# A FIELD OUTBREAK IN ILE-DE-FRANCE SHEEP OF A CARDIOTOXICOSIS CAUSED BY THE PLANT *PACHYSTIGMA PYGMAEUM*(SCHLTR) ROBYNS (RUBIACEAE)

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# **ABSTRACT**

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A field outbreak of *Pachystigma pygmaeum* intoxication in sheep is described. Noteworthy clinical signs were: respiratory distress, apathy and subcutaneous oedema of mainly the head. Gross changes included cardiomegaly, centrilobular hepatic necrosis and effusion of body cavities. Microscopically myocardial fibrosis, affecting predominantly the endocardium of the apex, left free ventricular wall and interventricular septum, was most striking in the majority of animals, whilst myofibre atrophy was present in 1 sheep.

#### INTRODUCTION

'Gousiekte' is a cardiotoxicosis of ruminants characterized by heart failure c. 3–8 weeks after the ingestion of certain rubiaceous plants (Kellerman, Coetzer & Naudé, 1988). Domestic ruminants are those mainly affected, and it appears that the springbok (Antidorcas marsupialis) is also susceptible (L. Prozesky, unpublished data, 1986).

While the majority of animals die peracutely without premonitory signs, a few animals may show clinical signs of congestive heart failure such as lethargy, dyspnoea and tachycardia a few days prior to death (Theiler, Du Toit & Mitchell, 1923; Jackson, Needham & Lawrence, 1968; Pretorius & Terblanche, 1967; Pretorius, Terblanche, Van der Walt & Van Ryssen, 1973). The underlying pathological lesion is a multifocal to diffuse myocardial fibrosis, usually accompanied by a mild to moderate round cell infiltration (mainly macrophages and lymphocytes) predominantly in the endocardium of the apex, left free ventricular wall and interventricular septum (Theiler et al., 1923; Smit, 1959).

The pathogenesis of 'gousiekte' is still unclear. Snyman, Van der Walt & Pretorius (1982) concluded that deviations of the myocardial energy metabolism may be primary or secondary in the genesis of the heart failure. Furthermore, a reduction in calcium uptake by fragmented sarcoplasmic reticulum *in vitro* and ultrastructural changes in the myofibrils were also considered possible causes of cardiac insufficiency (Pretorius *et al.*, 1973; Schutte, Els, Booyens & Pienaar, 1984).

This outbreak of 'gousiekte' is reported because of the unusual large number of animals that showed signs of congestive heart failure.

### FIELD OUTBREAK

A farmer from the Delmas district in the Transvaal highveld lost 37 out of 60 stud Ile-de-France sheep from March – May, 1986. Twenty 2–4-month-old lambs and 3 adult animals survived.

The animals were raised on artificial pastures and occasionally put on natural veld. No toxic plants were present in the camps, but *Pachystigma pygmaeum* (Fig. 1) was identified on the adjoining farm. According to the farmer, a few weeks prior to the death of the 1st sheep the animals managed to escape into the neighbouring farm and eat the lush green *P. pygmaeum* that was present among the tall grass.

On visiting the farm during April 1986 23 sheep had died and approximately 10 animals in the flock were

seen to be anorectic and apathetic. Varying degrees of subcutaneous oedema, particularly of the head and often extending to the sternal region, was present in 6 of the diseased sheep (Fig. 2). A few animals were in sternal recumbency with their necks stretched out, were dyspnoeic and made grunting noises during inspiration and expiration. On auscultation of a severely affected animal, tachycardia and a gallop rhythm were evident.



FIG. 1 Pachystigma pygmaeum



FIG. 2 Oedema of the head caused by congestive heart failure

# MATERIALS AND METHODS

Four adult ewes that showed signs of cardiac failure were necropsied: 1 sheep died on the farm while being handled; 2 ewes with signs of respiratory distress died when loaded onto a truck; and 1 animal was slaughtered. Specimens of various organs were collected in 10 % buffered-formalin, routinely processed and stained with

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haematoxylin and eosin. Additional staining techniques applied to myocardial sections included the Masson's trichrome stain for collagen, the Hotchkiss periodic acid-Schiff for mucopolysaccharides and the Marchy scarlet blue stain for fibrin (Anon., 1968).

#### RESULTS

# Gross pathology

The carcasses were cyanotic, and in 2 animals the subcutaneous oedema of the head and neck extended to the sternal region. A mild to moderate hydrothorax occurred in all the sheep, and the straw-coloured fluid coagulated on exposure to air. Apart from lung congestion and a prominent oedema, the ventral lung borders were atelectatic from hydrothorax. Oedema of the mediastinum and associated lymph nodes accompanied the lung lesions.



FIG. 3 Myocardial dilatation as a result of 'gousiekte' (left) and a normal heart (right)

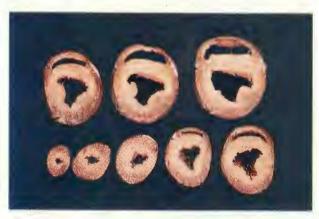




FIG. 4 & 5 Coronal sections of the myocardium. Note the pale endocardial zone

A mild to moderate hydropericardium was evident and the hearts were prominently enlarged and pale-brown (Fig. 3). The ventricles were dilated, the free ventricular walls thinned and the apex appeared more round than normal. Rigor mortis of the heart was practically absent approximately 5 h after death, and the free ventricular walls had a rubbery consistency. Coronal sections of 3 formalin-fixed hearts revealed a pale endocardial zone extending to the middle of the free ventricular walls and involving almost the entire interventricular septum (Fig. 4 & 5). This discoloration was more conspicuous in the left than in the right side of the heart.

Ascites  $(c.\ 2\ \ell)$  occurred in all the animals. Apart from a prominent hepatomegaly the surface of the liver was uneven and the lobulation accentuated. The cut surface of the organ had a nutmeg appearance, and multiple large areas of congestion were scattered throughout the parenchyma (Fig. 6). The gall bladder wall was oedematous.

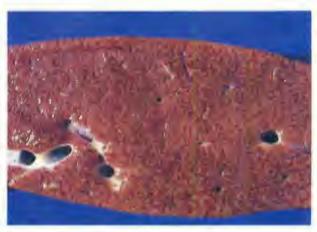


FIG. 6 The liver has a nutmeg appearance and accentuated lobulation

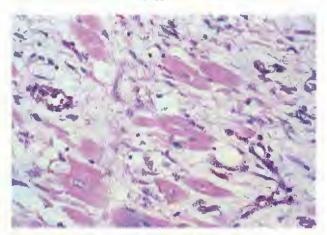


FIG. 7 Myocardial fibrosis predominantly affecting the endocardium  $HE \times 300$ 

The kidneys of 2 animals were markedly swollen, were light-brown and the congested medulla was clearly demarcated from the pale cortex. In 3 animals there was a mild to moderate splenomegaly, and the white pulp was hyperplastic. The abomasal folds were mildly oedematous in 1 sheep, and blood was present in the large and small intestines of 2 animals.

# Microscopical pathology

Myocardium: Focal disseminated to coalescing fibrosis, affecting predominantly the endocardium of the apex, left free ventricular wall and interventricular septum, was most striking in 3 animals (Fig. 7). Individual

or small groups of round cells (mainly macrophages and lymphocytes) were often present in areas of fibrosis, particularly in the right free ventricular wall. In both the left and right free ventricular walls the lesions were less striking towards the auricles. Single or small groups of affected myofibres, characterized by a loss of striation, hyalization of the sarcoplasm, the presence of contraction bands, binucleation and anisonucleosis, were seen in areas of fibrosis. Some myofibres appeared to be atrophic.

Apart from hyaline degeneration of a few haphazardly scattered myofibres, atrophy of most of the myocytes was the only other change in the myocardium in 1 animal (Fig. 8). Although it was difficult to demarcate the affected areas, it seemed that the endocardium of the left and right ventricles were mainly involved.

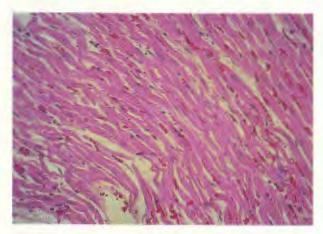


FIG. 8 Myocardium: Atrophy of myocytes: HE × 300

In 2 animals, a diffuse or occasionally segmental hypertrophy of the tunica media and/or deposition of a fine fibrinoid material (Fig. 9) in the tunica intima of the medium and larger arteries was notably predominant in areas of fibrosis. Other changes included hypertrophy of endothelial cells and occasionally fine vacuolation of the tunica media of small and medium arteries.

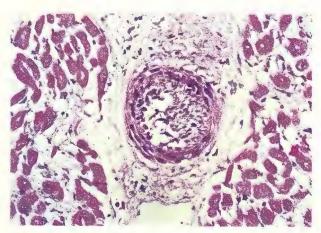


FIG. 9 Segmental thickening of the intima of a coronary artery characterized by the deposition of a fine fibrinoid material:  $HE \times 300$ 

In 1 animal with myocardial fibrosis, Ashoff bodies (small groups of lymphocytes, macrophages, multinucleated cells, a few eosinophils and Anitschkow's cells) were mainly associated with larger blood vessels in the epi- and endocardium (Fig. 10).

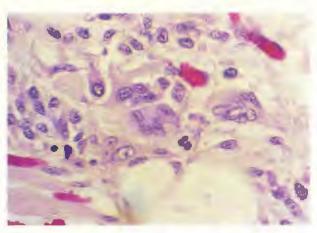


FIG. 10 Ashoff body in association with larger blood vessels:  $HE \times 1200$ 

Other organs: The livers in all the animals showed a mild to moderate centrilobular coagulative necrosis, whereas the remaining hepatocytes were affected by vacuolar degeneration. The sinusoids were dilated and congested.

Vacuolar degeneration of the tubular epithelial cells of the kidneys and proteinaceous material in Bowman's space were observed in 2 animals. The lung lesions in all the animals were characterized by congestion, oedema and scattered alveolar macrophages.

## DISCUSSION

This outbreak of 'gousiekte' was unusual in that many animals showed signs of congestive heart failure, such as respiratory distress and oedema, mainly of the head.

According to Theiler et al. (1923), dilatation of both ventricles and thinning of the free ventricular walls were present in the majority of intoxicated animals. Other workers claim that more frequently the hearts of animals with 'gousiekte' are normal in size and the ventricular walls thin and tough in consistency (Newsholme & Coetzer, 1984). We found that the detection of mild degrees of myocardial dilatation was extremely difficult and subjective, and the majority of animals that died of 'gousiekte' showed various degrees of ventricular dilatation and thinning of the free ventricular walls. No morphometric study on the myocardial changes associated with 'gousiekte' has yet been made.

In South Africa there are many cardiotoxic plants that affect stock (Kellerman et al., 1988). Except for 'gousiekte' intoxication all the other cardiotoxic conditions have a short course. Apart from occasional cases of 'gousiekte', where atrophy of myocardial fibres may be the most striking light microscopical lesion, atrophy is not a feature of any of the other plant poisonings (L. Prozesky, unpublished data, 1986).

In this outbreak, cardiac dilatation occurred in animals with either extensive myocardial fibrosis or mainly myofibre atrophy, indicating that overstretching of both affected myofibres and connective tissue may play a role in cardiac dilatation.

Hypertrophy of the tunica media and thickening of the tunica intima of medium and large coronary arteries were present in 2 of the sheep with myocardial fibrosis. This change has not previously been associated with 'gousiekte', but similar changes have been described in humans with endocardial fibrosis (Andrade & Teixeira, 1973). At this stage it seems unlikely that the vascular lesions are of primary importance in the pathogenesis of the intoxication.

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