TRAUMATIC PERICARDITIS OF CATTLE.

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Introduction.

Perforation of the pericardial sac by a sharp foreign body which has been swallowed and which penetrates the reticular wall and the diaphragm is a well-known disease, particularly among dairy cattle. Diagnosis of the disease is usually quite simple although endocarditis, pleurisy and mediastinitis may present very similar clinical symptoms. Surgical treatment for the condition has been recommended in the past but considerations of economy and practicability usually prevent this. Fifteen cases which were treated with sulphonamide drugs and penicillin and six which were not treated are recorded here. All of the patients were dairy cows presented to the large animal clinic of the faculty over a period of three years, during which a total of 30 cases of pericarditis and 180 cases of traumatic reticulo-peritonitis were attended amongst the same cattle population. Factors involved in the production of reticular perforation have been discussed elsewhere (Blood and Hutchins, 1955).

Clinical Findings.

The first diagnostic sign of pericarditis is the pericardial friction sound caused by the early inflammation of the epi- and pericardial surfaces. Until this is observed during the development of a case, a diagnosis of reticulo-peritonitis is all that can be made. In our series pericarditis was observed to develop in five cases and friction sounds were heard usually on the third day after the initial symptoms of reticulo-peritonitis had appeared (one day in one case, three days in three cases and four days in one case).

However, serious involvement was suspected if the cow was in the last three months of pregnancy (of 20 affected animals, 10 were in the last three months of pregnancy, two were in early pregnancy and eight were empty) and if the temperature was above 105°F. and the heart rate above 90 per minute at the first examination. Under these circumstances an examination of the white cell count and differential count was made and added further evidence. The friction rub is quite characteristic but may be confused with an endocardial bruit caused by a valvular lesion or a pleuritic friction sound especially when the pleural surface of the pericardium is the main site of inflammation. The endocardial bruit is softer and the pleuritic rub much louder than the pericardial sound. Fluid accumulation in the pericardial sac followed the initial inflammation in a period varying from one to ten days and was accompanied by signs of congestive heart failure. Jugular vein engorgement was the earliest sign, followed by oedema of the brisket, underline, neck and throat. In one severe case, with the animal grazing on pasture, marked oedema of the conjunctiva was apparent, the soft folds of palpebral conjunctiva hanging over the eyelids like bunches of grapes. Ausculation revealed an increased area of cardiac impulse and a muffling of the heart sounds. Other abnormal sounds could be heard especially splashing sounds if gas was present or tinkling or ticktack sounds. The occurrence of such abnormal sounds in the absence of signs of congestive heart failure may indicate the presence of fluid in the pleural sac rather than pericarditis. A considerable increase in the heart rate was characteristic (12 of our series of 14 cases had heart rates of more than 100 per minute with an average of 115 per minute and all were over 90 per minute). At these rates the pulse amplitude was very small and in some cases the pulse was impalpable.

Body temperatures were often high (up to 107°F.) and varied with the stage of the disease but pyrexia was not a diagnostic feature. In the cases described here the average of the highest temperatures observed during the course of the disease was 103.5°F. The high temperatures were maintained over a long period or receded quite quickly.

Severe thoracic pain was generally apparent. Firm palpation with the thumb pushed between the ribs in the pericardial area caused the animal to grunt. If peritonitis was also present percussion of the hypogastrium also caused

grunting. Abduction of the elbows was common in severe cases and respiration was usually increased in rate (30 to 60 per minute), shallow and often grunting. Such signs of thoracic pain may be evident in other thoracic diseases especially when there has been perforation of the pleural sac or the lung by a foreign body. Where congestive heart failure occurred the respiratory movements were usually very much increased and the animal often became dyspnoeic.

Alimentary tract symptoms were variable depending on the stage of the illness. When reticular perforation had occurred only a day or two previously ruminal movements were absent or weak and anorexia was complete. Several days later at the stage when pericarditis was evident the rumen had regained some motility (usually one moderate movement per one or two minutes) and the appetite returned a little. At a still later stage when congestive heart failure was present severe scouring occurred possibly due to back pressure in the portal venous system. The faeces were watery and offensive in smell.

The general appearance of the cows was suggestive of severe illness. The animal was dull, tended to lie or stand about in the one position, did not graze and lost condition rapidly. Exertion usually caused severe dyspnoea.

Clinical Pathology.

It has been suggested previously (Blood and Hutchins, 1955) that haematological observations are of value in the early diagnosis of reticular perforation and that a total leucocyte count of more than 12,000 per cmm. and a total neutrophile count of more than 8,000 per cmm. were indicative of serious complication. Lack of time and facilities prevented examinations on most animals and they were for the most part carried out in the early stages of the illness when confirmatory evidence was required. Details of serial observations on four animals are given below (Table 1). These serial observations were of little value as an aid to prognosis although use of the above criteria was of assistance in predicting whether or not pericarditis was likely to occur in the next few days.

Cases 1, 2 and 3 were all early cases showing symptoms of acute local peritonitis with sufficient increase in the pulse and temperature rates to warrant a haematological examination.

All haematological observations were carried out on jugular vein blood collected into potassium-ammonium oxalate bottles and examined within two hours of collecting. The longitudinal method of differential counting described by Dacie (1950) was used after staining with Leishman.

Treatment.

It had become fairly obvious from clinical and post-mortem examinations of previous cases that two factors combined to produce symptoms—congestive heart failure due to interference with heart movement by fluid and toxaemia from the bacterial invasion of the pericardial sac. In each case one of these two factors might be much more important than the other. In

TABLE 1.

Traumatic Pericarditis.

Serial Haematological Observations.

Case Ident.	Observation	Days of Illness						Outcome
		1-2nd	3-4th	5-6th	7-8th	9-11th	12-14th	
1	Tot. Leucocytes	11,400	—	—	10,082	—	3,720	
	Tot. Neutrophiles	7,524	—	—	7,556	—	1,116	Survived
2	Tot. Leucocytes	12,440	15,000	—	5,200	4,300	—	Died fif-
	Tot. Neutrophiles	8,832	10,275	—	3,448	2,218	—	teenth day
3	Tot. Leucocytes	12,000	—	15,200	12,840	—	—	Died six-
	Tot. Neutrophiles	9,840	—	11,020	9,858	—	—	teenth day
4	Tot. Leucocytes	—	—	—	16,500	14,500	—	Died
	Tot. Neutrophiles	—	—	—	10,972	11,600	—	twelfth day

the early stages of the disease the interference with heart movement is caused by the accumulation of inflammatory fluid often under considerable pressure. This compressive effect, when allied with probable myocardial weakness resulting from the bacterial toxaemia could be sufficient to cause death. Constrictive pericarditis of the later stages is due to the development of adhesions and fibrous tissue contraction and could be more compatible with life because the bacterial toxaemia has subsided. An animal in this stage is not likely to be an economical dairy unit but it is unlikely that any animal affected with pericarditis will be.

Bearing in mind these factors it was decided to attempt treatment of some animals with survival as the sole aim. Return to productivity was considered unlikely. Because previous investigations indicated that the perforating body was unlikely to be in its perforating position rumenotomy was considered unnecessary and was carried out on a few cases only and mainly for purposes of observation.

In the 15 cases in which treatment was attempted several drug regimes were used all of which were aimed at controlling the pericardial sac infection. Strict immobilisation by tying up on an inclined plane was enforced in all cases of recent origin to assist in controlling the spread of peritonitis. Sulphamezathine was administered at the rate of 1 g/15 lb. body weight per day once daily as a powder by mouth. Penicillin was used as a watery injection of procaine penicillin intramuscularly at 48 hour intervals at a standard dose rate of three million units per dose. The results are indicated in Table 2. For comparison a group of comparable animals not treated for economic reasons is included.

The surival rate with treatment was most satisfactory. However, the end-result left much to be desired. A period of convalescence of up to four months followed treatment and although complete follow-up data are not available at least some of the cows are indifferent producers and uneconomic as dairy animals. Penicillin appeared to have little effect on the results and this is thought to be due to the narrow range of organisms controlled by the drug. The number of animals treated was too small to determine whether the time at which the animal was first seen had any effect on the mortality rate. Of six animals in which treatment was begun during the first three days of their illness, four recovered and two died. Of nine animals in which treatment was instituted after the third day, four recovered and five died. In those animals which died the course of the disease did not appear to be significantly altered, death occurring on the 8th to 19th day (average—15 days). In the untreated control group deaths occurred on the 6th to 24th day (average—15 days). Exploratory rumenotomies were performed in three cases but had no influence on the outcome of the disease. In each case the foreign body was found lying free in the reticulum.

Autopsy Findings.

Autopsy findings were standard with an accumulation of foul-smelling serous fluid or thin creamy pus distending the pericardial sac in all cases. Heavy deposits of fibrin were usual and varying degrees of associated pleurisy and lung abscess of a similar appearance occurred in some cases. Bacteriological examination in several cases showed *Cor. pyogenes* and unidentified Gram-negative organisms to be present. The accumulation of serous fluid in the pleural and peritoneal cavities and in the subcutaneous tissues varied. A surprising feature was the absence of peritonitis in all but one case. This animal died after a 19 day illness and had acute diffuse peritonitis at autopsy. It appears that when perforation occurs penetration into the thorax is effected either through an existing sinus or that adhesions form so quickly that the

TABLE 2.

Results of Treatment of Traumatic Pericarditis.

Treatment	Survived	Died	% Mort.
Sulphamezathine alone	4	2	33
Sulphamezathine plus Penicillin*	4	3	43
Penicillin alone	—	2	100
Totals	8	7	47
No Treatment group	—	6	100

*One cow in this group responded so satisfactorily that she was released from immobilisation against our advice and died suddenly two weeks later with gangrenous pneumonia following breakdown of a pleuritic adhesion. At autopsy the pericardial infection was apparently well under control.

peritonitis has subsided by the time the animal dies of pericarditis.

Discussion.

Although pericarditis is relatively simple to detect attention must be given to the differentiation of three other disease entities. Valvular endocarditis presents an almost identical clinical picture (Johns, 1947; Udall, 1954) but has not been observed in a living animal in our practice. Traumatic pleurisy, especially when the pleural surface of the pericardial sac is involved, may be accompanied by friction sounds synchronous with heart movements but the sounds will be louder and no venous congestion or oedema occur. One case of traumatic mediastinitis has been attended in our clinic. Clinically the condition was identical with pericarditis but at autopsy perforation had occurred between the two pleural surfaces of the ventral mediastinum and the subsequent gross inflammation and suppuration caused pressure on the heart without actually involving the pericardial sac.

The importance of late pregnancy in the production of pericarditis rather than peritonitis suggests that great care must be taken when hand-feeding animals at this stage.

The attempt to control the infectious process in the disease appeared to be reasonably successful. No attempt was made to relieve pericardial sac pressure by continuous or intermittent drainage although such a measure combined with the antibacterial treatment may improve the survival rate.

Summary.

The clinical and pathology findings in 21 cases of traumatic pericarditis are presented. Treatment with sulphamezathine and penicillin appeared to reduce the mortality rate. Sulphamezathine appeared to be the more satisfactory of the two drugs.

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NOTICE TO CONTRIBUTORS

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REPRINTS

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