INVITED REVIEW

STRESS AND SUDDEN DEATH IN VETERINARY MEDICINE

ESTRESSE E MORTE SÚBITA EM MEDICINA VETERINÁRIA

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SUMMARY

Stress syndrome affects domestic and wild animals. Due to the complexity of stress pathophysiology, it is difficult to recognize the specific responses to different stressor agents and, consequently, its diagnosis and control. It is a syndrome with no established biological parameters, with large interanimal variability, which requires species-specific studies in order to establish score cards to help veterinarians and animal handlers to recognize it. Studies about animals under stress are scarce and it is believed that the stress response among different species is very differentiated. This article reviews the pathogenesis and consequences, as well as the impact of stress on different animal species.


RESUMO

O estresse é uma síndrome que acomete animais domésticos e selvagens e cuja fisiopatologia é bastante complexa, o que dificulta o reconhecimento de respostas específicas aos diferentes agentes estressantes e, consequentemente, o diagnóstico e controle. Trata-se de uma síndrome sem parâmetros biológicos estabelecidos, com grande variabilidade interanimal e que requer estudos espécie-específicos de forma a estabelecer marcadores que possam ajudar veterinários e tratadores no seu reconhecimento. Os estudos sobre estresse em animais são escassos e acredita-se que a resposta ao estresse seja bem diferenciada em cada espécie animal. Revisa-se neste estudo a patogenia, as consequências e o impacto do estresse sobre as diferentes espécies animais.


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INTRODUCTION

Stress is a subject that has been prominently discussed in human medicine and psychology for a long time. Scientific studies have shown that stress leads to health problems, behavioral changes and affects interpersonal relationships (SELYE, 1956).

The concept of stress is universally understood, but even after several decades of research on the subject, there is not yet an established clinical setting. This happens because stress is an abstract concept and some important questions still remain, such as, the biological parameters that best indicate stress, the lack of non-specific responses that characterize each type of stressor, the interanimal variability response to stress and the inability to establish a correlation between stress measurement and its impact on animal welfare (PACHALY et al., 1999).

The pace of life today, with the technology and its consequences, creates problems not only for humans but also for animals. They now live completely different than what would be natural and, therefore, have to adapt to a new routine. Noise and air pollution, confinement, loneliness, management, transportation and many other difficulties are now part of the daily routine of animals (SCHOLZ & REINHARDT, 2007). Other situations, such as being in the presence of visits, bathing, trimming, not having contact with other animals, being handled, transported, being fed differently than if they were in nature, may all seem harmless, but they can generate multiple stress events, such as, fear, aggression, anxiety, irritability, lack of affection, reproductive and metabolic problems, as well as deviant behavior that may progress to death in extreme cases (SCHOLZ & REINHARDT, 2007; SCORZA et al., 2010).

Thus, to suppose that all these changes do not exert any impact of the lives of these animals would be at least speculative. Even knowing that every organism can compensate for some level of stress individually, the hypothesis that all animals, whether a pet, production or wild, may be suffering from stress and that this disease may have serious consequences should be considered.

STRESS

Stress is the sum of biological reactions to any adverse stimulus, be it physical or emotional, internal or external, that tends to disturb the homeostasis of the organism (BLOOD & STUDDERT, 2002). This is a state manifested by a set of specific responses triggered by the body and different types of agents that are called stressors (SEYLE, 1956). The term stress denotes the state resulting from the perception of the stimuli that causes emotional excitement and disturbs the homeostasis, triggering a process of adaptation characterized, among other changes by increasing noradrenaline production and release, thus resulting in several systemic manifestations with physiological and psychological disturbances (MARGIS et al., 2003).

Humans and animals respond to exposure to stress by mobilizing the neural, neuroendocrine and metabolic processes. Each stressor produces a specific neurochemical signature, involving central and peripheral mechanisms. These neurochemical changes occur together with physiological, behavioral and experiential changes (FINK, 2007).

Hans Selye, the Canadian physician that pioneered the study of stress over four decades of research, described a single and general adaptation of the body when submitted to stressors, which was named “General Adaptation Syndrome” and can be divided into three stages that differ over time. The first stage is called the alarm reaction and occurs when the animal faces the stressor. At this stage, there is general mobilization of the body in an attempt to get rid of the stressor, with the participation of the sympathetic nervous system in the stimulation of the adrenal medulla to release catecholamines (SEYLE, 1959; BONDAN & ORSINI, 2006).

The second stage, known as adaptation or resistance occurs as a result of the first, when the stressor stimulus is still maintained. At this stage, the sympathetic nervous system enters hyperactivity and there is intense stimulation of the neuroendocrine system to release glucocorticoids by the adrenal cortex (BONDAN & ORSINI, 2006). The body remains active, albeit in a less intense level, in order to keep the resources available to face the stressor (OLIVEIRA, 2006).

The last stage, called exhaustion, occurs when the stressor is maintained until the animal does not have the ability to adapt any longer. There is no rest or return to homeostasis (SEYLE, 1956). Energy reserves are depleted and the organism goes into exhaustion, becoming vulnerable, resulting in decreased ability to think, remember and act, as well as decreased immune system responsiveness (OLIVEIRA, 2006). The process evolves until animal death by multiple organ failure. This phase is not necessarily irreversible, it depends on the importance of the affected organs. Moreover, animal death may happen still during the alarm stage, caused by the process organic imbalance (BONDAN & ORSINI, 2006).

CAUSES OF STRESS

Stressors are considered to be any environmental, chemical, physical, nutritional, psychological or behavioral changes that stimulate neuroreceptors. The nervous system analyzes and processes the impulses from the receptors and triggers responses to the effector organs, inducing the occurrence of various reactions that have been previously described.

The stressors can be divided into four categories: somatic agents, which are agents that act directly on animal body, such as, strange sounds, images and smells, handling, heat, cold, pressure, abnormal stretching of muscles and tendons, effects of drugs or chemicals; psychological agents, which are apprehension feelings that can intensify to anxiety,
fear, terror, rage and frustration; behavioral agents, which are the lack of social interactions, food, natural stimuli, changes of biological rhythms and management mistakes in general; and mixed agents, which can be bad nutrition, prolonged confinement, the presence of infectious agents, parasites and toxins, burns, surgery, drugs being used and physical and chemical immobilization (PACHALY et al., 1993; BONDAN & ORSINI, 2006).

Physical restraint was identified as one of the main stress factors in collared peccary (Tayassu tajacu) (BATISTA et al., 2009). Under stress conditions by physical restraint, which is a necessary procedure for management practices, such as, identification, examination, collection of biological material, as well as transportation, these animals showed clinical manifestations of stress syndrome, malignant hyperthermia or capture myopathy, which will be discussed later, and can result in animal death.

Behavioral and metabolic changes of dogs subjected to air transport have also been described, correlating them to the fear and stress caused by this practice. Increasing concentrations of salivary and plasma cortisol, as well as tachycardia and behavioral changes have indicated that transport is a cause of intense stress in this species (SHANNON et al., 2002).

**STRESS PATHOPHYSIOLOGY**

When a stress situation arises, several hormones called stress hormones are released and many body functions are changed. To maintain the normal state called homeostasis, the body has several negative feedback mechanisms that check plasma levels of each hormone and activate or not their production. This means that from a certain plasma concentration, the hormone inhibits its own production and under normal circumstances ensures that plasma concentration is set at constant level (SCHOLZ & REINHARDT, 2007). The most released hormone during stress is cortisol controlled by the hypothalamus, the main center integrating the activities of visceral organs and a major contributor to body homeostasis. It also connects nervous and endocrine systems, and regulate many important body functions, such as heat, sleep-wake pattern, blood pressure, respiratory function, control of food intake, fat metabolism and water balance (SCHOLZ & REINHARDT, 2007).

Body stimulation by a stressor agent occurs through specific organs and systems. The response to receptor stimulation by a stressor can follow three neuroendocrine pathways: a voluntary motor one; another involving the autonomic nervous system and is related to acute stress; and lastly, one that involves the neuroendocrine system and is related to chronic stress (PACHALY et al., 1993).

**Voluntary motor pathway**

The nervous impulse generated in the body by the action of somatic stressor agent goes to the spinal cord and is transmitted to the thalamus and cortex, where it is processed and transmitted to the motor areas that, through the basal ganglia and spinal cord, pass the information on to the peripheral nerves (FOWLER, 1986). The thalamus relays the nervous impulses to the cerebral cortex and is responsible for the conduction of impulses to the appropriate regions of the brain where they are processed. The thalamus is also related to changes in emotional behavior (CUNNINGHAM, 2004).

Generally, animals respond to stressful situations in a pattern characteristic of each species. The attitudes observed in response to the action of the voluntary motor system may vary widely, including dodging, hiding, struggling, running, vocalizing, trying to escape harm or even being aggressive. The animals may also be defensive or protective (PACHALY et al., 1993).

**Autonomic nervous system pathway**

The stressor may also act on the neuroreceptors, generating impulses that after being processed by the central nervous system stimulate the sympathetic nervous system, which acts on the adrenal medulla and determines the release of large amounts of catecholamines in the blood. These catecholamines eventually induce a series of events in the body, determined by their interaction with the respective receptors in target organs. Thus, sympathetic activity causes alertness, thus increasing the frequency and strength of cardiac contraction, splenic contraction and reduced blood circulation to peripheral regions in an attempt to increase the distribution of blood to vital organs. Catecholamines also cause tachypnea, in order to increase oxygen availability to the body and glucose release by the liver in an attempt to increase both the energy available to the muscles and circulating lymphocytes in case any damage occurs (BONDAN & ORSINI, 2006). Therefore, the sympathetic autonomous nervous system, in general, encourages actions that mobilize energy, allowing the body to respond to stress situations (CUNNINGHAM, 2004).

**Neuroendocrine pathway**

This pathway is related to the chronic stimulation of the body by stressors, including the so-called chronic stress. Undesirable metabolic responses are the direct result of excessive cortisol production, observed in response to continuous stimulation of the adrenal gland cortex, and the animals experience physical or somatic, psychological and behavioral changes (PACHALY et al., 1993).

The brain, particularly, the hypothalamus and related structures of the limbic system, regulate the internal and external stimuli and pituitary, which in turn controls the function of other endocrine glands through tropic hormones that act directly on body functions. The feedback of glands and target tissues is received in the form of biochemical and neural messages and provides the internal stimuli that affect the brain and hypothalamus regulatory controls (CUNNINGHAM, 2004).

Contrary to the effects of the autonomous nervous system, the hormones secreted in the neuroendocrine
system have a lasting effect on the body. The somatic changes related to chronic stress may include clinical signs such as, weakness and muscle atrophy, tremors, weight loss, increased abdominal size, hypertension, impairment of wound healing, polyuria and polydipsia. Moreover, there is a decrease of the immune response and higher probability of infections and vaccine failure as well. In addition, chronic stress specifically affects white blood cell counts, suppressing the activity of lymphocytes and eosinophils (FOWLER, 1986).

**Figure 1**: Neuroendocrine pathways through which stressor agents affect the body (FOWLER, 1986).
The impact caused by stress on domestic animals has been underestimated over time. Only in recent years we began to question how much stress an animal can bear before presenting health and behavior problems (SCHOLZ & REINHARDT, 2007).

As the commercial herds multiply, it has been observed the emergence of reproductive problems due to confinement and management practices that sometimes lead to chronic stress, triggered by a series of hormonal changes. For example, high plasma concentration of cortisol for a few hours or days near ovulation cause hormonal changes, reducing LH levels, cystic follicles are formed and the female can remain in constant estrus (nymphomania) or anestrus (LEITE, 2002).

Some studies show that some stress factors in domestic animals lead to production loss, reproductive and behavioral disorders and important physiological changes (BROOM et al., 2001; BROOM & CORKE, 2002; LEITE, 2002). Heat stress leads to decrease of milk production due to lower dry matter intake by the animal, resulting in prolonged negative energy balance postpartum and increasing intervals between births. Moreover, fertility rate drops, as evidenced by the absence of estrus and decrease of a dominant follicle (RENSIS & SCARAMUZZI, 2003).

It was observed that heat stress in dairy cows induce several metabolic and behavioral changes, especially increasing body temperature and respiratory rate. Additionally, there were reductions of food intake, reproduction, pregnancy, lactation and consequently, production efficiency (SILVA et al., 2010).

Meat pH of sheep transported smoothly on quiet roads was measured twenty four hours after the slaughter, and the pH was less acidic compared to meat of sheep transported in more abrupt conditions (BROOM et al., 2001), thus showing how stress caused by transportation affects meat quality of livestock. The pH changes meat characteristics such as color, water retention capacity and tenderness, and acid pH can significantly lower the commercial value of the product (DANTZER & MORMEDE, 1983).

The confinement and large amount of food offered to broilers aiming at fast growth, also result in stress and health problems for the animals. Most problems are caused by the lack of movement that result in reduced ability to walk, tibial dyschondroplasia, femoral head necrosis and valgus and varus syndrome (BROOM & CORKE, 2002). Egg production and quality were also affected when chicken were under stress. The birds that lived in cages were more susceptible to heat stress, since behaviors that would aid heat loss were difficult or even prevented. Furthermore, the higher density of birds in cages helped to maintain the heat generated by the animals (MIRANDA & SILVA, 2009).

Although all species can be affected by stress, pigs appear to be the most susceptible (DELLALIBERA et al., 2009). In stressful situations or even as a result of administering volatile anesthetics (markedly halothane), they present clinical signs such as, porcine stress syndrome, acute muscle necrosis, malignant hyperthermia and soft, pale and exudative muscle syndrome (DELLALIBERA et al., 2009).

Malignant hyperthermia in pigs was described for the first time by Hall et al. (1976), who showed that it was a genetic disorder that occurs in both humans and animals, caused by an increase of myocardial metabolism caused by the stimulation of beta-adrenocceptor. Dellalibera et al. (2009) described it as a non-infectious myopathy related to intensification of breeding processes that leads, in some breeds, to selection of individuals markedly hypertrophic and highly susceptible to stressful conditions. The clinical signs of this syndrome manifest commonly as tremors and muscle rigidity, tachypnea, tachycardia and hypertermia. Clinical and pathological changes may include metabolic acidosis, myoglobin, hyperkalemia and hyperglycemia, often followed by circulatory collapse and death (FOX et al., 2002; McGAVIN & ZACHARY, 2007). Various stressful situations on the farm, such as mating, vaccinations, fights, transportation to other places or to the slaughterhouse can trigger this condition (SOBESTIANSKY & BARCELLOS, 2007).

In the United States in 1950, the introduction of management practices that were not usual for the animals (high protein feeds and antibiotics) coincided with the incidence of gastric ulcers in pigs. These and other innovations such as confinement, changed animal temperature and behavior and contributed to incidence of stress in this species (BISPO & PEREIRA, 1994).

Sudden death as the name says it, occurs suddenly, within maximum of one hour from the onset of clinical signs or the first twenty-four hours after the last time the animal was seen alive. The causes can vary largely, and include heart or gastrointestinal disease, bleeding, malnutrition, dehydration, respiratory problems, sepsis, among others (VON EYE, 2004).

A retrospective study (OLSEN & ALLEN, 2000) determined that the causes of sudden death in dogs were: heart problems (21.9%), followed by toxicity (16.6%), digestive problems (13.2%), trauma (12.6%), hemorrhage not associated with trauma (6.6%), malnutrition or dehydration (5.3%), respiratory problems (4%), urogenital problems (3.3%), nervous system diseases (1.3%), peritonitis (1.3%), pancreatitis (1.3%) and undetermined causes (12.6%).

Another retrospective study (OLSEN & ALLEN, 2001) determined that the main cause of sudden death in cats was trauma (39.2%), followed by heart problems (20.3%), bowel disease (7.6%), respiratory problems (6.3%), urinary tract disease (5.1%), problems related to feline leukemia virus (3.8%),
meningoencephalitis (1.3%), hepatic necrosis (1.3%), sepsis (1.3%), hemorrhage not associated with trauma (1.3%) and undetermined causes (12.7%).

Platt (1982) showed that the causa mortis in horses is due to gastrointestinal problems, pointing bloat, intussusception and gastric rupture as main factors, followed by heart disease, trauma and non-apparent infections. In this study, the cause of sudden death could not be determined in 13% of the cases. Brown (1988) also reported gastrointestinal problems as the main cause of sudden death in horses, followed by respiratory, cardiovascular and nervous central system problems and several others, among them, toxicity, infection and injury by firearms. In 30% of the horses, it was not possible to determine the cause of death.

Sanford (1994) studied 38 pigs of commercial herds that died suddenly and were autopsied in order to determine the cause of death. In 71% of the animals, it was found twisting or gastrointestinal problems, followed by toxemia and fetal retention (10.5%), heart problems (2.6%), pneumonia (2.6%), pyelonephritis (2.6%), alveolar fibrosis (2.6%), endocarditis (2.6%). *Streptococcus sp.* infection (2.6%) and undetermined causes (2.6%). In addition, many identified stressor agents could be related with mortality of these animals, such as high temperatures, fights, confinement, transportation, lack of exercise, among others.

A close analysis of the statistical data of the retrospective studies cited shows that in a large part of the evaluated cases, it was not possible to determine the cause of death and so one can conclude that much remains to be studied with respect to the unknown causes of sudden death. Most of these animals may have had stress related complications that were not detected during the autopsy. Therefore, the biochemical markers of stress are important, both macroscopic and microscopic findings of the autopsy, in order to create a protocol containing these pathognomonic changes that can help to diagnose this syndrome.

### DEATH BY STRESS

Sudden death associated with stress has been reported in several studies with humans (CRITCHLEY et al., 2005; LATHERS & SCHRAEDER, 2006; FINESCHI et al., 2010) and some with animals (SWAYNE & SAIF, 1990; PINSON, 1997; CERETTA, 2009). It is usually associated with cases of extreme agitation, resignation, fear or anxiety. In veterinary, the agents somatic, psychological, behavioral and mixed, as described previously, are the stressor agents responsible for acute or chronic stress and can potentially lead the animal to death (PACHALY et al., 1993).

In veterinary, the best known cause of death resulting from stress occurs due to restraint procedures (PACHALY et al., 1993; BATISTA et al., 2009). The restraint procedures, either by chemical or physical means, can lead to potentially fatal reactions, and may be preceded by loss of consciousness and should be regarded as extremely alarming because it can lead to death. Among the various causes of unconsciousness, state of shock, ventricular fibrillation, cholinergic bradycardia, anoxia, hypoglycemia, hypothermia, brain concussion and bruises should be mentioned. Among the most common causes of death by restraint, acidosis, ventricular fibrillation, cholinergic bradycardia and capture myopathy should be mentioned (PACHALY et al., 1993).

### Acidosis

The acid-base imbalance is very common in carnivores, resulting either from relative or absolute excess of organic and inorganic acids that were not expelled by the kidneys or lungs, or reduced base in the body. It manifests itself by acidemia with absolute or relative reduction of bicarbonate concentration. The pathophysiology may be understood by increasing acids and decreasing bases (bicarbonates), decreasing partial pressure of carbon dioxide in arterial blood (when attempting to offset or compensate) and decreasing bicarbonate/carbonic acid ratio (FANTONI & CORTOPASSI, 2010).

### Ventricular Fibrillation

Ventricular fibrillation is characterized by a completely irregular heart rhythm with variable potential of chaotic atrial fibrillation, indicating disorganized ventricular depolarization and risk of death. It may indicate an imminent cardiopulmonary arrest, when it is necessary immediate restoration of the heart rhythm in order to preserve life (TILLEY & GOODWIN, 2002).

The primary cause of ventricular fibrillation leading to death is the release of adrenaline and noradrenaline during the alarm or flight reaction under stressful situations (PACHALY et al., 1993). Chatecolamines promote tachycardia as a physiological effect. The simultaneous presence of acidosis due to excessive muscular activity and hypoxia as well, leads to an increased myocardial sensitivity to those vasoactive amines. As a result, the fibrillation process is installed. Clinically, the struggling animal in the agonizing process is unfortunately often confused with simple resistance to restraint. Atrial fibrillation causes the ventricles to become unable to pump blood, which results in circulatory failure, followed by unconsciousness and death (PACHALY et al., 1993).

### Cholinergic bradycardia (Vagal bradycardia or syncope)

The stimulation of the hypothalamic centers that occurs in stressful situations often leads to considerable stimulation of the sympathetic nervous system, in addition to less stimulation of the parasympathetic nervous system, resulting in adrenergic response which is the typical alarm reaction, tachycardia and hypertension (SEYLE, 1956).

Moreover, during the restraint procedure, there may be excessive pressure on the eyeballs, carotid sinus and abdomen, causing intense hypothalamic stimulation.
that culminates in greater stimulus of the parasympathetic nervous system. In such situations, the cholinergic reaction triggered is greater than the adrenergic reaction, resulting in severe decrease of pulse and heart rate, significant hypotension, loss of consciousness leading to death due to hypovolemic shock (PACHALY et al., 1993).

Capture myopathy

Also known as stress or effort myopathy, capture myopathy is a degenerative muscle disease with extremely reserved prognosis, associated with the stress of capture, physical and/or chemical restraint, and transport of wild animals. It may present itself in the forms acute (1 to 12 hours), subacute (7 to 14 days) or chronic (weeks) and it is mainly observed in animals of the Family Bovidae, Equidae and Cervidae. Predisposing factors are considered to be fear and anxiety, hyperthermia, intense muscular effort, constant muscular tension and prolonged transport. The localized anoxia due to the contraction of certain muscles in abnormal positions, is another determinant for the occurrence of the disease (PACHALY et al., 1993).

The pathogenesis of capture myopathy involves two interrelated theories: the occurrence of tissue anoxia and the change in pH, leading to death of muscle fibers and subsequent release of potassium, myoglobin, lactate, substances that play an important role in the genesis of the disease. Potassium acts on heart muscle producing fibrillation and the hyperkalemia would explain the death of patients with heart failure. On the other hand, hyperhemoglobinemia due to the extreme nephrotoxicity of myoglobin, leads to acute tubular necrosis, which in turn, causes acute renal failure. Regarding acidosis, the bicarbonate is not sufficient to compensate for the increasing lactate serum levels causing the pH to fall to 6.9 and 7.0 instead of the standard 7.35 to 7.45. This leads to a state of shock, and the inability to adequate tissue perfusion generally leads to organ failure (DIAS, 1993).

Commonly observed clinical signs include difficulty in maintaining station, stiffness and pain on palpation of limbs, weakness that can lead to paralysis, prostration and recumbency. Dyspnea and tachycardia are also observed. The main laboratory changes are acidosis, high levels of creatine phosphokinase and lactic dehydrogenase, and less frequently hyperkalemia (DIAS, 1993).

The occurrence of myopathy has been reported during the capture of an emu that struggled a lot during transport and where it was placed. Two days after the incident, the emu developed lameness in one limb and became prostrate, recumbent and finally died. The autopsy showed a large hematoma in the thigh area, but without fracture. The other organs showed no macroscopic visible changes. Histopathology showed multifocal areas of degeneration and necroses of the muscle tissue examined (MUNHOZ et al., 2007).

These and other causes have been described in veterinary medicine as leading to sudden death related to stress. Pinson (1997) described the case of dog sudden death after an episode of extremely aggressive behavior, which happened 24 hours after bathing and grooming procedure. The autopsy of the dog was required, since there were no macroscopic lesions that could help to clarify the cause of death. The histological examinations of several organs showed small lesions such as liver congestion and renal medullary mineralization foci. Significant tissue damage was observed in the heart tissues and the myocardium had foci of coagulation necrosis and inflammation. Cause of death was attributed to a lethal arrhythmia associated with myocardial necrosis and histological examination of the time of injuries was consistent with the episode of aggression and stress that occurred 24 hours prior to death.

Another case of sudden death due to stress happened to laboratory mice that were subjected to immobilization stress. The death of these animals happened in the fifth or sixth hour from the onset of stress. Severe morphological changes were found in the cardiac muscle, lungs and kidneys (TOTH, 1990). The clinical history of physical restraint associated with morphological changes found in the necropsy allowed to relate the cause of death with a stressful situation, which may have been preceded by acidosis, ventricular fibrillation, cholinergic bradycardia and myopathy in general.

The sudden, unexpected death rate in a commercial turkey farm has also been reported (SWAYNE & SAIF, 1990). The death toll occurred one to two days after handling and transporting of the animals. Some animals underwent autopsy and it was observed congested lungs, hepatomegaly and decreased spleen. The more relevant histological lesions were the presence of edema, pulmonary hemorrhage, congested veins and capillaries in several organs and viscera. The pattern of increased sudden mortality, the lack of clinical signs and pathological changes observed were considered consistent with a diagnosis of sudden death syndrome.

Increased incidence of unexpected deaths of healthy broilers when submitted to stressful activities such as blood sampling, weighing as well as routine handling and transport during management practices has also been reported by Olkowski (2008). Thus, reinforcing the theory that physical restraint is one of the main stress causes in several animal species.

Another case of animal autopsy that died in a pet shop during bathing and grooming procedures was described by Ceretta (2009). The author describes the incidence of deaths in pet shops as more and more common and reveals that there is intense agitation of the animal before death. The main changes found in autopsy of dogs and cats that died during pet shop procedures and the like have also been reported (MARIÁ, 2010). All studied animals underwent procedures such as bathing, grooming, training, transfers, stay in hotels and other similar situations at the time of death and showed very similar histological changes.

Another case of sudden death of thousands of birds that fell dead from the sky, just before midnight on
New Year’s Eve has also been reported in the literature (ZEFERINO, 2010). Experts have hypothesized that the fireworks of New Year may have led these birds to death due to stress.

**CONCLUSION**

Stress is a subject extensively studied in human medicine and it is becoming a major issue in veterinary medicine as well. It is known that the knowledge about it can ensure the well being and quality of life of animals and help elucidate the biological and psychological impacts of the life style changes that have been imposed on them. Since the concept of animal welfare is not yet fully understood and the parameters used for its evaluation are not very specific and difficult to apply, to recognize stress by clinical signs (through behavioral change evaluation) and laboratory tests (by measuring levels of corticosteroids, glucose, catecholamines, among others) can improve health, quality of life and even the production of animals intended for this purpose, without failure of the adaptive process and consequently, exhaustion and death.

**REFERENCES**


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