.B. Anesthetic overdose of xylazine can be reversed by using yohimbine hydrochloride @ 0.1mg/ kg b wt I/Vly.

2. Anesthetic protocol 2

Triflupromazine/ Atropine/ Thiopentone or Xylazine/ Atropine/ Thiopentone

Premedication:

- Triflupromazine @ 1mg/ kg b wt or Xylazine @ 1mg/ kg b wt
- Atropine @ 0.04mg/ kg b wt

Note: the combination of Xylazine-Ketamine-Thiopentone is not considered safe for old, weak, and young patients and it is recommended that Protocol 2 be used only by an experienced vet.

Induction:

Thiopentone @ 25mg/ kg b wt I/V.

(Note Perivenous administration of thiopentone sodium will cause severe local reaction and must be treated with local infusion of at least three times the volume of sterile saline; this risk can be reduced by the use of a 2.5% solution and by ensuring that thiopentone sodium is given by intravenous route only. Facility of artificial ventilation using oxygen with the help of AMBU bag must be in reach while following this protocol).

Maintainance:

I/V Thiopentone at half the induction dose may be repeated as small I/V boluses but will lead to prolonged anesthesia and longer recovery time.

Fluid therapy and Respiration:

Same as Protocol 1.

The surgeon should use the anesthetic technique with which he is most familiar.

Sterilization Surgery: General Considerations

The choice of surgical approach is at the discretion of the veterinary surgeon.

As with all surgery, great attention must be paid to ensure that Halstead's surgical principles are diligently followed which includes

- Complete asepsis
- Accurate hemostasis
- Careful tissue apposition
- Gentle tissue handling
- Obliteration of dead space
- Post operative rest

The timing of operation

The dogs can be sterilized at any stage of estrous cycle because it is easy to grasp the ovaries due to enlarged size. However, since estrogen can delay blood clotting it is important to provide proper hemostasis for female dogs that are operated in estrous.

Surgical procedure for female dogs

A. Right flank approach (not recommended for pregnant and pyometra cases)

- The right flank method of surgery has been considered as the ideal and preferred method for spaying.
- The dog is positioned lying on its left side and the abdominal cavity is entered through the right flank with the ventral aspect of the dog directed towards the surgeon.

Location of incision site for flank spaying

- In adult bitches incision is made about 4cms behind the most caudal curve of the last rib, parallel to spine and about 9cms ventral to the transverse processes of the lumbar vertebrae.

- The incision often falls at the cranial end of the fold of skin connecting the stifle to the abdominal wall. In young bitches (under 6 months), the incision is placed more caudally. Failure to do this in young dogs results in difficulties in exteriorizing the uterine body near the bifurcation/ cervix to allow identification and removal of the second uterine horn.
- **Note:** The right ovary is more closely adhered to the right kidney and body wall than the left ovary and thus easier to exteriorize if incision is made on the right flank.

Tissues incised

Skin, Subcutaneous tissue or fascia, external abdominal oblique muscle, Internal abdominal oblique muscle and Transverse abdominal muscle to which the peritoneum is often attached.

The skin is cut with a scalpel. Subsequent layers are separated using scissors and blunt dissection. Incising the three muscle layers can cause hemorrhage. Splitting the muscles along their fibers reduces bleeding, causes less trauma and faster healing, but may result in a smaller aperture in which to work.

Inexperienced surgeons often find gaining entry to the abdominal cavity the most challenging part of this approach. Cutting these muscle layers is easiest if they are located using Allis tissue forceps by an assistant and if the surgeon's scissors are held perpendicular to the body wall.

The steps of spaying by this approach are as follows

1. locating the uterine horn and ovary
2. clamping the ovarian blood vessels
3. securely tightening ligature in place around the ovarian vessels
4. clamping the uterus and blood vessels just above the cervix.
5. securely tightening ligature over the groove made by the clamp
6. the ovarian vessels are cut from the ovary
7. ovary and uterus are removed taking care
8. wound closure

Advantages of flank approach

- The wound is under less tension than with midline incision since the three separate muscle layers each individually sutured (catgut can safely be utilized in this space). Wounds are not under the weight of abdominal contents.
- Post operative checking and dressing can be carried more easily in difficult animals.

- Should wound breakdown occur following release of the patient, life threatening complications is unlikely unless a lengthy incision was made.
- Less tension in incision area and increased vascularity can reduce healing time.
- In young lean animals the spay can easily be performed through a very small incision.
- Animals can be released earlier than with midline.

Disadvantages of flank approach

- Approach is more traumatic (i.e. through three muscle layers) rather than midline, and therefore increased post-operative pain is possible.
- Access to the left ovary or cervix may be more difficult, especially if the initial incision was incorrectly placed.
- Retrieval of a dropped ovary or bleeding ovarian stump or pedicle is difficult: if this occurs, the recommended procedure is to quickly suture the skin wound and prepare the dog for exploratory laparotomy via a ventral midline approach. Once the problem is addressed, the procedure is completed via the midline and then the flank incision is closed in layers as normal.
- Cutting through the three muscle layers can cause bleeding which may be sufficient to obscure the surgical field and can lead to increased risk of post-operativer infection
- Severe reaction to catgut can occur. Degradation sometimes produce swelling within the muscle and needs to be monitored. As it is a favorable site for infection.

Midline Spay Technique

Approach

- Tissue incised skin, subcutaneous tissue, linea alba – white fibrous tissue (aponeurosis) and peritoneum.
- The incision extend from one inch caudal to the umbilical scar.
- Precaution taken not to make incision paramedian.
- Spaying is done as in flank.
- Closure is done in one layer using vicryl as catgut degrades too quickly and linea alba heals slowly. Nylon sutures are recommended for skin closure.

Advantages

- Less hemorrhage
- Less post-operative pain

- Incision can be extended in case of complication like hemorrhage or dropped pedicle.
- Most familiar technique to the surgeon.

Disadvantage

- Failure to give incision on exact midline makes the incision paramedian.
- The wound is more inaccessible for post-operative care in fearful animals.
- Longer convalescence period because of slow healing.
- Incision is under the full weight of the abdominal organ and increased risk of wound break down and herniation.

Clinical Complications

1. Hemorrhage

- Hemorrhage occur during tearing of ovarian vascular complex while stretching/ breaking suspensory ligament.
- Bleeding can occur due to tension on the uterine body.
- Indisual ligation of the large vessels (fat dog)

2. Recurrent sign of estrous or heat

- Occurs due to remnant of ovarian tissue.

3. Uterine stump pyometra

- If any portion of uterus is not removed this complication occurs when it is recommended to do complete ovariohysterectomy rather than tubectomy or ovariectomy.

Surgical Procedure of castration in male dog

Approach

- Males are castrated through a single pre-scrotal incision.
- One testicle is advanced cranially and skin is incised over the tensed testicle.
- In sub-cutaneous tissue, dartos and external spermatic fascia are incised.
- The testicle within the spermatic sac is grasped and pulled free.
- The spermatic sac is then incised at its most ventral part.
- The vaginal tunic is reflected revealing the testicle and associated structure.

- The vaginal tunic is separated from the tail of the epididymis by breaking the ligamnetous attachmen leaving the tecsticle attached to the spermatic vessels in one bundle and ductus deference by the mesorchium.
- The ductus deference and spermatic vessel clamped and ligated. Once the vessels are ligated the testicle is severed from them. The spermatic vessels usually retract after severance.
- The contra-lateral testicle is now advanced in to skin incision and removed as usual.
- It is a good practice to suture the vaginal tunics of the two testicles so that potential opening in to the abdominal cavity is closed. Skin is sutured as usual.
- Particular must be paid to ensure hemostasis, to reduce the chance of post-operative hematoma, considerable post-operative bruising and swelling is common.
- The technique in other pet animals like cat, mongoose, guinea pig and rabbit are similar after proper anesthesia.

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ADVANCES IN CANINE CLINICAL NUTRITION

P.K. Dehuri, Dean
College of Veterinary Science & Animal Husbandry
Bhubaneswar-3

Good nutrition and a sound, balanced diet are essential to the health, performance and well being of all animals. The goal of nutrition for dogs in times past was simply to provide adequate calories, protein, fat, vitamins and minerals, but over the years, researchers have discovered that nutrition play an important role in management of diseases.

Recent clinical observations reveal that combining modern veterinary medicine with clinical nutrition may give better response than does either approach alone. Clinical nutrition enhances healing by providing the cells with better environment for regeneration and overcoming stress caused by injury or diseases. Although nutrients tend to work more slowly than drugs, in the long run they enhance metabolic processes and help restore function and balance to the body.

Clinical nutrition is a practical approach that can be incorporated to any clinical condition or disease protocol.

NUTRITIONAL MANAGEMENT OF GASTROINTESTINAL DISEASE

Gastrointestinal diseases can be broadly classified into acute and chronic cases. In case of acute gastrointestinal diseases, it is recommended to with hold food for 12-24 hours (bowel rest). Providing bowel rest has traditionally been considered important in order to reduce the amount of unabsorbed nutrients that could result in osmotic diarrhea and bacterial overgrowth and to reduce the antigenic load to the mucosa.

In early refeeding period glutamine may be helpful. Glutamine helps in stimulating intestinal mucosal growth and maintain intestinal barrier. Current recommendation is to provide 500-1000ig glutamine/ 100kcal enterally during first 7-10 days while recovery from acute G.I. disease. (Remillard, 1998). When animal starts eating again, a low fat diet is preffered to minimize the secretory effects of malabsorbed fatty acids and bile acides.

It is advisable to feed only one or two protein sources initially when intestinal permeability may still be high, in order to prevent development of new food sensitivities. Feeding of totally different protein sources during the first few weeks may minimize future problems in case the animals become sensitized to a protein that is dietary staple.

In case of chronic gastrointestinal diseases, the ideal diet should be highly digestible, low in fat and lactose, gluten free, isotonic and contains only one protein source. The diet should also be

nutritionally balanced and palatable since animal with gastrointestinal diseases often have reduced appetite. The diet should be moderately low in fat (<15% DM) to limit the effect of fat malabsorption.

Boiled rice is commonly used as carbohydrate source in dogs with GI disease, since rice starch is highly digestible and gluten free. The diet should be gluten free since gluten sensitivity may occur. Gluten is a component of wheat, oat, barley and rye which should be avoided. GI disease lowers the lactose activity. So diet of dogs with GI disease should be low in lactose and dairy products are in general best avoided.

The diet should contain moderate amount (<7%) of soluble fibre, since they are highly fermentable and produce large amount of short chain fatty acid, which increases mucosal cell proliferation and G.I hormone release. Inclusion of insoluble fibre is advisable to help maintain normal faecal consistency. Several commercial G.I recovery diets now contain beet pulp as a fiber source since this has both fermentable and non-fermentable component.

Dietary supplementation with fructo-oligosaccharides (FOS) is currently receiving attention as a means to discourage pathogen growth and promote mucosal health through metabolism to short chain fatty acid (SCFA). FOS are not digestible by the host and remain intact in small intestine, but are fermented completely to SCFA by specific bacteria in colon. The selective nature of this fermentability has resulted in dietary FOS supplementation being used as a prebiotic to manipulate the colonic flora and induce selective proliferation of non-pathogenic bacteria that are considered beneficial (Gibson & Roberforid, 1995). Manufactured FOS are currently added to some commercial pet food as an alternative to soluble fibre.

Then use of probiotic is also being evaluated. Probiotics are live microbial feed supplements that beneficially affect the host animal by improving its intestinal microbial balance. There may be direct inhibition of enteropathogen growth as well as nonspecific stimulation of host immune response. (Lewis & Freedman, 1998)

Increasing the amount of dietary ω-3 polyunsaturated fatty acid (PUFA) may be helpful in inflammatory intestinal disease. Fish oil is the commonly used source of ω -3 PUFA. ω -3 PUFA lower serum vitamin E concentration and additional Vitamin E should be given to dogs receiving fatty acid supplementation.

NUTRITIONAL MANAGEMENT OF HEPATIC DISEASES

Acute and chronic liver failure is associated with major metabolic abnormalities, malnutrition and reduced detoxification. The requirement for energy, protein and certain micronutrients are probably greater, because of the needs of regenerating hepatocytes coupled with impaired absorption and storage of nutrients. So to ameliorate the above condition the dietary management should be aimed at

1. To supply adequate energy and other nutrients to fulfill basic requirement and to support hepatic regeneration.
2. Correct metabolic abnormalities.
3. To reduce work load of diseased liver

Energy Requirement

The diet should supply adequate non-protein calories to prevent the use of amino acids for energy, reduce the need of gluconeogenesis and provide for repletion of hepatic glycogen stores. Normally energy is best supplied in form of fat, since it increases palatability and has greater energy density. It may be tempting to feed extra calories to put weight back on the debilitated animal, but this should be avoided in severe hepatic dysfunction since the excess calories will only put an extra burden on the diseased liver.

Protein Requirement

Protein levels are often incorrectly restricted in animals with liver disease in order to manage hyparammonaemia. In fact protein requirements are often increased and this practice may lead to protein malnutrition with increased breakdown of endogenous protein, weight loss and hypoalbuminaemia. Dietary protein requirement for dogs with liver disease have been derived from experimental studies specifying at least 2.1g/kg b.wt of high biological value protein. It is essential to feed high quality, high digestible protein that have amino acid content close to animal's requirement. Generally proteins of animal origin are of higher quality than proteins of plant origin. A study in dogs suggested that they did better on a milk diet than on a meat based diet and a casein based diet was helpful in the management of clinical patient with liver disease. (Compton 1971; Strombeck, 1983)

Lipid Requirement

Fat restriction is only recommended in patient with severe steatorrhea and in case of near bile duct obstruction. In such cases replacement of long chain triglycerides with medium chain triglyceride that are readily absorbed is advised. Some long chain triglycerides still must be given in order to supply adequate essential fatty acid.

Micronutrient Requirement

Vitamins

Vitamin K is the main fat soluble vitamin that needs to be supplemented. Deficiency treated with 2-3 doses of vitamin K_1 (0.5-1 mg/kg s/cevery 12 hours). Vitamin E is important in the protection against ongoing free radical or oxidant injury in the liver including acute and chronic hepatitis, fibrosis and cholestasis. (Feher et al, 1998). Vitamin E supplementation at a does ranging between 10-100 IU/kg per day is recommended in cholestasis, where its absorption is impaired.

Minerals

Zinc supplementation may reduce lipid peroxidation and has anti-fibrotic properties. Zinc acetate (2mg/kg/day), gluconate (3mg/kg/day) or sulphate(2mg/kg/day) divided into 2 or 3 daily doses can be used as dietary supplement. Dietary supplementation with zinc also protects against hepatocelluar damage associated with copper accumulation. Eating of food high in copper (eg-shell fish, organ meats and nuts) should be avoided.

NUTRITIONAL MANAGEMENT OF CARDIAC DISEASE

Recent research has shown that nutrition actually modulates cardiac disease, either by slowing the progression, minimizing the number of medication, improving the quality of life, or in rare cases actually curing the disease.

Maintaining optimal weight by avoiding obesity and cachexia is key to optimal management of cardiac disease. As early as 1960's authors proposed restricted protein diet for dog with congestive heart failure (CHF), but there is no evidence that protein restriction is necessary for dog. Unless severe renal dysfunction is present, high quality protein should be fed to meet canine maintenance requirement.

Most dogs with dilated cardiomyopathy (DCM) do not have taurine deficiency, but low taurine concentration have been found in some breeds of dog with DCM, most notably cocker spaniel. Supplementation with taurine (500mg bid-tid) and carnitine (1 mg bid-tid) is recommended in dogs with documented taurine deficiency. Dogs with CHF have been shown to have plasma fatty acid abnormalities, including decreased concentration of Eicosapentanoic acid (EPA) and docosahexanoic acid (DHA) compared to normal dogs. Dogs with DCM and CHF, fish oil supplementation at a does of 25mg/kg EPA and 18 mg/kg DHA normalized these fatty acid abnormalities.(Freeman et al, 1998)

Although healthy animals can easily excrete excess dietary sodium in urine, this response is blunted in animal with cardiac disease. It is unnecessary to institute severe Na restriction at a early stage when cardiac murmer is detected. As heart disease progresses and CHF ensues Na restriction becomes important in animal with cardiac disease without CHF, sodium (Na<0.1gm/ 100kcal) and with early CHF, Na<0.09gm/100kcal and in severe CHF, Na<0.04gm/100kcal is recommended. Vegetables, fruits and low salt commercial pet treats can be given.

Use of furosemide can cause hypokalemia and hypomagnesemia due to increase urinary losses. If hypomagnesemia is detected in patient with arrhymias supplementation should be instituted. CoenzymeQ10 has been proposed as a possible cause for DCM, but this association has not been proven. The current recommended dose is 30mg bid. In case of cardiac cachexia, supplementation of fish oil, which is high in ω -3 PUFA can decrease cytokine production in dog with CHF and improve cachexia. (Freeman et al., 1998)

NUTRITIONAL MANAGEMENT OF RENAL DISEASES

Contrary to popular myth, diets rich in proteins do not cause kidney damage. Additionally documented research on dogs indicates that reducing dietary protein in old dogs is unwise. Restricting dietary protein may be helpful to patient whose BUN value are rather high (>75mg/dl) and that are already in advanced kidney failure.

Acute renal failure

Fluid therapy is essential to the correction of hyperkalemia, metabolic acidosis and hypovolaemia. Especially for hyperkalemia, insulin therapy (0.25unit/ kg) rapidly drives K^+ back

into the cell; glucose should be provided simultaneously to avoid hypoglycemia. Hypokalemia results in worsening renal failure and potassium supplementation of the fluids during polyuric maintenance phase is often required to maintain normal serum concentration (Lane & Grauer, 1994). In case of hyperphosphatemia, feeding diet with decreased phosphate content and with phosphate binders is recommended.

Chronic renal failure

Management of Impaired Phosphorus Homeostasis

Excess intake of dietary phosphorous contributes to CRF due to development of nephrocalcinosis or other effects of hyperphosphatemia, including renal secondary hyperparathyroidism. (Brown et al, 1990). Dietary phosphate restriction (34-42mg/kg/day). If normal concentration is not reached, then phosphate binders like Al or Ca containing salts are used. Al containing salts are associated with osteodystrophy, while Ca salt associated with hypercalcemia which subclinically stimulate release of parathyroid hormone (PTH). Oral calcitriol therapy (0.7-1.4 mg/kg/day) will lower PTH in most, but not all dogs with CRF (Brown & Finco, 1994).

Management of Glomerular hyperfilteration, and hyperfilteration and hypertrophy

Dietary supplementation with ω -PUFA may prove beneficial in lowering intraglomerular pressure and intrarenal inflammation. (Brown et al, 1993). Although data are inconclusive, one of the proposed goal is to achieve ω -6/ ω -3 ratio between 0.2:1 and 5:1. A general rule of thumb would be 0.5-1 gm of ω-3PUFA/ 100kcal of food.

Management of acidosis

Metabolic acidosis leads to enhanced renal ammoniagenesis which can activate complement cascade, leading to tubulointestinal injury (Nath et al, 1985). Protein of animal origin are rich in sulpher containing aminoacids, whose metabolism leads to H^+ generation which must be excreted by kidney, if acid-base balance is to occur. So vegetable protein such as soya protein which is neutral or alkaline is used.

NUTRITIONAL MANAGEMENT OF ARTHRITIS

Dietary measures have a role in management of both inflammatory and degenerative form of arthritis in dog. Weight control is one important aspect of dietary management and in some obese patient may be the only measure to control the disease. A variety of dietary supplements including ω-3 fatty acid, chondroitin sulphate, glucosamine, antioxidant & green lipped mussel products have been used with varying degree of success in management of arthritis. Dietary supplementation with fish oil which is rich source of EPA & DHA is often advocated in management of arthritis.

Parenterally administered semisynthetic glycosamineglycan such as polysulphated glycosamineglycan and pentosan polysulphate have analgesic, anti-inflammatory and chondroprotective effects and their efficacy in dogs is favourable.

Glucosamine:

Glucosamine is a major precursor for the synthesis of glycosaminglycan (GAG) in cartilage matrix and synovial fluid. It is a small molecule which is rapidly and completely absorbed from G.I tract in dogs. Chondrocutes can synthesize glucosamine from glucose and glutamine, but this constitutes a rate limiting step in GAG and proteoglycan synthesis. So exogenous supply of preformed glucosamine is important at times of demand when cartilage turnover is accelerated following traumatic injury or in osteoarthritis.

Chondroitin sulphate: They are major glucosaminglycans in articular cartilage. They are chondroprotective and stimulate matrix production by providing additional substrate for proteoglycan synthesis but may also inhibit degradative enzyme activity in cartilage (Buci, 1994; Basleer, 1992).

Antioxidants & other micronutrients

Toxic oxygen radicals have been implicated in the pathogenesis of osteoarthritis (Greenwald, 1991). Diet derived antioxidants may be beneficial in reducing cumulative oxidative damage and may therefore have a role in management of osteoarthritis. Low serum zinc levels have been found in osteoarthritic patients (Grennan et al,1980), Pyridoxine may be an essential nutrient for connective tissue matrix formation (Masse et al,1994). Silicon is a trace nutrient which has an integral role in collagen and GAG formation in cartilage matrix and may be important in management of osteoarthritis (Carlise,1988). Manganese is an essential cofactor in proteoglycan synthesis, which is required for normal cartilage development (Hanson et al, 1997) and has been included as a component of glucosamine-chondroitin sulphate supplement.

Green Lipped mussel (GLM)

Among some indigenous coastal cultures, shell fish supplements have been used as traditional remedy for arthritis and in recent years, interest has focused on potential benefit of a nutritional supplement prepared from the New Zealand green lipped mussel, perna canaliculus. Recently, a lipid rich extract of stabilized GLM has been shown to be potent, but relatively slow acting antiflammatory agent, with the highest anti-inflammatory activity being found in the PUFA component of mussel (Whitehouse et al, 1997). It may be effective in treatment of inflammatory and immune based condition such as rheumatoid arthritis. A series of clinical trails conducted at WALTHAM have demonstrated the efficacy of GLM in alleviating arthritic signs in dogs. (Bui et al, 2000)

NUTRITIONAL MANAGEMENT OF DIABETES MELLITUS

Diabetes mellitus is a common endocrine condition of dogs characterized by relative or absolute deficiency or insulin. The stabilization of diabetic patient depends upon balance of three factors-insulin, diet and exercise.

Many canine diabetics may benefit from being fed high fibre diet, but those who are underweight may require diet with high energy density. Certain diets which are rich in simple sugars or that have high levels of fat are contraindicated in diabetes mellitus. Most diabetic dog that are of correct weight will have an energy requirement of about 50-70kcal/kg/day. Diabetic dogs that are underweight should receive atleast 150% of basal metabolic requirements for their ideal body weight. Diabetic dog that are overweight will benefit from 30% reduction in energy intake.

Dietary recommendation for a diabetic dogs are usually for a diet in which 50-55% of the energy content is derived from fat. Protein is not normally restricted and increased dietary fibre is advocated. Diet high in complex carbohydrate and dietary fibre have been shown to help glycaemia control. (Ihle,1995). Dietary fibre is thought to achieve this effect by decreasing gastric emptying time and slowing the absorption of the products of carbohydrate and protein digestion. All diabetic dog should be provided with ample quantities of water as hyperglycaemic episodes can occur sporadically even in otherwise stable animals.

NUTRITIONAL MANAGEMENT OF SKIN DISEASES.

Nutritional imbalance and various environmental factors can have a major impact on skin and coat condition. The nutritional factors mostly affecting the skin are-

Proteins- It is estimated that one-quarter of the protein consumed daily is utilized by the skin in production of new hairs and epidermis. (Scott,1990). Signs of protein malnutrition include depigmentation of the skin and hair due to a deficiency of tyrosine, tryptophan and cystine (Glatti et al, 1973). In addition sulpher rich aminoacid, cystein and methionine are important in production of keratin and a deficiency of this results in loss of hair. So, it is important to provide sufficient protein such as meat, egg and milk.

Lipids- Essential fattyacids are vital for maintaining normal skin structure and function. Dietary deficiency may occur in dog consuming lowfat dry foods or inappropriately formulated home-prepared diets. There are two series of e.f.a important to health of skin and coat, omega-6 (derived from linoleic acid) and omega-3 series (derived from ά-linoleic acid). Currently there is debate over the ratio of ω-6 to ω-3 fatty acid ratio in the dietary management of inflammatory disease (Scott et al, 1997). The chief indicator for dietary PUFA supplementation are pruritic skin diseases associated with hypersensitivity reaction in dogs.

Micronutrients

Zinc-Zinc deficiency may be due to reduced availability of dietary zinc through nutrient interaction or absorption of zinc. Oral zinc supplementation together with dietary correction brings resolution of clinical signs. Supplementation with $ZnSo_4$(10mg/kg) is usually adequate but life long therapy is normally required. In the UK, historically, most cases of zinc responsive skin disease in dogs have resulted from feeding soya or cereal based diet, the effects of which could be exacerbated in animals already subject to inherent defects of zinc absorption (Thoday,1989)

Vitamin A- Vitamin A responsive dermatosis is rare, seen exclusively in cocker spaniels that appear to have been fed a nutritionally adequate diet. (Miller, 1989). The condition responds to oral supplementation with Vitamin A at 10,000IU/day. This dose is in excess of normal dietary requirement and it is important that other causes are eliminated before therapy is initiated.

Vitamin E- The dietary requirement for Vitamin E is linked to the amount of PUFA in the diet. (Putnam and Comben, 1987). High fat diets can induce relative deficiency of Vitamin E leading to scale formation, erythema and hairloss (Scott and Sheffey, 1987)

DIETARY SENSITIVITY

Dietary sensitivity describes an adverse reaction to food and may be further classified as either food intolerance or food allergy. Food intolerance can result from an impaired ability to digest the food or from pharmacologic, metabolic or toxic reaction. But food allergy is an immunologically mediated response to specific dietary antigen. Most cases of dietary sensitivity manifest as skin or gastrointestinal disorder. The commonly recognized causes in dogs are beef and dairyprotein, other meat proteins, egg, lactose and gluten. The most useful and reliable method of diagnosing dietary sensitivity is to feed an elimination diet, based on previous dietary history, followed by dietary challenge with test meal. (Wills and Harvey, 1993)

Elimination diet and diagnosis

1. Identify the component of the diet and decide on a replacement diet. The food should be home prepared and should be composed of different ingredients from noprmal diet. No diet other than water is allowed. Water, a carbohydrate (boiled rice) and a source of protein (soya, lamb, chicken) are required. The duration of this diagnostic stage should be atleast 6 weeks.
2. If the disease goes to remmision, it suggests dietary sensitivity, challenge with original diet. This helps to demonstrate a relationship between diet and disease and it also gives an indication of time of relapse to occur.
3. If no relapse occur within 3 weeks, then dietary intolerance is not considered further-any improvement was coincidental.
4. If relapse occur, then reinstate the restricted diet. If the disease again goes to remmision, then a relationship with dietary component is probable and challenge studies may take place.
5. Maintain on a restricted diet and challenge with individual component of the original diet in order to identify this component. (elimination phase). It may be appropriate to transfer the animal to a commercially prepared, balanced, restricted (hypioallergic) diet. Individual components of the original diet are added to about 10% volume to restricted diet.
6. Advise on a balanced diet for a long term control.

The modulation of nutrients in the clinical practice is not meant to replace the use of drugs and surgery, but to work synergistically with the best protocols and tools at veterinarian's disposal. If properly incorporated into veterinary practice, the use of clinical nutrition can bring superior result.

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DOPPLER AND CONTRAST ULTRASONOGRAPHY

S. Prathaban
Professor and Head, Department of Clinics
Madras Veterinary College, Chennai –600 007

Doppler dramatically increased the diagnostic capabilities of cardiac ultrasound. This modality allows detection and analysis of moving blood cells and tells us about the direction, velocity, character and timing of blood flow. The haemodynamic information provided by Doppler echocardiography allows definitive diagnosis in most cardiac examination.

Three types of Doppler may be used during an echocardiographic examination

1. Pulsed wave (PW)
2. Continuous wave (CW)
3. Color flow (CF)

Pulsed wave Doppler:

Pulsed wave Doppler is site specific in which it can be directed and set to sample flow at specific places within the heart. However PW Doppler is limited in its capacity to detect higher frequency (velocity) shifts. Continuous wave Doppler (CW) can detect high frequency shifts and therefore high flow velocities with virtually no limits. Because sound is transmitted and received continuously it is not possible and interrogate at specific depths within the heart. Although this may sound disadvantageous, the information provided is valuable. Colour flow Doppler (CF) is a form of pulsed wave Doppler, frequency shifts are encoded varying lines and intensities of colour. Flow information is graphic and detection of abnormal flow is easier with CF Doppler however quantitative information is limited.

Doppler shift:

Christian Johann Doppler an Austrian physicists and mathematician was first to describe the Doppler effect. He found all types of waves (light, sound etc) change in wave length when position between the source of the wave and the receiver of the wave changes. For example if we move towards sound source the pitch are frequency of that sound increases and if we move away from that sound source the frequency decreases. The change in the frequency between sound that is transmitted and the sound that is received is the Doppler shift when the source and reflecting surfaces both are stationary the transmitted and reflected wave lengths are equal. When the reflecting structure is moving toward the sources sound waves encounted more often increasing the number of waves (high frequency) reflected back towards the source. When the reflecting structure is moving away from the source sound waves travels ahead of the transmitted wave

front and are encounted less frequently, decreasing the number of sound waves (Low frequency) reflected back to the source. The Doppler shifts that we use are in ultrasound in is the difference in frequency transmitted by the transducer and received frequency reflected from blood cells.

Doppler tracing:

Doppler derived frequency shift (fd) is equal to the reflected frequency minus transmitted frequency therefore objects moving towards the source result in the positive frequency shifts where as the object moving away from the source result in negative frequency shifts. The site (gate) for the Doppler flow interrogation is selected by the examiner and is represented on the Doppler display as a line (base line). Positive frequency shift (flow moving towards the transducer) produce wave forms up from the base line, whereas negative frequency shifts (flow moving away from the transducer) producing downward deflection on the Doppler tracings. These images are called spectral tracings.

Pulsed Wave (PW) Doppler:

Pulsing the sound waves allows a transducer to act as a receiver for the signal only during the time interval specified by a sample depth. With PW Doppler the transducer will receive frequency shifts only during the time interval dictated by the depth of the sample site ignoring all the returning the echoes. New sound waves will not be transmitted until the transducer has received echoes from the previous burst. Range resolution is the ability to measure velocity within a normal cell at a specified depth along the ultrasound beam. The gate is the site at which sampling is set to occur. The examiner usually sets gate while watching a two dimensional image.

Continous Wave (CW) Doppler :

CW Doppler continuously sends out and continuously receives sound. It is not possible to range gate CW Doppler because the transducer as no way of detecting the depth of the reflected signal. CW Doppler detects frequency shifts all along the ultrasound beam with no range resolution and is steered in one of two wave imaging CW systems use a cursor as colour representing the Doppler sound beam. The cursor is placed over the two dimensional image and frequency shifts are calculated all along beam in imaging CW system non imaging CW system use a dedicated CW pulse without a two dimensional image. These systems require recognition of characteristic flow profiles.

Velocity alone the beam varies and a full spectrum of frequency shifts are detected with a CW Doppler. When CW Doppler is used properly the highest velocity along line of interrogation are recorded. The highest flow velocity are what is of interest and diagnostically important. Lower velocity flows are hidden within the higher flow profiles. Flow patterns for the various valves and vessels in the heart are characteristic and usually are identified easily with both PW and CW Doppler.

The Doppler equation:

Doppler ultrasound can determine blood cell velocities within the heart or peripheral vessels based on the Doppler shifts. Accurate measurements of velocity are affected by transducer frequency and intercept angle.

Effect of Doppler frequency:

PW Doppler measures the frequency shifts location within the heart. Just like two dimensional and M– mode imaging, the reflected single must be received before the next pulse is transmitted or the recorded singles will be ambiguous. The time interval between the pulses is also referred to as pulse repetition frequency (PRF) must be two times the sample depth. The time between pulses must increase as sample depth increases, resulting in decrease PRF. Decrease PRF decrease the Doppler frequency shift that can be measured accurately. The sampling rate must be atleast two times the frequency shift for the transducer to receive unambiguous flow of information. Maximum Doppler shift that can be recorded accurately is equal to one half the PRF

Doppler shift = ½ PRF

One half of the PRF is referred to as niquist limit. When the niquist limit is exceeded, the flow profile wraps around the image, this can be corrected moving the baseline up or down on the monitor allowing the entire profile to be recorded accurately. When the niquist limit is exceed by large degrees the aliased signal no longer displays the characteristic flow profile and direction can no longer be determined. Switching to CW Doppler allows flow velocities that exceeded the niquist limit to be recorded accurately. The maximum velocity that can be recorded without aliasing is inversely proportional to depth for any given transducer frequency the niquist limit is exceeded for sooner at deeper gates for a given interrogation frequency.

Normal blood flow is typically laminar for all blood cell within a vessel out flow tract are chamber move in the same direction with similar flow velocity. Vessel and chamber walls create friction for the blood cell moving adjacent to wall surfaces and velocities are some what slower along the periphery of the flow stream than in the centre of the flow stream. PW Doppler appears hollow within the spectral broadening when flow is laminar intercept angle close to zero and the niquist limit is not exceeded spectral broadening in the filling of typically hollow waveform. Spectral broadening a pulse wave signal may be result of improper gain settings large intercepts angle are non laminar flow (turbulent). When flow becomes abnormal it is turbulent. Turbulent flow has blood cells moving in many directions at various velocities. This kind of flow is seen with stenotic lesions, shunts and valvular regurgitation. Doppler singnals produce from turbulent flow have a lot of spectral broadening because of many velocities and flow direction present in the jet. CW Doppler shows spectral broadening even when flow is laminar because flow velocities detected all along the transmitted sound beam vary tremendously .

Colour Flow Doppler :

CF Doppler is a form of pulse wave Doppler real time images and colour flow mapping are done at the same time with alternating scan lines dedicating towards real time image generation and Doppler signals. Blood flow in colour mapping is perceived by the machine either moving towards the transducer or moving away from it via negative or positive frequency shift. Flow moving towards the sound source is plotted in hues of red and flow moving away from the transducer is mapped in shades of blue. No flow generates no frequency shift has no colour is assigned. Enhanced colour maps available in most equipment display flow velocity information as well as

direction. Colour range from deep red for slow blood flow to bright yellow for rapid red flow towards the transducer. Slow blood flow away from the transducer is mapped in deep blue colours and whereas rapid blood flow away from the transducer is mapped in shades of light blue and white. CF Doppler depends on two important factors. Pulse repetition frequency and frequency of the transducer. As spectral Doppler the sound source frequency dictates the maximal velocity but can be accurately mapped at any given depth before aliasing occurs. Aliasing in CF Doppler involves a reversal of colour resulting in a mosaic of mixing of the red and blue hues. Aliasing in CF Doppler involves a reversal of colour while using high frequency transducer when in activating there is normal flow and the aliasing is only a function of transducer frequency. A aliasing would be eliminated if a lower frequency transducer was used.

Many ultrasound machines have variance maps. These machines map turbulent flow in hues other then blue or red.

Frame rate refers to the number of times a real time or colour flow image is generated per minute . A frame rate of atleast 15 times per minute is required for smooth transition and the appearance of a continuously moving image as a sector. Frame rate in CF Doppler is equal to PRF divided by scan lines per colour sector. The operator can alter the width of the sector . Decreasing the colour wedge decreases the amount of time necessary for sampling and increases the frame rate. The operator also can eliminate the real time image that extends beyond the width of the colour sector. Decreasing the colour wedge decreases the amount of time necessary for sampling and increases the frame rate. The operator also can eliminate the real time image that extents beyond the width of colour sector. This also decreases the time necessary for image generation and enhances colour flow mapping.

Many machine allow the operator to decrease the depth of the colour wedge . This has no effect on the frame rate, because total image depth is unchanged . It merely decreases the information the mind has two process.

The number of time the line of sound is sampled is referred as its packet size. Increasing packet size improve the image quality and fills in the colour display but at the expense of frame rate. Packet sizes usually can be selected by the operator. Decreasing packet size will increase the frame rate, but decrease sampling time. Information may be lost with short sampling times . However, this may be necessary width rapid heart rate. Increasing packet size will increase the time required for sampling and will decrease the frame rate. But it results in greater accuracy in measuring velocity and map colour.

S

ECG AND ITS INTERPRETATION

S. Prathaban
Professor and Head, Department of Clinics
Madras Veterinary College, Chennai –600 007

Electrocardiography is established as an atraumatic, relatively inexpensive and extremely useful technique for gaining information about the heart and it is accepted as a necessary part of the cardiac examination in a dog or cat. As early as 1912, the electrocardiogram was considered an important method of studying the heart. Augustus D. Waller was the first to demonstrate that the electrical impulses of the heart could be recorded from the surface of the body.

In 1895, Einthoven introduced the terms P, Q, R, S and T for the electrocardiographic deflections; where P correspond to atrial depolarization or contraction, QRS correspond to ventricular depolarization or contraction and the T wave represented ventricular repolarisation or relaxation. Waller was the first to use the term electrocardiogram.

The electrocardiogram is a graph of the variations in voltage produced by a mass of associated cardiac muscle cells or bundle of muscle fibers. The electrocardiograph as a galvanometer has a delicate writing instrument that will indicate a single positive or negative charge. An electrocardiogram should always be obtained without chemical restraint as any drug may influence the heart rate or rhythm: (Dukes McEwan 2000).

The electrocardiograph can be used to look at the electrical activity of the heart from different angles to get a complete picture. Each different angle or pair of electrodes is called a lead. The following lead systems are necessary to view the heart from different directions.

I. Bipolar Standard Limb Leads:

1. Lead I – Right arm (-) to the left arm (+).
2. Lead II – Right arm (-) to the left leg (+).
3. Lead III – Left arm (-) to the left leg (+).

II. Augmented Unipolar Limb Leads:

Lead avR (augmented vector right)-Right arm (+) compared to left arm and left leg (-).

Lead avL (augmented vector left) - Left arm (+) compared to the right arm and left leg (-).

Lead avF (augmented vector – frontal)- Left leg (+) compared to the right and left arm (-).

III.UNIPOLAR PRECORDIAL LEADS (EXPLORATING LEADS)

Position of explorating electrodes in different leads is as follows:

Lead V 10 – Over the dorsal spinous process of the seventh thoracic vertebra.

Lead CV6LL – Sixth left intercostal space near the edge of the sternum.

Lead CV6LU – Sixth left intercostal space at the costochondral junction.

Lead CV5RL – Fifth right intercostal space near the edge of the sternum. The standard leads are especially useful for studying abnormalities in the P, Q, and R, S and T deflections, diagnosing cardiac arrhythmias and determining the mean electrical axis.

Electrocardiography is a useful tool in two major areas:

1. Diagnosing most cardiac arrythmias since the electrocardiogram can determine the source of the rhythm and the frequency with which the impulse arises ; and
2. Providing information on the electrocardiographic tracing is often altered by either pathologic or physiologic factors.

Indications for an electrocardiogram:

1. Cardiac arrhythmias.
2. Acute onset of dyspnea
3. Shock.
4. Fainting or seizures.
5. Cardiac monitoring during and after surgery.
6. Cardiac murmurs.
7. Cardiomegaly found on thoracic radiographs.
8. Cyanosis.
9. Pre operatively in older animals.
10. Evaluating the effect of cardiac drugs – especially digitalis, quinidine and propanolol.
11. Electrolyte disturbances, especially potassium abnormalities.
12. Systemic diseases that affect the heart.
13. Serial electrocardiograms as an aid in the prognosis and diagnosis of cardiac disease.

Normal Canine Electrocardiographic Values

Rate

70 to 160 beats /min for adult dogs

60 to 140 beats /min for giant breeds

Up to 180 beats /min for toy breeds

Up to 220 beats /min for puppies.

Rhythm

Normal sinus rhythm
Sinus arrhythmia
Wandering SA pacemaker

Measurements (lead II, 50 mm / sec, 1 cm = 1mv)

P wave

Width: maximum, 0.04 sec (2 boxes wide).

maximum, 0.05 sec (2 ½ boxes wide) in giant breeds.

Height: maximum, 0. 4 mV (4 boxes tall).

P-R interval

Width: 0.06 to 0.13 sec (3 to 6 ½ boxes).

QRS complex

Width: maximum, 0.05 sec (2 ½ boxes wide) in small breeds.

maximum, 0.06 sec (3 boxes) in large breeds.

Height of R wave*: maximum, 3.0 mV (30 boxes) in large breeds.

maximum, 2.5 mV (25 boxes) in small breeds.

S-T segment

No depression: not more than 0.2 mV (2 boxes).

No elevation: not more than 0.15 mV (1 ½ boxes).

T wave

Can be positive, negative or diphasic

Not greater than one fourth amplitude of R wave; amplitude range $\pm$ 0.05 – 1.0 mV (1/2 to 10 boxes) in any lead.

Q-T interval

Width: 0.15 to 0.25 sec (7 ½ to 12 ½ boxes) at normal heart rate; varies with heart rate (faster rates have shorter Q- T intervals and vice versa).

Electrical axis (frontal plane) : + 40° to + 100°

Sinus rhythm:

A sequence of beats originating from the sino atrial node forms a rhythm, known as the sinus rhythm. there are four common sinus rhythms.

1. **Normal sinus rhythm:**

   In normal sinus rhythm, the stimulus originates regularly at a constant rate from the SA Node, depolarizing the atria and ventricles normally and producing a co ordinate atrioventricular contraction. The ECG shows a normal P wave followed by normal QRS and T wave. The rhythm is regular (constant) and the rate is normal for age, breed and species.

2. **Sinus arrhythmia:**

   In the case of sinus arrhythmia the stimulus originates from the SA Node, but the rate varies (increases and decreases) regularly. The ECG shows normal P wave followed by normal QRS and T wave. The rhythm varies in rate often associated with respiration. The rate is normal for age, breed and species.

3. In **Sinus tachycardia**, the SA Node generates an impulse and depolarization occurs faster than normal. The ECG shows a normal sinus rhythm but at a faster rate than normal.

4. In **Sinus bradycardia**, the SA Node generates an impulse and depolarization occurs more slowly than normal. This can be a normal feature in some giant breed dogs and in athletically fit animals. The ECG shows a normal sinus rhythm but at a slower rate than normal.

Ventricular premature complexes (VPC) are a common finding in dogs and cats and arise from an ectopic focus or foci within the ventricular myocardium.

The QRS complex morphology is abnormal. The complex is usually a) abnormal in shape, (bizarre) and slightly widened (prolonged). The T wave of VPC is often large and opposite in direction to the QRS wave. A run of three or more VPC is known as Ventricular tachycardia.

ECG interpretation essentially involves four main steps:

1. Calculation of the heart rate;
2. Determination of the heart rhythm;
3. Measurement of the complex amplitudes and intervals;
4. Measurement of the mean electrical axis.

Calculation of the heart rate

The simplest way to calculate the rate from an ECG is to mark a second strip on a representative part of the tracing, count the number of complexes which appear within this time frame and multiply this figure by 10. If the P wave rate and QRS-T complex rate differ, these should be recorded separately.

Electrical alternans refers to an alternation in the size of the QRS amplitude that occurs nearly every other beat.

Artifacts include electrical interference, muscle tremor artifacts and movement artifacts.

ECG ABNORMALITIES

A good understanding of the electrical activity of the heart is key to the accurate interpretations of ECG (Martin, 2002).

1. Right atrial enlargement

P wave grater than 0.4 mV (4 boxes) .Tall, slender and peaked P wave.

Eg. Bronchitis/ Pneumonia/ Congenital defects.

2. Left atrial enlargement

P wave more than 0.04 sec (2 boxes).

3. Right ventricular enlargement

S wave in L.I. greater than 0.05 sec (1/2 box).

4. Left ventricular enlargement

Tall 'R' waves in L II.

QRS duration- Wide more than (2 ½ box) 0.05 sec.

ST – coving.

5. T wave abnormalities

Should not be greater than 1/4 of the R wave.

Sharply pointed (or) Notched – Electrolyte imbalances

Associated conditions

Myocardial hypoxia (O2 deficiency).

Myocardial infarction.

Right or Left Bundle branch block.

Ventricular enlargement.

Hyperkalemia – Large and Spiked.

Hypokalemia – Small and diphasic.

Digitalis Quinidine – Toxicity.

Myocardial infarction

Notched R wave

Sudden deviation of ST segment

Tall peaked T wave.

Wandering pacemaker

It is normal in dogs Wandering pace maker is a shift of the pace maker from the S A node to AV node or from within the SA node .Change in the configuration of the P wave which becomes positive / negative or diphasic. It is an irregular, multiform, supra ventricular rhythm with changing P wave morphology.

Q – T interval

Normal: 0.15 – 0.25 sec (7 ½ - 12 ½ Boxes)

Prolonged QT interval

1. Hypocalcemia due to hypothyroidism renal failure
2. Hypokalemia - Metabolic and respiratory alkolosis

Shortened QT interval

1. Hypercalcemia, Degitilisation, Hyperkalemia

ST segment abnormalities

ST segment depression 0.2 mV (2 Boxes)

ST segment elevation 0.15 mV – in Lead I

S-T elevation is seen in animals with: Pericarditis, Severe ischaemia/infarction (eg, full wall thickness).

ST segment depression

Myocardial ischemia

Myocardial infarction

Hyper and Hypokalemia

Trauma to the heart.

Right bundle branch block

Wide S waves – in Rt. Bundle Branch block

QRS complex greater than 0.08 sec.

Left bundle branch block

QRS complex in greater than 0.08 sec. duration

QRS complex in wide and +ve.

Atrial premature complexes

'P' represents the premature complex

'P' wave is superimposed on the T wave of the preceding complex seen in atrial diseases.

Congenital cardiac defects (Mitral insufficiency) or PDA.

Atrial flutter (F waves)

Rapid, regular atrial rhythm at a rate varying usually from 300 to 500 beats / min ' P ' waves – replaced by saw tooth waves (Called F waves).

Atrial fibrillation

Fibrillation means rapid, irregular small movements of fibres. In atrial fibrillation, one of the most common arrythmias seen in small animals, depolarization waves occur randomly throughout the atria.

Normal QRS morphology, the R-R interval is irregular and chaotic and the QRS complexes often vary in amplitude. There are no recognizable P waves preceding the QRS complex. Fine irregular movements of the base line – known as F waves are seen as a result of the atrial fibrillation waves. Associated conditions include Sinus arrhythmia and wandering pacemaker – frequent in the dog.

Ventricular fibrillation is nearly always a terminal event associated with cardiac arrest. The depolarization waves occur randomly throughout the ventricles. The ECG shows coarse (larger) or fine (smaller) rapid, irregular and bizarre movement with no normal waves or complexes. (Martin, 2002).

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S

LABORATORY DIAGNOSIS OF CANINE AND FELINE DISEASES

S. K. Panda, Assoc. Professor & Head,
Veterinary Pathology, OUAT, Bhubaneswar

Veterinary laboratory diagnosis has become an integral part in modern pet animal practice. Laboratory tests yield useful information and provide definite evidence regarding the alterations during the disease process.

Basic clinical pathology or practitioners laboratory is a small self contained laboratory aiding in the diagnosis of certain diseases in a clinic or dispensary. It deals with routine screening of samples collected by the clinician. Generally examination of wet film of blood, blood smear, blood, urine, faecal sample, skin scrapping, nasal washing and cytological preparations are carried out in such laboratory. Tests could be easily and quickly performed in such laboratory. Complete diagnostic laboratory deals with clinical pathology, biochemical, parasitological and microbiological examination of the clinical samples collected from the diseased pet animals.

Diagnosis of the pet animal diseases mostly depend upon the proper collection of the samples, procedure of the tests and interpretation of the results.

Hematological Examination (Complete Blood Count)

Blood to be used for hematologic tests collected from cephalic vein which is the most commonly used site in dog. Ear vein can be used in the cat, small dog. A marginal ear vein on the dorsal side of the ear is usually selected. Toe or toe nail can be used in small dog and puppies. The capillary bed of the nail is cut into just short of the base of the nail. Femoral, saphenous or tibial vessels used in the dogs and cat. Recurrent tarsal vein can be used for dog (situated at lateral side of hock joint in hind limb). For collection of blood the needle and syringe must be dry since presence of water hemolyses the RBC. The needle should be sterile. Clip the area from where the blood is to be drawn. Apply Tr. Iodine and allow it to dry. Raise the vein by pressure. Use a large (bore) needle that has been sterilized and dried. Insert the needle into the vein; draw into the dry syringe the required quantity of blood. This engages the needle, insert the syringe into the specimen tube containing the anticoagulant to very near its bottom and allow the blood to flow out. Thoroughly mixed with anticoagulant. Never shake the blood vigorously otherwise froth will form, RBC may rupture and erroneous results may be obtained. In collecting blood care should be taken to ensure that needle is of sufficient diameter. A needle of too small gauge may cause disruption of RBC and damage WBC. If it is necessary to transfer the blood from a syringe into a test tube, the needle should be removed, as forcing blood through the needle may damage cells.

A great variety of anticoagulants may be used for hematological examination. Dipotassium and Disodium salt of Ethylene diamine tetracetic acid (EDTA is mostly recommended for routine hematological procedures may be used in either a liquid or in dry form.1mg of powder per ml of blood is required. If liquid is available, 1 drop of a 10% solution is sufficient for 5ml of blood. Care must be taken not to exceed the recommended level of EDTA as it adversely affects the hematological results. Heparin is also commonly used in liquid or dry form (in a test tube). A syringe may be rinsed with a stock solution (1%)of heparin & will contain sufficient anticoagulating activity to preserve 5ml of blood. 1-2 mg (0.2ml of 1% solution) is required for 10 ml of blood.

Taking too much time in obtaining the blood so that the sample has already started to clot before being mixed with the anticoagulants. Failure to agitate the blood sample immediately after placing it in vial and filling the vial to the top cause clotting, as the anticoagulant is not properly mixed with the blood. It is usually safe to take about 0.5 ml blood/Kg body wt. in all spp. Blood should be collected when the animal is at rest and without under excitation.

It is always advisable to start the examination of blood as soon as possible after its collection. But if delay is expected the blood samples must be kept in refrigerator at 4^0C for 24 hours without much alterations. The refrigerated blood sample should be taken out at least 30 min before the start of exam & the sample should be mixed properly with gentle shaking.

Interpretation for Hemoglobin (Hb)

Decreased Hb in anemia (Hypochromic) which is caused by blood loss**,** hemolytic anemia**,** auto immuno hemolytic anemia**,** nutritional**,** parasitism-anaplasma, piroplasma, bacteria-leptospira**,** Chemical-lead, phenothiazine, copper**,** Deficiency-copper,cobalt,vit-B12,folic acid, riboflavin, pyridoxine.

Increased Hb in hemoconcentration, diarrhoea, vomition, excess sweating, burn, polyuria, shock.

Interpretation of Total Leucocyte Count (TLC)

Leucocytosis in generalised infection, localised infection, intoxications including those produced by metabolic disturbances, chemicals, drugs and venoms, rapidly growing neoplasms, acute hemorrhage particularly into one of the body cavities (thoracic peritoneal Joint), sudden haemolysis of erythrocyte, leukemias, trauma, from parenteral administration and excess secretion of adrenal corticosteroids

Leukopenia: The general causes of leukopenia are 4D which are related to alteration in bone marrow. 4D are Degeneration, Depression, Depletion, Destruction. If any of these alterations occurs in the bone marrow, the no. of leukocytes mostly nutrophils in the peripheral circulation is decreased. Viral infections such as canine distemper, infectious canine hepatitis, feline panleukopenia are often accompanied by a decrease in TLC. The decrease is usually observed during the early stage of disease-secondary infection following viral diseases are usually accompanied by a leukocytosis. Overwhelming bacterial infections also result in leukopenia that is commonly accompanied by a diminution of mature neutrophils in the peripheral blood. Early stages of an infectious disease or of a localized severe infection where there is depletion of the pheripheral

blood of its leukocytes with concentration in the area of the infection resulting in a leukopenia until the bone marrow catches up with the production. Endotoxins from Gram-ve bacteria produce a significant leukopenia, large doses of endotoxin have severe effects on formed elements of the blood particularly leukocytes & thrombocytes both of which is decreased. Leukopenia is also associated with cachectic and debilitated states and old age that may be caused by lack of certain nutritional factors or by exhaustion of the marrow. In Vitamin B12 and folic acid deficiency there will be mild chronic leukopenia. Physical agents such as x-rays and radioactive substances produce a leukopenia by destroying the cellular elements of the bone marrow. Anaphylactic shock and early stages of a reaction to foreign protein may produce a leukopenia. Bone marrow abnormalities also produce leucopenia. Bone marrow hypoplasia which may be due to metabolic disorders, Ionizing radiation. Chemical agents, cause leucopenia Bone marrow dysplasia caused due to subleukemic or aleukemic Leukemia, Bone marrow displacement (myelophthisic) may cause leucopenia. Rickettsial diseases like ehrlichiosis and protozoan diseases like toxoplasmosis also cause leucopenia. Chemical agent may also produce leuckopenia those are some includes some of the antibiotics–(Chloramphenicol, penicilin streptomycin and oxytetracyline), Analgesics(phenacitin,,Antipyrine and aminopyrine), Antifungal–(griseofulvin), Antihistamines (pyribenzamine), Anticonvulsants (primidone), antithyroid (thiouracil), hemopoetic depressant (cytoxan, 6-mercaptopurine),cortisone products, sulfonamide, inorganic chemicals (lead Benzene, bismuth, mercury, Arsenic, Thallium and others (DDT, Barbiturates, chlorpromazine).

Interpretation for TEC

Decreased TEC occurs in anaemia, esp. in toxic, aplastic, haemolytic and chronic haemorrhagic anaemia.

Increased TEC occurs in polycythemia**,** haemoconcentration due to loss of fluid, diarrhoea. vomiting. excess sweating, severe burns, polyuria, shock.

Interpretation for PCV

In haematocrit a column of PCV shorter than normal indicates anaemia while one taller than normal indicates hemoconcentration. An anaemic animal with severe hemoconcentration due to loss of fluid from the body (diarrhoea, vomition) may have a PCV falling within normal limits. So while interpreting PCV, the water balance of the animal should be taken into consideration. Approximate leucocyte counts can be made from the buffy coat at the top of the packed erythrocyte layer in the wintrobe tube. The first millimetre equals to 10000 and each 0.1 mm thereafter equals 2000 leucocytes/microliter.(1mm equals 20000) If lower, it is leucocytopenia and if higher, it is leucocytosis. This approximation should never be used as a substitute for TLC since it is possible to be misled by a marked thrombocytosis or the variability in size.

Interpretation for ESR

Increased ESR in acute generalised infection, acute localized infection of serosal membranes like peritonitis, pleuritis, pericarditis. chronic localised infection like suppurative conditions, skin alterations like inflammation, hypothyroidism and hyperadrenocorticism, tissue injury, malignant tumors where considerable reaction, vascularity and tissue breakdown, pregnancy.

Specific conditions characterised by an increased ESR are dog-distemper, leptospirosis, pyometra, chronic interstitial nephritis, radiation injury, filariasis, acute and subacute bacterial endocarditis, myocarditis, myocardial degeneration, pneumonia, pleuritis, peritonitis, ICH, salmon diseases, hypothyroidism, hyper adrenocorticism, bone fracture, trypanosomiasis.

Blood smear

Examination of a smear of peripheral blood is one of the most informative laboratory procedures. It should be made within 15 min. after the collection, but the best smear are made immediately from fresh blood. Inspect the blood smear under low power to note distribution of cells & select a portion of the smear near the thin end where the erythrocytes do not overlap. Switch to the oil immersion objective for making the rest of the examination.

Erythrocytes should be inspected in regard to

i) Size – Variation(Anisocytosis)- Normocytic, Macrocytic or Microcytic)

ii) Shape – Variation(Poikilocytosis)-target cells,sperocytes, acanthocytes, leptocyte)
[Toxic disease may cause change in size or shape of RBC]

iii) Colour – Normochromic, Hyperchromic, hypochromic

iv) Erythrocytic inclusions(Polychromasia)- Howell-Jolleybodies,Basophilic stippling as in Lead poisoning, hinz bodies

v) Polychromatophilia, Nucleated erythrocytes, reticulocytes in brilliant crysyl blue stain or new methylene blue.[N.B- Reticulocytes are immature RBC & larger in size & non nucleated. Since this cell represents a more immature form of erythrocyte, its presence in periphery blood is of importance in assessing bone morrow response.

Bacteria & parasites : Parasites likeTryps, Anaplasma, Piroplasma, Micro filaria, and bacteria in septicemia.

***Thrombocytes or platelets* :-** Estimate the number present as normal, increased or decreased or none. [Four or more per oil immersion field is normal]. Note variation in size and morphology. Mostly seen in clusters but may occur singly or in small clumps. Giant forms are occasionally seen with a distinct cell membrane surrounding light colored cytoplasm and deep reddish purple staining granules. Greatly increased no. would suggest increased activity of bone marrow.

***Leukocytes* :- Interpretation of DC**

Neutrophilia in **Non – infectious** conditions like neoplasia, foreign body**,** trauma**,** sterile abscess**,** tissue manipulation uremia (toxemia)**,** stress and **Infectious** conditions like bacterial (acute infection), mycotic, protozoan or parasitic.

Neutropenia in overwhelming bacterial infection, viral infections, rickettsia, chronic infections, malignancy, deficiency of Vit-B-12 & Folic acid, per – acute bacterial infection, bone marrow depression

Eosinophilia in skin allergies Eosinophilic pneumonia, enteritis, parasitism, and leukemia

Eosinopenia in any stress (release of corticosteroids), administration of ACTH or corticoids

Basophilia in basophilic granulocytic leukemia. But it is rare.

Lymphocytosis in neutropenia may have relative lymphocytosis, Lymphocytic leukemia, recovery stage of certain infections, adrenocortical insufficiency, following vaccination, chronic infection, hyperthyroidism, lymphadenitis, blood parasites (Babesia, trypanosoma), excitement

Lymphocytopenia in some viral disease like canine distemper, ICH, parvo, pan leucopenia etc., stress – due to glucocorticoids, Injection of ACTH and hyperadrenocortcism, Ionizing radiation and mmunosuppressive drugs.

Monocytosis in chronic diseases, granulomatous reaction, fungal infection, T.B, brucellosis, protozoan infection, erysipelas, listeriosis, monocytic leukemia, acute stress, hyper adenocorticism, recovery / late phase of diseases.

Wet Blood Smear can be employed for microfilaria

Urine Examination

Interpretation of urine examination

When there will be pathophysiological change of the body there may be alteration in the quantity and quality of the urine excreted. Hence examination of urine helps in assessing the health of the body. A critical examination of urine is an important diagnostic procedure.

The urine must be collected in a clean container from the mid stream of the morning sample. Urine can be collected by catheterization, manual compression of urinary bladder. Urine analysis should be completed as quickly as possible after collection as chemical and cytological changes occur rapidly in urine. Bacteria multiply rapidly. If urine to be preserved then kept in refrigerator and added with the preservatives like toluene, thymol etc.4-6 drops of formaline(40%) can be added but gives false reaction for sugar. Thymol(0.1gm/100ml)may be used but false reaction for albumin. A pinch of camphor can preserve 30 ml of urine. In leptospire either directly examine the slides under dark ground illumination. In case of parasite in urine 2 drops of 40% formalin is added to 30-40 ml of urine and dispatch.

Interpretation of physical changes:

Volume: urine volume is dependant upon several physiological factors including water and fluid intake, environment, diet, activity of animals. Increases in urine volume (polyuria) may be present transiently owing to diuretic therapy or increased fluid intake and followimg parental administration of fluid or administration of corticosteroids. **Pathologic polyuria** associated with acute and chronic generalized nephritis, diabetes mellitus, insipidus, nephrogenic diabetes insipidus, diduretic phase of toxic nephrosis, primary renal glucosuria, pyometra. renal amyloidosis, pyelonephritis, compulsive polydypsia and some liver diseases. **Urine volume will decrease** (oliguria) with decreased fluid intake, high environmental temperature, hyper ventilation. Oliguria seen in dehydration, decreased BP, acute nephritis, prolonged fever, circulatory dysfunction.

Colour and Transparency: Normal urine is pale yellow and clear. Colour becomes red in hematuria amd hemoglobinuria, greenish yellow in jaundice, green when given methylene blue(urinary antiseptic)brown or coffee in hemoglobinuria, dark yellow in acute nephritis, fever, dehydration

etc. Turbid(thick) urine indicates pyogenic process in urinary tract and epithelial cells(protein). Pathologically cloudy urine may be observed when any of the following like leucocytes, erythrocytes, epithelial cells, bacteria. Transparency is tested by viewing against light in a test tube. In congenital porphyria, a faint pink. Pink colour also in medication of phenothiazine.

Specific gravity: Increased specific gravity seen in acute interstitial nephritis, cystitis, diabetes mellitus, dehydration, vomition, diarrhoea, fever, reduced fluid intake, hypovolumeic shock, burns. **Decreased** specific gravity seen in increased fluid intake, interstitial nephritis, advanced state of uremia, diabetes inspidus, hyperadrenocortisim, pyometra.

Odour: It is not diagnostic although the urine of males of feline and has an especially strong odour. An odour of ammonia may appear if urea is being converted to ammonia by bacterial action. Strong ammonia smell suggest cystitis due to proteus organism. Ketone bodies impart a sweetish and fruity odour and may be detected in urine and associated with pregnancy disease, acetonemia, diabetes mellitus. Rotten grapes like smell of urine suggest diabetes mellitus.

Foam:if shaken after collection, normal urine produes a white foam that is limited in quantity. if there is proteinuria excess foam produced and slowly disappear. if bile-foam is green,yellow,yellow brown, if hemoglobin-foam is red to brown.

Interpretation of chemical examination:

Reaction of urine PH: Acid urine or aciduria seen in normally carnivore's urine, nursing calves if diets with excess protein but **pathologically** seen in starvation, fever, acidosis, administration of acidic salt like NaCl, ammonium chloride,CaCl2, proteineuos diet. **Alkaline urine or alkalanurea** seen normally in carnivoress with veg diet.**,** but **pathologically seen** in cystitis**,** retention of urine**,** administration of alkaline salts like acetate, bicarbonate, citrate or nitrite of sodium or potassium, rapid adsorption of transudates.

Glucose(sugar): Glycosuria is seen in emotional states-fear, excitement, where there is sudden release of adrenaline which causes hyperglycemia with resultant glycosuria, heavy meal of carbohydrate, diabetes mellitus, after general anaesthesia, hyperthyroidism due to rapid absorption of glucose from the bowel, chronic pancreatitis, acute pancreatic necrosis, hyperpituitarism, overactivity of adrenal cortex, shock, intravenous glucose administration, chronic liver diseases.

Ketone Bodies(acetone): Ketonurea seen in diabetes mellitus, starvation, high fever, cachetic condition.

Protein: Proteinuria found in **physiological conditions** like excess proteinous diet intake, excess muscular excretion, emotional stress, in convulsion and **pathological conditions** like nephritis by increased permeability of the glomerular filter. In acute interstitial nephritis—protein with cast in urine. In chronic interstitial nephritis—slight protein with casts. In pyelonephritis –marked proteinuria with leucocytes & RBC. In nephrosis due to poisioning by phenol, arsenic, lead, phosphrous, mercury, sulphonamide,turpentine,ether,bismuth, salicylic acid -excess proteinuria. In amyloidosis, renal infraction & neoplasms—proteinurea. In renal congestion due to cardiac congestion, pressure on abdominal veins due to ascites, tumor—slight protenuria. In postrenal condition when protein

enters urine after it leaves the kidney tubules like cystitis, vaginal or preputial discharges,prostitis,pyelitis,uretheritis,ureteritis,urolithiasis,trauma with haemorrages –proteinurea observed.

Blood: Hematurea is differentiated from hemoglobinurea by centrifuging the urine. In hematuria sediment examined for presence of RBC and supernetent is colourless. In hemoglobinuria the fluid is red or coffee colour and erythrocytes are lacked**. Hematurea** seen in pyleonephritis, acute nephritis, ureteritis, cystitis, pyelitis, urolithiasis, passive congestion of kidney, infarction of kidney, neoplasm of kidney, bladder, or prostrate, abscess of kidney, during estrus, at postpartum in females, prostitis, severe infection in leptospirosis, Rubarth's disease, trauma to urethra during improper catheterization, toxic chemical agent-copper, phenol, sulfonamide, mercury, arsenic and thalium poisoning, sweet clover poisoning, shock-capillary hemorrhage, parasite- Dictyophyma renale,Dirofilaria,Capillaria plica. If blood found in last drops of urine then source is bladder, if urine is red throughout source of blood is kidney and if first portion of urine is red then source of blood is some urethral lesions. **Hemoglobinuria** found in leptospirosis, postparturient hemoglobinuria, babesiosis, photosensitization, bacillary hemoglobinuria-clostridium hemolyticum infection, Chemical hemolytic agents like copper,mercury and sulfonamides, severe burns, hemolytic disease of the new born, incompatible blood transfusion, drinking large volume of water, plant poisons

Myoglobinurea (azoturea) when urine contains myoglobin and absence of erythrocytes in the sediment. When muscle pigment myoglobin is found in urine, the condition is called as myoglobinurea. it is suggestive of severe destruction of muscle fibres. Myoglobinurea also occurs in electric shock and snake venum.

Calcium: normally very small amount present. Decreased in Bovine Hypocalcemia, tetany, hypothyroidism, osteomalacia. **Increased** in renal osteodystrophy,hyperthyroidism,hypervitaminosis.

Urobilinogen: Normally small amounts are excreted in urine. Decreased amount or absence of urine urobilinogen in obstruction of biliary passages, decreased RBC destruction, impaired intestinal absorption, diarrohea, nephritis and some times due to polyuria causing dilution. **Increased** urine urobilinogen seen in hepatitis, cirrhosis, hemolytic jaundice.

Bile:Presence of excess bilirubin in urine is bilirubinurea. Found in hepatocellular disease, Infectious canine hepatitis, cirrhosis, neoplasm, toxicity, obstruction of bile duct, jaundice, acute enteritis and intestinal obstruction.

Interpretation of microscopical examination:

For microscopical examination sediments of centrifuged urine always taken.

Epithelial cells- Squamous epithelial cells are laregest and having small round nucleus. Transitionalbepithelial cells are smaller,granular and having small round nucleus. Renal epithelial cells are smallest with a nucleus. **Pathologically** large number of renal epithelial cells found in acute interstitial nephritis. Transtitional epithelial cells found in cystitis and polynephritis.

Erythrocytes- Indicates hemorrhage in some part of genitourinary tract and in faulty catheterization.

Leucocytes (pus cells)- larger than erythrocytes and smaller than epithelial cells and granular cytoplasm. Increased leucocytes in urine is pyuria seen in nephritis, pyelonephritis, pyelitis, ureteritis, cystitis, vulvitis, vaginitis, balanitis

Casts- Hyaline cast indicates a mild form of renal irritation. These are composed of protein as homogenous, semitransparent, colourless, cylindrical structure having rounded ends. **Granular casts** indicates severe type or renal disease in tubular epithelium. Granular casts are hyaline casts contain granules. **Epithelial casts** derived from desquamated epithelial cells. It indicates desquamation of epithelial cells. **Waxy Casts** similar to hyaline being homogenous but appear more opaque than hyaline. it indicates advanced or severe nephritis. **Fatty casts** contain small droplets of fat as refractile bodies.It indicates deposits of fatty material in tubules. **Blood casts** are homogenous cylindrical masses having a deep yellow to orange colour. Erythrocytes within these casts degenerate. This indicates glomerulitis, haemorrhges. **Leucocyte casts** means presence of many pus cells. it indicates nephritis, pyelonephritis, kidney abscess etc. **Mucus thread** indicates presence of contamination from the genital secretion. **Parasites**:ova seen in urine like Dictyophyma ranale-giant kidney worm of dog, Capillaria plica-bladder worm of dog,cat,fox. **Crystals**:if acid urine-amorphous urates, uric acid, calcium oxalate and hippuric acid. If alkaline –triple phosphate, amorphous phosphate, calcium carbonate, ammonium urate.

Amorphous phosphate-Powder, Calcium oxalate-diamond/Envelope, Triple phosphate-Prism, Sulfa crystal-Long prism, Uric acid-Radiating needle are some of the morphological pictures. **Bacteria**-seen in cystitis, pyelonephritis, genital tract infection. **Yeast and fungi**-only contaminatants. **Fat** seen in diabetes mellitus, high fatty diet, obesity, hypothyroidism, contamination.

Biochemical examination:

Blood serum or plasma can be tested for various enzyme levels which are elevated and depressed in various disease conditions. The results can be correlated with diseases.

Parasitological Examination:

The presence of ova or larvae of endo and ecto parasites can be easily demonstrated by direct examination or examining centrifuged faecal samples, skin scrapings etc. Faecal examination requires centrifugation after mixing with water, saline etc. and digestion of skin scrapping in 10% KOH or NaOH.

Microbiological Examination:

Bacteriology: Smears prepared from clinical samples can be stained for identifying Gm+ve or Gm –ve bacteria, acid fast bacilli etc. Cultural examination and antibiotic sensitivity test can be utilized for identification of the bacteria and to determine the susceptibility of bacteria to antibiotics.

Virological methods: Some commonly used virological methods employed in disease diagnosis are virus isolation, HI, ELISA, immunofluorescence, virus neutrasilation, electron microscopy, molecular methods like PCR etc.

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DIAGNOSIS,CHEMOPROPHYLAXIS AND TREATMENT OF MANGE IN DOGS

M . R. Das
Associate Professor,Dept. of Clinical Medicine
College of Veterinary Science & Animal Husbandry
O.U.A.T,Bhubaneswar-751003

Canine demodectic mange (also known as red mange, follicular mange or puppy mange) is caused by *Demodex canis.* It is worldwide in distribution. The mite is a normal inhabitant of the canine skin. Under favourable condition, it causes severe infection with dermatological disorders with superimposed bacterial and fungal infection. The parasites are normal commensals on canine skin but are capable of producing generalised disease if mite numbers proliferate.

Pathogenesis and Transmissions:

The pathogenesis of generalised demodicosis due to D. canis is incompletely understood. It is believed that disease arises through an inability of the host to regulate mite numbers rather than an increase in the virulence of the mite itself. Whether or not Demodex causes harm to a dog depends on the animal's ability to keep the mite under control. It is not a disease of poorly kept or dirty kennels. It is generally a disease of young dogs that have inadequate or poorly developed immune systems or older dogs that are suffering from a depressed immune system mostly due to endoparasitic load. One hypothesis is that the disease results hereditary, specific T cell deficiency or in conjuction with other immunosupressive conditions. Two distinct age groups have been identified with the development of generalised demodicosis ie. juvenile-onset disease which occurs in dogs upto 18 months of age and adult- onset disease generally occurs older than 4 years of age with no previous history of disease. It may surprise that demodectic mites of various species live on the bodies of virtually every adult dog and most human beings, without causing any harm or irritation. The small(0.25mm) 'alligator-like' mites live inside of the hair follicles (i.e., the within the skin through which the hair shaft comes through),hence the name is termed as follicular mange. In humans, the mites usually are found in the skin, eyelids and the creases of the nose. The demodectic mite spends it's entire life on the dog's body. Eggs are laid by a pregnant female mite, hatch and then mature from larvae to nymphs to adults and the entire life cucle is believed to take 20-35 days.

Clinical Signs:

Individuals that are sensitive to the mange mites may develop a few isolated lesions or they may have generalized mange, in which case, there are more than five lesions involving the

entire body or region of the body and most lesions in either form develop after four months of age. The lesions and signs usually involve hair loss,crusty, red skin with a greasy or moist appearance. Though the mites prefer to live in the hair follicle, hair loss is the noted sign which begins around the muzzle, eyes and other areas of head. The disease may be present as a squamous form in which there is little erythema or exudative discharge, patchy alopecia with excessive scale formation without bacterial infection(folliculitis and furunculitis).

The second form of the disease is postural demodicosis which is characterized by extensive bacterial infection causing folliculitis and furunculosis. The skin is exudative with plaque formation and extensive crust and scale accumulation.Peripheral lymphadenopathy may be marked and less severely affected areas particularly at the edge of purulent lesions may show the hyperpigmented skin. Pustural demodicosis may be painful and if, extensive, may lead to septic changes causing anorexia, lethargy and depression.

Diagnosis:

In most instances the diagnosis of demodicosis is made easily by a deep skin scrapping where the skin is squeezed and scraped until capillary oozing is seen and the selected site should be from the edge of the expanding lesion. Applying paraffin oil to the skin helps for better collection of epithelial debris from the skin. The resultant material should be examined through direct method or sedimentation method.

(a)Direct method: Collected skin scrapings are transferred to clean glass slides and a few drops of 10% potassium hydroxide (KOH) solution are added to it. The slide is flame heated till evaporation is starting. Then after cooling the slide is examined under low power microscope after putting a cover slip.

(b)Sedimentation method: The skin scrapings are placed in a clean specimen test tube containing 2ml of sodium hydroxide (10%NaOH)sol. and kept at room temp. for 8hrs.The suspension is flame boiled for 5minutes and after cooling, the suspension is transferred into a centrifuge test tube and centrifuged for 3minutes at 3000rpm.The supernatant is discarded and with the help of a pipette, one drop from sediment fluid is taken and placed on a clean glass slide and examined under low power microscope after putting a cover slip.

Treatment:

Identification and elimination of concurrent pyoderma is critical to successful treatment of demodicosis.The treatment of demodicosis is usually accompanied with lotions, dips and shampoos. Fortunately, 90% of demodectic mange cases are localized, in which only a few small areas are involved and can be treated topically. Bathing periodically with a benzoyl peroxide shampoo and feeding a high quality diet and a multivitamin with fatty acid may also help some dogs. Most of these localized lesions will heal on their own and do not require overly aggressive treatment.

If a dog develops generalized demodicosis, more aggressive treatment is usually required. Studies show that between 30% and 50% of dogs that develop the generalized form will recover

on their own without treatment, but treatment is still always recommended for generalized form. The treatment of choice continues to be ivermectin orally @600 microgm/kg/day for 18weeks which cured 87% after 10 months duration along with Amitraz dips applied twice a week which require 4-14 dips. Amitraz should be applied with care. Humans should always wear rubber gloves when applying it to their dogs, and it should be applied in an area with adequate ventilation. It is recommended that longhaired dogs be clipped short, so that the dip can make good contact with the skin. Prior to dipping, the dog should be bathed with a benxoyl peroxide shampoo for flushing purpose which help remove oil and cellular debris. After the first three or four dips, a skin scrapping should be performed to determine if the mites have been eliminated. Dips should continue until there have been no mites found on the skin scrapings taken after two successive treatments. Toy breeds in particular are sensitive to amitraz.So,half strength dips should be used on these sensitive animals. Ivermectin should not be given to Collies and Old English Sheepdogs due to idiosyncratic reactions. Another drug, Milbemycin oxime has also been used successfully to treat demodicosis @2mg/kg/day for a period of 60-180 days which gives 90% cure.

Dogs those have generalized demodicosis, often have underlying skin infections. So antibiotics are often given for the first several weeks of treatment. In addition, good multivitamin containing fatty acids (omega 3&6) should be supplemented with diets though demodex flourishes on dogs with a suppressed immune system. It is wise to check for underlying causes of immune system disease, particularly if the animal is older developing the condition. It is advisable to the owners that individuals have a history of demodectic mange, their parents and siblings should not be bred

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