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Clinico-physiological manifestations of heat stroke in in exotic and cross bred cattle

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Abstract
A study was conducted with the objective to study Clinico-physiological manifestations of heat stroke in exotic and cross bred cattle. Sixteen cattle were included in the study on the basis of history, clinical signs and blood smear examination to rule out the presence of haemoprotozoan and rickettsial parasites. Affected animals were evaluated on the basis of Clinico-physiological observations including Clinical signs, temperature, pulse and respiration rate. Ten apparently healthy animals were taken to serve as control group. The clinical signs of heat stroke suffered cattle revealed hyperthermia, panting, tachycardia, hyper salivation, congested mucous membranes, ataxia, muscle tremor and severe dehydration in cows. There was highly significant increase ($P< 0.01$) in temperature, pulse rate and respiration rate.

Keywords: Heat stroke, exotic cattle, clinico-physiological

Introduction
Heatstroke, the deadliest of heat illnesses, and is defined by a core body temperature above 40°C (104°F) (Lee-Chiong and Stitt, 1995) [14]. Heatstroke is a life-threatening condition characterized by hyperthermia, central nervous system abnormalities and varying degrees of organ dysfunction. It occurs when the normal thermoregulatory system fails (Tayeb and Marzouki 1990; Drobatz and Macintire 1996; Barrow and Clark 1998; Bouchama and Knochal 2002; Grogan and Hopkins, 2002) [21, 10, 3, 5, 11]. Affected cows attempt to reduce heat load by reducing exercise, feed intake and lactation. They actively seek shade and wet areas (Wolfenson *et al*., 2000) [25].
Heat stroke affects almost all systems of the body include the CNS, gastrointestinal, cardiovascular, hepatobilary, renal/urologic, hematologic and muscular (Walters, 2002) [24]. Acute renal failure is common especially in the dehydrated animal; it can manifest as oliguric or polyuric renal failure and can be fatal. Central nervous system injury can occur as a result of direct thermal injury, respiratory alkalosis, thrombosis, or hypoperfusion from shock. Disseminated intravascular coagulation and hyperthermia lead to interparenchymal hemorrhage that can result in seizures, thus sustaining hyperthermia (Roger, 2005) [18]. Direct thermal injury damages the gastrointestinal barrier. Hypo perfusion and hypoxemia results in necrosis, death, and sloughing of the mucosa, leading to bacterial translocation. Bacterial translocation can cause sepsis and systemic inflammatory syndrome. Direct thermal injury and hypoxemia from hypo perfusion also damage the liver. Heat stroke damages the muscles of the body, especially if it is the result of excessive exertion (Roger, 2005) [18].
Rhabdomyolysis, due to muscle necrosis, is common and can exacerbate the acute tubular necrosis via dehydration, hypo perfusion, and pigment deposition (Walters, 2002) [24]. The thermal damage of the GI tract will predispose the patient to translocation of bacteria that can develop into systemic inflammatory response syndrome (SIRS) or septic shock (Davis, 2004) [9].

Materials and Methods
The present study was conducted on 16 clinical cases of heat stroke affected exotic and crossbred cattle presented at TVCC, College of Veterinary and Animal Science, Bikaner, due to high environmental temperature and high relative humidity during summer of 2015 with highest prevalence during June to August. Temperature-humidity index (THI) describes the effect of environment on animal's ability to dissipate heat. During drought, the ambient temperature and relative humidity frequently exceed the critical comfort level of temperature humidity index (72) resulting in elevated body temperature and panting.

Screening criteria for cattle suffered with heat stroke to be included in the present study were screening criteria for cattle suffered with heat stroke to be included in the present study were history, clinical signs and examination of blood smear. Blood samples were collected from ear vein of all 16 heat stroke suffered cattle for detection of any haemoprotozoan and rickettsial infection. Clinical examination was carried out to record rectal temperature, respiration rate and pulse rate. A group of 10 healthy cows were taken as a control and clinical examination was carried out for comparison.

Results

The blood smears were negative for tick born haemoprotozoan and rickettsial infection. Based on history, clinical signs and laboratory findings the cases were tentatively diagnosed to be suffered by heat stroke. The living environment of the study in June was average environmental temperature of 40.8 ^{0}C, average relative humidity 39 per cent and temperature humidity index 89.33. In July average environmental temperature was 36.9 ^{0}C, average relative humidity 58 per cent and temperature humidity index 88.97 whereas in August average environmental temperature 36.09 ^{0}C, average relative humidity 61 per cent and temperature humidity index 88.5. The clinical signs of heat stroke in present study included hyperthermia, panting, tachycardia, hyper salivation, muscle tremor, incoordination, mild to moderate ataxia, severe dehydration, congested mucous membranes, marked decrease in appetite, marked reduction in milk production and severe depression(Table 1). Clinical manifestations recorded in the present study are in agreement with those reported by Vermunt and Tranter (2010), Chandrabhan *et al.* (2013) and Randhawa *et al.* (2014) [23, 8, 17].

The mean± SE value of temperature, pulse rate and respiration rate in heat stroke suffered cattle are presented in table-2, The mean± SE values of temperature, pulse rate and respiration rate in heat stroke suffered cattle were 107.52±0.1450 ^{0}F, 113.69±2.9892 per minute and 115.50±2.4859 per minute, respectively. Corresponding mean± SE values for healthy control group are given in table-2. The mean ± SE values of temperature, pulse rate and respiration rate in healthy control group were 101.94±0.1950 ^{0}F, 62.20±1.2792 per minute and 24.50±0.9618 per minute, respectively.

There was highly significant increase ($P< 0.01$) in temperature, pulse rate and respiration rate (Table-2) in heat stroke suffered cattle as compared to healthy control cattle. Similar findings were recorded by Kelly (1974), Srikanda and Johnson (2004), Roger (2005), Radostits *et al.* (2007), Temizel *et al.* (2009), Vermunt and Tranter (2010), Randhawa (2012) and Chandrabhan *et al.* (2013) [18, 17, 13, 20, 16, 22].

Table 1: Major clinical manifestations of heat stroke suffered cattle

S. No.	Clinical manifestations	Suffered cattle (N=16)	Frequency (%)
1.	Hyperthermia	16	100
2.	Panting	16	100
3.	Tachycardia	16	100
4.	Congested mucous membrane	10	62.50
5.	Hyper salivation	16	100
6.	Ataxia	6	37.50
7.	Muscle tremor	5	31.25
8.	Severe dehydration	10	62.50
9.	Milk loss	16	100
10.	Decreased appetite	16	100

Table 2: Mean ± SE values of temperature (^{0}F), Pulse rate (per minute) and Respiration rate (per minute) in healthy control and heat stroke suffered cattle

S.No.	Parameters	Healthy control (N=10)	Heat stroke Affected cattle (N=16)
1	Body Temperature** (^{0}F)	101.94±0.1950^a	107.52±0.1450^b
2	Pulse (Rate/min)**	62.20±1.2792^a	113.69±2.9892^b
3	Respiration** (Rate/min)	24.50±0.9618^a	115.50±2.4859^c

* ($P< 0.05$) ** ($P< 0.01$)

Means with different superscripted letters in the same row differ significantly.

Discussion

Heat stroke is a medical emergency requiring rapid diagnosis and treatment. This potentially life-threatening condition occurs mostly when environmental temperatures and relative humidity are high. The high rise in rectal temperature observed in present study may be attributed to exposure to high environmental temperature and humidity. Low humidity and air movements are important to allow evaporation of sweat and convection of heat (Bricknell, 1995; Bruchim *et al.*, 1999-2004 and Al-Tamimi, 2007) [6, 7, 1]. Dehydration and increase of electrolyte concentration in the body fluid of cattle exposed to heat reduces their thermoregulatory evaporation and allowing the body temperature to rise. This readjustment in thermoregulation has been observed in both panting and sweating and appears to be a regulated response that allows the dehydrated animal to save water. In species that both pant and sweat, such as the cattle, progressive dehydration leads to suppressed sweating and increased panting (Silanikove, 1987) [19]. At high environmental temperature and humidity, an increased respiratory rate is an important way of increasing heat loss by cattle and is usually the first visible sign of heat stress (Mc Dowell, 1972) [15]. Tachycardia may be associated with translocation of blood from the central circulation to the periphery in an attempt to get rid of heat, or it might have resulted from the increased production of nitric oxide (Howorth, 1995 and Alzeer *et al.*, 1999) [12, 2].

In the present study, all the affected cattle showed neurological signs including mild to moderate ataxia, incoordination and muscular tremors. These neurological findings could be attributed to metabolic disarray, cerebral edema or ischemia (Boersma, 1998) [4].

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