RUMINAL, CARDIORESPIRATORY AND ADRENOCORTICAL SEQUELAE OF Na2EDTA-INDUCED HYPOCALCAEMIA IN CALVES

D.J.-M. DESMECHT, A.S. LINDEN AND P.M. LEKEUX
Departments of Pathology, Bacteriology, and Physiology, Faculty of Veterinary Medicine, University of Liege, B42-43 Sart Tilman, 4000 Liege, Belgium

ABSTRACT


A study was undertaken to provide further information on the ruminal, cardiorespiratory and hypothalamo-pituitary-adrenocortical (HPAC) physiological sequelae of hypocalcaemia in dairy calves.

The functional picture observed in standing calves experiencing Na2EDTA-induced progressive hypocalcaemia showed a biphasic pattern. During the first phase (Ca*+ varying between 1.20 ± 0.09 and 0.64 ± 0.15 mmol/L, mean ± SD), the animals became dull and lethargic, shifting their weight from one hind limb to the other, with cool extremities and hypersalivation. Their ventilation was slightly increased but their heart rate, thoracoabdominal pressure, pulmonary mechanics, haemoglobin and temperature remained constant. Conversely, their systemic arterial pressure (SAP) and the amplitude of their ruminal contractions (RCA) were severely decreased. During the second phase (Ca*+ < 0.64 ± 0.15 mmol/L), there was restlessness, tachycardia, hypertension, polycythaemia and, finally, inability to stay upright. It is suggested that the diminished Ca*+ availability caused smooth-muscle and myocardial dysfunctions which could explain the RCA and SAP changes recorded during the first phase, whereas neural and/or humoral sympathetic discharge probably accounted for the reversal in SAP and heart rate when Ca*+ was decreased further. Serum cortisol increased regularly and remained significantly correlated with Ca*+ in each animal. Moreover, regression of Δcortisol/ΔCa*+ on ΔCa*+/ΔNa2EDTA was significant (p < 0.001).

It was concluded that mild asymptomatic hypocalcaemia severely impairs ruminal function, which will progressively worsen the Ca*+ deficit; that the inability to maintain posture in hypocalcaemia is not due to hypotension; and that the higher the HPAC response to hypocalcaemia, the higher the resistance to its effects. An asymptomatic periparturient cow with barely detectable ruminal activity may merit preventive calcium borogluconate therapy. Also, the physiological role of hypotension in explaining the clinical picture may be less important than other processes, such as neuromuscular failure. Finally, the present results imply a possible HPAC exhaustion in cows with periparturient paretic hypocalcaemia.

Keywords: calf, cortisol, hypocalcaemia, pathophysiology, rumen

Abbreviations: A-aDO2, alveolar–arterial O2 difference; AVP, arginin vasopressin; C1,dyn, dynamic lung compliance; ECG, electrocardiogram; EEZF, end expiratory zero flow; HbTOT, total haemoglobin content; HPAC, hypothalamo-pituitary-adrenocortical; HR, heart rate; Na2EDTA, disodium ethylene-diaminetetraacetate; O2CT, total O2 content; PaCO2, arterial CO2 tension; PaO2, arterial O2 tension; PH, periparturient hypocalcaemia; Ppl, pleural pressure; Ppr, intraruminal pressure; PTH, parathyroid hormone; PpTOT, plasma total protein; RCA, amplitude of ruminal contraction; RCD, duration of ruminal contraction; RCF, frequency of ruminal contraction; RL, total pulmonary resistance; RR, respiratory rate; SAP, systemic arterial pressure; T E, expiratory time; T i, inspiratory time; Tc, core temperature; Vr, respiratory airflow; V E, minute volume; VP, tidal volume
INTRODUCTION

Significant strides have been made in understanding the regulation of ionized calcium (Ca\(^{2+}\)) in the blood of mammals. Much of the information can be applied to ruminants. However, ruminants are the only animals that experience crippling episodes of periparturient hypocalcaemia (PH). Animals that develop PH are unable to meet the demand for Ca\(^{2+}\) which is brought about by the sudden loss of this mineral from the blood to form milk. The plasma calcium content in such cows falls below that normally observed at parturition, and this leads successively to ataxia and restlessness, to recumbency and hypothermia, and finally to loss of consciousness, complete muscle flaccidity and death (Yates and Hunt, 1990). Parturient hypocalcaemia has been shown to reduce the mean productive life of dairy cows by 3.4 years (Yates and Hunt, 1990).

Notwithstanding the 8–10% incidence and the plethora of clinical descriptions of the disease, there have been remarkably few experimental studies on the cascade of physiological events which underlies the clinical picture. Ruminal and abomasal tone and motility were depressed in 7 dairy cows (Daniel, 1983); cardiac output and arterial blood pressure were reduced in 4 Jersey cows (Daniel and Moodie, 1978); and smaller motor unit potentials emanating from the peroneus tertius muscle were qualitatively detected in 2 cows (Bowen et al., 1970). Despite a number of studies on the endocrine control of Ca\(^{2+}\) mobilization at parturition in the cow (reviewed by Braithwaite, 1976), only Horst and Jorgensen (1982) have measured cortisol concentrations.

The following study was undertaken to provide additional information on the ruminal, cardiorespiratory and hypothalamo-pituitary-adrenocortical physiological sequelae of hypocalcaemia.

MATERIALS AND METHODS

Animals

Seventeen male Friesian calves were studied. The age and the body weight of the animals were 332 ± 15 days (mean ± SD, range 311–369 days), and 292 ± 22 kg (range 260–325 kg). The calves were cared for in accordance with the most recent law devoted to the protection of experimental animals (Belgian Ministry of Agriculture, 1994). At one month of age, the right carotid artery was exteriorized in the mid-cervical region and maintained in a subcutaneous location by closing the underlying muscles, using xylazine (Rompun, Bayer, St-Truiden, Belgium), gaiacolate (Gujatal 10%, Aesculaap, Ghent, Belgium) and thiopental (Pentothal, Abbott, Louvain-la-Neuve, Belgium) anaesthesia. In order to make the calves comfortable and to prevent injury, they were kept on thick straw in wooden stalls which allowed them free movement yet gave them mechanical support in case of exhaustion. They were accustomed over several months to handling, to being fastened to the stanchion and to the instrumentation for cardiorespiratory testing, and were fasted for 8–12 h prior to blood collection. On the evening preceding the study, two catheters were inserted percutaneously by the Seldinger technique under local analgesia (Lignocaine, Kela, Hoogstraten, Belgium),