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EFFECTS OF ACEPROMAZINE ON CAPTURE STRESS IN ROE DEER (*CAPREOLUS CAPREOLUS*)

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ABSTRACT: The aim of this study was to evaluate effect of a short-acting neuroleptic (acepromazine) on capture stress response in roe deer (*Capreolus capreolus*). Sixteen roe deer were captured by drive-nets in the winters of 1998, 1999, and 2001. Roe deer were divided into two groups: animals in the treatment group received an intramuscular injection of acepromazine (0.093 mg/kg±0.003 SEM; $n=8$) while animals in the control group ($n=8$) did not receive tranquilizer. Heart rate and body temperature, as well as hematologic and biochemical indicators of stress, were used to evaluate effect of the neuroleptic over 3 hr. Heart rate decreased over time after capture in both groups ($P<0.05$), but stabilized sooner in the treated roe deer (75 min after capture) than in the controls (105 min after capture). Body temperature decreased over 45 min and then stabilized in both groups ($P<0.05$). Comparisons of blood parameters revealed significantly lower red blood cell count (RBC), lymphocyte count, hemoglobin concentration, packed cell volume (PCV), and serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), creatine kinase (CK), and lactate dehydrogenase (LDH) activities in tranquilized animals compared with controls (at least $P<0.05$). A reduction in PCV, lymphocyte count, and serum cortisol concentrations (at least $P<0.05$) and an increase in serum creatinine levels ($P<0.05$) were recorded over time in control animals, while a reduction in RBC and hemoglobin concentration (at least $P<0.05$) and an increase in serum urea concentrations ($P<0.05$) over time were observed in the treated group. Finally, a decrease in serum lactate and potassium levels and an increase in CK, AST, ALT, and LDH activities were recorded over time in both groups. Results obtained showed the suitability of using acepromazine in capture operations in order to reduce stress response and prevent its adverse effects in roe deer. The beneficial effect was not only due to the sedative effect of acepromazine, but also to peripheral vasodilatation.

Key words: Acepromazine, *Capreolus capreolus*, capture, neuroleptic, roe deer, stress.

INTRODUCTION

Capture and handling is one of the most stressful events that happen to wild ungulates and is sometimes associated with considerable mortality. During the capture and restraint of free-ranging wildlife, a variety of negative stimuli can lead to prolonged exertion, exhaustion, stress, and hyperthermia. Collectively these factors can cause physiologic disorders including lactic acidosis and initiate development of myositis, muscle necrosis, depressed immune function, and possibly death (Spraker, 1993; Williams and Thorne, 1996). As a result of increased concern for conservation of wildlife species and increased game translocation operations, numerous studies have been conducted to measure stress responses of a variety of species, particularly in relation to their capture and confinement (Kock et al., 1987a, b; Hattingh et al., 1988).

Hematologic and biochemical measurements reveal significant differences in certain variables in relation to methods used for animal capture and handling (Kock et al., 1987b), and it has been suggested that these changes may vary with species, type of stress, and the individual's previous experience (Price, 1985). Ability to measure stress accurately is particularly important for determining the least stressful capture and handling method in order to reduce mortality and improve the well-being of wild species (Morton et al., 1995).

Physical restraint in excitable wild ungulates can result in exertional myopathy, also called exertional rhabdomyolysis or capture myopathy. Exertional myopathy is

one of the main adverse consequences of stress in wild animals. Capture myopathy is a syndrome that occurs in wild (free-ranging and captive) mammals and birds. It is associated with the stress of capture, restraint, and transportation. In ungulates the syndrome is characterized clinically by depression, muscular stiffness, lack of coordination, paralysis, metabolic acidosis and death. Pathologically, capture myopathy resembles the myodegenerative disorders of domestic cattle, sheep, horse, and swine, and it is mainly characterized by muscular and renal lesions (Williams and Thorne, 1996).

The roe deer (*Capreolus capreolus*) is a valuable wild ungulate because of its ecologic function, because it acts as an undergrowth cleaner, and it is economically importance as a game animal. Roe deer distribution is expanding and it is increasingly involved in reintroduction and restocking operations (García-Ferré et al., 1995) that require capture and handling. There are few studies on capture stress in roe deer (Meneguz et al., 1996) and none dealing with the use of neuroleptics to reduce stress and its adverse effects in this species. However, there are several studies in farmed red deer (*Cervus elaphus*) dealing with the effect of long-acting neuroleptics over management practices (Diverio et al., 1993, 1996a, b). Otherwise, it is widely believed that short-acting tranquilizers are ineffective when animals are highly stressed at the point of administration, particularly in free-ranging animals, although this needs to be further explored (Diverio et al., 1996b).

Acepromazine is a phenothiazine short-acting neuroleptic agent. While exact mechanisms of action are not fully understood, phenothiazines block post-synaptic dopamine receptors in the central nervous system and may also inhibit release of, and increase the turnover rate of, dopamine. They are thought to depress portions of the reticular activating system that assists in the control of body temperature, basal metabolic rate, emesis, vasomotor tone, hormonal balance, and alertness. Additionally, phenothiazines have varying degrees of anticholinergic, antihistaminic, antispasmodic, and α-adrenergic blocking effects. The onset of action is fairly slow, requiring up to 15 min following intravenous administration (Plumb, 2002). The recommended dose for deer species is 0.05–0.1 mg/kg (Arnemo et al., 1993).

In this study we evaluate the acute stress response caused by physical capture in roe deer and the effect of acepromazine on this response by using clinical, hematologic, and biochemical parameters.

MATERIAL AND METHODS

Sixteen roe deer, four males (three adults and one fawn, <1 yr old) and 12 females (11 adults and one fawn, <1 yr old) captured by means of drive-nets in the National Game Reserve of Alt Pallars-Aran (47°22′N, 3°48′E, north-eastern Spain), the Controlled Hunting Area of Vall d'Aran (47°35′N, 3°15′E, north-eastern Spain), and a private hunting area (44°40′N, 8°30′E, north-western Italy) were used in this study. Eight randomly selected animals, two adult males and six adult females received 2.5 mg (0.093 mg/kg±0.003 SEM) of acepromazine (Calmo Neosan®, Smithkline Beecham, Madrid, Spain) in a volume of 0.5 ml intramuscular in the femoral area, while eight animals, two males (one adult and one fawn) and six females (five adults and one fawn) acted as controls receiving the same volume of saline. The mean liveweight of animals was 24.31±0.77 kg (range 20–26.5).

Twenty capture operations carried out in the winters of 1998, 1999, and 2001 were necessary to obtain the 16 roe deer. Drive-trapping was conducted by a line of beaters, each one within sight of the next, and went on for approximately 30 min. Once in the net, the animals were initially restrained by using the net to wrap them, blindfolded, their legs restrained, and finally introduced in a transport sack net (Ziboni Ornitecnica, Bergamo, Italy), where they were maintained for 3 hr. At the end of the capture operation, the right thoracic and the left precordial areas were clipped in order to install telemetric heart rate recording equipment (Polar Vantage NV®, Polar Electro Oy, Kempele, Finland). Also a telemetric body temperature recording device (Mätman datalogger®, Chipsobits Eltex AB, Sweden) was introduced in the rectum.

Adequate records for heart rate analysis were obtained from 14 (seven per group) deer. Heart

rate was measured at 60 sec intervals for 2 hr. Arithmetic mean of heart rate values was calculated for every 5 min period for statistical analyses. Adequate body temperature records were only obtained from 11 free-ranging (five in the treated group and six in the control group) for 1 hr. Rectal temperature was measured at 60 sec intervals. The arithmetic mean of rectal temperature values was calculated for every 15 min period for statistical analyses. Ambient temperature during capture operations was between 0–12 C.

Blood samples were taken at capture (time 0) and one each hour thereafter for 3 hr (time 1, 2, and 3 respectively). Blood samples (10 ml) were collected from the jugular vein using disposable syringes and 0.8×25 mm needles. A 2.5 ml subsample was placed in a commercial tube with anticoagulant (EDTA K_3) for hematologic analyses. The remainder was placed in a serum collection tube, allowed to clot at room temperature, and used for biochemical analyses. Serum was kept at −18 C until analyses were completed. Hematologic examinations [red blood cell (RBC) count, hemoglobin concentration, mean corpuscular volume (MCV), mean corpuscular hemoglobin concentration (MCHC), mean corpuscular hemoglobin (MCH), and white blood cell (WBC) count] were performed by means of a semiautomatic analyzer (Sysmex F-800, Toa Medical Electronics, Japan). Packed cell volume (PCV) was measured by the standard microhematocrit method with a hematocrit centrifuge (Micro-Hematocrit Centrifuge, Hawksley, Lancing, UK) at 11,000 rpm for 5 min to adjust values obtained with the analyzer. Differential leukocyte count was performed with blood smears stained with commercial Diff-Quick® stain (Química Aplicada S.A., Amposta, Spain). Biochemical analyses were performed by means of an automated analyzer (COBAS MIRA®, Roche, Nutley, New Jersey, USA) except for sodium and potassium concentrations that were measured by flame photometry (Corning 410C®, Corning Medical, Medfield, USA) and serum cortisol, which was determined using an ELISA commercial kit (DRG Cortisol EIA-1887, DRG Diagnostics, Germany).

Statistical analyses were performed by means of a repeated measures analysis of variance using the PROC MIXED procedure of the SAS® statistics software package (SAS Institute Inc., Cary, North Carolina, USA). The main factor was treatment (acepromazine or saline) and the repeated factor was time. Also sex, age, and interactions between factors were included in the statistic model. Type 1 autoregression (AR(1)) structure for the covariance matrix of the repeated measures was used. When statistical differences between treatment groups at time 0 were obtained, values were expressed as a time 0 ratio in order to evaluate the effect of acepromazine regardless of initial values. Least square means (LS MEANS) were used due to the unbalanced distribution of animals between groups. In all cases, accepted significance level was at least $P<0.05$.

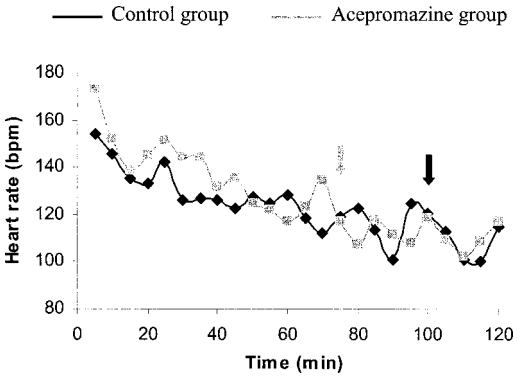

FIGURE 1. Heart rate of control ($n=7$) and treated ($n=7$) roe deer over a 2 hr period after capture. Heart rate decreased over time in both groups (at least $P<0.05$). Arrows indicate when heart rate stabilized in each group (i.e., no statistical differences were found between these values).

RESULTS

After capture and fitting with heart rate recording devices, roe deer heart rate (Fig. 1) decreased over a 2 hr period in both groups ($P<0.05$). However, it stabilized earlier in the treated roe deer (75 min after capture) than in the controls (105 min after capture). Body temperature (Fig. 2) decreased over time in both groups ($P<0.05$) and it stabilized 45 min after capture, as no statistical difference was found between temperature values at minute 45 and those at minute 60.

Red blood cell count (Fig. 3a) and hemoglobin concentration (Fig. 3b) of treated animals decreased over time and were significantly lower than those obtained from the control group (at least $P<0.05$). Packed cell volume also was significantly lower in the treated group (Fig. 3c), but differences over time were found in the control group (at least $P<0.05$). Lymphocyte count (expressed as a time 0 ratio) decreased over time in the control group

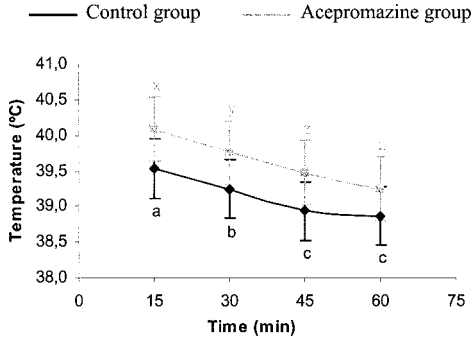

FIGURE 2. Body temperature (mean±SEM) of control (n=6) and treated (n=5) roe over 1 hr after capture. Values with different superscripts are significantly different from each other in the control group (at least $P < 0.05$). Values with different superscripts are significantly different from each other in the treatment group (at least $P<0.05$).

(at least $P<0.05$), while it did not change in treated animals (Fig. 3d). The control group also showed lower lymphocyte numbers than the treated group at time 3 ($P<0.01$).

Serum creatine kinase (CK), aspartate aminotransferase (AST), lactate dehydrogenase (LDH), and alanine aminotransferase (ALT) activities increased over time in both groups (at least $P<0.05$) due to physical stress caused by exercise and restraint. However, they were significantly lower in the treated group (at least $P<0.05$) from time 2 onwards (CK and AST) and at time 3 (ALT and LDH) compared with control animals (Fig. 4a, b, c, and d, respectively).

Serum creatinine concentrations increased significantly over time ($P<0.05$) in the control group (Fig. 5a). Serum urea concentrations increased at time 3 ($P<0.05$) in treated roe deer but no changes were observed in control animals (Fig. 5b). Serum lactate levels decreased faster (at least $P<0.05$) in treated roe deer than in controls (Fig. 5c), although when expressed as a time 0 ratio lactate levels did not statistically differ between treatment groups (Fig. 5d). No differences were

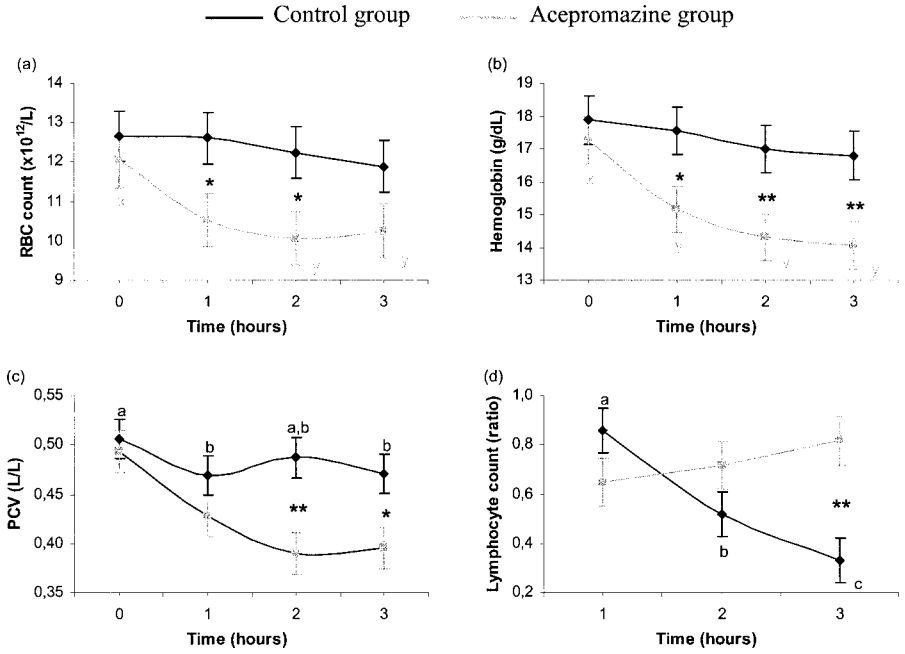

FIGURE 3. (a) Red blood cell (RBC) count, (b) hemoglobin concentration, (c) packed cell volume (PCV) (mean±SEM), and (d) lymphocyte count (expressed as a time 0 ratio±SEM) of control and treated roe deer over a 3 hr period after capture. Values are significantly different between groups (*$P<0.05$; ** $P<0.01$). Values with different superscripts are significantly different from each other in the control group (at least $P<0.05$). Values with different superscripts are significantly different from each other in the treatment group (at least $P<0.05$).

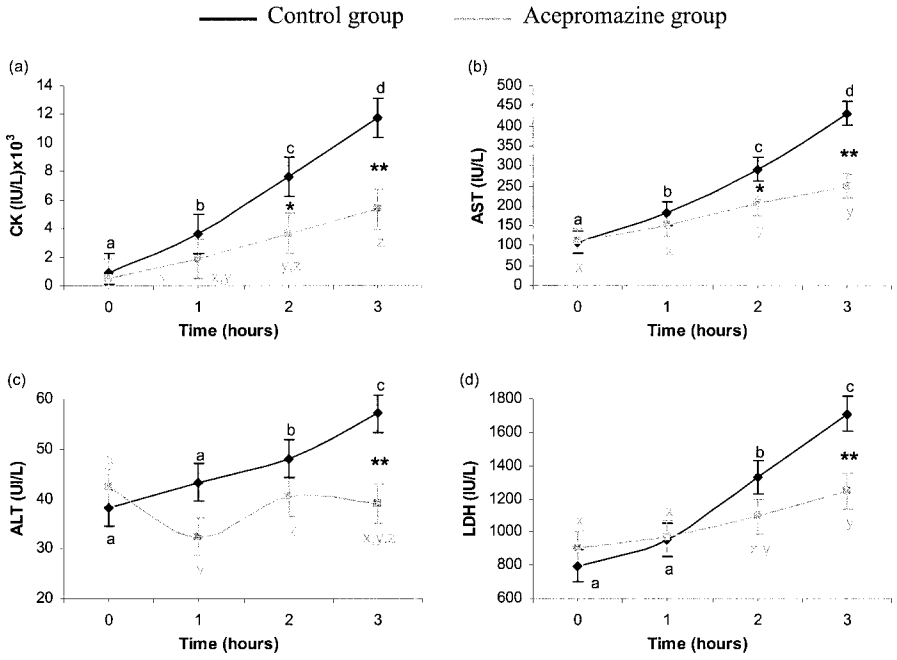

FIGURE 4. (a) Serum creatine kinase (CK), (b) serum aspartate aminotransferase (AST), (c) serum alanine aminotransferase (ALT), and (d) serum lactate dehydrogenase (LDH) activities (mean±SEM) of control and treated roe deer over a 3 hr period after capture. Values are significantly different between groups (*$P<0.05$; ** $P<0.01$; **** $P<0.0001$). Values with different superscripts are significantly different from each other in the control group (at least $P<0.05$). Values with different superscripts are significantly different from each other in the treatment group (at least $P<0.05$).

found in total protein and sodium (Fig. 6a, b). Serum potassium levels decreased over time in both groups (at least $P<0.05$), but the decrease was faster in control roe deer (Fig. 6c). However, no differences were found between treatment groups when expressed as a time 0 ratio (Fig. 6d). Finally, serum cortisol concentrations reached the maximum level at time 1 in control roe deer ($P<0.05$), while no differences were observed in the treated deer and between treatment groups (Fig. 7a, b).

DISCUSSION

Heart rate is one of the most widely used acute stress indicators (Broom and Johnson, 1993) and is considered to be an objective way of assessing the autonomic nervous system response to psychological stressors (Hopster and Blockhuis, 1994). Heart rate can be a useful measure of the emotional response of an individual to short-term problems, provided a distinction is made between the metabolic and emotional effect, and the measurement itself does not cause too much disturbance (Broom and Johnson, 1993). In our study, the lack of differences in the absolute values of heart rate between treatment groups could be due to a reflex tachycardia secondary to hypotension caused by acepromazine (Plumb, 2002) or to the high interindividual variability normally found in this parameter (Hopster, 1998). Kock et al. (1987a) did not find significant differences in heart rate between normal bighorn sheep (*Ovis canadensis*) and those considered stressed. Likewise, Read et al. (2000) did not find statistical differences in heart rate between wapiti (*Cervus elaphus*) treated with a neuroleptic (zuclopenthixol acetate) and controls. Moreover, Diverio et al. (1996b) found a greater increase in heart rates in red deer treated with a long-acting phenothiazine than in untreated deer during the 30 min imme-

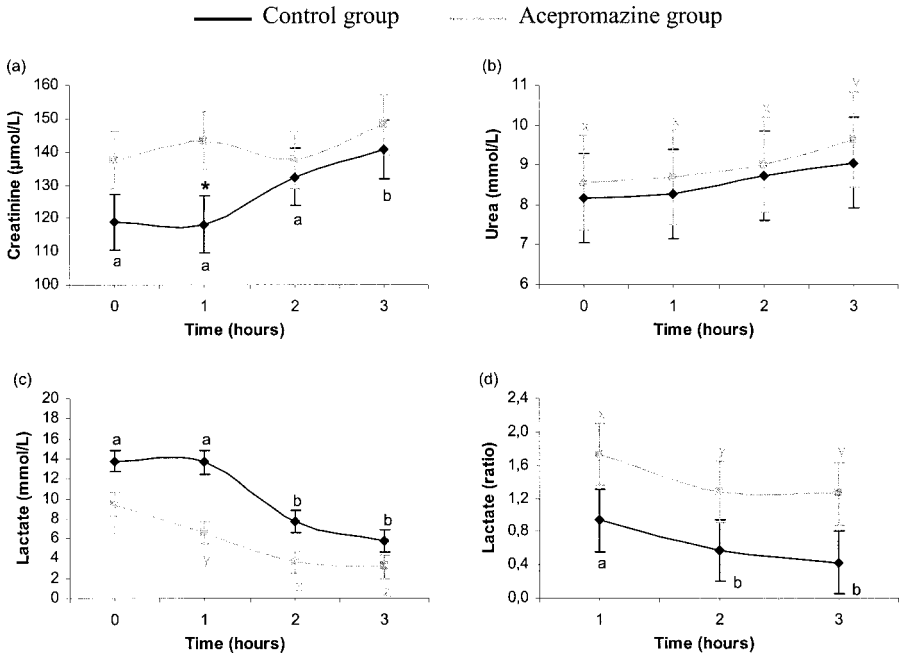

FIGURE 5. (a) Serum creatinine concentrations, (b) serum urea concentrations, (c) serum lactate concentrations (mean±SEM), and (d) serum lactate concentrations (expressed as a time 0 ratio±SEM) of control and treated roe deer over a 3 hr period after capture. Values are significantly different between groups ($P<0.05$). Values with different superscripts are significantly different from each other in the control group (at least $P<0.05$). Values with different superscripts are significantly different from each other in the treatment group (at least $P<0.05$).

diately following stressor application, which was attributed to a reflex tachycardia. Earlier stabilization of heart rate recorded in treated roe deer in comparison with controls was also observed in the previously cited work. This difference could be explained by the tranquilizing effect of the drug.

Porges (1985) proposed that, compared with heart rate level as such, the physiologic variability in heart rate may be a better indicator of both the status of the nervous system of the individual and its capacity to respond to environmental demands. The coefficient of variation of heart rate, used as a measure of heart rate variability (Hopster and Blokhuis, 1994), was not significantly altered by acepromazine action (control group: 23.23±2.38%; treated group: 25.95±2.67% [Mean±SEM]).

Increases in body temperature in certain stressful situations cannot only be explained by physical activity, but also there is another component called stress-induced hyperthermia (SIH) (Moe and Bakken, 1997; Bakken et al., 1999). Stress-induced hyperthermia is a regulated shift of the thermoregulatory set point (Briese and Cabanac, 1990) mediated by prostaglandin E and interleukins 1 and 6 (Le May et al., 1990; Kent et al., 1993). A correlation has been found between SIH, the sympathoadrenal medullary system, and the hypothalamic-pituitary-adrenal axis, which indicates that SIH is a stress-mediated response (Groenink et al., 1994). It has been suggested that SIH may be elicited in response to the anticipation of a known or unknown unpleasant event, indicating that SIH may reflect a state of anticipatory anxiety (Lecci et al., 1990). Zethof et al. (1994) stated that in mice SIH is time dependent, that it takes 10 min to reach a (stable) high level, which is 1–1.5 C higher than the baseline, and that it takes 60 min to return to baseline. Moe (1996) found

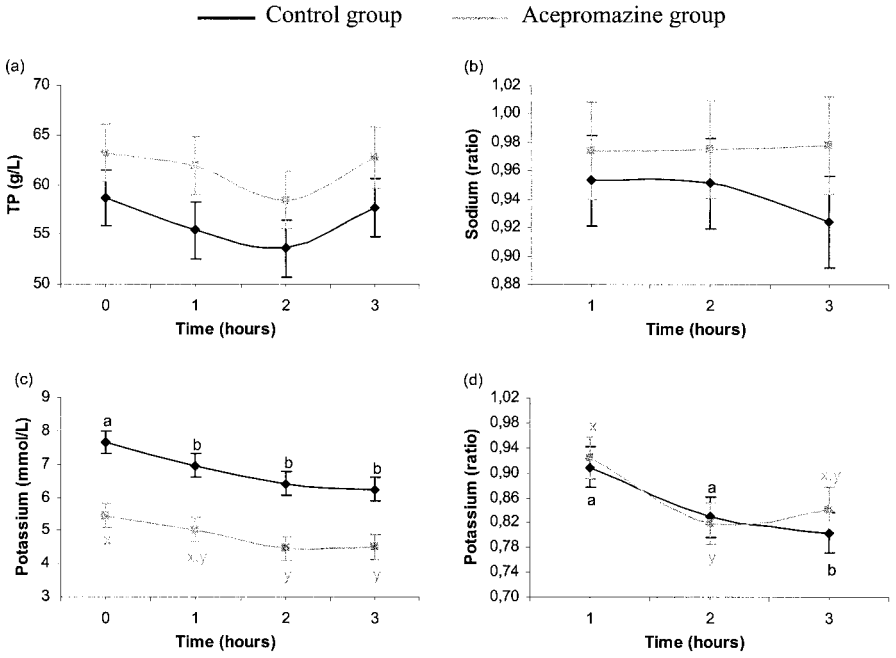

FIGURE 6. (a) Serum total protein (TP) concentrations (mean±SEM), (b) serum sodium concentrations (expressed as a time 0 ratio±SEM), (c) serum potassium concentrations (mean±SEM), (d) serum potassium concentrations (expressed as a time 0 ratio±SEM) of control and treated roe deer over a 3 hr period after capture. Values are significantly different between groups (*$P<0.05$; ** $P<0.01$). Values with different superscripts are significantly different from each other in the control group (at least $P<0.05$). Values with different superscripts are significantly different from each other in the treatment group (at least $P<0.05$).

that SIH in farmed silver foxes (*Vulpes vulpes*) lasts 60–90 min after a short stressor presentation. Therefore, the changes in body temperature observed in our study resemble those of a SIH response. Acepromazine did not have any effect on body temperature, although hypothermia is a well described non-desired effect of phenothiazines (Plumb, 2002). Olivier and Miczek (1998) indicate that stress-induced hyperthermia can be suppressed by administering benzodiazepines and serotoninergic agonists but not with phenothiazines. However, SIH can not be easily prevented in physical capture operations, where the anticipatory anxiety response is

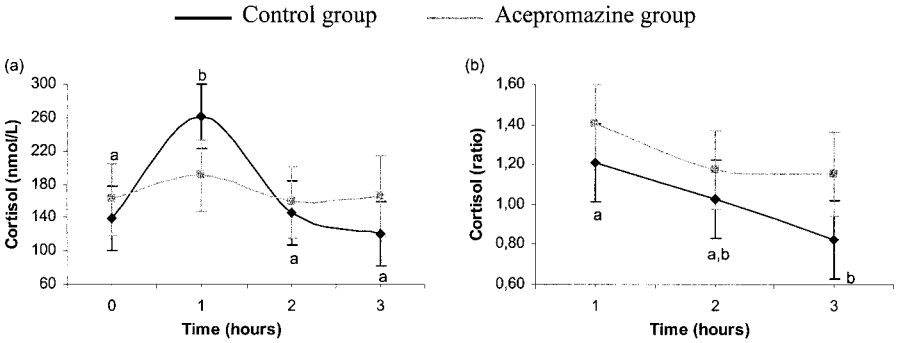

FIGURE 7. (a) Serum cortisol concentrations (mean±SEM) and (b) serum cortisol concentrations expressed as a time 0 ratio of control and treated animals over a 3 hr period after capture. Values with different superscripts are significantly different from each other in the control group (at least $P<0.05$).

elicited before any drug can be administered.

The first step in stress response is the activation of the sympathetic nervous system, stimulating the adrenal medulla and releasing catecholamines. Increases RBC, hemoglobin concentration, and PCV are associated with splenic contraction caused by the effect of catecholamines on α-adrenergic receptors located in the splenic capsule (Ganong, 2002), and partly to a reduction in plasma volume (Wesson et al., 1979; Cross et al., 1988). In our study, the lower values in RBC and hemoglobin concentration in treated animals can be explained by the α-adrenergic blocking effect of acepromazine. This brings about the relaxation of the spleen and the consequent splenic sequestration of erythrocytes (Jain, 1993). Hemodilution caused by acepromazine due to lowering of blood pressure can be ruled out because in that case total protein and sodium concentration would also have decreased (Fig. 6a, c).

Total and differential leukocyte counts may be altered by stress events, such as capture and handling. Epinephrine-induced physiologic leukocytosis in ruminants is characterized by neutrophilic leukocytosis with increases in lymphocytes and monocytes with a mild eosinopenia. Corticosteroid-induced changes include a mature neutrophilia, lymphopenia, eosinopenia, and monocytosis (Taylor, 2000). In domestic animals, neutrophilia and lymphopenia peaks appear after 4–8 hr of exposure to stress (Jain, 1993; Duncan et al., 1994). In our study, lymphopenia associated with stress leukograms was not observed during the study period in animals that received acepromazine. Thus, stress-induced lymphopenia could have been delayed or suppressed by acepromazine.

Muscle enzyme activity increases during capture and handling operations due to increased muscle cell permeability or muscle cell damage (Duncan and Prasse, 1986). These enzymes (ALT, AST, CK, and LDH) appear elevated in many stressed wild ungulates and in those suffering from capture myopathy (Kock et al., 1987a; Vassart et al., 1992), although some authors found that CK and AST levels are the most sensitive indicators of muscular disorders (Chapple et al., 1991). When muscle activity begins, blood flow increases but is intermittent. Blood flow decreases as muscle contracts because of the compression of vessels and increases during relaxation, a process called the muscle pump (Guyton and Hall, 1996). The muscle pump is active when the animal is running but it is inactive when it is immobilized by physical or chemical restraint. In most situations, the muscles of frightened animals that are not running are in a relatively isotonic state of contraction, which hinders blood flow into muscles. This leads to poor tissue perfusion, decreased heat dissipation, and hypoxia (Spraker, 1993). Acepromazine may cause vasodilatation in striated muscle arterioles by blocking the α-adrenergic receptors or by stimulating the β_2-adrenergic receptors and, thus, increase muscle blood flow (Booth, 1982; Guyton and Hall, 1996). Results obtained in our study indicate that acepromazine exerts a protective effect against muscle damage, probably due to its vasodilative effect, and suggest that acepromazine can be used as a preventive treatment of rhabdomyolysis, as previously indicated for horses (Beech, 1994).

Serum creatinine levels can be used to assess renal function. However, in some ungulates an increased creatinine concentration due to muscular activity and a decrease in renal excretion because of vasospasm in the kidney produced by catecholamines has been described (Harthoorn, 1976). Epinephrine (40%) and norepinephrine (20%) cause a decrease in renal blood flow, thus predisposing to renal hypoxia (Guyton and Hall, 1996). Ischemic renal failure has been produced by injecting epinephrine in laboratory animals (Finco, 1997). The differences observed in our study in serum creatinine concentrations between treatment groups can be ex-

plained by the α-adrenergic blocking effect of acepromazine over renal arterioles, where it promotes vasodilatation and, thus, allows creatinine to continue filtering (Jarvik, 1970). Moreover, this implies that oxygen supply to kidneys was not impaired, thus preventing renal hypoxia and subsequent renal ischemic necrosis.

Processes which increase protein catabolism will tend to result in increased levels of serum urea (Knowles and Warris, 2000). It has been described that the capture stress response causes an increase in serum urea concentrations (Gibert, 1991). This increase may be due to physical exercise, diminished renal perfusion, and to the effect of glucocorticoids over protein catabolism (Finco, 1997). In our study, however, serum urea only increased in the treated group, which is not correlated with the increase in serum creatinine levels. Urea excretion in ruminants is governed by nitrogen intake. Animals that are on a nitrogen deficient diet excrete almost all blood urea via the gastrointestinal tract and very little via the kidneys (Duncan et al., 1994), which probably happens in free-ranging roe deer during winter. This fact could account for the differences in the trend of serum urea and creatinine concentrations.

Hattingh et al. (1988) found an increase in lactate levels due to capture and handling in wild impala (*Aepyceros melampus melampus*) compared with control values from impalas shot in the brain in the early morning. Our results showed a decrease in serum lactate concentrations, which probably indicates that lactate levels were returning to baseline. However, this decrease was faster in the treated roe deer (1 hr after capture) than in the controls (2 hr after capture). In horses, intravenous administration of acepromazine 20 min before exercise resulted in lower serum lactate concentrations after exercise than when horses did not receive the drug. This was related to a protective effect of acepromazine because of its vasodilative action (Freestone et al., 1991; Beech, 1994).

The capture stress response causes hyperkalemia (Gibert, 1991). During exercise, potassium is released from working skeletal muscles (Van Beaumont et al., 1973). Following the completion of exercise potassium quickly declines, controlled by extrarenal factors including insulin, catecholamines, glucocorticoids, and acid-base balance (Bia and DeFronzo, 1981). However, in our study, serum potassium levels decreased faster in control animals compared to treated ones. It has been suggested that by influencing electrolyte movements, acepromazine may alter neuromuscular excitability and impair development of exertional rhabdomyolysis (Freestone et al., 1991).

Glucocorticoid levels rise in response to many short-term challenges in life and their measurement gives valuable information about the welfare of animals (Broom and Johnson, 1993). Glucocorticoid hormones, produced in and released from the cortex of the adrenal glands in response to an extremely wide range of stressors, play a major role in mediating the physiologic response to stress, but because of the role of the brain in release of glucocorticoids, they are widely interpreted as a measure of an animal's psychologic perception of a situation, in addition to the extent of its physiologic reaction. Many authors have used plasma cortisol as an indicator of stress associated with capture and handling in wildlife species (DelGiudice et al., 1990; Hastings et al., 1992; Morton et al., 1995). In our study, serum cortisol reached the maximum level 1 hr after capture in control roe deer. However, no changes were observed in treated animals. Hartmann (1988) found a peak in cortisol concentrations 30 min after an intravenous injection of ACTH in calves. Therefore, the blood withdrawal timing might have influenced the results obtained. Moreover, the lack of differences between groups in serum cortisol levels could be due to the fact that sedative effects do not have a consistent effect on plasma cortisol concentrations. Brearley et

al. (1990) found that at a similar depth of sedation, xylazine suppressed the cortisol response to stress while acepromazine had a slight enhancing effect. It has been suggested that chlorpromazine causes systemic release of epinephrine, which may result in an increase in adrenocorticotropic hormone (ACTH) release and hence cortisol release (Bruss, 1980). Other reasons that could explain the lack of differences between groups could be the great interindividual differences found in stress-induced plasma cortisol concentrations (Moberg, 1985) together with the low number of individuals per group available in this study, the existence of ultradian, circadian and seasonal rhythms in cortisol secretion (Ingram et al., 1999), and the disturbance caused by the sampling method itself.

Results obtained show the suitability of using acepromazine in capture of roe deer to reduce the stress response and prevent its adverse effects. However, caution must be taken in hypovolemic or shocked animals, because of decreased blood pressure when using phenothiazines. It is suggested that the beneficial effect is not only due to the sedative effect of acepromazine but also to peripheral vasodilatation. This vasodilation has a protective effect against the muscular and renal damage that can arise from stress episodes in wild animals, which is directly involved in the pathogenesis of capture myopathy. Thus, animal welfare is partly improved by preventing and reducing adverse effects of stress, although acepromazine had no effect on stress-induced hyperthermia.

ACKNOWLEDGMENTS

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