

THE CLINICAL PATHOLOGY OF HEARTWATER. II. STUDIES ON CARDIAC AND PULMONARY FUNCTION IN 4 CALVES WITH EXPERIMENTALLY-INDUCED HEARTWATER

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ABSTRACT

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Studies to evaluate cardiac and pulmonary function were undertaken in 4 calves suffering from experimentally-induced heartwater. There was a marked variation in the course of the disease. Three of the calves recovered spontaneously after developing clinical signs. These included a rectal temperature in excess of 40 °C, anorexia and listlessness but no neurological signs. The remaining calf died 2 days after developing a fever and neurological signs. In the 3 calves that recovered, a mild hypoxemia developed during the acute stage of the disease. Arterial CO₂ tension remained within normal limits, but there was a tendency towards an alkalosis. Increases in pulmonary dead space and fluctuations in venous admixture were observed.

The calf that died showed similar mild changes in blood gas parameters, despite the presence of a marked reduction in minute volume, and a lung oedema was demonstrated on post-mortem examination. No marked changes in systolic and diastolic blood pressures and in right cardiac intraventricular pressures were observed. Terminally, however, there were marked decreases in stroke volume and cardiac output. These changes were associated with a sharp increase in heart rate. No primary cardiac pathology was observed on clinical and post-mortem examinations.

INTRODUCTION

As pathological changes commonly involve the lungs and the thoracic and pericardial cavities in clinical cases of heartwater, evaluation of possible functional changes in both the heart and lungs are important. Pathological changes include a hydropericardium, hydrothorax and both an interstitial and alveolar oedema. The degree of these changes will vary according to the stage and severity of the disease (Prozesky, 1987). For this reason it is quite conceivable that functional changes in these organs may be very variable. No correlation studies have been undertaken between pathological and functional changes in clinical cases of heartwater. Clark (1962) and Owen, Littlejohn, Kruger & Erasmus (1973) described aspects of the pathophysiology of heartwater as related to heart and lung function. This was reviewed by Van Amstel, Guthrie, Reyers, Bertschinger, Oberem, Killeen & Matthee (1987). Changes in cardiovascular function described by these workers include a progressive drop in both systolic and diastolic blood pressures. These are associated with a drop in blood volume and possibly a central nervous system-induced sympatholysis (Clark, 1962). During the early stages of the clinical disease there is an increase in cardiac output resulting from an increase in stroke volume. As the condition progresses, stroke volume diminishes but cardiac output is maintained by an increased heart rate, and terminally a marked drop in cardiac output occurs. The decrease in stroke volume and cardiac output is probably associated with a developing hydropericardium (Owen *et al.*, 1973). As no electrocardiographic (EKG) abnormalities could be demonstrated, the myocardium and tissues of the specialized conduction system were probably not affected (Owen *et al.*, 1973).

Owen *et al.* (1973) found that there was an increase in respiratory minute volume during the acute stage of the disease, which was caused by an increase in both tidal

volume and respiratory rate, and that there was a tendency towards CO₂ retention and respiratory acidosis. These changes were accompanied by an increase in the ventilation equivalent (VEO₂), which indicated that an increased volume of air was required to extract a given amount of O₂. These authors' conclusion that the observed changes in respiratory function were probably associated with the presence of a pulmonary oedema was confirmed at autopsy.

The exact mechanism of pulmonary dysfunction in heartwater still needs to be elucidated. The interstitial oedema of the lungs which occurs during the early stage of the disease probably does not cause a reduction in the diffusing capacity of O₂ between the alveolar gas and the blood, leaving the pulmonary capillaries even if the oedema is severe (Nunn, 1969). Prozesky & Du Plessis (1985) state that the lung oedema present in the more advanced stage of the disease is a mixture of both interstitial and alveolar oedema. Interstitial oedema, even if severe, is probably not responsible for a reduction in the pulmonary diffusing capacity (Nunn, 1969). On the other hand, alveolar oedema may seriously impede gas exchange thus resulting in arterial hypercapnia and hypoxaemia (Nunn, 1969).

Hypoperfusion of normally ventilated alveoli results in increased alveolar dead space (Nunn, 1969). This pathophysiological change often accompanies pulmonary hypotension. Systemic hypotension has been associated with heartwater (Clark, 1962; Owen *et al.*, 1973) and thus it is conceivable that pulmonary hypotension may occur in this condition.

Blood that enters the pulmonary veins without passing through ventilated alveoli constitutes venous admixture (Nunn, 1969). Pathological venous admixture has been recorded as occurring in several conditions, including chronic obstructive pulmonary disease in the horse (Littlejohn, Bowles & Maluleka, 1982) and contusion and oedema of the lungs in humans (Nunn, 1969). In diseases, such as heartwater, in which there is severe lung oedema (both interstitial and alveolar), adequate gaseous exchange is not possible because of the flooded alveoli. This would contribute to venous admixture.

The object of this experiment was to study cardiac and pulmonary function during the course of experimentally-

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induced heartwater and to assess the possible role played by any changes in the clinical outcome of the disease. It was hoped that such an assessment would supply valuable information which would lead to more efficient supportive treatment.

MATERIALS AND METHODS

Experimental animals

Four healthy 6–10-month-old Friesland calves (identified as 0549, 0605, 1066 and 1067) were used. To facilitate the collection of arterial blood samples a portion of the left common carotid artery was sub-cutaneously relocated in the jugular groove, using the technique described by Butler (1962). Complete healing of the surgical wounds occurred during a 3-week period.

Heartwater was induced in the calves by inoculating each of them intravenously with a single dose (5 ml of infected blood) of the vaccine strain of *Cowdria ruminantium* (Ball 3 isolate) by intravenous injection. Rectal temperatures were measured once daily until death or clinical recovery. A temperature of 40 °C was regarded as an indication of the onset of clinical disease. All 4 calves developed a temperature above 40 °C from 14–24 days post-infection. Calf 1066 died on Day 18, whilst the other 3 calves recovered without treatment and were regarded as clinically normal on Day 25.

Each of the 4 calves served as his own control, as serial recording of physiological parameters were carried out during the course of the disease.

Experimental procedures

Blood and gas sampling and analysis

The following blood and gas samples were collected once 4 days before infection and then once daily from Days 15–25 post-infection, except from calf 1066 which died on Day 18 post-infection: arterial blood (a); mixed venous blood (V); mixed expired gas (E) and end-tidal gas (ET).

Arterial blood was collected via a 20 G hypodermic needle introduced percutaneously into the relocated portion of the carotid artery.

Mixed venous blood (V) was collected after the placing of a polyethylene tube in the right ventricle via percutaneous puncture of the jugular vein. Blood samples were collected anaerobically in 5 ml of heparinized disposable syringes, which were sealed and stored in ice water. Blood gas analysis was performed within an hour of sample collection, using an ABL3¹ semi-automated blood gas analyser.

Mixed expired gas (E) was collected from the calves by placing a face mask over the animal's muzzle. The face mask consisted of a plastic 5 l household detergent bottle from which the bottom had been removed and the cut edge padded with cotton wool. The point of contact between the cut edge and the animal's skin was made airtight by sealing with petroleum jelly. Flow-directed valves were used to direct environmental air into the face mask and the expired gas to a 200 l latex meteorological balloon. The volume of expired gas was determined with a Collins 600 l spirometer.

End-tidal gas (ET) was collected by placing a 15 G hypodermic needle percutaneously into the trachea in the mid cervical region, and attaching the needle to a 50 ml glass syringe.

The collection of blood and gas samples took place simultaneously and continuously over a period of 3 min.

Electrocardiograph (EKG) and pressure recordings

EKG tracings were recorded using a Mingograph 62² six channel recorder on which leads I, II, III, aVR, aVL and aVF were recorded simultaneously. Electrodes consisted of atraumatic crocodile clips, and a drop of Elema-Schönander electrode cream was used to moisten the skin surface at the sites of electrode application.

The P-wave duration, P-R interval, QRS duration, RR interval, P-wave amplitude, QRS complex amplitude and T-wave amplitudes were determined for leads I–aVF.

The presence of any arrhythmias was noted.

A Statham P50³ pressure transducer in combination with a electromanometer 863⁴ attached to the mingograph 62 was used for the measurement of the systolic and diastolic blood and right intraventricular pressures.

Carotid pressures were recorded by attaching the transducer to a 20 G needle placed in the translocated carotid artery.

For the recording of intraventricular pressures, a 120 cm length of polyethylene tubing with a 1.2 mm outer diameter and 0.85 mm inner diameter⁵ attached to a blunted 20 G needle was used. This catheter was aseptically placed through a 15 G needle into the jugular vein and then into the heart chambers. All readings were made with the transducer at the level of the shoulder joint.

EKG and pressure recordings were made on the same days as blood and gas samples were collected.

Calculations of physiological data

All ventilation volumes were corrected to BTPS (body temperature, pressure, saturated) and gaseous exchange volumes to STPD (standard temperature, pressure, dry).

Cardiovascular parameters

1. The cardiac output was measured using the Fick principle with oxygen as the indicator substance.

The minute volume (MV) was calculated from the spirometer readings. The true oxygen fraction (O_2F) was calculated from the mixed expired PO_2 and PCO_2 , according to the method of Severinghaus (1966). The oxygen consumption (VO_2) is then equal to the product of the minute volume and the true oxygen fraction. Cardiac output (ℓ/min) = VO_2 (ml/min)/ $CaO_2 - CvO_2$ (ml/ℓ)

where VO_2 = oxygen consumption,

CaO_2 = arterial blood oxygen content,

and CvO_2 = mixed venous blood oxygen content.

2. Stroke volume was calculated using the formula, Stroke volume (ml) = [Cardiac output (ℓ)/Cardiac rate] \times 1 000

3. The venous admixture was calculated as a percentage of the cardiac output according to the formula used by Nunn (1969). Venous admixture (%) = $[Cc'O_2 - CaO_2]/[Cc'O_2 - CvO_2] \times 100$

where $Cc'O_2$ = pulmonary end capillary oxygen content,

CaO_2 = arterial oxygen content,

CvO_2 = mixed venous oxygen content.

² Elema-Schönander

³ Gould Statham Instruments Inc., Hato Rey, Puerto Rico

⁴ Siemens-Elma AB, Solna, Sweden

⁵ Intramedic No. 7425, Clay Adams, New Jersey, USA

¹ Radiometer, Copenhagen

TABLE 1 Results of the bloodgas analysis on arterial blood

Day post-infection	Number of calves		PO ₂ (mm Hg)	PCO ₂ (mm Hg)	HCO ₃ (mmol/l)
-4	4	Mean range	96,22 (91,9-97,8)	33,15 (30,0-37,0)	18,32 (14,9-22,4)
17	4	Mean range	90,17 (73,7-113,0)	43,82 (41,2-45,9)	25,5 (22,0-27,7)
18	4	Mean range	77,02 (57,9-87,7)	42,10 (37,3-49,8)	26,3 (24,8-27,2)
19	3	Mean range	81,53 (75,7-91,4)	38,63 (34,6-41,9)	24,93 (23,8-26,1)
20	3	Mean range	84,63 (79,5-89,9)	40,13 (39,2-40,9)	24,43 (23,9-25,2)
21	3	Mean range	91,96 (87,6-94,9)	39,43 (37,6-42,7)	22,8 (19,3-25,3)
22	3	Mean range	92,1 (79,4-99,4)	40,36 (38,7-43,3)	24,06 (22,8-24,9)
23	3	Mean range	82,76 (78,4-86,1)	40,53 (37,9-42,9)	25,5 (24,1-26,6)
24	2	Mean range	95,1 (83,4-106,8)	33,75 (29,9-37,6)	23,65 (22,2-25,1)

TABLE 2 Results of the heart rates

Day post-infection	Number of calves		Heart rate/min
-4	4	Mean range	107,25 (96-122)
15	4	Mean range	109,25 (94-125)
16	4	Mean range	92,5 (84-98)
17	4	Mean range	88,75 (81-105)
18	4	Mean range	104 (82-124)
19	3	Mean range	100,66 (90-106)
20	3	Mean range	98,66 (76-127)
21	3	Mean range	98 (92-104)
22	3	Mean range	96 (76-112)
23	2	Mean range	94 (77-111)

The Cc'O₂ is indirectly derived from the alveolar oxygen tension (PaO₂), which is represented by the end tidal oxygen tension (PetO₂).

Pulmonary parameters

1. Alveolar dead space was calculated according to the formula of Severinghaus & Stupfel (1957).

$$\text{Alveolar dead space } VD(\text{alv}) = \frac{\text{PaCO}_2 - \text{PetCO}_2}{\text{PaCO}_2}$$

PetCO₂ = endtidal carbon dioxide tension
PaCO₂ = arterial blood carbon dioxide tension

2. Physiological dead space was calculated according to the Bohr equation as described by Nunn (1969).

Physiological dead space

$$VD(\text{phys}) = \frac{\text{PaCO}_2 - \text{PeCO}_2}{\text{PaCO}_2}$$

Where PaCO₂ = arterial blood carbon dioxide tension and PeCO₂ = mixed expired gas carbon dioxide tension

RESULTS

Course of the disease

There was a wide variation in the course of the disease in the 4 calves. Calf 1066 developed a temperature reaction in excess of 40 °C on Day 16 post-infection and died on Day 18. On the day that it died it developed neurological signs which included hyperaesthesia, ataxia and hypermetria, followed by lateral recumbency and paddling. On post-mortem examination both a moderate to severe lung oedema and an hydropericardium were present.

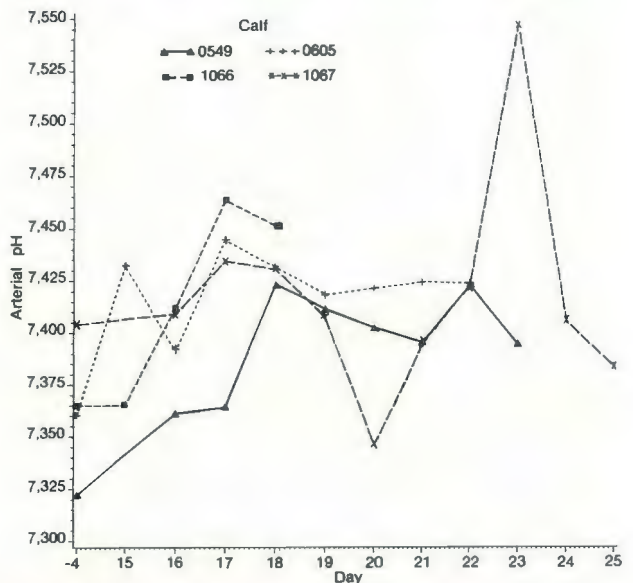


FIG. 1 Results of the arterial pH

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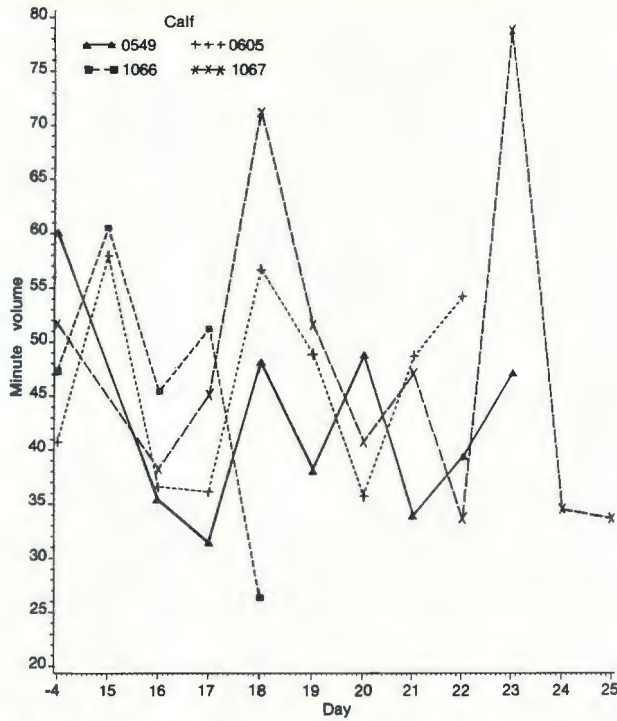


FIG. 2 Results of the minute volume in litres

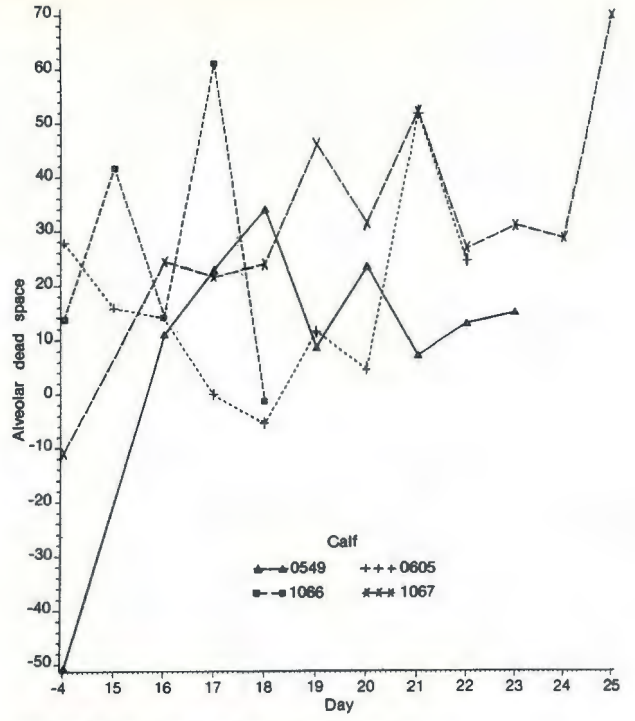


FIG. 4 Results of the alveolar dead space in per cent

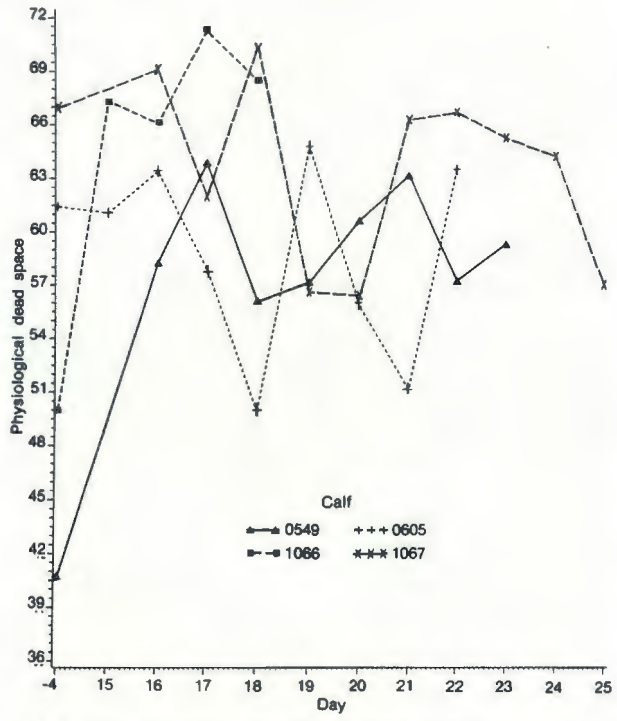


FIG. 3 Results of the physiological dead space in per cent

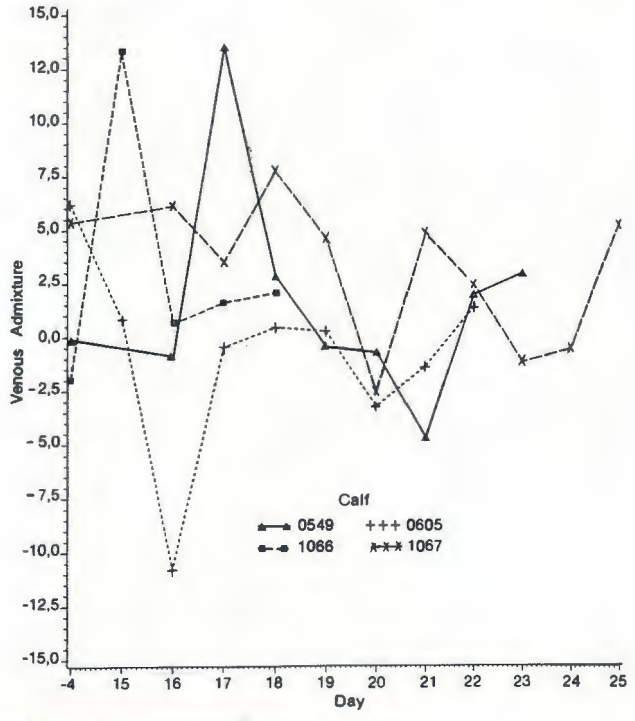


FIG. 5 Results of venous admixture in per cent

None of the other calves developed neurological signs. Calves 0605 and 0549 reached the peak of their clinical course between Days 14–20 post-infection. This was characterized by a pyrexia in excess of 40 °C, partial anorexia and listlessness. Calf 1067 only reached the acute stage of the disease from Days 18–24 post-infection.

Acid-base

Fluctuations in arterial blood pH are graphically shown in Fig 1. There was a general increase in blood pH during the acute stage of the disease, and this ten-

dency towards an alkalosis was present even in calf 1066 that died. It was marked in calf 1067 in which on Day 23 post-infection it coincided with the acute stage of the disease. In calf 1067 the peak arterial pH was associated with an increased minute volume (Fig. 2), increased PO₂ (106,8 mm Hg) and low PCO₂ (29,9 mm Hg), suggesting that there was no interference with pulmonary gas diffusion.

The results of the blood gas analysis are shown in Table 1. A general slight metabolic acidosis 4 days prior to infection was present. The reason for this was not immediately apparent. In addition, a general moderate

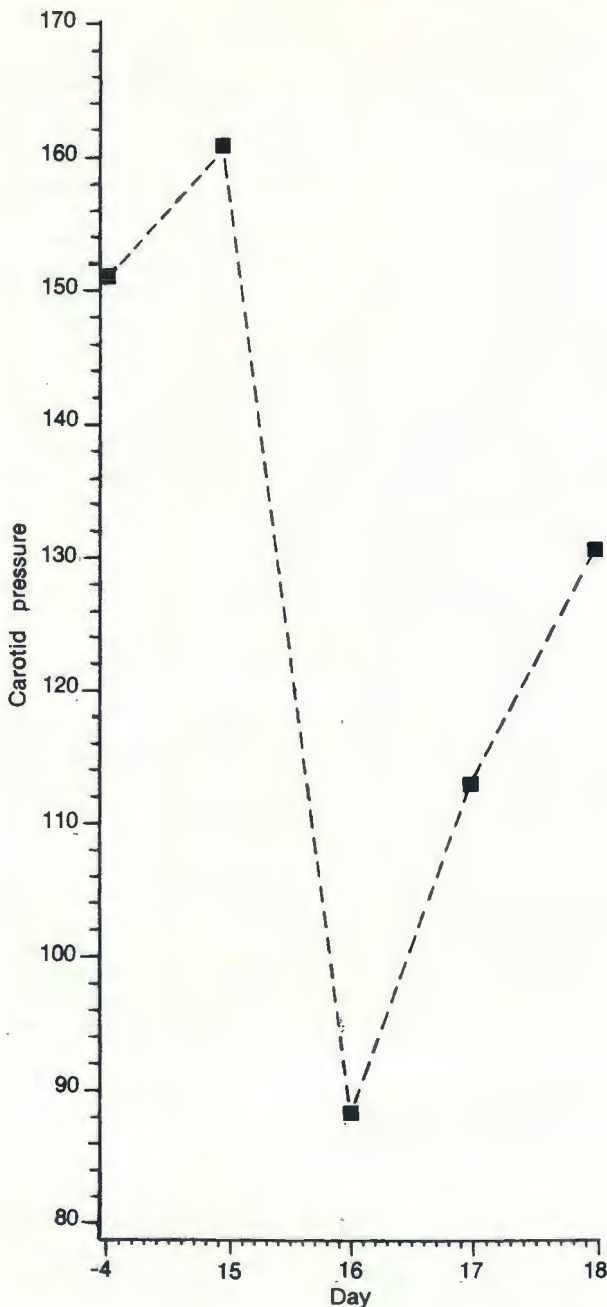


FIG. 6 Results of the systolic carotid pressure of Calf 1066 (mm Hg)

decrease in arterial PO_2 occurred during the acute stage of the disease, whereas the PCO_2 and HCO_3 remained within normal limits. In contradistinction to the fluctuations in the bloodgas of calf 1067 referred to above, calf 0549 showed a marked drop in PO_2 (57.9 mm Hg) and a moderate rise in PCO_2 (49.8 mm Hg) during the acute stage of the disease. This was accompanied by a steep rise in its recorded venous admixture, indicating that it may have been suffering from lung oedema at the time. Although there were increases in both its physiological and alveolar dead space, these parameters did not differ from those of the other calves at that time (Fig. 3 & 4). There was, however, an apparent drop in the minute volume of calf 0549 (Fig. 2).

Despite the presence of a severe lung oedema, as revealed during the post mortem examination, the PO_2 level of calf 1066 was only moderately depressed (77.1 mm Hg) and the PCO_2 was within normal limits (41.3

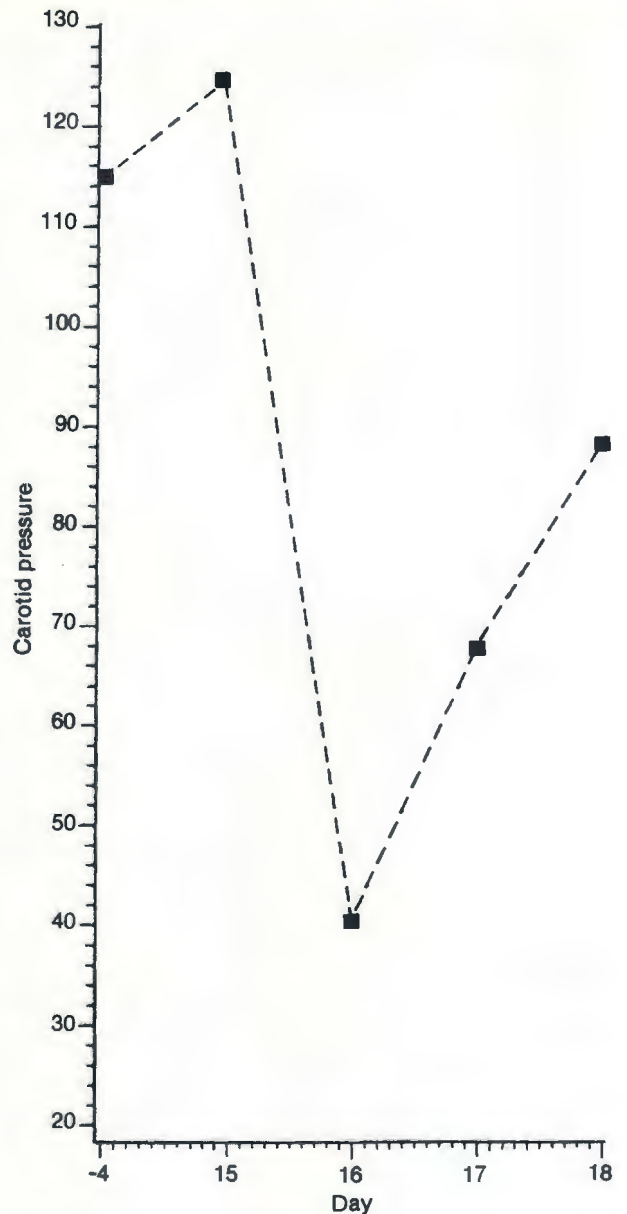


FIG. 7 Results of the diastolic carotid pressure of Calf 1066 (mm Hg)

mm Hg), an indication that no severe impedance in gas diffusion had occurred. The minute volume was depressed on the day it died (Fig. 2). Hypoventilation results in a marked reduction of the anatomical dead space which limits the fall of alveolar ventilation resulting from small tidal volumes (Nunn, 1969). This, in part, may account for the limited change in gas diffusion present in Calf 1066, despite the presence of the lung oedema. However, Figs. 3 & 4 show that on the day of its death, there was a reduction in the alveolar dead space, while the physiological dead space was still large. The anatomical dead space is equal to the physiological dead space minus the alveolar dead space, according to Nunn (1969).

From the above it is clear that wide variations occurred in the arterial PO_2 and PCO_2 during the course of the disease in the different calves, although there was a tendency for a lowered PO_2 . The arterial respiratory alkalosis and high PO_2 seen on Day 23 in Calf 1067 were apparently associated with an increase in minute volume. The exact reason for this increased minute volume is not clear. As it occurred during the acute stage of the disease

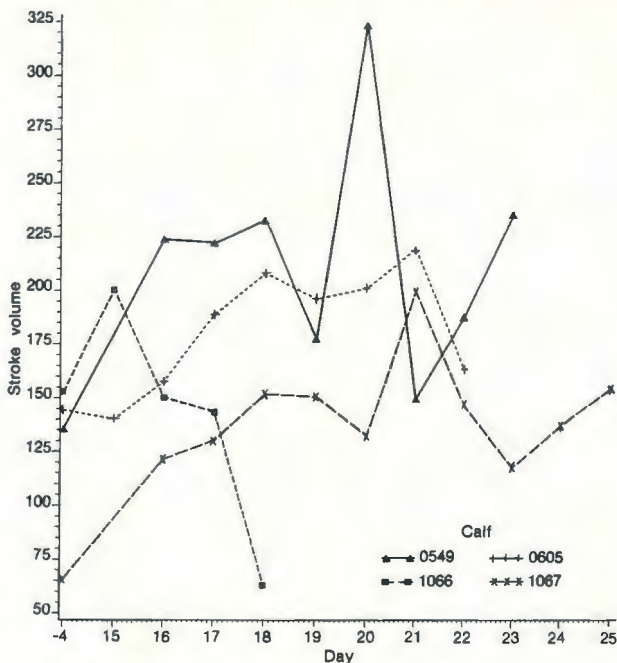


FIG. 8 Results of the stroke volume (ml)

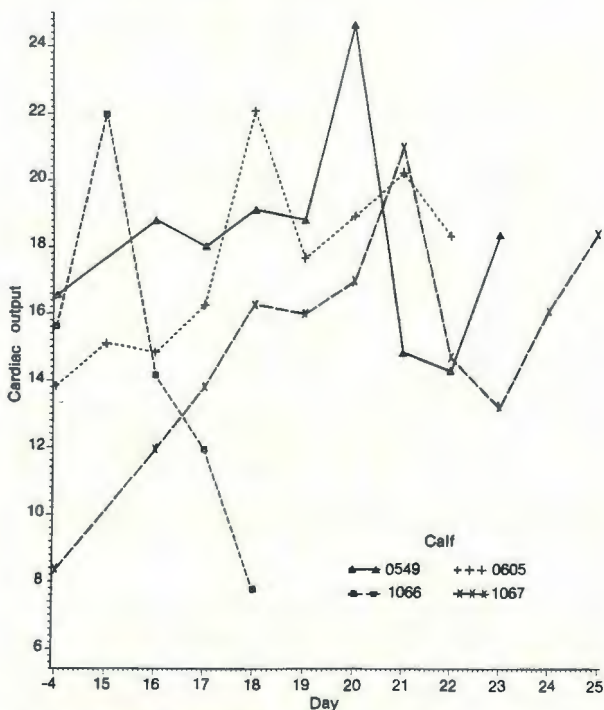


FIG. 9 Results of the cardiac output (l/min)

in this animal, it may have been associated with the fever and possibly also the handling.

Calf 0549 showed a severe drop in arterial PO_2 and a rise in PCO_2 . This was associated with an apparent reduction in minute volume. The latter was also seen in Calf 1066 on the day it died, but without such marked changes in the arterial PO_2 and PCO_2 as compared with that in Calf 0549. However, the venous admixture was high in Calf 0549, possibly indicating the presence of lung oedema or pulmonary vascular shunting (Nunn, 1969).

The remaining calf (0605) did not show any marked changes in blood gas analysis during the course of the disease.

Respiratory function

The results of the respiratory parameters measured are shown in Fig. 2, 3, 4 & 5. Initially, the mean respiratory minute volume (Fig. 2) dropped from 50.2 l on Day 4 before infection to 40.2 l on Day 17 post-infection. The initial minute volumes recorded in these calves, however, may not be a true reflection of their normal status as a moderate metabolic acidosis was present at the time (Table 1) and these calves were clearly trying to compensate by reducing their arterial PCO_2 levels by means of hyperventilation (Table 1). This apparent reduction in minute volume through Day 17 post-infection may thus not have played a major role in the blood gas changes as described. If the foregoing hypothesis is accepted, then calves 1067, 0605 and 0549 were clearly hyperventilating on Day 18, while Calf 1066 showed a definite reduction in minute volume (26 l) on that day which was the day of its death. This reduction may have been associated with an existing brain oedema, the presence of which was shown clinically in Calf 1066 (S. R. van Amstel, unpublished data, 1987).

From Fig. 2 it is evident that the minute volume did not manifest a consistent pattern during the course of the disease and it appears that individual peaks, as seen in Calf 1067 on Day 18 and 23, may well have been iatrogenic in nature.

Fig. 3 & 4 reveal a tendency towards increases in both physiological and alveolar dead space during the acute stage of the disease. This may be associated with lung oedema and could explain the moderate reduction in PO_2 seen during the acute stage of the disease.

Results of the calculated venous admixture are shown in Fig. 5. There appears to be some correlation between the venous admixture and alveolar dead space in individual calves suggesting that peaks in venous admixture observed may in part be due to a decreased ventilation/perfusion ratio (Nunn, 1969). The effect of frank vascular shunt on the results of the venous admixture is not known.

Cardiovascular function

Heart rate

The results of the heart rate counts of the calves are shown in Table 2. There was a progressive decrease in heart rate up to Day 17, which was probably associated with the animals adapting to handling procedures. As all 4 calves had rectal temperatures in excess of 40 °C from Days 16–18 post-infection, this parameter did not seem to have a major effect on heart rate. The most dramatic change in heart rate was seen in Calf 1066 that died. Its heart rate increased from 83/min on Day 17 to 124/min on Day 18, when it died. This increase in heart rate was apparently not associated with blood pressure changes, as both the systolic and diastolic blood pressure had been rising for the 2 days immediately prior to its death and was within normal limits the day it died (Fig. 6 & 7). A marked reduction in both stroke volume and cardiac output occurred concomitantly with a marked elevation in heart rate (Fig. 8 & 9). It is probable that the observed tachycardia was a compensatory response to maintain cardiac output.

Blood pressure

Wide fluctuations in both systolic and diastolic blood and right ventricular pressures occurred during the course of the disease. As with heart rate, there was an initial drop in mean blood pressure from the first recording 4 days before infection until Day 17 post-infection. This was probably also caused by the calves' adaptation to handling. The blood pressure changes in the calf

(1066) that died are shown in Fig. 6 & 7. There was only a slight drop in its mean right ventricular pressure (13 mm Hg 4 days prior to infection to 9 mm Hg on the day it died).

Stroke volume and cardiac output

The results of the stroke volume and cardiac output of the experimental calves are shown in Fig. 8 & 9. A progressive increase in both stroke volume and cardiac output were recorded during the acute stage of the disease in the 3 calves that survived. In calf 1066 there was an initial rise followed by a progressive and marked drop in these parameters.

EKG findings

No abnormalities were found in the EKG parameters recorded. No arrhythmias were noted in any of the EKG tracings.

DISCUSSION

The wide variation in the results in these 4 calves suffering from experimentally-induced heartwater is probably attributable to the difference in the onset and severity of the disease. Although some general trends in cardiac and lung function are apparent from the results, the most meaningful results with respect to the objectives of this study were obtained from those of the calf that died.

Acid base studies showed a tendency towards the development of a hypoxemia during the acute stage of the disease. The arterial CO₂ tension, however, remained within normal limits and there was a tendency towards a mild alkalosis. These results are consistent with those expected with a mild to moderate lung oedema. In the latter situation there is soon impedance to O₂ diffusion, but not to that of CO₂, as the latter is approximately 20 times as soluble as O₂ (Nunn, 1969). Owen *et al.* (1973), in their study on experimentally-induced heartwater in sheep, described the presence of a hypoxia as well as CO₂ retention with a concomitant respiratory acidosis. A lung oedema was found at post-mortem, but the severity of this lesion was not described.

Despite the presence of a severe lung oedema as revealed during the post-mortem examination of Calf 1066, the PaO₂ level was only moderately depressed (77.1 mm Hg) and the PaCO₂ was within normal limits (41.3 mm Hg). These findings raise the question of the degree to which disturbances in acid-base homeostasis contribute to the cause of death in clinical cases of heartwater. It should be noted that the last analyses of blood gas parameters were made approximately 12 h before the death of this animal (calf 1066) and thus may not be representative of the findings just prior to death.

In this study, no clear pattern emerged as to the exact mechanism of the respiratory dysfunction. The minute volume in the calf that died showed a marked terminal reduction. This could possibly be associated with brain oedema, the presence of which was confirmed clinically in this animal by means of electroencephalography (S.R. van Amstel, unpublished data, 1987). No clear pattern emerged from pulmonary dead space and venous admixture parameters, although there was an indication that there was an increase in physiological dead space. This increase in physiological dead space was probably associated with the development of lung oedema. The contribution of ventilation/perfusion mismatching and true vascular shunting to venous admixture was not investigated in this study and needs to be fully investigated. Interpretation of the analyses based on the measurement of mixed expired gas concentrations is compounded by the fact that there may have been some contamination of

the mixed expired gas with gas eructated from the rumen.

No marked changes were observed in the systolic or diastolic blood or right intraventricular pressures. In the calf that died the blood pressure was within normal limits on the day it died. Clark (1962) and Owen *et al.* (1973) reported a reduction in systolic and diastolic pressures in experimentally-induced heartwater in sheep. Lowering in diastolic pressures was particularly severe. Changes in blood pressure need to be further investigated, as supportive treatment in this regard may play an important role in the management of clinical cases.

There was a marked increase in heart rate in Calf 1066 that died. This was associated with marked reductions in both stroke volume and cardiac output. This tachycardia was probably reflexly induced in an attempt to maintain adequate circulation.

The 3 surviving calves showed a progressive increase in stroke volume and cardiac output during the acute stage of the disease which were not correlated with tachycardia.

Absence of any EKG abnormalities suggests that no primary cardiac damage occurred and that conduction remained normal.

The results of this study seem to indicate that reduced cardiac function is a major contributing factor to the death of animals suffering from heartwater. The reduced cardiac function probably occurs secondary to the development of a severe hydropericardium, as was present in the fatal case. Medical management of hydropericardium should be investigated as possible rational supportive therapy in clinical cases of heartwater.

CONCLUSIONS

Cardiac and pulmonary function in clinical cases of heartwater needs further study. More meaningful results would probably be obtained if the course and severity of experimentally-induced heartwater was better controlled. This could be achieved by using a higher dose of the vaccine strain or by using a more virulent strain of *Cowdria ruminantium*.

Discrepancies in blood pressure changes seem to exist between this study and those of Clark (1962) and Owen *et al.* (1973). Should the marked drop in stroke volume and cardiac output be a general finding in severe cases of heartwater, then special attention should be given to the pathogenesis and management of hydropericardium.

The medical significance of the lung oedema may be less than is generally accepted, as it appears that the lung retains its capability to adequately oxygenate the blood. To fully validate this finding indices of pulmonary function need to be studied in the 12 h just prior to death. The contribution of pulmonary hypotension, ventilation/perfusion mismatch, true vascular shunting and venous admixture in the pathogenesis of pulmonary dysfunction needs to be investigated.

Control of cardio-pulmonary dysfunction is largely dependent on demonstrating the causes of vascular permeability changes which develop during the acute stage of the disease (Clark, 1962). The mechanism of this increased permeability is unknown. Prozesky & Du Plessis (1985) found only mild morphological changes in the alveolar walls of sheep and goats suffering from experimentally-induced heartwater. In the same study, ultrastructural examination of endothelial junctions revealed that the occurrence of gaps at such junctions occurred infrequently. Prozesky & Du Plessis (1985) speculated that the underlying mechanism in the formation of the lung and pericardial effusion may be an endotoxin liberated by *Cowdria ruminantium*. This possibility needs to be investigated.

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