

AN INTRODUCTION TO VETERINARY ACUPUNCTURE

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HISTORY

The origin of veterinary acupuncture can be traced to the primitive society (2200-3000 BC) in China (Yu, 1984). Acupuncture is still widely used in both animals and people in China. In the United States, acupuncture is now receiving greater interest and more acceptance.

BASIS

Acupuncture may be defined as the stimulation of a specific point (acupuncture point) on the body with a specific method, resulting in a therapeutic or homeostatic effect. Thus, there are 3 major components of acupuncture process: (1) acupuncture point (acupoint); (2) stimulating methods; (3) acupuncture-inducing physiological effects.

Recent researches show that most of acupuncture points are located in the areas on the skin of decreased electrical resistance or increased electrical conductivity. A point finder, acupoint detector, or AC dermometer is based on this assumption. The morphologic basis of this bioelectrical phenomenon has been attributed to neural or vascular elements in the dermis or hypodermis (Hwang and Egerbacher, 1994).

There are 173 major acupuncture points in horses (Xie, 1994) and 361 points in peoples. Acupuncture points correspond to 4 known neural structures (Gunn, 1977). Type I acupoints, which make up 67% of all acupoints, are considered motor points. The motor point is the point in a muscle which, when electrical stimulation is applied, will produce a maximal contraction with minimal intensity of stimulation. Motor points are located near the point where the nerve enters the muscle. For instance, SI-9 is located at the junction of musculi deltoideus and triceps brachii and supplied by axillary and radial nerves. Type II points are located on the superficial nerves in the sagittal plane on the midline dorsally and ventrally. For instance, Bai-hui (GV-20b) lies in the depression between the spinous processes of the last lumbar and the first sacral vertebrae in the dorsal midline and is supplied by the dorsal branch of the last lumbar nerve. Type III points are located at high density loci of superficial nerves and nerve plexuses. For example, GB-34 is located at the point where the common peroneal nerve divides into

the deep and superficial branches. Type IV points are located at the muscle tendon junctions where the Golgi tendon organ is located. Recently, histological studies have revealed that an acupuncture point is located at the high density of small microtubules including free nerve endings, arterioles, lymphatic vessels and mast cells.

According to Traditional Chinese Medicine (TCM), the most commonly acupuncture points are located on the pathway of meridians. The meridians are the TCM energy flow system. There are 14 major meridian systems: Lung (LU) meridian, Heart (HT) meridian, Pericardium (PC) meridian, Spleen (SP) meridian, Kidney (KI) meridian, Liver (LV) meridian, Large Intestine(LI) meridian, Small Intestine(SI) meridian, Triple Heater (TH) meridian, Stomach (ST) meridian, Bladder (BL) meridian, Gallbladder(GB) meridian, Governing Vessels (GV) meridian and Conception Vessels (CV) meridian. Chi is an important concept in TCM. Chi, or a whole body energy flow, moves throughout the body in the meridians. The energy cannot be seen. However, a number of recent scientific studies have been done that demonstrate some of the physiological events that take place during acupuncture. Acupuncture points occur along the 14 major meridians and can be identified electrically in the same locations the ancient Chinese documented in their acupoint chart 1,000 years ago.

MECHANISM

1. Inhibition or reduction of transduction and transmission

The perception of pain involves 4 phases: transduction, transmission, modulation and perception. Acupuncture analgesic effect may be related to one or more of these 4 pain processing phases.

One hypothesis proposed that stimulation of specific acupoints can block pain sensations before they reach the central nervous system (Snader, 1993). Numerous studies have shown that the lightly myelinated A δ fiber is consistently considered the most dominant in mediating acupuncture, followed by unmyelinated cutaneous C fibers (Kendall, 1989). A δ and C fibers are also considered nociceptors (nociceptive afferents), or noxious receptors. The repetitive stimulating pain depends on A δ fibers, which can be blocked by pressure. The intense prolonged pain depends on C fibers, which can be blocked by local anesthetic. The afferent nociceptive A δ and C fibers project to lamina I of the spinal cord. The acupuncture signal, on the other hand, is transmitted to lamina I of spinal cord by A δ and C fibers at the same time when acupuncture is used for a pain-producing disease. A segmental spinal inhibition of nociceptive inputs based on the gate control theory results in an immediate, short-lasting, segmental and non-opioid analgesia (Ernst and Lee, 1987).

Yonehara et al (1992) found that the tooth pulp stimulation (TPS) evoked an increase in release of immunoreactive substance P (iSP) in trigeminal nucleus caudalis (Vc-I,II) in rabbits. This increase was inhibited by EA at ST-36 in 9 out of 13 rabbits. The potentials evoked by TPS consisted of fast and slow components. The slow potential, reflecting

the excitation of A δ fibers, was significantly inhibited by CP-96,345, an SP antagonist. The slow potential evoked by TPS was also inhibited by EA stimulation in 8 out of 11 animals. The trigeminal nucleus is considered to be a relay for impulse transmission from the tooth pulp afferents, including A δ and C fibers, to the tract cells of the ascending pathways to higher levels of the brain. SP might be involved as a primary afferent transmitter in the transmission of dental pain messages (Yonehara et al, 1992). Thus, Acupuncture may inhibit SP release from the primary sensory afferent fibers and relay neurons and consequently block onward transmission of the pain impulse to the high levels of the central nerve system.

Acupuncture stimulation at ST-36 induced a decrease in sympathetic renal nerve activity (RNA) and mean arterial blood pressure (MAP) in rats under the deep anesthetic condition (Ohsawa et al, 1995). But, acupuncture stimulation at the skin of acupoint alone did not induced any change of MAP and RNA. This suggested that anatomic structure of acupoint consists of the deeper tissues rather than skin.

2. Neuroendocrine response modulating pain

Endogenous morphine-like substances: The endogenous opioid peptides were discovered by Hughes in 1975 (Hughes, 1975). Since the acupuncture effect can be reversed by naloxone, a specific opiate antagonist (Mayer et al, 1977), the endorphin system has been generally considered a possible pathway for acupuncture analgesia. Cepeda and Carr (1993) reviewed the neuroendocrine response to pain. ACTH and β -endorphin are found to be derived from the same precursor molecule, pro-opiomelanocortin (POMC). POMC undergoes a series of ordered proteolytic cleavages and modifications in the corticotrophin of the anterior pituitary to yield ACTH and β -lipotropin (β -LPH). β -LPH, in turn, gives rise to daughter molecules, including β -endorphin (Cepeda and Carr, 1993). It appears that at least 3 separate endogenous opioid neuronal systems are present in the brain: an enkephalin family with components similar to those found in the adrenal; a β -endorphin family, and a dynorphin family (Watson et al, 1982). β -endorphin might produce its analgesic effects by suppressing substance P (SP) release in the spinal cord. Methionine enkephalin blocks release of SP, which may be related to what closes the gate in the spinal cord pain transmission system (Lumb and Jones, 1984).

Acupuncture was found to induce an increase in endogenous morphine-like substances (EMLS) in the cerebrospinal fluid (CSF) drained from the cerebroventricle in patients suffering from brain tumors (Zhang et al, 1980), and in serum EMLS in patients with soft tissue pain, acute appendicitis and peri-arthritis (Xi et al, 1983). In a study using the lip twitch, a form of acupuncture known as acupressure, β -endorphin release was investigated in the horses. The use of lip twitch resulted in a doubling of plasma β -endorphin levels in as little as 5 minutes after application of the twitch (McCarthy et al, 1993). There was a linear correlation between increases in β -endorphin and the body's ability to tolerate pain (Hamra et al, 1993). EA stimulation induced large amount of β -endorphin released into the peripheral blood (Malizia et al, 1979). Similar to micro-

iontophoretic opiates, morphine and etorphine, EA produced a strong inhibition on the spontaneous activity of the periaqueductal gray (PAG) neurones, and this inhibition could be reversed by iontophoretic naloxone (He and Dong, 1983). Pain threshold (PT) was increased significantly in rabbits after 10-minute acupuncture stimulation at LI-4 and TB-5. At the same time, acupuncture produced a significant increase in the release of leucine-enkephalin and β -endorphin in the preoptic area (Wu et al, 1995).

Injection of protein-A purified antibodies against Met-enkephalin or β -endorphin into the periaqueductal gray (PAG) was shown to decrease the analgesic effect of EA in rabbits (Han et al, 1984). Antibodies of Met-enkephalin were found to be active at the spinal level whereas antibodies against β -endorphin were without effect, which is in agreement with a rich enkephalinergic innervation and absence of β -endorphin-containing fibers in the spinal cord (Han et al, 1984).

Both acupuncture and morphine analgesic effects increased progressively during ontogenetic development in rabbits (Zhou et al, 1982). Acupuncture analgesia effect was not found in 2-, 7- and 14-day-old rabbits. However, significant acupuncture analgesia was found in 21-day-old rabbits and acupuncture analgesia in 28-day-old rabbits was almost the same as that in the adults. Morphine analgesia was not found in 2- and 7-day-old rabbits, but was found in 14- and 28-day-old rabbits. Both acupuncture analgesia and morphine analgesia was reversed by administration of naloxone.

Plasma levels of β -endorphin, β -lipotropin and ACTH increased significantly in the volunteers after EA stimulation at the acupoints St-36, LI-4 and P-7, while conventional needles at the same acupoints without electric stimulation did not change these parameters (Nappi et al, 1982). This result coincided with the clinical findings in that EA was more effective than conventional acupuncture (Liang, 1982).

D-phenylalanine (DPA), an enkephalinase inhibitor, is known to block the activity of carboxypeptidase, an enzyme which degrades enkephalins. DPA was found to potentiate the pain relief induced by acupuncture in animals and humans and produce naloxone-reversible analgesia in mice (Ehrenpreis, 1985). Kitade et al(1988) found DPA a potentiated acupuncture-induced increase in PT in healthy volunteers. DPA increased acupuncture analgesia results by 26% in 30 patients with chronic low back pain and by 35% in 18 patients with tooth extraction (Kitade et al, 1990). A pressor blood pressure response was elicited by strong electric shock stimulation at the front paw in rabbits. EA inhibited the pressor response and this inhibition was readily blocked by naloxone (Wang et al, 1994).

ACTH and cortisol: Stimulation of acupoints triggers the pituitary gland to release adrenocorticotrophic hormone (ACTH), which stimulates the adrenal glands to release cortisol into the blood stream. Serum cortisol is a natural steroid anti-inflammatory agent which acts to reduce inflammation and pain in affected areas (Smith, 1992). EA stimulation induced large amounts of ACTH to be released into the peripheral blood (Malizia et al, 1979).

EA produced a 33% increase in PT in rats, and after dexamethasone injection, the acupuncture-induced increase in PT dropped by 11% (Liu et al, 1988). Thus, dexamethasone administration reduced acupuncture analgesic effect. Dexamethasone could block acupuncture analgesia by inhibiting release of β -endorphin from the pituitary. Some evidences demonstrated that dexamethasone inhibits secretion of both β -endorphin and ACTH from the pituitary. Takeshige (1985) found out that acupuncture analgesia was naloxone reversible, non-acupoint analgesia was dexamethasone reversible, and stress-induced analgesia by low frequency electrical shock was both naloxone and dexamethasone reversible. All 3 types of analgesia were related to the descending pain inhibitory system, a common pathway for analgesia. This pathway was found in the arcuate nucleus (dopaminergic), ventromedian nucleus of the hypothalamus, raphe nucleus (serotonergic), reticular gigantocellular nucleus (noradrenergic) and reticular paragigantocellular nucleus.

Substance P (SP): SP seems to be important in mediating effects of EA. Injection of antibodies against SP into the PAG caused decrease of the acupuncture analgesia whereas intrathecal administration of Fab-fragment SP antibodies caused a marked potentiation (Han et al, 1984). However, repeated EA at UB-11 and UB-54 significantly increased SP, neuropeptide Y and neurokinin A in the hippocampus and neuropeptide Y in the occipital cortex in rats (Bucinskaite et al, 1994).

Other endocrine responses: PT was increased significantly in rabbits after 10-minute acupuncture stimulation at LI-4 and TB-5. At the same time, acupuncture produced a significant decrease in release of noradrenaline in preoptic area (Wu et al, 1995).

Acupuncture stimulation was found to increase the blood concentration of free tryptophan, the serotonin precursor, in healthy volunteers subjected to ketamine (Costa et al, 1982). Serotonergic neurons could be involved in analgesia by acupuncture. These serotonergic neurons consists of the so-called @negative feedback-loop system@ , which could activate a descending system with inhibitory effects on spinal cord pain-transmitting neurons, resulting in analgesia (Scherder and Bouma, 1993).

Takeshige and Sato (1996) used the reduction of twitch height of tetanized gastrocnemius in guinea pigs as a model of pain model. The recovery of twitch height was considered as pain relief. Muscle pain relief might be induced by recovery of circulation due to the enhanced release of acetylcholine as a result of activation of the cholinergic vasodilator nerve endings innervated to the muscle artery. Needling at the tetanized gastrocnemius muscle facilitated recovery from the reduced twitch height due to tetanic stimulation. This needling recovery effect was abolished by intravenous injection of atropine. Although a simple cut of the sciatic nerve innervated to the gastrocnemius muscle did not influence the needling effect, denervation 2 weeks after the nerve cut, abolished the needling effect. The needling effect was also abolished by capsaicin, which depletes SP and the calcitonin-gene related peptide (CGRP). Intraarterila injection of either SP or CGRP recovered the reduced twitch height. These

results indicate that needling to the muscle most likely stimulates many kinds of the sensory nerve endings such as CGRP containing nerve endings. These CGRP containing nerve endings might innervate and stimulate the cholinergic nerve endings to accelerate the release of acetylcholine and dilate the artery in the muscle by axon reflex, since denervation and atropine abolished this needling recovery effect (Takeshige and Sato, 1996).

A main pathway of acupuncture analgesia (AA) is via the release of endogenous opioid peptides. The exogenous opiate has a potentiating effect on AA, while some peptides, including cholecystokinin (CCK), angiotensin II and orphanin FQ (OFQ) have anti-AA effect. OFQ is a newly discovered 17-amino-acid peptide, which is sometime called Anociceptin® (Zhu et al, 1996). Intracerebroventricular or intrathecal administration of OFQ induced hyperalgesia in rat tail-flick model and showed a dose-dependent antagonizing effect against AA. OFQ anti-AA effect was abolished by Intracerebroventricular administration of antisense oligonucleotide (ASO), which could block the synthesis of OFQ receptor. ASO was also found to enhance AA (Zhu et al, 1996).

CLINICAL APPLICATION

According to Traditional Chinese Medicine (TCM), acupuncture points on each meridian (Channel) communicates with a specific organ and reflects the conditions of that organ. When an organ is afflicted with pathological charges, the related acupuncture points may exhibit tender or other signs. For example, Zusanli (St-36) may become tender when a person has stomach problem. When the acupuncture points are stimulated by needles(or moxa, or massage), Chi (flow of energy) will be activated and move to the "sick place", resulting in therapeutic effect. Therefore, acupuncture is used both diagnostically and therapeutically in equine clinic.

Clinical Indications

***Musculoskeletal and Neurologic Conditions**

- Cervical stiffness and wobbler's syndrome
- Seizure
- Hip dysplasia
- Arthritis, degenerative joint diseases
- Intervertebral disc diseases
- Tendon or ligaments problems
- Facial paralysis, laryngeal hemiplegia Laminitis, navicular disease

***Oncology and tumor**

***Skin Problems**

***Gastrointestinal Disorders**

Diarrhea, inflammatory bowel diseases (IBD)
 Constipation, megacolon, abdominal pain or colic
 Vomit, gastric ulceration, stomach problems

***Reproductive Disorders**

Anestrus & infertility in mares
 Infertility in stallion
 Infertility in dogs

***Respiratory Disorders (herbal medicine)**

Cough & cold
 Pneumonia
 Heaves, COPD, asthma
 Exercise-induced pulmonary hemorrhage
 Non-sweating

Clinical studies: a review

Table 1 Acupuncture for treatment of back pain

Acupuncture	Clinical Results			Reference
	Total cases	Improved	failure	
Conventional needling	15	13	2	Martin & Klide, 1991
Aquapuncture using vitamin B1 etc.	155	139	16	Martin & Klide, 1987a
	50*	49	1	Lu, 1983
	50*	50	0	Tang, 1983
	8	7	1	Gansu Institute, 1976a
Pneumo-acupuncture	12	11	1	Gansu Institute, 1976b
Electro-acupuncture	14	14	0	Pei, 1981
Fire-needling	51*	49	2	Zhao, 1982
Vinegar-Liquor hot Moxibustion	45*	45	0	Wang, 1992
	5*	5	0	Fu, 1992
Laser	15	11	4	Martin & Klide, 1987b
Total number of cases	420	393 (94%)	27(6%)	

*: Mixed animals

Table 2 Acupuncture for treatment of paralysis and paresis

Location of paralysis	Type of acupuncture	Clinical results (# of case)			Reference
		Improved	Failure	Total	
Radial nerve	EA**+AquA**	4	1	5	Wang & Hu, 1983
	AquA	5	0	5	Wang, 1991a
Suprascapular	PneumoA***	16	1	17	Liang, 1980
Hindlimb	EA	1	0	1	Dan et al, 1995
Glossopharyngeal	EA	2	0	2	Shi, 1984
Bladder	EA	5	3	8	Sichuan-shehong 1976
Facial nerve	EA	9	0	9	Guo, 1984
	EA + AquA	4	1	5	Wang&Hu, 1983
	AquA	3	0	3	Bai, 1987
	A-shi + Herb	9	0	9	Wang, 1991a
	Embedding	1	0	1	Ma, 1981
Total numbers of cases		59 (91%)	6 (9%)	65	

* EA: electro-acupuncture

**AquA: aquapuncture

PneumoA: pneumo-acupuncture

Table 3 Acupuncture for treatment of lameness due to chronic arthritis or soft tissue injuries

Type of Rheumatism	Type of acupuncture	Clinical results (# of case)				Reference
		Sound	Improved	Failure	Total	
Four limbs ⊥	EA + CA*	13	11	1	25	Liang, 1982
	EA	174	21	3	198	Li, 1993
Hindlimb ⊥	VLHM** +AP***	0	122	0	122	Meng et al, 1993
Hindlimb in horses	Warm-needling	0	8	0	8	Tao & Wang, 1984
	AquA	0	6	0	6	He et al, 1982

General rheumatism in horses	CA + SS****	9	3	2	14	Liang, 1984
	CMM*****	0	19	3	22	Ji & Du, 1990
In cattle	AquA	0	20	1	21	Tan, 1984
Limb&neck	VLHM +CA	0	19	2	21	Zhu, 1987
Cervical	EA	23	0	4	27	Zhang, 1980
Rheumatism ⊥	EA	143	0	10	153	Sichuan-shehong, 1976
Total numbers of cases		362 (59%)	229 (37%)	26 (4%)	617	

⊥: Mixed animals * CA: Conventional acupuncture ** VLHM: vinegar-liquor hot moxibustion
 *** AP: acupressure ****SS: sodium salicylate *****CMM: Chinese medicine moxibustion

Table 4 Acupuncture for treatment of lameness due to acute injuries

Point contusion	Type of acupuncture	Clinical results (# of case)				Reference
		Sound	Improved	Failure	Total	
Shoulder or hip/stifle	He-Ne laser	0	36	2	38	Yang, 1984
	HA*+Herbs	34	0	1	35	Guo & Li, 1983
	EA	6	0	0	6	Jiangxi-xingzi, 1976
Shoulder	CA+massage	1	0	0	1	Wang, 1986
Hip+stifle	HA		7	0	7	Jing et al, 1988
Carpus	Laser+AquA	1	0	0	1	Xie & Wang, 1991
stifle + metacarpus	Laser	4	0	0	4	Zhao et al, 1983
Fetlock	HA	0	106	0	106	Li, 1986
	CA+Herbs	6	0	0	6	Liu, 1991
	HA	0	63	0	63	Zhang, 1988b
Forelimb	AquA+HA	83	2	0	85	Sun & Gao, 1989

	AquaA	0	61	1	62	Chen, 1989
Forelimb or hindlimb	AquaA+Herb	22	0	1	23	Ming & Gao, 1989
	AquaA	37	0	10	47	Bai et al, 1989
	AquaA+Herbs	136	15	10	161	Liu et al, 1990
	EA	2	0	1	3	Zhang, 1980
Total numbers of cases		332 (51%)	290 (45%)	26 (4%)	648	

* HA: hemo-acupuncture

**CM: conventional medicine .5%prednisolone and 2% procaine

Table 5 Acupuncture for treatment of lameness due to muscular atrophy

Location of muscular atrophy	Type of acupuncture	Clinical results (# of case)				Reference
		Sound	Improved	Failure	Total	
Shoulder in horse	PneumoA	46	0	4	50	Su, 1982
	EA+PneumoA	1	0	0	1	Xie & Wang, 1991
	Cupping	0	1	0	1	Zhang, 1988a
Shoulder in cattle	PneumoA	7	0	1	8	Zhang, 1981
	PneumoA	17	0	5	22	Chen, 1984
Hip in horse	EA+PneumoA	1	0	0	1	Zhang, 1986b
Total numbers of cases		72 (87%)	1 (1%)	10 (12%)	83	

Table 6 Acupuncture for treatment of tendinitis and laminitis in horses

Type of lameness	Type of acupuncture	Clinical results (# of case)				Reference
		Sound	Improved	Failure	Total	

Tendinitis	Soft-burning	1	0	0	1	Xie & Wang, 1991
	Magnetic	2	0	0	2	Bai et al, 1982
Laminitis	HA	39	0	6	45	Wang, 1990
	EA, HA+AquA	0	0	3	3	Xie (unpublished)
Total numbers of cases		42 (82%)	0	9 (8%)	51	

Table 7 Acupuncture for treatment of colic in horses

Type of acupuncture	Clinical results (# of case)			Reference
	Clinically relief	Failure	Total	
EA	89	11	100	Zhu, 1979
HA+/orJiang-ya	284	22	306	China Agriculture Academy Vet Inst, 1960
HA +/or Jiang-ya	878	65	943	Lu, 1973
EA	16	5	21	Gao, 1987
Fire-needling	27	8	35	Zhang, 1989
EA	8	0	8	Zhou, 1992
HA + EA	285	0	285	Sun, 1986
HA +/or Jiang-ya	51	6	57	Yang, 1983
Total numbers of cases	1638 (93%)	117 (7%)	1755	

Reference

- Bucinskaite, V.; T. Lundeberg; C. Stenfors; A. Ekblom; L. Dahlin and E. Theodorsson. 1994. Effects of electro-acupuncture and physical exercise on regional concentrations of neuropeptides in rat brain. *Brain Research*. 666:128-132.
- Cepeda, M.S. and D.B. Carr. 1993. The Neuroendocrine response to postoperative pain. In: Ferrante and VadeBoncourer (Ed). *Postoperative Pain Management*. P79-123. Churchill Livingstone.
- Costa, C.; F. Ceccherelli; f. Ambrosio; P. Baron; A. De Antoni; S. Vanzan; G. Allegri and G. Manani. 1982. The influence of acupuncture on blood serum levels of tryptophan in healthy volunteers subjected to ketamine anesthesia. *Acupuncture & Electro-Therapeutics Res*. 7: 123-132.
- Ehrenpreis, S. 1985. Pharmacology of enkephalinase inhibitors: animal and human studies. *Acupuncture & Electro-Therapeutics Res*. 10: 203-208.
- Ernst, M. and M.H.M. Lee. 1987. Influence of naloxone on electro-acupuncture analgesia using an experimental dental pain test, review of possible mechanisms of actions. *Acupuncture & Electro-Therapeutics Res*. 12:5-22.
- Guidelines for Alternative and Complementary Veterinary Medicine. 1996. *JAVMA*, Vol 209, No. 6, 1027-1028.
- Gunn CC. 1977. Type IV acupuncture points. *Am J Acupuncture*. 5:51-52.
- Hamra J.G.; S.G. Kamerling; K.J. Wolfsheimer and C.A. Bagwell. 1993. Diurnal variation in plasma ir-beta-endorphin levels and experimental pain thresholds in the horses. *Life Sciences*. 53(2):121-129.
- Han, J.S.; G.X. Xie; Z.F. Zhou; R. Folkesson and L. Terenius. 1984. Acupuncture mechanisms in rabbits studied with microinjection of antibodies against β -endorphin, enkephalin and substance P. *Neuropharmacology*. 23(1): 1-5.
- He, L.F. and W.Q. Dong. 1983. Activity of opioid peptidergic system in acupuncture analgesia. *Acupuncture & Electro-Therapeutics Res*. 8: 257-266.
- Hughes, J. 1975. Isolation of an endogenous compound from the brain with pharmacological properties similar to morphine. *Brain Res*. 88; 295-308.
- Hwang, Y.C. and M. Egerbacher. 1994. Anatomy and classification of acupoints. In: AM Schoen (Ed). *Veterinary Acupuncture: Ancient Art to Modern Medicine*. P19-31. American Veterinary Publications.

- Kendall, D.E. 1989. A scientific model for acupuncture: part I. *American Journal of Acupuncture*. 17(3): 251-268.
- Kitade, T.; Y. Odahara; T. Ikeuchi and T. Sakai. 1990. Studies on the enhanced effect of acupuncture analgesia and acupuncture anesthesia by D-phenylalanine (2nd report)-schedule of administration and clinical effects in low back pain and tooth extraction. *Acupuncture & Electro-Therapeutics Res.* 15: 121-135.
- Liang, H.J. 1982. Effect of acupuncture treatment on rheumatic pain (bi-syndrome) in animals. *Chinese Journal of Traditional Veterinary Science*. (1): 36-37. (In Chinese)
- Liu, J.Z.; Y.H. Huang and P.J. Hand. 1988. Effects of dexamethasone on electro-acupuncture analgesia and central nervous system metabolism. *Acupuncture & Electro-Therapeutics Res.* 13: 9-23.
- Lumb, W.V. and E.W. Jones. 1984. *Veterinary Anesthesia*. P 43-73. Lea & Febiger.
- Malizia, E.; G. Andreucci; D. Paolucci; F. Crescenzzi; A. Fabbri and F. Fraioli. 1979. Electro-acupuncture and peripheral β -endorphin and ACTH levels. *The Lancet*, 2: 535-536.
- Mayer, D.J.; D.D. Price and A. Rafii. 1977. Antagonism of acupuncture analgesia in man by the narcotic antagonist Naloxone. *Brain Research*. 121: 368-372.
- McCarthy, R.N.; L.B. Jeffcott and I.J. Clarke. 1993. Preliminary Studies on the use of plasma B-endorphin in horses as an indicator of stress and Pain. *Journal of Equine Veterinary Science*, 13(4): 216-219, 1993.
- Nappi, G.; F. Facchinetti; G. Legnante; D. Parrini; F. Petraglia; F. Savoldi and A.R. Genazzani. 1982. Different releasing effects of tradition manual acupuncture and electro-acupuncture on proopiocortin-related peptides. *Acupuncture & Electro-Therapeutics Res.* 7: 93-103.
- Ohsawa H.; K. Okada; K. Nishijo and Y. Sato. 1995. Neural mechanism of depressor responses of arterial pressure elicited by acupuncture-like stimulation to a hindlimb in anesthetized rats. *Journal of Autonomic Nervous System*. 51: 27-35.
- Scherder, E.J.A. and A. Bouma. 1993. Possible role of the nucleus raphe dorsalis in analgesia by peripheral stimulation: theoretical considerations. *Acupuncture and Electro-Therapeutics Res.* 18: 195-205.
- Smith, F.W. 1992. Neurophysiologic basis of acupuncture. *Problems in Veterinary Medicine*, 4(1): 34-52.

Snader, M.L. 1993. *Healing Your Horses: Alternative Therapies*. Howell Book House. MacMillan Publishing Company, 866 Third Ave., New York, NY 10022.

Takehige, C. 1985. Differentiation between acupuncture and non-acupoints by association with analgesia inhibitory system. *Acupuncture Electro-Therapeutics Res.* 10: 195-203.

Takehige, C. and M. Sato. 1996. Comparisons of pain relief mechanisms between needling to the muscle, static magnetic field, external qigong and needling to the acupoint. *Acupuncture Electro-Therapeutics Res.* 21 (2): 119-131.

Wang, M.Z.; W.F. Xu and L.F. He. 1994. Mu opiate receptor antagonist blocks electro-acupuncture inhibition on noxious blood pressure response in rabbits. *Acupuncture & Electro-Therapeutics Res.* 19: 3-9, 1994.

Wu, G.C.; J.M. Zhu and X.D. Cao. 1995. Involvement of opioid peptides of the preoptic area during electro-acupuncture analgesia. *Acupuncture & Electro-Therapeutics Res.* 20: 1-6.

Xi, Z.F. and Q.S. Li. 1983. Changes in serum levels of morphine-like substances during acupuncture therapy on patients with pain and its relationship to the curative effects. *Shanghai Journal of Acupuncture & Moxibustion.* 1:12. (In Chinese).

Xie, H. 1994. *Traditional Chinese Veterinary Medicine*. Beijing Agricultural University Press.

Yonehara, N.; T. Sawada; H. Matsuura and R. Inoki. 1992. Influence of electro-acupuncture on the release of substance P and the potential evoked by tooth pulp stimulation in the trigeminal nucleus caudalis of the rabbit. *Neuroscience Letters.* 142: 53-56.

Yu, C. 1984. *Chinese Veterinary Acupuncture*. China Agricultural Publishing House. (In Chinese)

Zhang, A.Z; X.P. Pan; S.F. Xu; J.S. Cheng and W.Y. Mo. 1980. Endorphins and acupuncture analgesia. *Chinese Medical Journal.* 93:673. (In Chinese)

Zhou, G.Z; M.P. Chen and S.F. Xu. 1982. The relationship between the ontogenetic development of opiate receptors and acupuncture analgesia in rabbits. *Acupuncture & Electro-Therapeutics Res.* 7: 7-16.

Zhu, C.B.; S.F. Xu; X.D. Cao and G.C. Wu. 1996. Antagonistic action of orphanin FQ on acupuncture analgesia in rat brain. *Acupuncture & Electro-Therapeutics Res.* 21: 199-205.

CORAL SNAKE ENVENOMATION IN THE DOG

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SNAKE CHARACTERISTICS

The coral snake belongs to the family Elapidae, which is also represented by the cobras, mambas, kraits and tiger snakes. The venom of the coral snake is the most toxic per milligram of dried weight of that of any snake in the U.S.

Two genera of coral snakes are found in this country. *Micrurus* (including the eastern and Texas varieties) and *Micruroides* (the Sonoran or Arizona variety), the latter found only in southeastern Arizona and southwestern New Mexico. There are two subspecies of *Micrurus fulvius* native to the U.S.: 1) *M. f. fulvius*, found in the geographic area extending from eastern North Carolina to the southern tip of Florida and from the Gulf coastal plain to the Mississippi River; 2) *M. f. tenere*, the Texas coral snake, found west of the Mississippi River in Louisiana, Arkansas and Texas. These subspecies look very much alike to the layman and are differentiated by the amount and arrangement of black pigment within the red rings on the snake's body.

The adult coral snake (*M. fulvius*) may range between 50-100 centimeters in length, has a black snout, and yellow, black, and red bands encircling the body. The red and black rings are wider than the interposed yellow rings. The harmless scarlet king snake can be easily confused with the coral snake, but the yellow and red rings on their bodies are separated by black rings. In contrast to the pit vipers (rattlesnakes, copperheads, cottonmouths), coral snakes have round pupils and lack facial pits. Coral snakes are secretive and rarely bite anyone unless disturbed or handled. The fangs, unlike those of the pit vipers, occur as one or more pairs that are short, rigid and immovable thereby requiring a chewing motion to accomplish the injection of venom.

VENOM EFFECTS

Coral snake venom is chiefly paralytic (neurotoxic) in action, and usually only minimal-to-moderate tissue reaction and pain occur at the puncture sites which most often involve the lips. It is difficult, however, to determine which afflicted patients will develop severe signs of envenomation because there is often a delay of several hours before the onset of clinical signs. This is in contrast to crotalid (pit viper) bites, in which moderate-to-severe envenomation can be predicted by rapid onset of local and systemic effects.

Neurotoxicity caused by coral snake venom is characterized by central nervous system depression, vasomotor instability, and muscle paralysis. The neurotoxins are primarily those of basic polypeptide, and most are post-synaptic, non-depolarizing neuromuscular blocking agents with curare-like action. The effects occur gradually over 18 hrs after

envenomation and can last as long as 7-10 days. The amount of neurological impairment can range from mild skeletal muscle weakness to complete bulbar paralysis with tetraplegia, thus causing the patient to be in a completely helpless and potentially life-threatening situation. Aspiration pneumonia may be a major complication in the subacute period. Marked salivation is almost always present and is due to dysphagia. Dyspnea and dysphonia may be followed by hyporeflexic spinal reflexes. Absence of local signs can be potentially misleading as fatal envenomation can occur without any significant local tissue reaction.

Hematologic changes induced by the coral snake venom include hemolysis and red blood cell morphological changes ("burring" phenomena). An interesting finding in most dogs treated at the University of Florida is the presence of severe hemoglobinuria secondary to hemolysis. Little information is available regarding hematologic toxicity induced by coral snake venom. Although the exact components of the elapidae venom remain unknown, some fractions have been isolated and studied in detail. These include the enzymes phospholipase-A, hyaluronidase, proteinase, ribonuclease, deoxyribonuclease, and phosphodiesterase.

THERAPEUTIC MEASURES

Recommendations for treating poisonous snake bites have been the subject of controversy amongst clinicians and laboratory investigators. The difficulty in initially identifying the envenomated patient is further complicated by the delay in onset of systemic signs. Prompt antivenin therapy in suspected cases of envenomation is thus advised.

Local (first aid) therapy involves immediate immobilization of the animal and thorough cleansing of any visible wound using copious irrigation along with germicidal soap. This limits the spread of venom into the surrounding tissues and removes any residual venom from the skin surface. There is no experimental proof that incision and suction is of any value in coral snake bites, although some investigators still advocate its early use.

Unfortunately, the FDA has removed the coral snake antivenin from our suppliers because of purity problems. I will proceed to describe the product anyway in case it does become available in the near future. Antivenin (Antivenin (Micrurus fulvis), Equine Origin, North American Coral Snake Antivenin, Wyeth Laboratories, Inc., Marietta, PA 17547) treatment is recommended with the findings of one or more fang marks from which blood can be expressed or with a history of the snake hanging by its mouth on the patient. It is a refined, concentrated, and lyophilized preparation of serum globulin obtained by fractionating blood from healthy horses that have been immunized with eastern coral snake (Micrurus fulvius fulvius) venom. Cross-neutralization tests indicate that it will protect against the venoms of the eastern and the Texas coral snakes, but will not neutralize the venom of the Arizona or Sonoran coral snake. Since the possibility of

anaphylaxis always exists whenever horse serum is administered, epinephrine and intravenous fluids should be available for immediate use if necessary.

A skin test for detection of sensitivity to the antivenin is performed in human patients prior to administration although a negative test does not guarantee against a severe reaction. A 1:10 dilution of antivenin at 0.02 to 0.03 ml is injected intracutaneously. A positive reaction occurs within thirty minutes and is manifested by a wheal with or without pseudopodia and surrounding erythema. If the skin test is strongly positive, administration of antivenin may be dangerous and the risk of administration must be weighed against the risk of omission.

Anaphylaxis is a possible complication of *Micrurus* antivenin because of its horse serum origin. Any early signs of anaphylaxis (vomiting, salivation, restlessness) should be immediately treated with epinephrine (use 1:1000 concentration and administer 0.01 mg/kg IM or SQ; can repeat Q 15-20 min). The continued administration of the antivenin is allowable along with simultaneous epinephrine injections at the above dosage so long as the patient remains stable. Because of several previous adverse reactions to this antivenin, I will now give the epinephrine at the beginning of treatment as a prophylactic measure. Diphenhydramine can also be given at a dose of 1.1 mg/kg IM.

To date, there have been no human studies to define a dose-response relationship between antivenin administration and coral snake envenomation. Large coral snakes can inject up to 20 mg of venom, four to five times the human lethal dose. Since a single vial of antivenin can only neutralize 2 mg of venom, up to ten vials may be indicated in bites from larger snakes. It is best given within the first four hours from the time of envenomation. Unfortunately, the prohibitively high cost of antivenin (> \$400.00 per vial) might preclude this aggressive approach in most dogs and cats. We recommend the administration of at least one to two vials initially followed by repeated doses 4 to 6 hours later if necessary.

ANCILLARY TREATMENT

A broad spectrum antibiotic should be used for a period of 7 to 10 days to treat any bite-induced bacterial infection. *Clostridium tetani* is a rare inhabitant of the snake's mouth and none of the dogs in my experience have ever developed tetanus. Therefore, the use of tetanus antitoxin is probably not necessary. The use of corticosteroids is controversial and current treatment recommendations do not include them. Ventilatory assistance must be used in the setting of progressive respiratory failure ($\text{PaCO}_2 > 60$ mmHg) and is accomplished by intubation and mechanical ventilation. Due to the increased risk of aspiration pneumonia, close monitoring is essential. Serial arterial blood gas evaluations to assure the adequacy of pulmonary ventilation and perfusion should be performed.

Coral snake envenomation should be included in the differential diagnosis for acute flaccid quadriplegia in the dog and cat in the appropriate geographic setting. If the

history of snake bite is absent or fang marks cannot be found, it may be difficult to differentiate coral snake envenomation from polyradiculoneuritis, tick paralysis, botulism and myasthenia gravis. However, unexplained ptyalism, vomiting, hemoglobinuria and paralysis involving the cranial nerves, the accessory nerves of respiration and the limbs strongly suggest the occurrence of coral snake bite.

INTEGRATIVE MEDICINE: RECENT FINDINGS

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A new movement today in medicine is to incorporate modern, Western medicine with the best of other forms of healing into a single more expansive, integrative medical system. This allows a broader basis of diagnostic and therapeutic modalities that can be applied to a given medical problem. In effect, it makes our medicine bag bigger.

Western medicine is great at diagnosing and treating acute disease. On the other hand, it is not always the best at preventing disease. Certainly, judicious use of vaccinations has helped protect against diseases of early life; but, short of this, modern medicine has not yet embraced methods to keep most diseases from happening, particularly chronic diseases like auto-immune disorders and cancer. Only now are diet, exercise and nutritional supplements being considered as part of health and physicians are beginning to encourage patients to seek help from less "traditional" medical systems.

Veterinarians have lagged behind this movement in human medicine toward integrative care. Of course there are a number of veterinarians who practice non-traditional forms of medicine; but most of these veterinarians do not practice conventional medicine as well as complementary medicine. This leads to a division in veterinary care rather than integration of this care. Hopefully, the movement toward integrative medicine will bridge the gap and bring both sides of traditional and complementary veterinary medicine together. Rather than to argue who has the best way to treat a patient, veterinarians can focus on how best to resolve any current disease and, then, how to keep the patient healthy in the future. This is, to me, the goal of integrative medicine. The controversy seems to be based upon the more recent desire to make medicine scientifically based and to remove the "art" from medicine. Unfortunately, you cannot do this. The science still needs the art and the art still needs the science for medicine to be great and to allow medicine to be responsive to both the client's and patient's needs. We can look at current trends in human medicine to see the need to consider integrating medicine and to work together to provide excellence in patient care.

Being natural or ancient does not make medicine good, but it also does not make it bad. As research is applied to the old medicines, we often find that they work exactly as originally described. Recent studies have confirmed the basic tenants of acupuncture. Using sophisticated function magnetic resonance imaging (fMRI), a test which was clearly not available to the ancients, researchers have shown that stimulation of acupuncture points (AP) specifically stimulates various regions of the brain. Moreover, the stimulation is spatial, temporal and dependent upon the time of simulation used. While not all AP have been examined, fMRI was able to show that the AP used for analgesia results in activation of the brainstem regions involved in pain mechanism

while non-analgesic AP did not activate these regions. So, we are now beginning to understand how acupuncture works on a neuroanatomic and neurophysiologic level because we finally have the tools to test how it works. Most Western practitioners accept transcutaneous nerve stimulation (TNS) as a valid treatment modality even though it is very difficult to use it in animals because of their hairy skin. Acupuncture, if nothing more, is a form of TNS (when coupled to electrical simulators) that gets around the problem of the animal's hair-coat.

The importance of regular aerobic exercise in the prevention of chronic degenerative diseases should not be overlooked. Many studies in human beings have demonstrated improved muscle performance, memory and cerebral blood flow in patients who undertake aerobic exercise. Many of the goals of treatment in chronic neurodegenerative diseases are obtainable through regular exercise. Two forms of exercise seem the most useful: walking and swimming. Both have their merits and they may not be exclusive. A number of owners have reported that swimming assists dogs beyond the exercise of mere walking. Swimming generally increases muscle tone and allows movement without stress on joints. Walking, on the other hand, helps build strength, since gravity is involved. In older patients, particularly those with arthritis, gradually building the exercise program is important. In addition, allowing a day of rest between heavy workouts can help the patient recover faster from the exercise.

I recommend starting with 5-10 minutes of walking or swimming every other day for 2 weeks. Then, increase the length of exercise time to a goal of 30 minutes twice a week and a long walk of 1 hour once a week. If your dog already exceeds this limits, that is fine. However, remember to provide a day of easier exercise between vigorous workouts. This is particularly important as the patient gets older. It is sustained exercise which is important, walking in the backyard is not adequate. Many patients with chronic spinal disorders have remained functional because of exercise alone. We use to think that hospitalization was harmful to patients. We now know this is the lack of exercise which is harmful. Make sure your pet gets their exercise if they are hospitalized or kenneled for any period of time.

Dietary and dietary supplement management of dogs has not received great attention. IN veterinary school most of us were taught that dogs should eat dog food and nothing but dog food. This is probably wrong for most pets, since they can lead much healthier lives if they are given the choice for balanced real food. Diet may have a powerful influence on the development of chronic degenerative diseases and new information suggests that dietary regulation might play a more significant role in the progression and development of diseases like Multiple Sclerosis (MS). Elimination of toxins from pre-processed food may assist in preventing a number of immune-related disorders. Diet might help in correcting this defect and allow the immune system in dogs to stabilize. Wild dogs were not meat eaters. They ate bodies, including intestinal contents (often laden with plants and plant materials). Dogs have evolved so that eating animal fats and protein do not cause them to suffer the same problems as human beings when eating these sources of saturated fats. Moreover, today's dogs have evolved with human

contact for over 18,000 years. During most of that time, dogs ate what we ate and have evolved around the food sources available. Commercial dog food is a fairly recent development, similar to fast-food outlets for human beings. We now know that fast-food is one factor in creating modern diet-related problems.

The human diet paradigm has recently been called into question, since for many people the Atkins' diet was shown to be more effective at maintaining a good to bad cholesterol ratio better than the American Heart Association's low-fat diet. By eating high levels of carbohydrate, human being suffer from fat accumulation which eventually leads to numerous vascular diseases, damages the immune system which leads to inflammatory diseases or cancer, develop wide fluctuations in serum glucose levels which can lead to diabetes, and suffer all the effects of obesity. Commercial dogs foods, particularly dry food are based upon the outmoded human diet, in spite of the fact that dogs have no carbohydrate requirement if their diet is adequate in protein content. The only low-carbohydrate foods available are those for treating cancer, once it has developed. Does is not seem that low-carbohydrate food might also help prevent cancer development? Those are questions that beg to be answered.

Dietary supplementation is also relegated to the back of the shelf. We recognize that supplementation with chondroprotectant can help reduce clinical signs of oseteoarthritis. There are studies to support this. There are also studies to suggest that balanced levels of antioxidants (including vitamin E, vitamin C, vitamin A or beta carotene, selenium and vitamin B complex) can be healthy and help control many chronic degenerative disorders. The current approaches to the treatment of chronic sinusitis and ear problems in humans are now being questions; since, although there is improvement initially, they continue to recur soon after the treatment. Now, investigations into the underlying causes are being sought in hopes of providing real long-term care. In many cases, changing the diet to eliminate allergens (like milk and milk products) and assisting the immune system to work properly are what appears to be needed. These same principles should be applied to chronic animal diseases.

Recently, veterinarians have begun to question our "ancient" vaccination regimens. As a result, new data clearly shows that there is little evidence for many of our current practices. The three main diseases in dogs, parvovirus, distemper virus and hepatitis virus, are probably the only vaccines that are needed by most dogs. If a puppy is appropriately vaccinated against these diseases (and develops a neutralizing titer), then no further vaccination with them is needed for the rest of the dog's life. Remember that the diseases themselves provide life-long immunity. Those dogs who do not respond probably remain at risk for life. Other vaccinations may not be very effective at preventing diseases. *Leptospira* vaccination even if given every 6 months may not fully protect from disease. On the other hand, *Leptospira* vaccination often causes complications from the vaccination process. Most other vaccines are even less effective. Depending upon the region of the country or state the dog lives determines the risk versus benefit of vaccinating or not. Rabies vaccination is, however, a legal issue. Although the current rabies vaccines may be effective for many years, the results

of current studies have not led to changes in legal vaccine requirement. In conclusion, vaccine practices need to be reviewed in light of recent findings and recommendation.

By opening our medicine bags widely, we can provide optimal veterinary care utilizing emerging new technologies while maintaining connections with older healing arts. Not all veterinarians can be trained in all new fields and specialization is needed. However, it is important for all veterinarians to be aware of advances in modern medicine, so that these new options can be made available to their patients by those who are trained to offer them. On the other hand, it is equally important that all veterinarians be aware of older healing methods so that they can offer their clients choices in care, when the alternative methods have evidence to support their usefulness in the animal's condition and when they are available from people trained to deliver them properly. Integrative medicine is about choices. In some cases, Western medicine is clearly superior to older medicine; but, in many cases, it is not. Our continuing role is to know the times when each medicine is indicated, what the chances of success with each medicine is likely to be, and where to find the best medicine available. Working together as a network of veterinary care providers, we can offer clients and patients the best of both worlds through a comprehensive, integrated health care program.

EASTERN DIAMONDBACK ENVENOMATION IN THE DOG

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Crotalus adamanteus, one of the more dangerous snakes in the United States and the most dangerous snake in Florida, is indigenous to the southeastern United States (North and South Carolinas, Georgia, Florida, Alabama, Mississippi, and Louisiana). Crotalids characteristically have two elongated canalicular upper maxillary teeth that fold back against the roof of the mouth. When striking a victim with its mouth wide open, the snake's maxilla and fangs are rotated forward about 90° and thus become effective stabbing instruments. Other characteristic identifying features of the crotalids include vertically elliptic pupils, a deep pit (hence, the name *pit viper*) between the eye and the nostril, which functions as a heat receptor organ, and a somewhat triangular head. Crotalus adamanteus can grow as long as seven feet and can live longer than 22 years.

In general, most snake bites occur between the months of June and October and are rare between December and March. Most incidents involving humans occur between 3 PM and 6 PM, with 80% occurring between 9 AM and 9 PM. More specifically, most incidents of rattlesnake bites involving humans occur in the late afternoon during the hot summer months (July and August).

Pit viper bites cause one or two fang puncture wound(s). The snake can strike and envenomate its victim in less than 1 sec. Penetration and envenomation are rapidly followed by the onset of swelling, hemorrhage, and pain around the wound. A deeply penetrating fang puncture and subsequent envenomation can result in shock within minutes.

Not all poisonous snakebites result in envenomation. From one third to one half of all human victims showed little or no evidence of envenomation. Variables that influence the severity of a snakebite include (1) the location, depth, and number of bites, (2) the amount of venom injected, (3) the species and the size of the snake involved, (4) the age and the size of the victim, (5) the victim's sensitivity to venom, (6) the microbes present in the snake's mouth, and (7) the type of first aid treatment and subsequent medical care. Bite wounds to the head and neck are particularly dangerous because subsequent soft tissue swelling can obstruct respiration. In addition, the increased vascularity of the head and neck areas facilitates rapid entry of the venom into the systemic circulation, and tourniquets therefore cannot be applied easily to this area. The aftereffects of head wounds are particularly relevant for dogs; 80% of the dogs treated by me were struck in the face. This incidence differs markedly from statistics on humans, which indicate that the majority of snakebites have involved the distal extremities.

The amount of venom released depends on when the snake last ate. If a snake has not eaten recently, a large volume of venom will be available for release at the time of the

strike. In addition, a large snake can usually inject a greater amount of venom than a smaller snake. Human victims that are either very young or old are particularly susceptible to the toxic effects of envenomation. Furthermore, the toxic effects are inversely proportional to a victim's size.

Venom Toxicity

Familiarity with the components and effects of venom and an understanding of the pathophysiology of poisonous snake venoms are essential for effective treatment. Most poisonous snake venoms have direct or indirect toxic effects on the victim's blood cells, heart, blood vessels, and respiratory and nervous systems. The enzymes found in venom include phospholipase A, phosphatases, exopeptidase, hyaluronidase, and L-amino oxidase. Proteases and amino acid ester hydrolases (endopeptidase) are also found in crotalid and viperine venoms. The combined effects of these enzymes contribute to the overall toxicity of the venom.

Phospholipase A penetrates nervous tissue, where it destroys or alters certain phospholipids; it also causes hemolysis and contributes to the cardiotoxic effects of venom. Proteases account for the anticoagulant effect of venoms. Crotalus adamanteus venom has low proteolytic activity, which enhances its procoagulant effect. Hyaluronidase contributes to the swelling and edema at the site of the bite wound. Amino acid (L-arginine) esterases are common in crotalid venom and cause procoagulant activity and bradykinin release.

Poisonous snake venom also contains nonenzymatic polypeptides known as *hemorrhagins*, *cardiotoxin*, and *neurotoxin*, all of which exert profound clinical effects. Hemorrhagins, which are commonly found in crotalid venom, are vasculotoxic and cause rapid hemorrhage and edema at the wound site as well as extensive systemic hemorrhage, which contributes to hypovolemic shock. Damage to the blood vessels results from disruption of the endothelial cell junctions and basement membranes. The marked hemorrhagic edema in the dogs that I have treated clearly illustrates the combined effects of the hemorrhagins and the enzymatic components of poisonous snake venom.

Cardiac arrhythmias can occur in as many as 50% of dogs that are severely envenomated. Cardiac dysfunction results from the combined effects of cardiotoxin and impaired myocardial perfusion.

Mental depression is the only sign in the dog that might reflect the effects of the neurotoxin. In humans, however, the neurotoxin found in Crotalus adamanteus venom causes paresthesia, tetanic contractions, and fasciculation.

Adverse hematologic side effects commonly occur and include hemolysis, anemia, defibrination without overt hemorrhagic diathesis, and defibrination associated with hemorrhagic diathesis. Because of the high incidence of hemolysis and the possible need for transfusion, a snakebite patient should be crossmatched with blood donors as

soon as possible after admission. Anemia can result singly or from a combination of hemolysis and extravasation of blood into the soft tissues, which may be massive enough to necessitate whole blood transfusion.

Studies have shown that Crotalus adamanteus venom contains an amino acid esterase that exerts thrombin-like activity. This enzyme acts directly on fibrinogen in vivo (and in vitro) apparently without affecting any other protein or the platelets involved in blood coagulation. The procoagulant effect of this thrombin-like enzyme causes inappropriate fibrinogen cleavage, with subsequent formation of soft friable microclots, fibrinogen degradation products, and fibrinogen depletion. Fibrinogen degradation products inhibit normal fibrin polymerization, and their identification in clinical patients indicates activation of the fibrinolytic system. Defibrination caused by a thrombin-like enzyme aggravates the bleeding produced by the hemorrhagins. In humans, the true syndrome of disseminated intravascular coagulation (DIC) rarely is documented after Crotalus adamanteus envenomation because the snake venom esterase seldom causes platelet aggregation, does not activate and consume factors V and VIII, and shows minimal if any response to heparin treatment.

In humans, defibrination with grossly anticoagulant blood may persist for days without any signs of bleeding. Dogs can also show abnormal coagulation times in the absence of bleeding.

First Aid

Common first aid measures for poisonous snakebites of humans include (1) immobilizing the patient and the affected limb to slow the spread of venom, (2) applying a light-constricting tourniquet, and (3) performing local incision and suction.

A tourniquet should be applied at least 10 cm proximal to the fang marks and cause only light constriction to obstruct the superficial lymphatic and venous flows; the tourniquet should be released every 30 minutes for 60 to 90 seconds. A tourniquet is most effective when applied within 30 minutes of envenomation. Some authors, however, object to the use of a tourniquet because it prevents dilution of the venom and decreases tissue perfusion, thereby promoting ischemia and tissue necrosis. Furthermore, there is no evidence supporting a reduction in morbidity or mortality when a tourniquet is used. Tourniquets are seldom (if ever) useful in dogs because the majority are struck in the head. In addition, the elapsed time between the strike and the owner's awareness of the incident might exceed 30 minutes. Incision and suction are effective only when done immediately.

Treatment

After a dog is bitten by a snake, the following considerations are warranted:

1. Establish whether the dog was bitten by a nonpoisonous or a poisonous snake.
2. Decide whether envenomation has occurred.
3. Institute measures to prevent or limit severe tissue damage.
4. Provide life support measures if severe envenomation has occurred.

Dogs that are moderately affected by envenomation should receive a thorough medical evaluation and intensive medical treatment, including a hemogram, urinalysis, clotting evaluation, and an electrocardiogram. These tests should be repeated regularly during hospitalization to assess the adequacy of the patient's response to treatment as well as to detect the onset of delayed complications (especially severe anemia and cardiac arrhythmia). Urine output and blood urea nitrogen or serum creatinine levels should also be measured to detect any renal failure resulting from ischemia or hemolysis.

Essential treatments are intravenous fluids, antivenin, and antibiotics. Because hypoproteinemia and anemia are common results of soft tissue extravasation, fresh whole blood should be given if the packed cell volume and total protein drop below 20% and 5 g/dl, respectively. Analgesic drugs should be used if necessary. Lidocaine by CRI can be given at a loading dose of 1 mg/kg IV and then a maintenance dose of 50 µg/kg/min. Buprenorphine at 0.02 mg/kg IV push can also be given every 6 hours.

The use of polyvalent crotalid antivenin is the mainstay of therapy for moderate to severe envenomation; however, the cost of antivenin limits its feasibility as a treatment for dogs. In humans, early intravenous antivenin treatment of victims with moderate to marked symptoms has been stressed repeatedly. Antivenin should be given within four hours after the snakebite. Although the full beneficial effect diminishes when antivenin is given after this period, it is still recommended up to 24 hours after envenomation. If the bite results in the injection of a large quantity of venom deep into well-vascularized soft tissues or directly into a vessel, death may occur despite vigorous antivenin treatment.

Anaphylaxis is a possible complication of antivenin treatment because of its horse serum origin. Any early signs of anaphylaxis (vomiting, salivation, restlessness) should be immediately treated with epinephrine (use 1:1000 concentration and administer 0.01 mg/kg IM or SQ; can repeat Q 15-20 min). The continued administration of the antivenin is allowable along with simultaneous epinephrine injections at the above dosage.

Most of the dogs treated with antivenin at the University of Florida have received an average total dose of 42.7 ml; one leading authority recommends administering larger doses similar to those used on humans (80 to 100 ml). Dogs, unlike humans, are not particularly susceptible to immediate allergic reactions from antivenin treatment. In situations where this might occur, all drug package inserts provide instructions for hypersensitivity testing, desensitization procedures, and the treatment of allergic reactions.

In humans, the benefits of glucocorticoid treatment of poisonous snakebite victims are controversial. Several authorities find no proof of efficacy and find its use contraindicated. In fact, many experts state that glucocorticoid might even antagonize the effects of antivenin. Most of the dogs in my experience have done well without steroid treatment.

Because fang wounds can be accompanied by inoculation of bacteria into the victim's tissues, broad-spectrum antibiotic treatment is recommended. In humans, the administration of tetanus antitoxin, tetanus toxoid, or both is routine. Indications for these compounds in dogs are minimum because of low species sensitivity. Communications over periods of weeks to months between University of Florida staff and the owners of surviving dogs that did not receive tetanus antitoxin revealed that these dogs had done well after being discharged from the hospital, with none showing any signs of tetanus.

ALLERGY UPDATE

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ABSTRACT

Dogs with environmental allergies often have concurrent allergies (e.g. food and flea allergies) and are prone to relapsing skin and ear infections that significantly contribute to the level of discomfort of the animal. Management of both secondary infections and concurrent allergies is crucial to reduce itching. Combination of several systemic and topical therapies may be necessary to control clinical signs. For many years steroids have been the main therapy available for affected animals. Serious adverse effects, increased incidence of infections and decreased efficacy overtime are major limitations to the chronic use of steroids. A great effort of research has been done in recent years to identify effective and safe alternative treatments. The results of recent clinical trials evaluating these new therapies are presented.

IMPORTANCE OF ADDRESSING ALL THE FACTORS

Concept of pruritic threshold and co-factors involved in allergies

According to the theory of threshold a given individual can tolerate a variety of stimuli without developing clinical signs as long as the threshold is not reached. All these stimuli have an additive effect and once the threshold is reached itching and other clinical signs become evident. The threshold varies from patient to patient and can be lower by stress. This concept has important consequences on the management of canine allergies. Most of dogs with environmental allergies also have other allergies thus control of concurrent allergies is crucial to decrease the severity of clinical signs. Some allergic stimuli (e.g. flea and food) are easier to control than others (e.g. pollen) and effort should be devoted to decrease parasite load or exposure to offending foods. Most environmental allergens are absorbed through the skin, some is swallowed or inhaled. Recent studies have demonstrated that oral challenge with house dust mites in sensitive dogs can cause the same lesions as environmental exposure. Frequent bathing helps to decrease allergen exposure.

How can I diagnose a food allergy?

Food allergy is not very common. However it can be extremely itchy and much easier to treat than other allergies therefore it should be considered in all dogs that have non-seasonal problems. Unfortunately there are no tests that can reliably predict food allergy in dogs therefore a food trial with a novel source of protein and carbohydrate is required to diagnose food allergy. Several commercial diets have been shown to be suitable for food trials although home-cooked diets are the only ones that can

completely eliminate the possibility of an allergic reaction to preservatives. Hydrolyzed diets (e.g. Z/d) have been introduced on the market for dogs and cats. They may help in some cases but may not be able to diagnose all cases of food allergy.

How can I eliminate fleas in Florida?

Although challenging, this can be done with the products available on the market. Since resistance to adulticides has been increasing it is important to combine various products with different mechanisms of action and to combine flea control on the animal with environmental control.

The importance of treating secondary infections

In addition dogs with environmental allergies are prone to secondary skin infections that significantly contribute to the level of discomfort. Resolution of skin infections may dramatically decrease the level of pruritus. Thus the management of a patient with environmental allergies should include control of all concurrent allergies, limitation of allergen exposure, and control of secondary infections. Additional therapy is then directed to decrease inflammation and/or to modulate the allergic reaction.

Clinical presentation of environmental allergies in dogs

Canine allergies have a strong breed predilection. They are common in Terriers, Setters, Retrievers (Golden and Labrador), Beagle, Miniature Schnauzers, Shar-Pei, Dalmatians, Lhasa Apsos, Cocker Spaniels, Boxers, and Bulldogs. The typical age of onset is between 1 and 3 years of age. Progressive worsening of the disease is typical. Clinical signs are initially seasonal (worse in the spring and summer) and with time become all year round. The first clinical sign is pruritus without any primary lesion. Skin lesions are due to self-trauma and secondary pyoderma. Pruritus is usually responsive to glucocorticoids at the beginning of the disease. Face, feet, ears, axillae and groin are commonly affected areas. Sometimes pruritus is generalized. Erythema (redness), excoriations, hyperpigmentation (dark skin), and lichenification (thick skin) are secondary changes due to chronic inflammation and self-trauma. Chronic recurrent otitis externa and erythema of the pinnae is also a common presentation.

Diagnosis

Diagnosis is based on history, clinical signs, and the exclusion of other diseases. Positive allergy testing is considered secondary criteria. When dermatitis has a distribution of lesions and pruritus suggestive of flea allergy and/or the patient has non-seasonal pruritus, good flea control and a poor response to 8-12 weeks of hypoallergenic diet may be required to confirm a diagnosis of environmental allergies.

The most important reasons to consider allergy testing are to institute the practice of avoidance measures and hyposensitization (allergy vaccine). It is important not to base a diagnosis of environmental allergies just on a positive skin test or serology test, as normal animals or animals with other pruritic diseases may have positive reactions. A positive reaction on a test only indicates that the animal has IgE antibodies for that allergen: it does not imply that the clinical signs are caused by that allergen. Skin testing has the advantage of testing the organ that is directly affected by the disease.

The incidence of false positive results is low compared to other types of tests. Disadvantages with skin test include the influence of drugs (e.g. steroids and antihistamines) and stress on the results. Suggested withdrawal times for drugs prior to IDT are 2 weeks for both antihistamines and topical steroids and 2 months for systemic steroids. There are some animals with strong clinical signs of environmental allergies that can have a negative skin test. Reasons for false negative skin test include subcutaneous injections, too little allergen, outdated allergens, testing with mixes, drug interference, estrus, pseudopregnancy, stress, and off season testing (testing more than 2 months after the end of the season).

In vitro serology testing is commonly done but it lacks specificity and has a high incidence of false positive results. Correlation between positive results of IDT and serology testing is poor. One explanation for this is that the two tests measure different things. Intradermal testing detects IgE antibodies in the skin while serology testing measures circulating IgE antibodies and direct correlation may not exist.

Hyposensitization or Allergy vaccine. For severe cases or for cases with clinical signs all year round, hyposensitization may be the best long-term therapy as symptomatic therapy decreases in efficacy overtime and avoidance may not be feasible for most allergens. Vaccine is effective in 60-80% of cases and is especially indicated in young animals. Vaccine should be made including the positive results of the allergy test. Choice of allergens is based on the history of each case (e.g. seasonality, plants are more prevalent in the patient's environment). More than one vaccine may be necessary in cases with numerous allergies. Side effects are not common and include increased itching after the injection, urticaria, and rarely anaphylaxis. Response to hyposensitization is not usually evident for the first 3-4 months and it may take up to a full year before full efficacy is evaluated. If sudden improvement is noted after the first few weeks, it is most likely due to other concurrent forms of therapy (e.g. antibiotic therapy, flea control). If adverse effects are seen, schedule may need to be adjusted. It might be useful to pre-medicate the patient with antihistamines one hour before the injection. Once the maintenance dose is achieved the interval of administration is judged based on the individual case. As an example, some dogs may be relatively symptom-free for the first 2 weeks after the injection and then pruritus may progressively increase. In these cases the injection should be given every 2 weeks. In other cases a longer interval may be appropriate (usually 3 weeks). Decrease of the frequency of relapse of skin and ear infections is a sign of improvement, even if occasional antibiotic therapy is still required.

Oral Drugs

Antihistamines. Antihistamines are helpful to decrease the inflammation of the skin and work better as prevention than as treatment. They may be the only therapy for animals with seasonal signs or for mild to moderate cases. As efficacy varies among individuals, several antihistamines (each one for 14 days) should be tried to find the one that is most effective in each individual case. As a general rule, antihistamines are

helpful in 40-50% of cases. The most commonly used antihistamines are first generation or H1 blockers. Sleepiness and excitability are possible side effects. Diphenhydramine (Benadryl®) is recommended at 2.2 mg/kg 3 times/day (TID) by mouth (PO). If no response is seen, Hydroxyzine (Atarax®) HCl can be tried at 2.2 mg/kg TID PO. Chlorpheniramine (ChlorTrimeton®) has been reported to be effective at 0.2-0.8 mg/kg TIP PO in dogs. Trimepazine tartrate (Temaril®) may be tried at 2.5-5 mg/dog TID PO. In cases with a behavioral component, amitriptyline hydrochloride (Elavil®) (tricyclic antidepressant with antihistamine activity) may be useful at 2.2mg/kg PO BID.

Essential fatty acids (EFA). The word “essential” implies that it is a fatty acid that cannot be synthesized by the animal thus it has to be supplied in the diet. Essential fatty acids modulate inflammation, composition of lipids in the skin and lymphocyte functions. Commonly used sources of EFA are cold water marine oil, evening primrose, borage, and black currant oil. Suggested dose of eicosapentaenoic acid is 180mg/10 lb of body weight. Marine oil is rich in eicosapentaenoic acid. Side effects are rare and include diarrhea and pancreatitis. Fatty acids have a sparing effect on the dose of corticosteroids and a synergistic effect with antihistamines.

Glucocorticoids. Oral administration is preferred over injections; it can be better regulated, it is safer for long-term therapy. Reported side effects in dogs include increased drinking and urinating (polyuria, polydipsia), increased appetite (polyphagia), behavioral changes, panting, diarrhea, pancreatitis, muscle wasting, increased risk of infections (both skin and urinary tract) demodicosis (red mange), hairloss, thin skin, comedones (blackheads), calcinosis cutis, osteoporosis, diabetes mellitus and decreased synthesis of thyroid hormones. Use of oral steroids should be limited to 4-5 months/year.

Cyclosporine. Cyclosporine A (Cs, Neoral, Atopica) at 5mg/kg once daily is as effective as glucocorticoids for moderate to severe cases of allergies in people and dogs. Reported adverse effects with Cs use include vomiting, diarrhea, increased risk for bacterial infections, wart like dermatitis and changes in the coat color and length. At higher doses Cyclosporine can also include bone marrow suppression and cause kidney disease.

Cyclosporine is available in capsules (25 and 100mg) or in an oral solution (100mg/ml). Cyclosporine is fairly pricey. Soft gel capsules (Sandimmune®) and microemulsion formulations (Neoral®) are not interchangeable with the latter having a higher bioavailability. Studies reported for canine AD utilized the microemulsion formulation. Concerns have been expressed in human medicine regarding the long term use of cyclosporine and development of lymphoma. Limited information exists in dogs.

Pentoxifylline (PTX, Trental,® at 10mg/kg twice daily has been used for canine allergies with good response. No adverse effects were reported. In people adverse effects are occasionally reported and include vomiting, diarrhea, nausea, and rarely

angina, agitation, dizziness, headache, and tremors. They are usually dose-related and are decreased by lowering the dose. It is clinical impression that the efficacy of PTX in dogs is dose related with higher doses ($20\text{-}25\text{mgkg}^{-1}$) being more effective than lower doses (10mgkg^{-1}). It appears to be well tolerated even at higher doses. Due to the rapid elimination in dogs, PTX should be administered TID. Pentoxifylline can be safely combined with antihistamines to improve efficacy. Pentoxifylline is particularly helpful in dogs that have contact allergy to plants of the Wandering Jew family.

Topical therapy

Topical therapy is very beneficial in dogs with allergies because it reduces the amount of allergen absorption through the skin. Several products can be used to decrease pruritus. Oatmeal is antipruritic, soothing, often combined with pramoxine, a topical anesthetic (Dermalsoothe®, Relief®). The exact mechanism of action of oatmeal is unknown. It is available in shampoos, cream rinses and leave on conditioners. Topical glucocorticoids may be helpful. Hydrocortisone leave on conditioner at 1% has no effect on chemistry panels and adrenal function. Lime sulfur (Lym Dip®) is a strong anti-pruritic agent (4-6oz/gallon of water, q5-7 days). It is keratolytic, antifungal and anti parasitic. It dries the coat with prolonged use. Topical capsaicin (active ingredient of chili pepper) at 0.05% has been reported to decrease itching in dogs with allergies. Worsening may be noted after the first week of therapy. Once improvement is achieved, relief persists for 1-2 weeks after discontinuation of therapy. The most recent topical therapy for allergies in humans is called Tacrolimus (Protopic®) and belongs to the same family of cyclosporine. It is extremely expensive (\$80/tube) but has great potential for refractory cases, especially when pruritus is localized.

CONCLUSIONS

Allergies are a life-long disease and long-term management is essential. All concurrent allergies and secondary infections should be addressed. Combination therapy is required in most cases to decrease the need for glucocorticoid therapy.

THE CHANGING FACE OF VETERINARY PAIN MANAGEMENT

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One of the biggest advances in veterinary medicine over the past decade has been our understanding of pain in animals. These fall into three main categories:

1. Assessment and “measurement” of pain
2. Treatment of acute pain
3. Treatment of chronic pain

Assessment of pain in dogs – how do we know if they hurt?

The assessment of pain in animals is essential to successful pain management, but is not an easy task. We must ask “What is pain?” In humans it is defined as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage.” This definition of pain, assumes that animals are capable of emotions in order to feel pain. Emotions are described by language, something that is obviously unavailable to animals. The nervous system of dogs is very similar to ours therefore it is reasonable to claim that they do experience pain even if they cannot “voice” their discomfort in human terms. We must accept that if it would hurt us, it will hurt them. In humans it is now accepted that “the inability to communicate in no way negates the possibility that an individual is experiencing pain and is in need of appropriate pain relieving treatment”. As veterinarians we are in a similar situation to paediatricians and those working with non-verbal adults who may be in pain.

All pain is subjective and no one can “feel” another person’s pain. Even after an identical injury in humans there is a wide range in the pain that is experienced and reported by individual patients and there is no reason to doubt that this is also true in dogs. However, in humans, pain is what the patient says it is and in animals it is what we (the caregiver) say it is. Obviously there is more room for error when we assess pain in animals and if we get it wrong, they may suffer unnecessarily.

We can only claim to treat pain effectively if we can measure it. We can measure many things in our patients for example temperature, heart rate and blood pressure, but “pain” is more elusive and there are no “pain meters”! At this time there is no gold standard for assessing pain in animals. Many different scoring methods have been published, but the issue in animals is complex because we must consider differences in many factors including gender, age, species, breed, and environment. I am sure no one disagrees that Labradors behave differently to Arctic breeds when it comes to voicing their “opinion”! Any assessment system must also account for different types of pain, such as acute pain associated with surgery or chronic pain related to osteoarthritis.

Acute pain (surgery, trauma)

We now know that the best way to judge pain in dogs is by studying their behaviour. This has been done by observing dogs or videotaping them before surgery to see what

their normal behaviour is then repeating this after surgery and looking at how this changes and which drugs restore normal behaviour. Developing these systems has been a daunting task; 166 possible pain behaviours associated with spay surgery in bitches have been identified. However, a very robust system called the Glasgow Composite Measures Pain Score has emerged which seems to be very accurate in detecting pain in dogs. This is the system we use at the University of Florida and it is easy and quick to use and it lets us know when we need to give more pain relieving drugs after surgery.

Chronic Pain

Due to the nature of chronic pain, such as that associated with osteoarthritis or cancer the changes in behavior can be subtle and easily missed. In addition when we are dealing with chronic disease, we are looking at overall quality of life, not just pain. Some owners assume that certain changes in their dog are inevitable with advancing age. Preliminary data based on owner interviews showed changes in 32 types of behavior in dogs with chronic pain. It is important to note that this study showed that ***the owners are the best evaluators of their pet's pain***. Although still in the development phase, the Glasgow University Health-Related Dog Behaviour Questionnaire has identified some key indicators of chronic pain in dogs, including, but not limited to, decreased mobility, activity, sociability, and curiosity and increases in aggression, anxiety, daytime sleeping, and vocalizing.

Treatment of acute pain

The implementation of more complex surgical procedures can only proceed if we can provide excellent anesthesia and pain management to our patients to prevent suffering and guarantee a quick recovery and return to normal function. When interviewed before surgery humans are often more afraid of anesthesia and how much pain they will be in than the surgery itself. Pet owners should expect and demand that pain management is an important focus at their veterinarian's practice. Ten years ago, less than 30% of dogs received pain medications for "routine" surgeries but a recent survey shows that that this number is now about 80%.

The most common drugs used to relieve surgical pain are the opioids (narcotics). These include drugs such as morphine and hydromorphone that are given by injection along with a tranquilizer before anesthesia. These drugs also ensure a smoother anesthetic period and more stable heart rate and blood pressure during surgery. Other techniques that may be used include the use of local anesthetics to "numb" the surgical area. Epidural or spinal anaesthesia is commonly performed in dogs just as it is in humans. This method involves placing the drugs close to the spinal cord where they act – this allows for excellent pain relief without the "grogginess" we sometimes see when the same drugs are given through the intravenous (IV) catheter or into the muscle. This technique is used almost routinely for orthopaedic surgery such as repair of a torn cruciate ligament or for hip surgery. For major surgery many dogs will be given different drugs by IV infusion and they may be continued for several days after surgery and adjusted up or down depending on the dog's pain score. Another way to provide

continuous pain relief for several days is with a “patch” placed over shaved skin. The most commonly used one is the fentanyl (Duragesic) patch which is designed for humans with cancer pain. These can be very useful, last up to 3 days and avoids having to keep injecting the dog or maintaining an IV line – many dogs will be sent home with these in place. Some owners ask if their dog can become addicted to narcotics and the answer is “no”. Veterinarians only give dogs these drugs when they are in pain and for short periods of time and the problems sometimes seen in people just don’t happen in dogs.

Another group of drugs that is used for acute pain are the non-steroidal anti-inflammatory agents (NSAIDs). These are well known for treating chronic pain (see below), but it makes sense to use them after surgery because all surgical procedures are associated with inflammation. These drugs are not controlled substances and are available in liquid, chewable tablet and caplet formulations which are easy to give at home. With all the new drugs and techniques available to veterinarians, dogs should sail through surgery and recover quickly with minimal discomfort.

Treatment of chronic pain

The most common type of chronic pain seen in dogs is associated with osteoarthritis (OA) and some forms of cancer. However dogs with ear and skin problems and dental disease also suffer pain over long periods of time and relieving their pain should be an integral part of their medical management.

Arthritis is not a curable disease but we can make patients with this disease comfortable. Although drugs play a primary role in alleviating osteoarthritis pain, weight loss, nutritional supplements (for example glucosamine and chondroitin), controlled exercise, physical therapy and complimentary therapies such as acupuncture are also important. It is estimated that 1 in 4 dogs will develop arthritis and it is seen most commonly in older (>6 years) and larger (>20 kg) dogs. Unfortunately only about 50% of dogs with arthritis receive treatment because many owners assume that slowing down is inevitable with age and may not realise their dog is slowing down because it is in pain.

The most commonly used and effective drugs for decreasing the pain of OA are the non-steroidal anti-inflammatory drugs (NSAIDs). Currently there are several FDA approved drugs licensed for use in dogs such as Deramaxx (deracoxib), Rimadyl (carprofen), Metacam (meloxicam), Etogesic (etodolac) and Zubrin (tepoxalin). All these drugs can be effective and safe when used correctly. People respond differently to NSAIDs and I am sure what you use for a headache or soreness isn’t the same as the person sitting next to you and what works for you was found by trial and error. The same is true of dogs and just because a dog does not respond well to the first anti-inflammatory prescribed this does not mean that they will “just have to put up with the pain”, it just means it may take time to find the drug that works, or that additional therapies may be needed.

The NSAIDs can be associated with unwanted side-effects; the most common are related to the gastrointestinal tract (bleeding and ulceration), but liver and kidney problems are also reported. Before starting a dog on NSAIDs blood work should be performed to look at kidney and liver function. Animals with kidney disease should not be given NSAIDs. Because dogs will likely require these drugs for many years it is advisable to have their blood work checked at regular intervals to monitor for any potential problems. If a dog vomits, has bloody stools or goes off its food while taking these medications you should stop treatment and contact your veterinarian immediately.

There has been a lot of concern and bad publicity over the so-called “COX-2 specific” drugs in humans (for example VIOXX), and these relate primarily to cardiovascular side-effects such as stroke and “heart attacks”. Because dogs don’t have coronary artery disease or atherosclerosis (fat deposition in vessels) these problems are unlikely to surface in dogs that take COX-2 specific drugs (for example Deramaxx). However there have been reports of severe gastric ulceration and even perforations in some dogs. When these were investigated it was found that most dogs had been overdosed, been on steroids, the owners had given aspirin in addition to the prescribed NSAID, or had another underlying disease that had not been diagnosed. Therefore it is very important to follow the dosing instructions carefully and not to give any other drugs without consulting your veterinarian.

The NSAIDs can produce dramatic results but other measures may be required for optimal pain relief. Weight control and regular exercise (no weekend warriors!) are important for dogs and owners! Special arthritis or joint diets containing specific antioxidants and omega-3-fatty acids seem to be helpful. Nutraceuticals such as chondroitin and glucosamine have anecdotally helped some dogs, but there are not any large scientific studies to back this up. Adequan[®] (Polysulfated Glycosaminoglycan) is FDA approved for dogs and is given by injection – this product is thought to help repair damaged cartilage in arthritic joints. Acupuncture and acupressure or massage can also produce added benefits, and many owners can learn to massage “trigger” points. There are also several other drugs currently undergoing trials for relieving chronic pain in humans and dogs.

The good news is that we have made great advances in our understanding of pain in animals and have developed better ways to assess and treat this whether it is in the young healthy dog undergoing surgery or the older dog with chronic pain and most veterinarians have radically changed how they manage pain and are seeing the benefits. We still have work to do, but the days of dogs having to “put up with the pain” are over – it just requires patience and effort and a willing owner to achieve an optimal outcome.

Canine Communication & Aggression

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Wolves are the distant ancestors of dogs. As such, dogs engage in pack living. They are predatory and hierarchical. Disputes may be settled through ritual signaling or attacks that cause injury. Ritual signaling involves the use of ears, tail, head, lips, stance, eye contact, licking, and mounting.

Ears that are **up and forward** signal alertness or dominance; those that are **down and back** signal fear/submission or excitement/anxiety. It is important to remember that when entering into an attack, even dominant animals will lay their ears back to protect them from damage. A **Tail** that is **up** signals alertness or dominance; if it is **midlevel**, the dog is relaxed, attentive; a tail that is **down** is indicative of fear/submission. When a dog's **Head** is **up**, this is a signal of alertness or dominance; when it is **down or turned away**, this is a signal of fear/submission. **Lips - elevation of lips without retraction of the commissure** is a dominant aggressive threat; **retraction of the commissure** is a sign of submission; and **retraction of the commissure with exposure of the teeth** is a defensive threat. **Stance** – when a dog is **upright/leaning forward**, this is a sign of alertness or dominance; when **crouched**, the dog is fearful/submissive. Lying down is the most submissive position, short of rolling over. **Mounting** is a signal of dominance, not sexual behavior, with the exception of an intact male mounting an estrous female. **Rolling over** – Mothers roll their puppies over to clean them. This behavior continues into adulthood as a submissive signal. **Eyes** – dogs that **stare at** are signaling dominance; those that **look away, blink** are signaling submission. **Licking** - As puppies are being weaned, older wolves regurgitate partially digested food for them. Puppies solicit regurgitation by licking the lips of the older wolf. Licking remains in adults as a form of active submission. Dogs which are not allowed to lick faces may lick hands instead.

Metacommunication is a form of communication in which information is provided that modifies the meaning of subsequent communication. The **playbow** is a form of metacommunication. It means “what I do next is play”.

Diagnosis and Treatment of Aggressive Behavior in Dogs

Numerous considerations are involved, such as the human-animal bond, public safety, and euthanasia. When treating aggression in dogs, all of the following should be taken into account: the attitude of the owner, the presence of vulnerable individuals in the household, the size of the dog, the type of aggression, the intensity of the aggression, and special logistical issues for preventing bites (such as doors, fences, gates, collars, muzzles). With the treatment of any aggression, it is important to caution owners of the

unpredictability of any attempt to treat. **NO TREATMENT IS 100% EFFECTIVE.** Any dog may bite, whether they have done so previously or not.

Aggression Directed at Humans

Categories include dominance, fear, possessive, territorial/protective, maternal, and predatory. Remember the “ritual signals”... If the dog signals with its eyes, ears, head, body, tail and the threatening person doesn't go away, what's left? With *some* dogs: growling, snapping, and biting. If, at that point the person retreats, the behavior has been negatively reinforced and the dog is more likely to perform that behavior in the future. Not all aggression is the same. **A diagnosis is imperative!**

Dominance Aggression

It is a current issue and over diagnosed. In many cases it is presumed to be the cause of aggression when no diagnostic process has been conducted. In “Dominance Aggression Syndrome” there is persistent aggression (growling, snapping, biting), accompanied by multiple ritual dominance signals directed toward the owner. The most common signalment is a male (intact, at least when problem began) that is 1-3 years of age. The most common breeds represented are the German Shepherd, Doberman Pinscher, and Springer Spaniel. But it is important to remember that it can occur in any breed, in either sex, and at any age. If it is seen in a female, it's usually younger, less than 6 months old. A dominant dog will often block the owner's movements while in the home – by standing or lying in doorways, halls. The dominant dog may lie on the bed and growl when approached. *But so will the fearful dog, so you need to look at postures!* There is to be no punishment of the dominant dog, as the aggression is often exacerbated by physical punishment. Again, it is important to distinguish from fear aggression, which can also escalate when punishment is used. Dominance motivated aggression is a **problem of relationships**, so the aggression is **most likely directed at family members**. A dominant dog will resist submissive postures, and there may be a history of not learning the ‘down’ command in obedience class. Food guarding in the absence of ritual dominance signals or aggression to people in any other situation is *simply food guarding* (possessive aggression). Many food guarders are quite submissive and non-aggressive in all other situations.

Treatment:

Don't “teach the dog who's boss”, or “have it out” with the dog, as someone is likely to be bitten. There is never to be any “**alpha rolling**”. Treatment involves avoiding all situations in which the dog behaves aggressively. It is important to realize that **avoidance ≠ “giving in”**. In fact it is actually the person taking the power back by not allowing the dog to respond in an aggressive manner and denying him the opportunity to *practice*. There is a significant learning component to the behavior, so the dog needs to “re-learn”. If the dog is an intact male, neuter; however neutering an intact female may not help - there is evidence of a small effect of making the problem worse or more persistent if the dog is showing dominance aggression when a puppy. Behavior Modification involves giving a high frequency of low intensity dominance signals. It is important to identify what ritual signals you can **safely** exhibit to the dog, and do this

many times a day. In a structured situation, gradually train the dog to exhibit a progressively more submissive posture, e.g. 'down'. Institute the "nothing in life is free" program: the dog must obey a command, preferably 'down', to get anything it wants – food, petting, into the house, out of the house, etc. It is important to reward submissive postures. The **Gentle Leader**[®] head collar is designed to provide subtle dominance signals and helps to decrease overall level of anxiety. Any pharmacological treatment is extra-label use and will need owner consent. However, increased serotonergic activity should produce a decrease in affective aggressive behavior (i.e. non-predatory behavior), so pharmacological options include the tricyclic antidepressants such as clomipramine and the selective serotonin reuptake inhibitors such as fluoxetine (Prozac[®]) and paroxetine (Paxil[®]). Another option is the progestin, megestrol acetate (Ovaban[®]). The typical response is a decrease in aggressive behavior with mild sedation. The mild side-effects, which are common, include: increased thirst and hunger, increased urination, and mammary hyperplasia. The possible serious side-effects include: elevated blood glucose leading to diabetes and carcinomas. Progestins should generally be **restricted to cases in which euthanasia is highly probable** if there is not significant, rapid improvement. With long-term use, the **dog's life-span as an acceptable family pet may be extended**, but life-threatening medical complications are very likely to eventually occur

Fear Aggression

The most common motivation for aggression directed at people. It is characterized by aggression coupled with signals of fear and submission: avoidance, ears back/down, tail down, retraction of commissure of lip – "grin", looking away, turning away, and licking lips, yawning.

Treatment:

Do not punish! Avoid situations that are likely to trigger fearful, defensive behavior. Implement desensitization and counter-conditioning. Use of the Gentle Leader[®] head collar may help to decrease the overall level of anxiety. Avoid threatening gestures such as standing over, reaching out/over, loud voices, direct eye stare, etc. Interact in a non-threatening manner: pet under chin, chest – on dog's level, talk softly, etc.

Treatment: Desensitization & Counter-Conditioning

The animal is exposed to a stimulus that elicits a given response, but at such a **low level that the response is not elicited**. Over time and successive repetitions, **the intensity of the stimulus is gradually increased**, ideally without eliciting the response. A response is elicited which is *behaviorally* and *physiologically* incompatible with another response. The dog **cannot be relaxed and anxious at same time**. Teach the dog to relax and reward the dog for being relaxed – the dog "re-learns".

Pharmacological Intervention

Used to decrease the dog's level of anxiety so that it can learn what it needs to. Options include the tricyclic antidepressants and the SSRIs – again, off-label use. The

Dog Appeasing Pheromone - DAP[®] diffuser may also help to decrease the dog's overall level of anxiety.

Possessive Aggression

The dog defends specific items (food, bones, chewies, toys, etc.), but otherwise does not exhibit aggression or ritual dominance signals. The behavior is often fear-based.

Treatment:

If there is a limited number or type of items defended, remove them, otherwise, desensitize and counter-condition so that the dog re-learns what interactions mean - at the food bowl, with toys, etc.

Territorial Aggression

The dog protects an inappropriate location or protects an appropriate location in an inappropriate context. The aggression can be directed at humans, other dogs, other animals, or a combination of targets. The dog can be territorial of the house, the yard, its crate, its sleeping place, a confined place, the car. It may also protect an individual approach distance – a “mobile territory”.

Treatment:

Never leave dog outside alone “with a territory to protect”. The Gentle Leader[®] head collar can be useful in owner-control. The dog can be desensitized and counter-conditioned to people approaching its territory.

Protective Aggression

An extension of territorial aggression where the **dog perceives that the owner is threatened** when there is no *real* threat – such as with a stranger at door, when the dog is approached when in a car with the owner, when another dog approaches owner, when a person raises its voice to owner, or when a person hugs the owner.

Treatment:

Avoid situations where the dog believes it needs to protect the owner. Implement command control. DS&CC for approaches from strangers, dogs, etc. with the owner present, and the Gentle Leader[®] head collar.

Maternal Aggression

This is normal behavior that typically wanes as the puppies mature. Make sure that the prepartum bitch is familiar with whoever will be caring for her and the puppies postpartum.

Predatory Aggression

Canis familiaris is a predator. This type of aggression results in a number of fatalities each year, in addition to many injuries. Common targets include: joggers, bicyclers, and running children. Risk factors include: a loose dog and any history of predatory behavior

Treatment:

Appropriately contain the dog and implement command control. DS&CC to the object of predation *may* be effective. Use of the Gentle Leader® head collar **with a light basket muzzle** is recommended. In laboratory studies, serotonin and GABA have been found to have an inhibitory effect on predatory behavior. Therefore, theoretically, serotonergic medications and GABA agonists should reduce probability that this type of behavior will occur.

Inter-Dog Aggression

Can be status-related, fear-motivated, arousal-related, possessive, protective, territorial, redirected, and/or predatory.

Fear

Secondary to a lack of socialization or previous experience. Therefore, focus on the early history, upbringing, the time of adoption, and the interaction with other dogs. Look at the aggressor's body posture and the victim's body posture. It is important to get a description of both dogs' behavior before and after the fight – was it defensive? offensive? What was the context?

Treatment:

Consists of decreasing the dog's overall level of anxiety using a head collar, DS&CC to the other dog's presence, ± anxiolytic medication.

Arousal

Some dogs respond either more quickly or more intensely to a given stimulus than other dogs. The incidents may be situational – such as at feeding time, at owner arrival, going inside/outside, in a hallway, stairwell, on walks, etc.

Treatment:

Involves the anticipation of behaviors, redirecting the dogs, and rewarding appropriate behaviors. It is important to avoid certain situations and contexts. Use of a head collar will allow for better control, and the dogs can be desensitized and counter-conditioned to one another.

Possessive

Protecting food or other resource may be an ancestral adaptive behavior and food-related aggression is tightly coupled to canine evolutionary history - canids are binge-and-gorge eaters. Some dogs from large litters that are fed from one bowl may learn to be aggressive to each other to successfully compete for food. Free-ranging street dogs may get enough food only by successfully fending off competitors. With possessive aggression, the dog reacts when food or other resource is approached and/or reached for by growling, lip lifting, snarling, lunging, and/or biting. Generally, the higher the quality of the resource, the more pronounced the aggression. Some dogs exhibit possessive-related aggression only to other dogs and there may not be a problem with humans.

Treatment:

It is much easier to avoid the situations and their consequences than to treat the problem. Deny access to “illicit” items such as rawhides, bones, etc. Feed dogs separately or at a distance at which they are not aggressive. Desensitization and counter-conditioning.

Protective/Territorial

This type of aggression is most obvious when the dog is in the yard and another dog passes, or when the dog is inside and a dog passes outside. Most or all dogs will bark, as this is the normal first step in the sequence of behavior characterizing protection. The problem occurs when the dog refuses to stop barking on command and/or becomes defensive and aggressive, and/or when the dog persists in the behavior despite cues indicating a contrary context. Some dogs become protective of their crates, places they sleep and/or an individual approach distance. Confined spaces may intensify the response – such as cars, restrictive chains. *The hallmark of territorial aggression is that the dog is not aggressive when it is removed from the territory* – the dog is fine with other dogs off-property.

Treatment:

Involves avoiding situations that dog perceives that it must defend or protect. Never leave the dog outside alone, and realize that the dog can become more aggressive in a fenced area. It is important that the owner have command control. DS&CC to approaches by other dogs can be done. A head collar is recommended ± anxiolytic medication

Redirected

Often seen when a dog is yelled at, physically punished, or otherwise thwarted from pursuing another aggressive behavior. For example, a person stops a dog from chasing the cat and the dog redirects its aggression to other dog. The dog usually goes after the nearest individual who is not involved, so a person, another dog, cat, etc. It can be difficult to diagnose as the circumstances that precipitate it are not often witnessed, and therefore it is not always recognized by the owner for what it is. When

one dog is aggressive to another dog, the owner often thinks that the dog is jealous or being competitive for attention. However, few or no data exist to support this.

Treatment:

Involves identifying the primary source of the dog's initial upset, if possible. Avoidance is key. If possible, address the behavior that is interrupted in the first place, and separate the dog and target animal so that there is no chase to interrupt. Separate the individuals involved in the redirected aggression when unsupervised and make sure that the recipient of the aggression has the most freedom. It is important to reward the aggressor for ignoring the victim. Other things such as a head collar and a bell on the aggressor to warn the victim, may also help.

Predatory

Predation is a maintenance behavior in carnivores, and therefore not all behaviorists agree that it's a "true" aggression. It is necessary for survival. There are two classes of predatory aggression: 1) Dogs that stalk, stare at, or silently pursue small animals - birds, squirrels, cats, other dogs, and sometimes infants, and 2) Dogs that chase moving objects.

Treatment:

Dogs that are predatory to other animals should *never* be off lead, unsupervised, or confined in a fence that other animals might cross. There needs to be adequate control using a head collar and voice command.

Status-Related Inter-Dog Aggression

Intact males are aggressive mostly to other males and show more aggression than neutered males. Females are aggressive mostly to other females. Aggression between familiar dogs involves dogs in the same household. It is also called "Social Aggression", "Intraspecific Aggression", and "Sibling Rivalry". It is a manifestation of canid hierarchical conflicts and underlying anxiety. The dogs are uncertain of their role in the hierarchy. The aggression is most commonly limited to one pair of dogs – even if other dogs are present. It is common between same-sex dogs and early spaying/neutering may help. Intra-household aggression is more severe than aggression between non-housemates with female-female aggression being the most severe. The aggressor tends to be younger and tends to have arrived more recently in the household. The fights are less frequent but more injurious than fights between non-household dogs. Common triggers include excitement, feeding, walking, owner arrival, control over resources, physical proximity, confining areas (doorways, hallways, etc.), and the owner's presence (the dogs may compete for attention). The owner tends to support victim (subordinate) and punish the aggressor (dominant). This can increase the aggression if the victim perceives a "coalition" between itself and the owner causing it to act more confidently. In many cases, the *owner's presence and behavior exacerbates the instability between the two dogs and fights may occur when the owner is present.*

Rarely, the aggression may persist in the owner's absence and fights may occur then, also. Common triggers for fights include: owner interference when the dogs interact in an attempt to change an established hierarchy, the owner inadvertently or deliberately encouraging a subordinate dog to try to establish dominance over the higher-ranking dog. It is important that the two dogs be allowed to "talk" to one another. Often, there is not a problem between the dogs as to which will be dominant - they understand the hierarchy and signal each other accordingly and appropriately. But, if the "normal talk" is interrupted and/or punished, what is left? There can be an escalation of the communication with growling, snapping and biting.

Onset usually occurs when the younger dog reaches social maturity (18 – 24 months old) and the hierarchy is not clearly established. It can also occur in evenly matched dogs, or when the "dominant" dog is aging or ill. In this case, the advanced age and/or illness do not allow the dog to maintain its status and this can result in ↑irritability → ↓tolerance to conspecifics.

Treatment:

Involves separating the dogs when not supervised. It is important to establish owner control with head collars or harnesses ± muzzles. If the male dogs are intact, neuter them. It is imperative to stabilize the pack hierarchy by identifying and supporting the dominant dog. This dog is to get attention first, be fed first, be given access to preferred resting places, allowed outside/inside first, and walked in front of the other dog. It is important to allow and reward ritualized signaling – the dogs need to be allowed to "talk to one another". It may be difficult for the owner to comply, as they may have been punishing the aggressor and comforting the victim. Attempting to superimpose a system of equality may make matters worse. There is a misconception that the dog that has "seniority" should dominate the new dog. In fact, a dog's social rank is determined by its ability to defend priority access to resources and not by seniority *per se*. An older or sick dog may not be capable of defending these privileges, or, the other dog may not offer subordination. In most cases, the dogs are getting "mixed signals": Dog A is dominant to Dog B. Dog A knows it and Dog B knows it and they signal each other appropriately. However the owner reinforces Dog B as dominant. Dog B *knows* that is submissive to Dog A, but it is getting reverse signals from "BIG Alpha". This becomes a source of confusion and anxiety...

Bottom Line... The aggression typically occurs in situations that include competition over valuable resources and aims at establishing a dominance-subordination relationship. The owner should interfere if there are excessive dominance displays or if the aggressive displays do not cease when the subordinate dog defers. They should call the dominant dog away and diffuse aggression in an "upbeat and jolly" manner, so as not to add to the arousal. They are to give preferential attention to the dominant dog and reinforce their own status. The problem may not be resolvable with two evenly matched dogs that are strongly motivated to be alpha. They are likely to fight until one succeeds in injuring the other. In this case, the owner should withdraw privileges from *both* dogs and interrupt dominance displays by *both* dogs. They should randomize

order of feeding and handling and desensitize and counter-condition to each other's proximity. It is important to look for ritualized signals and reward them. The prognosis is poorer if the initiator is younger than target, if a person has been bitten, and/or if the aggression is unpredictable.

Storm Phobia
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Anxiety

In humans:

“The apprehensive anticipation of future danger or misfortune accompanied by a feeling of dysphoria and/or somatic symptoms of tension (vigilance and scanning, increased motor activity, etc.)”

The focus of anxiety can be internal or external

Fear

In humans:

“A feeling of agitation and anxiety caused by the presence or imminence of danger”

In animals:

The subjective state of fear is *presumed to exist* when they exhibit specific behaviors, e.g. avoidance, crying, etc.

Fear

“An *adaptive* response that prompts an individual to remove or protect itself from dangers or noxious stimuli and thus increase its chances of survival”

Most fears are learned and can be unlearned with gradual exposure

Phobia

Fear reaction that

is **persistent** over time

is **consistent** in terms of what causes the fear

is **learned, irrational, not adaptive**

May be, but is not necessarily intense (hysteria, catatonia, panic)

“Normal” Fear vs. Phobia

Experiencing fear when lightning hits a nearby tree is normal (run, hide)

Experiencing fear every time there is a dark cloud in the sky is a phobia

Identifying Fear

Subjective-Easy / Objective-Difficult

'Panting': heat or fear?

'Whining': arousal or anxiety?

Look for *combinations* and at *context*

Possible Causes of Noise Phobias

Fears and phobias associated with noise are common in dogs

Can be seen as early as 9 weeks old

Problems severe enough to cause owners to seek professional help occur in approximately 20% of dogs (Beaver)

Usually develop over an extended period

Examples include:

Thunder

Fireworks

Gun shots

More subtle noises:

Dishwasher

Plastic garbage bag

Toilet paper dispenser

Diagnosis

Straightforward

Usually fear-eliciting sound is loud and distinct

Owner can usually identify:

When it occurs

Animal's response

Thunderstorm Phobia

Can show anxiety, fear with wind, rain alone, thunder, lightning, etc.

May or may not have been inciting cause to explain current behavior

Treatment *may* be simple:

Bring the pet indoors during the storm

Provide television or radio "noise"

Pet may be ok as long as owner is present

Provide "safe hiding place"

Bathtub

Laundry basket

Closet

Prognosis

Varies greatly and depends on the:

Individual

Duration of the phobia

Ability to control the stimulus during TX

Success of finding an effective controllable stimulus for DS&CC

Management

#1 – exert control over the pet and the environment

Ideally, except during training sessions, pet should not be exposed to the fear-evoking stimulus

Treatment should occur off-season

Management Steps

Identify stimuli and thresholds

Start with rain

Move onto thunder

Increase the volume of each stimulus

Establish a gradient of stimuli

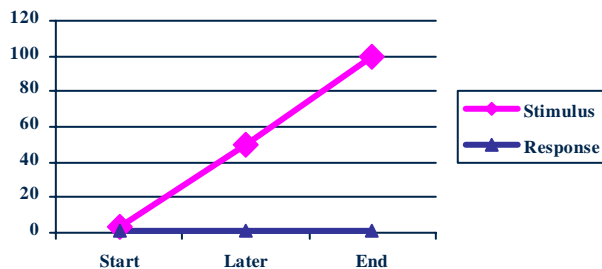
For desensitization & counter-conditioning (DS&CC)

Need to be able to control intensity

Audiotape/CD

Videotape

Desensitization



Retrain with rewards

Controlled situation

“sit/stay/relax”

Use highly motivating rewards

Counter-Conditioning

Flooding

Not generally recommended

Pet needs to be exposed to stimulus for time it takes to adapt and relax

Otherwise, technique can be reinforcing

Punishment

Under NO circumstances should punishment be used

Relaxation...

Keeping the pet calm and relaxed in general is an very important part of treatment

Sit/Stay/Relax exercises

Drug Therapy

Long term

Tricyclic Antidepressants (TCAs)

Clomipramine

Clomicalm™

Selective Serotonin Reuptake Inhibitors (SSRIs)

Fluoxetine

Prozac®

To decrease overall level of anxiety

Short term

Anti-anxiety Medications (Benzodiazepines)

Alprazolam

Xanax®

Tranquilizers

Acepromazine

*do not use alone, without an anti-anxiety medication such as

Xanax

To address the panic and motor activity

Other Treatments

Storm Defender Cape

Dogs sense the static charge buildup before a thunderstorm

“This cape has a metallic lining that discharges the dog's fur and shields him from this static charge buildup. It reduces his sensitivity to this charge”

DAP®

Dog-appeasing pheromone

“Mimics the properties of the natural pheromones of the lactating female”

Decreases overall level of anxiety

Anxiety Wrap

www.anxietywrap.com

Works by using gentle maintained pressure on the animal's body

Melatonin

Hormone that regulates sleep-wake cycle

Serotonin → Melatonin in pineal gland

0.1mg/kg/day

Prevention?

Early exposure of pet to as many different stimuli as possible

Habituation during early, sensitive periods *may* help prevent many of the fears and phobias seen in adult cats and dogs

Thunderstorm Phobia in Cats

No reason to assume that it doesn't occur

Cats tend to hide instead of exhibiting destructive behavior

Treatment?

Pyometra and Pyometra Treatment in the Bitch and queen : Where Do We Stand?

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Pyometra is a disease of the uterus mainly observed during the diestrus or anoestrus period of the bitch cycle. Cystic endometrial hyperplasia, mediated by progesterone and potentially aggravated by estrogens, was usually proposed as the initiating lesion in pyometra of dogs. CEH is characterized by degenerative tissues changes (cystic distention of glands, fibrosis, etc) that provide opportune conditions for establishment of uterine infections. The compromised uterus is then invaded by pathogens from the vagina which will extensively multiply mostly due to the excessive amounts of secretory fluids that have accumulated in its lumen and glands of the uterus. Early reports suggested that excessive or prolonged exposure to progesterone was responsible for the susceptibility to pyometra. However, if the importance of progesterone in the pathogenesis of the spontaneous disease is believed to lie in its suppression of immune responses, its stimulation of endometrial gland secretion which provides a suitable environment for bacterial growth, its functional closure of the cervix which inhibits drainage of uterine exudates and, perhaps most importantly, in its mediation of cystic endometrial hyperplasia. Recent work, suggests that this may not be so, and that a subtle (subclinical) uterine infection may occur first, providing the stimulus for excessive endometrial hypertrophy and hyperplasia. The associated increased glandular secretions would exacerbate the infection leading to increase secretion by endometrial glands and luminal epithelial cells progressing to pyometra. This hypothesis is supported by results of experiments done in dogs by Nomura and colls who have shown that during the luteal phase of the cycle, a variety of physical, biological, and chemical substances will cause the endometrium to proliferate. This observation is interesting as it may more easily than the classical theory explain the occurrence of unilateral or young animals pyometra.

The clinical signs include vaginal discharge, lethargy, polyuria Onset of signs is often polydipsia and occasionally vomiting

The traditional therapy for pyometra in both bitch and queen is surgical -ovariohysterectomy. This remains the recommended treatment in all cases except where the owner strongly desires to breed the bitch. Because of the insidious onset of the disease and its often equivocal clinical signs, patients are often in poor condition for anesthesia and surgery. Although treatment should not be unduly delayed, patients should be stabilized for surgery by the administration of broad spectrum antibiotics and intravenous fluids. Supportive measures should be continued during and after surgery;

antibacterial therapy should be continued for at least a week after surgery. In spite of these precautions, some complications like DIC may be expected.

Results of medical treatment of canine pyometra with prostaglandin F₂alpha (PGF₂alpha) have been reported to be encouraging in many cases. Apart from its luteolytic effect, PGF₂a is believed to mediate functional opening of the cervix to permit drainage of exudate and to promote myometrial contraction, facilitating this drainage, though experimental evidence for this is lacking in the bitch. The use of PgE₂ to open cervix before inducing uterin contraction is also nowadays documented and of practical interest. Furthermore, to fasten luteolysis and improve the overall immunologic condition of the animal the use of dopamine-agonist inhibiting prolactin secretion, therefore inducing luteolysis, has also been consistently described with significant results.

Great care should be exercised in calculating the dose as the therapeutic index is relatively small (LD₅₀ in dogs is approximately 5 mg/kg) and side effects are quite severe.

The condition of animals treated with PGF₂alpha does not improve during the first 48 hours after onset of treatment and may deteriorate, however substantial increases in uterine discharges should be observed rapidly in open pyometra, later in closed pyometra. DIC can be observed when treatment is begun in chronic cases and no significant responses are observed after a few days.

Initial response to PGF₂" treatment includes a transient increase in the amount of vaginal exudate, followed by a change in its character (to serous), and its eventual cessation. The leukogram returns to normal, although leukocytosis may be aggravated initially in some cases. In successful treatments, a 50% reduction of the uterine diameter is generally observed in 2 to 3 days. Observation of no significant changes in the uterine diameter after 2 to 4 days is generally of very bad prognosis. The treatment is discontinued when the uterine size is return to normal.

Concomitant broad spectrum antimicrobial therapy should be administered during PGF₂a treatment. Some bitches with pyometra are bacteremic and it is reasonable to suppose that increased uterine contractile activity may predispose bitches further to bacteremia or septicemia. Many different antimicrobials have been used successfully, but in vitro sensitivity studies and clinical evidence suggest that amoxycillin, amoxycillin plus clavulonic acid, cephalosporins or potentiated sulfonamides are good choices. It is recommended that the antimicrobial therapy be continued for 10 to 14 days minimum after the end of PgF₂alpha treatment. The bitch should be re-evaluated 2 weeks after completion of the treatment. Pyometra is prone to recur in treated bitches and is most likely to recur during subsequent periods of diestrus. Breeding should be attempted at the first estrus following treatment and at every subsequent estrus until the desired number of offspring have been obtained or until recurrence when retreatment or ovariohysterectomy should be considered.

More recently the anti-progestin agents have been proposed alone or in combination with PgF2 to treat pyometra. The results appear promising particularly in cases of close pyometra. Last the authors have recently developed a new transcervical endoscopic catheterisation technic (TECT) of the uterus which is associated with rapid resolution and treatment of the disease.

Tips and Tricks About Pregnancy Management in the Bitch

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In recent years, advances in small animal reproduction have allowed pregnancy management to evolve into a clinical service that has application from the beginning to the end of gestations. The advances provide for new and improved methods of pregnancy detection; improved use of ultrasound to determine gestational age, assess fetal well and predict the date of parturition; the use of progesterone assays to monitor luteal function and allow consideration of progesterone supplementation; use of progesterone assays and managed breeding information in the evaluation of prolonged gestations and in the timing of elective or interventional caesarian sections; use of fetal and uterine monitoring in management of critical pregnancies late in gestation; use of ancillary and supportive therapy in caesarian and induced deliveries; the potential to use anti-progestin therapy to induce pre-scheduled whelping in normal pregnancies;

Practical aspects of these different new points and technologies will be discussed during this presentation and include:

PREGNANCY FOLLOW-UP

Ideal pregnancy follow up should begin just after mating and/or AI. A perfect planning would allow for the prevention of the main problems related to pregnancy management including: early embryonic resorption, early detection of continuous estradiol production by follicular cysts, hypoluteism, fetal resorption and/or abortion, prolonged pregnancy, abnormalities of pregnancy, dystocia...

Many of the problems, diseases or troubles occurring during pregnancy can indeed be prevented by a proper pregnancy management which, even if expensive, would at the end prevent major and important economic or emotional losses.

A proper pregnancy follow-up should include at day 10-15 post LH surge a CL check by assessing progesterone and a total ovulation by a vaginal smear and/or estradiol assay. A 25-30 days post-LH surge is needed to realize an early pregnancy check allowing to confirm pregnancy, litter size and give reference data for the rest of the follow-up. A 30-35 days, progesterone check might be of major interest particularly in animals having had or susceptible to develop hypoluteism or early pregnancy losses. Then, in normal pregnancies, a X-ray control around day 60 is of interest to confirm litter size and

prepare parturition preventing major risks of dystocia. In abnormal pregnancies a more frequent follow-up may be needed.

PREGNANCY DIAGNOSIS.

Palpation of uterine swellings is possible by day 20, and easiest when they are of 3 cm diameter between days 28 and 32 and progressively more difficult after day 35 when individual swellings become less distinct from one another.

The currently available assays for the pregnancy-specific placental hormone relaxin are very accurate and definitive, since relaxin is not produced absent a pregnancy. The ELISA may detect relaxin as early as day 20-23 and the Witness test often detects pregnancy relaxin as early as day 26-30. False positives occur in cases of recent resorption or with retained placental tissue. False negatives as late as day 30 or 34 have been observed anecdotally, and negative results should be followed by U/S or re-assay one week later. U/S can detect pre-implantation vesicles by day 21 or earlier, embryonic masses by day 22-25, embryonic heart movement by day 24-27.

IMAGING. Ultrasound (U/S) exams can be very useful in pregnancy management. Its use to document pregnancy 3 weeks after the end of estrus (metestrus vaginal smear), or at day 26-28 after the estimated day of the LH surge can be a critical service. It is of major interest in bitches being judged infertile by ruling out early pregnancy losses and hypoluteism. It can also provide confirmation of normal development and a reasonable estimate of the stage of pregnancy and estimated whelping date in bitches in which the day of the LH surge was not determined. Accuracy of fetal number estimation decreases with smaller size and stage of pregnancy.

RESORPTIONS AND ABORTION. Multiple ultrasound studies have documented idiosyncratic embryonic and fetal losses that would have otherwise gone undetected, including partial as well as full resorption of litters as late as day 35. Even after day 35, fetal losses with related vaginal discharges may go undetected in fastidious bitches that lick away or ingest discharged materials. Ultrasound studies have also indicated that resorption may occur in 10 to 25 % of pregnancies, and total resorption in 5-10% of pregnancies. In one study, many instances of resorption were preceded by embryonic development retarded in relation to the expected time course.

PROGESTERONE SUPPLEMENTATION. There have been reports of instances of suspected luteal failure, luteal insufficiency, or "hypoluteoidism" in which peripheral concentrations of progesterone were observed to be exceptionally low in bitches which were confirmed to have resorbed or aborted their litter. Protocols have been suggested for progesterone supplementation in pregnant bitches in which progesterone falls before day 55, or declines more rapidly than expected in mid-gestation. The application of such progesterone replacement has involved successful pregnancies. However, excessive exogenous progesterone (or other progestin) or non-justified progesterone administration can compromise normal pregnancy and parturition and can result in abnormal sexual development (cryptorchidism, feminization, ..) or dead puppies that must be recovered by c-section.

PARTURITION. Normal parturition occurs as results of a rapid decline in progesterone from the 4-10 ng/ml observed in the preceding days to values below 2 ng/ml over an 12-24 hr period beginning 1-2 days prior to whelping. Monitoring and/or examining for the decline in progesterone can be an important tool in managing bitches with an apparent or presumed prolonged gestation, in evaluating bitches presented for dystocia, and in timing elective c'sections, especially in bitches in which the day of ovulation or day of the preovulatory LH surge was not estimated with accuracy. Temperature declines of 1°C or more during the 12-24 h prepartum, and is a parameter that can be monitored twice daily starting day 55-60 by owners who wish to be involved.

FETAL AND UTERINE MONITORING.

U/S detection of fetal HR below 180-200, often accompanied by bowel movements, is indicative of fetal distress and can be the basis of intervention in near-term bitches. U/S can also reveal fetal overgrowth or growth retardation, and other abnormalities including hydrocephalus, fetal anasarca, herniation of the ventral abdominal wall. Such case can then benefit form intervention. Uterine contraction activity has also been monitored with commercial devices designed for that purpose.

C-SECTIONS AND ADJUNCTIVE THERAPY.

In cases of pre-scheduled c'sections and when emergency surgery is required in bitches with obvious signs of dystocia at the expected whelping time, additional steps can be considered. Collection of a serum sample for progesterone analysis can be informative. "Term" dystocia in the absence of a normal prepartum decline in progesterone is unlikely. Whether small litters or other factors can result in a failure of the expected fetal-induced prepartum luteolysis bitches is not known. Singleton mummified fetuses can occur and result in prolonged gestation.

INDUCED PARTURITION. The availability of a progesterone antagonist as a veterinary drug marketed for termination of unwanted pregnancies has resulted in research and clinical application of the drug as a means to induced parturition. However, the values of any such therapy in normal pregnancy except for the possible convenience it might affords is not clear. It is difficult to recommend in light of anecdotal reports of such treatments yielding less timely or less successful results in some bitches with or without support of tocolytic doses of OT or PGF.

Neonate Care and Neonate Diseases

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Morbidity and mortality in Neonates

Normal neonates and Growth

Major causes of neonate Morbidity and Mortality

Preventive care and management of Neonate

Definitions

Morbidity

Mortality

Neonate and pediatric companion animals

Problems related to Neonates

Exquisitely Small

Dramatically sensible

Uncommunicative

1ST INDICATION OF TROUBLE IS OFTEN NOTICED WELL AFTER THE DISEASE IS ESTABLISHED

Clinically different from other members of their species

Response to routine therapies different than "routine fashion"

Not able to react against environment ==> Totally dependant

THE LIST OF EVALUATION OF STABILITY MUST BE EVALUATED FOR EACH PUPS/KITTENS

- EVERY 2-4 H. FOR THE FIRST 12-24 H.
- EVERY 6 H. FOR THE NEXT 3 DAYS
- EVERY DAY OR TWICE A DAY THERE AFTER

In High Risk litter this following up must be followed more closely

Regularly control vital functions

Regularly weight the animals: compare with prediction

Control sanitary condition of managment: sanitation, cleaning and traffic pattern in husbandery

Provide adequate nutrition to mother during pregnancy, parturition and lactation

Provide a quiet, warm and relaxing whelping cage: prevent overcrowding and related stress

Normal vital signs in the neonate

FIRST 24 hours

- TEMPERATURE: 34-36

- PULSE: 160 - 200 MIN.
- RESPIRATIONS: 10 - 20 / MIN. WITH SIGNIFICANT IRREGULAR RHYTHM AND NO ABDOMINAL EFFORT

24 hours to one week

- TEMPERATURE: 37 - A GRADUAL CLIMB
- PULSE: 200 - 220 / MIN.
- RESPIRATIONS: 16 - 35 / MIN. AND A REGULAR RHYTHM

After 1 week

- 37,5-38,5

Normal physical examination of the neonate (a)

Hair and umbilical cord

- HAIRCOAT : SLEEK AND COMPLETE
- SKIN COLOR: PINK
- FOOT PADS NORMAL
- UMBILICAL CORD: DROP AT 2 - DAYS, CLEAN, DRY , PINK

Muscle

- MUSCLE TONE PRESENT
- ABLE TO STAND AFTER 2 WEEKS
- HEAD RASING ARROUND 3 DAYS
- ABILITY TO CRAWL AT ONE WEEK

Urogenital function

- TESTES SHOULD BE DESCENDED AT WELPING THE LATEST AT 4 MONTHS

Cardiovascular system

- MUCOUS MEMBRANE: PINK, HYDRATATION: NORMAL
- PULSE QUALITY: GOOD - NO SINUSAL ARRYTHMIAS

Respiratory function:

- NOSE MUST BE MOIST, NARES SHOULD BE PATENT AND CLEAR: NO MILK, NO EXSUDATE, SEROUS DISCHARGE OR BLOOD PRESENT
- AIRWAYS FREE: LOUD SOUND NOT ROUGH

Gastrointestinal function:

- MUCOUS MEMBRANES HYPERHEMIC UNTIL DAY 3 - 4 PINK AND MOIST AFTER
- ABDOMEN NOT PAINFUL, ANUS MUST BE CHECKED: BLOOD, FEACES
- PERINEUM STIMULATION SHOUD INDUCE URINATION AND DEFECATION

Neurological examination

- MENACE REFLEX: AS EARLY AS 14 DAYS, NORMAL ONE MONTH
- ROOTING AND SUCKLING: AT BIRTH
- EXTENDED NECK REFLEX:
 - » kitten supported by the mastoid region,

from birth to 3 days flexor dominance: kitten should curl

from 4 days : extensor dominance: kitten should extend spine and limbs

Factors increasing the risk in a litter (a)

if present would place a litter into a high risk category

Management Problems

- POOR HYGIENE
- POOR REGULATION OF ENVIRONMENTAL TEMPERATURE
- MISMANAGEMENT OF THE PERIPARTUM PERIOD
 - » Prepartum queen's nutrition and vaccination status
 - » whelping
 - » nursing/dam's behaviour
- PARASITE CONTROL
- CHEMICAL CONTAMINATION – INSECTICIDES

Factors increasing the risk in a litter (b)

if present would place a litter into a high risk category

Infectious problems

- ENDOPARASITES: ROUNDWORMS/HOOKWORMS
- EXTERNAL PARASITES: FLEAS/TICKS/MITES
- BACTERIA: SKIN INFECTIONS / DIARRHEA
 - » Streptococci β hemolytic, E. Coli,
- VIRUSES

Congenital Abnormalities

- GENETIC
- TERATOGENIC
- MATERNAL MALNUTRITION

Neonatal mortality in kitten

	<u>USA 1978 (%)</u>	<u>Belgium 1990-92(%)</u>
<i>Nmber of kittens</i>	3468	540
<i>Number of litter</i>	790	159
<i>Mean kitten/litter</i>	4.4	3.9
<i>Death birth</i>	352 (10.2)	48 (8.9)
<i>Alive</i>	3116 (89.8)	492 (91.1)
<i>Death between</i>		
<i>0- 3 days</i>	412 (11.9)	39 (7.2)
<i>4 - 7 days</i>	63 (1.8)	9 (1.7)
<i>1 - 3 weeks</i>	84 (2.7)	3 (0.5)
<i>4 - 6 weeks</i>	143 (4.6)	5 (2.5)
Total	1072 30.9%	104 19.25%

Causes of Neonatal mortality

Death during pregnancy and parturition : 23.5 %

Congenital diseases: 20.6 %

Neonatal pathologies: 35.2 %

Unknown causes: 20.6%

Neonatal mortality: range: 10 to 40 %

Causes of Neonatal mortality

Abnormal maternal behaviour: 15 to 20 %

Lactation problems: up to 40 %

Mammary glands pathologies

Maternal pathologies preventing milking

- PLACENTAL RETENTION
- METRITIS
- HYPOCALCEMIA AND ECLAMPSIA

Fading neonate syndrome: up to 40 %

Cardiomyopathy

Hemorrhagic syndrome

Diarrheas

Causes of Neonatal mortality

Infectious diseases

- VIRUS
 - » Leukemia (FeLV)
 - » Peritonitis (FIP)
 - » Panleukopenia
 - » Herpes and rhinotracheitis
- BACTERIAS
 - » E. Coli, Salmonella, Streptococci β hemolytic
 - *Umbilical infection*
- PARASITES
 - » Ascaris
 - » Toxoplasma Gondii
 - » Coccidiosis
 - » Fleas, Ticks, ...

Signs of troubles in Neonates

Continuously crying

- NORMAL NEWBORNS WILL CRY WHEN COLD, AWAY FROM MOTHER OR HUNGRY BUT WILL STOP AFTER A FEW MINUTES

- SICK NEONATES WILL CONTINUE VOCALISATION FOR PROLONGED PERIODS, EVEN WHEN APPARENT CAUSE OF DISCOMFORT HAS DISAPPEARED

Continuous restlessness or listlessness

- NO RESPONSE TO STIMULATION

Inability to find or maintain nursing position

- SICK NEONATES CAN NOT OR WILL NOT SUCKLE

Continued flexor position after 4 days

Regurgitation, discharge from any orifice or any sign of infection

Maternal neglect of one member of the litter

Inefficient weight gain

- NORMAL PUP/KITTEN PRESENT AFTER 2 TO 3 DAYS AN EARLY LOSS OF WEIGHT
- NORMAL PUP/KITTEN DOUBLE IS WEIGHT BY TWO WEEKS

Inability to follow the dam/queens or the others littermate

Signs which should place a neonate in an critical status

PUPS/ KITTENS UNRESPONSIVE TO THE ENVIRONMENT, NOT ROUSABLE, MENTAL STATUS DETERIORED

Dehydration

- ORAL MEMBRANES SHOULD BE MOIST. IF TACKY, DRY THEN CIRCULATORY STATUS MAY BE IMPAIRED

Lip foam or body temperature below 31°C

Increased respiratory rate or abdominal respiration

- INDICATE CARDIOPULMONARY PROBLEMS

Blue or pale mucous membranes

- NORMAL MUCOUS MEMBRANES ARE HYPERAEMIC UNTIL DAY 4-7

Cachexia

- A NORMAL PUP/KITTEN SHOULD BE PLUMP AND ROLLY-POLY
- A BLOATED ABDOMEN IS A HEALTHY SIGN ONLY AFTER FEEDING
- OTHERWISE IT MAY INDICATE RETENTION OF URINE OR FAECES, AEROPHAGIA OR BLOATING

Specific problems in the neonate and paediatric patient

Hypothermia

Fading neonate syndrome

Hypoglycaemia

Dehydration

Hemorrhagic syndrom: numerous bleedings, numerous causes:

INFECTION, POOR GENERAL CONDITION, HERPES

Toxic milk syndrom - often associated with infections in the mother: uterus, mammary glands

CRYING PUPS, BIG ABDOMEN, RED RECTUM,
TREAT THE MOTHER!!

Swimming puppies

massage

Specific signs of troubles in the neonate and paediatric patient

Crying and abdominal pain

GI troubles: constipation, diarrhea, colic, abnormal GI bacterial content

Check mammary glands and milk

Hypothermia and hypoglycemia

Quiet not reacting pups, rejected by the mother

Umphalite => risk of peritonitis

Impetigo => antiseptia

Dead ==> always ask a necropsy!!

Hypothermia

Normal body temperature:

- AT BIRTH: 94-96
- AFTER 3-4 DAYS: OVER 96
- AFTER 1 WEEK: AROUND 99

kittens are heat seekers at about 2 days

As Kittens cool, their ability to move decreases

Neonates are unable to react to cool

- VASOCONSTRICTION OR DILATATION, INCREASE METABOLISM UNTIL ABOUT 1 WEEK OF AGE:
THEY ARE TOTALLY DEPENDANT ON EXTERNAL SOURCE OF HEAT

Crude estimate of body temperature:

- CHILLED NEONATE 27°C
 - Restless, continuous crying
 - increase respiratory rate: 40/min.
 - Increased heart rate: + 200/min.
 - Good muscle tone
 - red mucous membrane
 - Cool skin

Prevention of hypothermia

– OPTIMAL AMBIANT TEMPERATURE	
– DAYS POST PARTUM	ENVIRONMENTAL TEMPERATURE
1 - 5	29-32
6 - 20	27
20	GRADUALLY LOWER TO 21-24

IF PATIENT IS SICK MAINTIEN TEMPERATURE ON THE HIGH END OF THE RANGE
OR INCREASED TO 1°C ABOVE RECOMMENDED
SOME RECOMMENDED 2°C ABOVE THAT LIMIT

Heating lamps, incubators, warm bottles, hot water gloves, heating pads: caution: risk of burns !!

Rewarming a chilled neonate

Rewarming a chilled neonate must be done slowly particularly if it is less than 1 week of age

- KEEP THE COAT DRY
- KEEP HUMIDITY AROUND 55 TO 65%
- DON'T FEED ANY KITTEN IF TEMPERATURE IS LESS THAN 32°C
- LISTEN TO BOWEL SOUNDS, MOVE PATIENT OFTEN

Methods

- HEATING PADS, HOT WATER GLOVES, HOT WATER BOTTLES BUT SKIN WARMING BEFORE VISCERA AND RISK OF BURNS
- CONSULT VETS: INSPIRATION OF WARM AIR, WARM FLUIDS (NO MORE THAN 1°C OVER BT) IV, IP OR IO, WARM WATER ENEMAS

Fading neonate syndrome

Term used to describe a set of problems rapidly and irreversibly leading to death
Final pathway of a number of ethiology.

A fading neonate is born healthy

Numerous deregulators may lead to the condition:

- MANAGEMENT PROBLEMS
 - Poor hygiene
 - Poor regulation of environmental temperature

- Mismanagement of peripartum period
- Parasite control
- INFECTIONS AND TRAUMA
- CONGENITAL ABNORMALITIES

Usually recognised in the first week of life

Death occurs within 4 days after clinical signs

Symptoms

- PROGRESSIVE ANOREXIA AND/OR CACHEXIA THAT COMPROMISES AN INDIVIDUAL TO THE POINT OF MORBIDITY.

Incidence

- 25 TO 30 % SOMETIME MORE OR LESS IN SPF CONDITION

Causes

- IMMATURE PHYSIOLOGICAL PROCESSES,
- INBORN METABOLIC ERRORS
- ANATOMIC BIRTH DEFECTS AND DYSTOCIAS
- ANOXIA AND INADEQUATE NUTRITION
- INFECTION
- TRAUMA AND CANNIBALISM - ENVIRONMENTAL PROBLEMS.....

Pathway

- THE INITIATOR OF THE SYNDROME PREVENTS THE NEONATE FROM SEEKING HEAT ===> DEPRESSION, ANOREXIA
- THE NEONATE BECOMES HYPOTHERMIC
- THE NEONATE IS UNABLE TO NURSE AND IS DEPRIVED OF COLOSTRUM
- DEHYDRATION AND CIRCULATORY INSUFFICIENCY OCCUR
- HYPOGLYCAEMIA OCCURS
- THE IMMUNE STATUS DETERIORATES
- THE NEONATE SUCCUMBS TO INFECTION (β HAEMOLYTIC STREPTOCOCCI AND E. COLI)
- OR CARDIOPULMONARY FAILURE AND COLLAPSE OR SHOCK

EMERGENCY !!

Hypoglycaemia

Glucose reserve very poor: less than 24h.

Predisposing factors

- PREMATURE BIRTH
- POOR MATERNAL NUTRITION
- MATERNAL DIABETES
- LOW BIRTH WEIGHT

Clinical signs:

- NEUROLOGIC DYSFUNCTION
 - changes in mental status
 - tremors, seizures, irritability
 - polyphagia

Dehydration

Difficult to assess

Mucous membranes should be moist

- BECOME DRY AFTER 5 - 7% DEHYDRATION
- LOSS OF ELASTICITY AFTER 10%
- CIRCULATORY COLLAPSE OCCURS AT 12 - 15%

Rehydration is necessary: IP, IO

Methods of administration

IV IP IO