# DALMENY DISEASE. AN INFECTION OF CATTLE PRESUMED TO BE CAUSED BY AN UNIDENTIFIED PROTOZOON

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THIS PAPER DESCRIBES the clinical and pathological features of an outbreak of disease in a herd of dairy cattle. This disease was characterized clinically by fever, emaciation, anaemia, abortion and high morbidity and mortality in the adult cattle. The results of laboratory studies indicate that this condition was associated with the presence of a protozoan parasite morphologically similar to *Toxoplasma gondii.*

## HERD HISTORY

On December 11, 1961, the owner noticed two cows off feed and down in milk. He called his veterinarian who found an elevated temperature (104° F.) in these two animals. Three other cows had temperatures between 104° and 105° F. but no other symptoms. These five animals were given penicillin and streptomycin intramuscularly but showed no response. During the next six days all of the adult cattle in the main stable became affected. The main stable housed the bull, 20 cows, 3 heifers and a steer in two rows together with ten calves under nine months of age. In addition, there were three heifers and two horses in an adjacent but separate stable.

The symptoms seen were intermittent anorexia, drop in milk yield, rapid loss of condition, moderate pyrexia (up to 105° F.), and dyspnea. Diarrhoea was evident in one animal. Two eight-month-old calves had a transient temperature of 104° F. but no other clinical symptoms. One of the heifers in the horse stable went off feed on January 9, showed slight hypersalivation, a temperature of 104° F. for two days and recovered. As the outbreak progressed a haemorrhagic vaginitis developed in 13 animals and hypersalivation in 9. Ten of the 17 pregnant animals aborted during the outbreak. The aborting animals were in the last trimester of pregnancy and only one survived. In the salivating animals erosions were present on the tongue and buccal mucosa. Nasal hyperaemia was evident in a few animals.

Chronic cases were characterized by extreme emaciation, pale and occasionally icteric mucous membranes, extensive submandibular oedema, slight to marked exophthalmia, cessation of lactation and in most cases sloughing of the tip of the tail. As a state of cachexia was reached the animals became recumbent and showed fine muscular tremors over the entire body resembling those associated with hypocalcaemia.

Supportive and antibiotic therapy appeared to have no effect on the course of the disease. The bull died on December 19 and an additional 16 animals

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succumbed during the following eight weeks. Thus of the 25 animals affected, eight eventually recovered and three of these calved normally in March and April.

The outbreak occurred in a closed Holstein herd in an isolated location near the hamlet of Dalmeny in Eastern Ontario. There was no direct contact with other cattle. The stock on the premises was watered from a well. A V-shaped continuous wooden water trough ran along each of the two rows of stanchions in the main stable (Fig. 1). The remaining animals were watered individually.

FIGURE 1. View of stable showing affected animals and watering trough.

A group of 16 pigs housed in two pens in the main stable remained clinically normal throughout.

## GROSS PATHOLOGY

Post mortem examinations were conducted on four of the five animals that died and on 12 which were killed when moribund. The characteristic finding in the acute stage was severe and widespread haemorrhage. This haemorrhage varied from petechial to ecchymotic and was present in the subcutaneous tissues, perineural fascia, lymph nodes, mammary glands, larynx, trachea, lung, myocardium, thymus, spleen, kidney, oesophagus, vagina, cervix and the serosal and mucosal surfaces of the entire intestinal tract, gall bladder, urinary bladder and uterus. Haemorrhage was particularly severe in the myocardium and a "tiger striping" effect was evident in the rectum. The lymph nodes were generally enlarged and oedematous. The trachea was often filled with froth and the lungs were oedematous and emphysematous. Excessive fluid was present in the body cavities and the body tissues had an icteric discolouration. The urinary bladder frequently contained coffee-coloured urine.

Although haemorrhage was present in all animals it was much less severe in animals examined later in the outbreak. As the disease progressed oedema of the lymph nodes, larynx, mediastinum, mesentery, abomasal folds, submandibular tissues and brisket became more pronounced. Greater quantities of fluid were found in the body cavities. Punched out ulcers were present on the lips, gums, cheeks, dorsal and lateral aspects of the tongue, abomasal folds and colon. This ulceration involved the serrations of the palatine ridges and formed a linear interlacing pattern on the soft palate and relatively long continuous erosions

in the oesophagus. Metritis was present in the animals which had aborted and macerated foeti were found in two others. The lungs of several animals showed pneumonic areas. Excessive cerebrospinal fluid was found in the majority of chronic cases.

## HISTOPATHOLOGY

Tissues from the 16 animals necropsied were examined histologically. These tissues were fixed in 10 per cent formalin in physiological saline. Paraffin sections were routinely stained with hematoxylin and eosin.

A cyst-forming protozoon resembling *T. gondii*, was demonstrated parasitizing the endothelial cells of blood vessels in the lung, myocardium, kidney, spleen, brain, lymph nodes, thymus, mammary gland, uterine caruncles, pancreas, intestinal tract, gall bladder and submaxillary salivary gland. These cysts were found in 11 of the 16 animals.

The term cyst is used because the situation in this disease appears to be analagous to that described in chronic toxoplasmosis (1, 2). What appeared to be different stages in the development of these cysts were observed. The presumably immature stage showed an eosinophilic staining limiting membrane

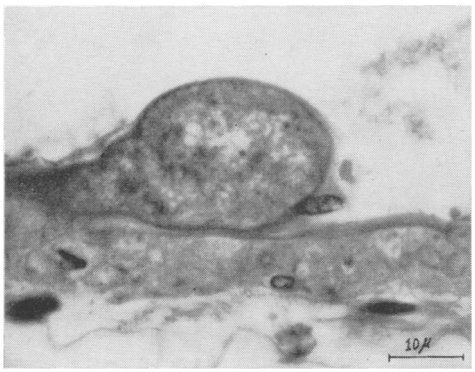


FIGURE 2. Immature stage of cyst formation in blood vessel in brain. H & E.

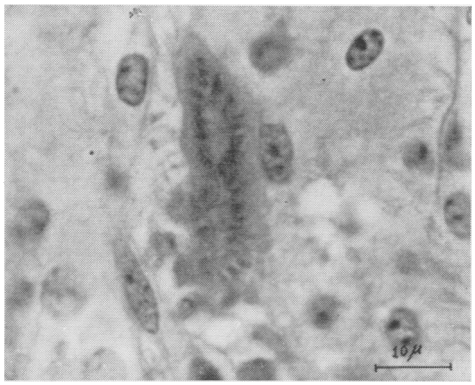


FIGURE 3. Cyst within renal medulla showing palisade arrangement of parasites. H & E.

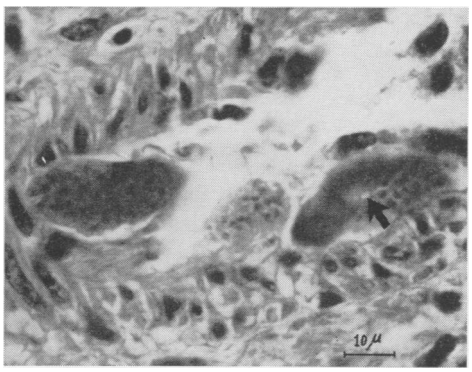


FIGURE 4. Cyst in follicular artery of spleen. Arrow indicates apparent lumen within cyst. H & E.

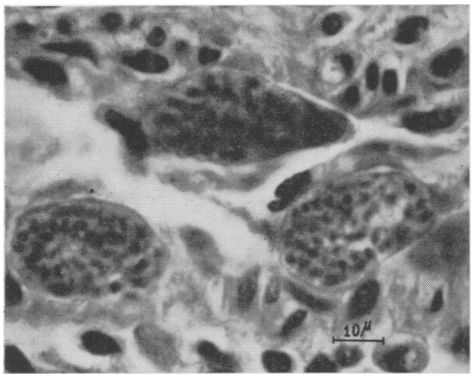


FIGURE 5. Mature cysts within follicular artery of spleen. H & E.

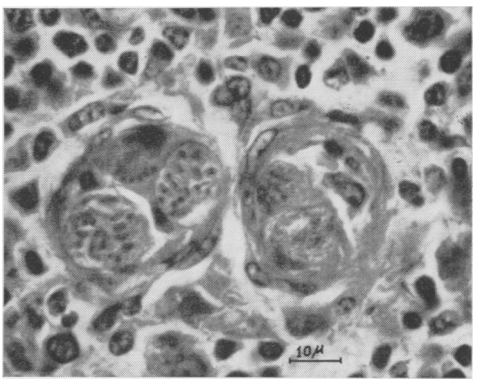


FIGURE 6. Lumen of vessel on left is completely occluded by presence of three cysts. Vessel on right contains mature (upper) and immature (lower) cysts. Spleen. H & E.

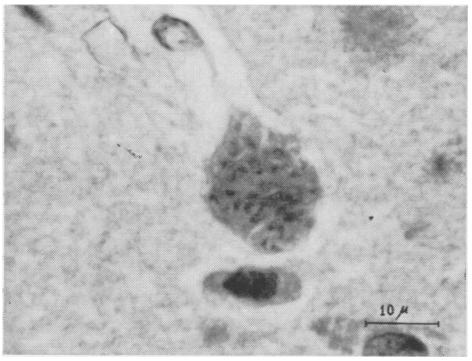


FIGURE 7. Cyst occluding cerebral capillary. H & E.

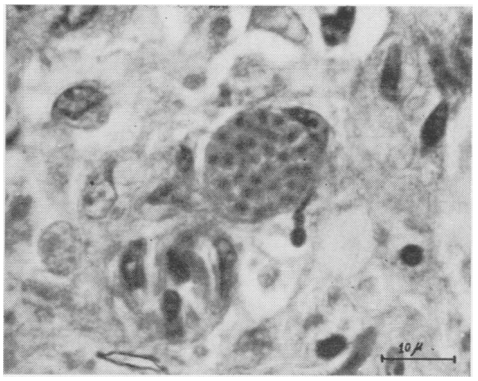


FIGURE 8. Cyst occluding capillary in uterine caruncle. H & E.

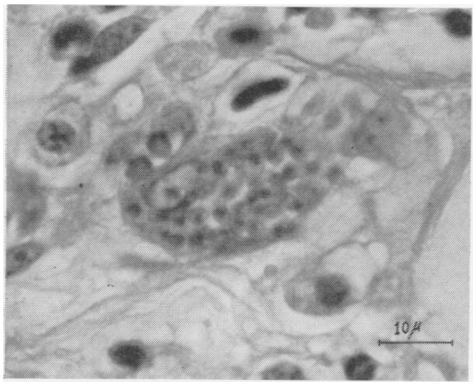


FIGURE 9. Cyst showing individual circular and oval parasites. Thymus. H & E.

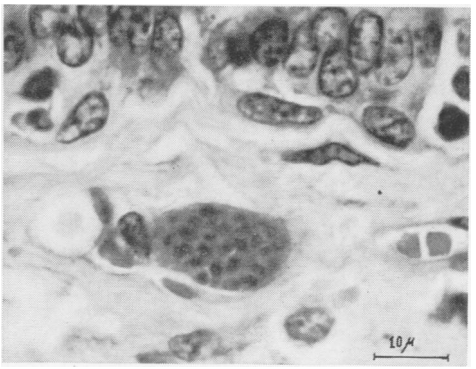


FIGURE 10. Mature cyst in endothelial cell of capillary. Mammary gland. H & E.

containing a basophilic granular dust and one or more larger eosinophilic bodies (Fig. 2). The next stage in maturation was characterized by the presence of individual parasites distributed in palisade fashion at the periphery of the colony leaving a central zone devoid of parasites (Fig. 3). In some cases this latter area appeared to form a lumen (Fig. 4). In the mature cyst the entire volume was filled with parasites (Figs. 5–10). Generally it was impossible to ascertain the exact morphology of the individual parasite in the mature cyst. Those found in the cyst in the palisade pattern were oblong in shape but where the parasites were not tightly packed they appeared oval or circular (Fig. 9). Both the cysts and individual parasites varied considerably in size and shape. The diameter of the cysts varied from 7.5 to 19.9 $\mu$, with elongated cysts measuring up to 37.2 $\mu$ in length. Oval parasites measured up to 2.1 $\times$ 3.0 $\mu$ whereas the fusiform type measured up to 1.9 $\times$ 4.7 $\mu$.

The parasites consisted of a basophilic nucleus and a pale eosinophilic cytoplasm but where compacted they appeared as a mass of basophilic dots on a deeply eosinophilic background. Little success was achieved in staining these organisms by P.A.S. or Giemsa methods but P.A.S. positive granules were found within the cysts. Cysts which parasitized small vessels tended to conform to the shape of the vessel and appeared elongated (Fig. 11), whereas those occupying larger vessels projected into the lumen assuming a more or less oval shape (Figs. 2, 4, 5 and 12). As in chronic toxoplasmosis, the intact cyst evoked no host reaction except where occlusion of the vessel occurred. An inflammatory reaction was present around recently ruptured cysts (Figs. 13–15). Cysts were numerous in animals dying in the acute stage but became more difficult to demonstrate in the chronic cases.

The lungs of animals in the acute phase showed congestion, petechial haemorrhage, oedema within the alveoli and interlobular tissues, alveolar and interstitial emphysema, focal infiltration of the alveolar walls by mononuclear cells with lymphocytes and plasma cells predominating, and mild lymphoid hyperplasia. Numerous cysts were present in the alveolar walls and occasionally they assumed a worm-like appearance where they conformed to the outline of the containing capillary (Fig. 11). Cysts were also found in parabronchial lymph

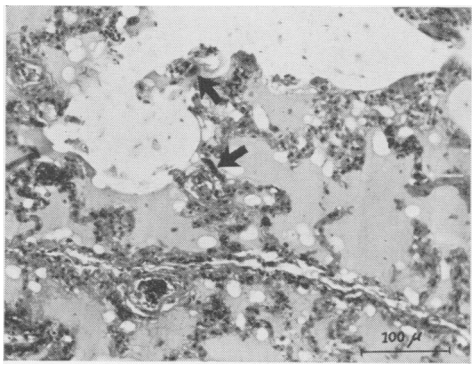


FIGURE 11. Acute reaction in lung. Arrows indicate cysts. Lower cyst has worm-like shape. H & E.

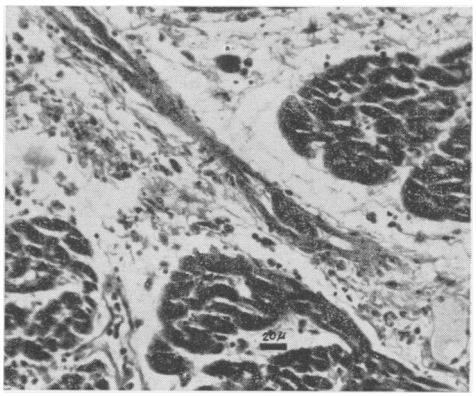


FIGURE 12. Reaction in myocardium. Note presence of cyst on vessel wall.

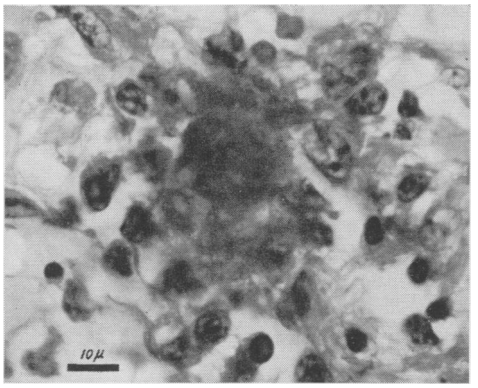


FIGURE 13. Rupturing cyst and accompanying reaction. Myocardium. H & E.

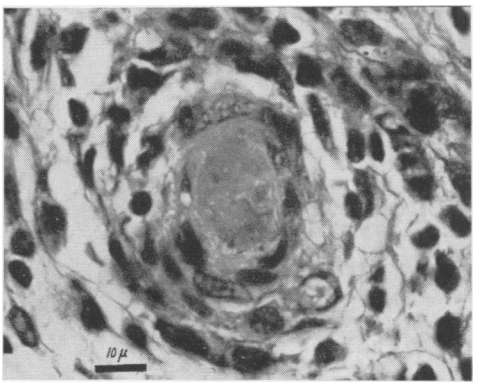


FIGURE 14. Occlusion of vessel by recently ruptured cyst within intestinal sub-mucosa. Note accompanying reaction. H & E.

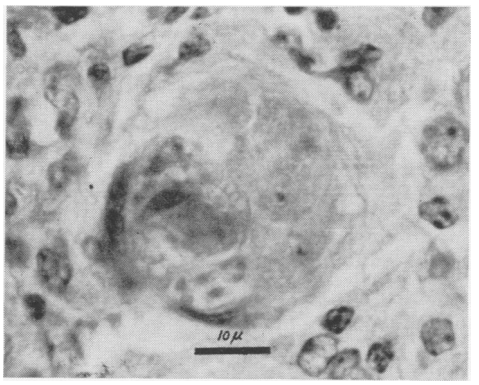


FIGURE 15. Blood vessel occluded by recently ruptured cyst and immature cyst (left centre). Pancreas. H & E.

nodules. Many animals showed pneumonic changes due to secondary bacterial infection. In the later stages the oedema and haemorrhage subsided and a large amount of blood pigment was seen in macrophages within the alveolar walls and parabronchial lymph nodules. The alveolar walls became thickened and staining by Van Giesen's method revealed an increase in smooth muscle. This muscle proliferation sometimes produced a picture not unlike that seen in the normal avian lung (Fig. 16). Focal accumulations of chronic inflammatory cells and lymphoid hyperplasia were present throughout the course of the disease.

The myocardium was extremely haemorrhagic particularly in the acute cases. Focal accumulations of leucocytes consisting principally of mononuclear cells, and an occasional polymorphonuclear cell were present (Fig. 12). These leucocytic foci were more prominent beneath the epi- and endo-cardium. Cysts were found in the endothelial cells of blood vessels and the odd recently ruptured cyst were seen (Fig. 13). The endothelial cells, especially those of the capillaries,

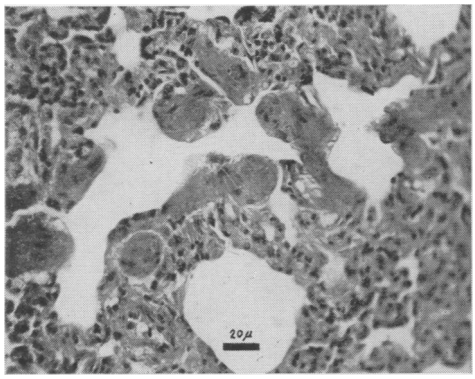


FIGURE 16. Hyperplasia of smooth muscle in lung of chronic case. H & E.

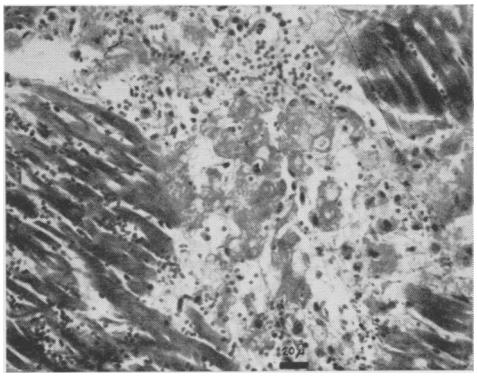

FIGURE 17. Myocardial degeneration and haemorrhage. H & E.

appeared swollen. The myocardium displayed foci of degeneration which showed up as areas of amorphous, billowy, pink-staining material (Fig. 17). As the outbreak progressed the degree of haemorrhage diminished but the focal areas of mononuclear cell infiltration and areas of myocardial degeneration became more distinct.

In the kidney numerous cysts were present in the glomeruli during the acute phase of the disease. The glomeruli appeared swollen and relatively bloodless. Cysts were also scattered throughout the renal medulla (Fig. 3). Bowman's spaces usually contained quantities of amorphous proteinaceous material. As in other organs in the acute phase of the disease haemorrhage was a characteristic feature. In a few cases complete necrosis of the glomeruli was noted. Perivascular, peritubular and periglomerular aggregations of mononuclear cells, not unlike those seen in leptospirosis were present to a varying degree in all kidneys examined. Haemosiderin was found free within the lumina of the tubules as well as within the cells of the tubular epithelium. In some cases pigment casts were formed. A focal embolic nephritis was present in one animal associated with an acute bacterial pneumonia. In the later stages the glomerular endothelium appeared hyalinized and periglomerular fibrosis was noted.

Large amounts of haemosiderin were present in the spleen and numerous cysts were found particularly in the follicular arteries (Figs. 4–6). In some cases the cysts occluded the vessel lumen (Fig. 6). Small areas of coagulation necrosis were often present. Megakaryocytes and active haemopoiesis were noted in the majority of spleens. In one of the animals affected with a secondary bacterial pneumonia an acute splenitis was observed.

No cysts were demonstrated within the liver proper but they were found in the wall of the gall bladder along with foci of lymphocytes and plasma cells. Bile stasis was a feature of the early cases while mild fatty metamorphosis and lymphocytic infiltration in the portal areas were noted in nearly all livers examined. The Kupffer cells were usually stuffed with haemosiderin and haemopoiesis was evident.

Cysts were found within the endothelial cells of vessels both in the brain (Fig. 7) and in the meninges. Small areas of gliosis with necrotic centres were

scattered throughout the brain (Fig. 18). Mild mononuclear cell infiltration of the cerebral meninges as well as mild perivascular cuffing and swelling of the endothelial cells was noted. Occasionally haemosiderin was present in the perivascular reaction. Virchow-Robin spaces were increased in size and what were assumed to be two calcified cysts were found in the brain of one of the last animals killed.

The various body lymph nodes showed a similar type of reaction although the supramammary, mediastinal, bronchial, portal, mesenteric and inguinal nodes were always more severely affected. Haemorrhage and oedema were again a characteristic finding in the acute phase of the disease. The germinal centres were oedematous and appeared indistinct. Both developing and mature cysts were

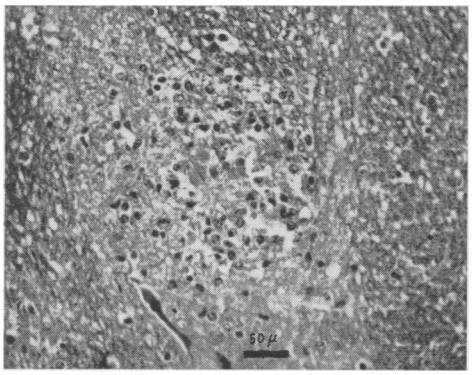


FIGURE 18. Glial reaction surrounding small necrotic focus within brain. H & E.

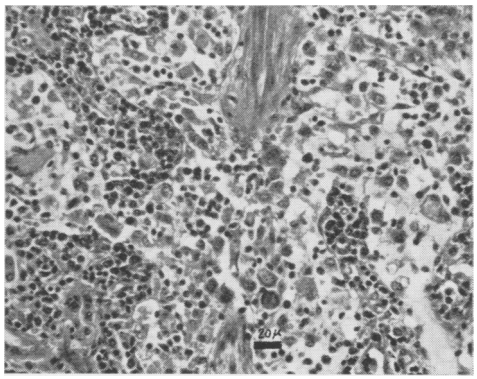


FIGURE 19. Paratrabecular sinus filled with pigment laden macrophages, mononuclear cells and oedema fluid. H & E.

numerous. The cortical and paratrabecular sinuses were filled with pigment-laden macrophages (Fig. 19). Mild erythrophagia was noted in some cases. As the disease progressed the germinal centres became extremely active with numerous mitotic figures being evident. In addition to the pigment-laden macrophages numerous plasma cells and lymphocytes, along with a few eosinophils, were present in the sinuses of the nodes. Neutrophils were seen in a few cases. Mitotic figures were present in the macrophages and the plasma cells. Some of the macrophages were multinucleated. The thymus gland as a rule exhibited changes similar to those found in the lymph nodes.

Focal haemorrhage and areas of interstitial mastitis characterized by the presence of lymphocytes, plasma cells and macrophages together with numerous mature and immature cysts were found in the mammary glands of the acute cases (Fig. 10). As the condition progressed the mammary gland became indurated and haemosiderin and a few eosinophils were found in the areas of interstitial reaction.

The uterine caruncles of aborting animals were haemorrhagic and exhibited areas of coagulation necrosis. Calcification of individual epithelial cells as well as

of the fibrous connective tissue stroma was evident. Numerous cysts were present (Fig. 8). Aborting animals killed late in the disease showed a chronic endometritis characterized by mononuclear cell infiltration. The last animal to be killed had an acute bacterial-type endometritis. Histological examination of foetal tissues, both aborted and those in utero, failed to reveal the presence of cysts or other significant pathological changes.

Both mature and immature cysts were found in the pancreas (Fig. 15), with an occasional focus of mononuclear cell infiltration being scattered throughout the parenchyma.

The reaction observed in the gastro-intestinal tract was uniform throughout. In acute cases, haemorrhage with formation of blood pigment and oedema, together with foci of mononuclear cells in the mucosa, were present. Oedema was particularly severe in the abomasal folds. A vasculitis was present where what were considered to be ruptured cysts occluded the lumen (Fig. 14). A few eosinophils formed part of the inflammatory reaction in the chronically affected animals. An ulcerative colitis, characterized by sharp punched-out ulcers which penetrated to various depths and surrounded by an acute inflammatory reaction was seen in a few of the animals. Cysts were found throughout the length of the intestinal tract. Ulceration involving the tongue, lips, gums, cheeks, hard and soft palate, oesophagus and abomasum became a striking feature of the chronic phase. These ulcers varied in depth and distribution and although no cysts were found in association with them, all were accompanied by an acute inflammatory reaction.

Sections of the eye and adrenal, pituitary and thyroid glands revealed no significant changes. Bone marrow was examined from one animal only and was found to be extremely hyperplastic.

## Other Laboratory Studies

### *Bacteriology*

Bacteriological examination of various tissues failed to result in the consistent isolation of any bacterial pathogens. *C. pyogenes*, *P. multocida* and *P. haemolytica* were present in some of the tissues examined but in all cases it appeared that these were secondary invaders.

### *Toxicology*

Because of the unusual character of this outbreak it was considered advisable to examine the water supply and various tissues from affected animals at the acute stage of the disease. These submitted specimens were analyzed (courtesy of Dr. W. Oliver, Ontario Veterinary College) for fluoride, heavy metals, alkaloids, nitrates and nitrites. All results were either negative or within the normal range.

### *Hematology*

During the course of this outbreak routine examinations were carried out on 70 samples from 26 animals. The differential counts, WBC and PCV estimations in five animals are summarized in Table I. This table reflects the general haematological picture seen in the herd. In all cases this was characterized by the development of varying degrees of leucopenia and anaemia, with a terminal leucocytosis in animals showing evidence of secondary bacterial infection (#3).

TABLE I

HAEMATOLOGICAL FINDINGS IN FIVE ANIMALS

| Animal (Age) | Date of Bleeding | P.C.V. % | Hb gms % | W.B.C. (per cu. mm.) | Differential Count | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | | | Immat. Neut. | Mat. Neut. | Lymph | Eus. | Mono |
| #1 | Dec 15 | 25.0 | 9.0 | 7,150 | 4 | 14 | 72 | 0 | 10 |
| (3 yrs.) | " 21 | 16.0 | 6.0 | 4,600 | 9 | 9 | 67 | 3 | 12 |
| | " 22 | 12.0 | 4.0 | 5,200 | 5 | 10 | 70 | 0 | 15 |
| | " 24* | 8.0 | 2.5 | 6,000 | 16 | 8 | 63 | 0 | 13 |
| #3 | Dec 15 | 35.0 | 12.0 | 6,700 | 0 | 22 | 71 | 7 | 0 |
| (3 yrs.) | " 21 | 34.0 | 11.5 | 6,100 | 4 | 22 | 70 | 4 | 0 |
| | " 22 | 32.0 | 11.5 | 5,800 | 8 | 20 | 60 | 3 | 9 |
| | " 27 | 32.0 | 10.5 | 5,300 | 16 | 2 | 78 | 1 | 3 |
| | Jan 17 | 31.0 | 10.5 | 11,000 | 5 | 28 | 63 | 0 | 4 |
| | " 19 | 33.0 | — | 27,450 | 2 | 68 | 27 | 0 | 3 |
| | " 22 | 36.0 | — | 29,950 | 5 | 75 | 17 | 0 | 3 |
| #4 | Dec 15 | 32.0 | 12.0 | 4,100 | — | — | — | — | — |
| (3 yrs.) | " 21 | 28.0 | 9.5 | 5,450 | 4 | 18 | 78 | 0 | 0 |
| | " 22 | 27.0 | 10.0 | 5,100 | 3 | 6 | 79 | 1 | 11 |
| | " 27 | 24.0 | 8.0 | 4,500 | 5 | 11 | 78 | 1 | 5 |
| | Jan 17 | 22.0 | 8.0 | 6,200 | 4 | 12 | 78 | 0 | 6 |
| | " 30* | 24.0 | — | 3,300 | 0 | 26 | 73 | 0 | 1 |
| #5 | Dec 15 | 32.0 | 11.5 | 5,000 | 0 | 10 | 87 | 3 | 0 |
| (3 yrs.) | " 21 | 32.0 | 11.0 | 4,350 | 8 | 8 | 84 | 0 | 0 |
| | " 22 | 27.5 | 10.0 | 4,650 | 18 | 13 | 59 | 1 | 9 |
| | " 27 | 25.0 | 8.0 | 3,800 | 27 | 13 | 50 | 0 | 10 |
| | Jan 5* | — | — | — | — | — | — | — | — |
| #7 | Dec 22 | 20.5 | 8.0 | 3,950 | 5 | 10 | 72 | 2 | 11 |
| (8 yrs.) | Jan 4 | 18.0 | — | 10,350 | 6 | 48 | 44 | 0 | 2 |
| | " 5 | 19.0 | — | 6,050 | — | — | — | — | — |
| | " 8 | 22.0 | — | 6,950 | — | — | — | — | — |

*Date of Euthanasia.

*Isolation and Transmission*

Efforts were made to isolate the causal agent and to transmit the disease in the following experiments:

1. A pregnant heifer and two pregnant goats were inoculated intraperitoneally and intramuscularly with a suspension of lymph node in pericardial fluid. In addition the heifer was given 200 cc. of citrated, febrile stage blood intraperitoneally.
2. Two pregnant heifers and two pregnant goats were inoculated intramuscularly and also drenched with a suspension of lymph node, brain and spleen.
3. The same suspension used in experiment 2 above was infused into the mammary gland of a lactating cow.
4. Another pregnant heifer was given a transfusion of 2,000 cc. of citrated blood collected from a clinically affected animal prior to euthanasia.
5. Five groups of three guinea pigs were inoculated intraperitoneally with various tissue suspensions.
6. Five groups of ten mice were inoculated intraperitoneally with tissue suspensions and a series of three blind passages at three–four day intervals were carried out.
7. Five groups of ten embryonating eggs were inoculated with tissue suspen-

sions into the yolk sac and two blind passages were made at eight-day intervals.

8. One group of ten mice was inoculated with aqueous and vitreous humor.
9. Three ferrets were fed affected lymph node for seven days.
10. Brain, salivary gland and foetal tissues were seeded onto cell cultures of bovine embryonic kidney.

All materials used in these trials were collected from animals in which large numbers of cysts were demonstrated. The tissue suspensions were prepared and inoculated immediately. All of the inoculated animals remained clinically normal and no cytopathic effects were observed in the tissue cultures. Histological examination of various tissues collected from the mice at the time of passage and from the ferrets when they were killed six months later failed to reveal the presence of the organism which had been observed in the naturally infected animals and no significant lesions were found.

*Serology*

A total of 69 serum samples were collected from the animals in this herd between December 18 and January 17 and the following serological tests were conducted:

1. Thirty-three samples were tested for brucellosis by plate and tube agglutination methods. All were negative.
2. Sixty-three samples were submitted to the complement fixation (CF) test using a psittacosis-lymphogranuloma group (EAE) antigen with negative results.
3. Ten samples tested for *Leptospira pomona* antibodies by the plate agglutination method were negative.
4. CF tests for anaplasmosis (10 samples), trypanosomiasis (5 samples) and Q fever (41 samples) were all negative.
5. Direct and indirect CF tests for toxoplasmosis yielded negative results on 41 sera. However 31 of these when tested by the modified direct CF test (7) revealed two positive and six questionable reactions. Most of the reactions were incomplete and many sera showed a low degree of non-specific reaction with the normal antigen control. There appeared to be no correlation between the observed clinical manifestations and the results of this test. For example, of the two positive animals one was a six-month-old calf which was never clinically affected. The other was only mildly affected and recovered.
6. Through the courtesy of Dr. N. A. Fish, Ontario Veterinary College, paired serum samples from six clinically affected animals were subjected to the Sabin-Feldman dye test with negative results. The first of these samples were collected ten days after the commencement of the outbreak and the second samples were taken two to four weeks later.
7. On January 8, 1963 intradermal tests using two *T. gondii* skin test antigens, one prepared at the National Veterinary Assay Laboratory, Tokyo (courtesy of Dr. K. Nobuto), and the other prepared at the Animal Diseases Research Institute, Hull, Quebec, was carried out on six animals. Five of these had recovered from the illness described and the other was a mature cow which had been purchased in May, 1962. No reactions were apparent at 24, 48, or 72 hours after inoculation.

## Discussion

Frenkel (3) states that "Morphologic identification of *Toxoplasma* is difficult due to its small size and simple organization. Biologic criteria, especially immunologic and serologic behaviour, host range and host response furnish more critical information than the morphologic data". Although the parasite observed in this outbreak resembles *T. gondii* morphologically it has not been possible to demonstrate an immunological relationship between the disease it apparently caused and toxoplasmosis.

Toxoplasmin skin tests in five recovered animals and Sabin-Feldman dye tests on the sera of six affected animals gave negative results. Direct and indirect CF tests were negative. Although some reactions were observed in the modified direct CF test it was not possible to correlate these with the clinical findings.

*T. gondii* is described as an obligate intracellular parasite capable of invading and multiplying in a wide variety of cell types. Cells parasitized include neurons, microglia, ependymal cells, mesothelial cells of the pericardium and serosa, endothelial and reticular cells, epithelium of the lung and glands, hepatic cells, Kupffer cells, leukocytes and cardiac, skeletal and smooth muscle cells (2, 4, 5, 6). The parasite involved here while widely distributed throughout the body was demonstrated only within the endothelial cells of blood vessels.

There is little information in the literature regarding bovine toxoplasmosis. Sanger *et al.* (8) reported toxoplasmosis in four widely separated herds in Ohio. The clinical symptoms described in adult animals by these authors bore little resemblance to the signs exhibited in this outbreak. In marked contrast to the present findings gross lesions were present in only one of the six animals in which Sanger *et al.* demonstrated Toxoplasma.

Despite the failure to transmit the condition described here it is felt that by causing widespread occlusion and damage to blood vessels, the parasite demonstrated was responsible for most if not all of the clinical and pathological findings in this herd. It is possible that some other etiological agent may have contributed to the production of this outbreak, however the negative results obtained in the isolation studies tend to lessen this possibility.

It is thought that the watering system on this premises may have influenced the epidemiology of the disease within the herd inasmuch as all fatalities occurred in animals watering from the communal troughs, while only a transient febrile reaction was observed in animals watered individually. The source of the infection remains unexplained. Since, in the authors' opinion, the clinical and pathological features of this outbreak do not appear to have been previously described, the name Dalmeny disease is proposed after the locality in which it occurred.

## Summary

This paper describes in detail the clinical and pathological aspects of a disease outbreak in a herd of Holstein cattle characterized by high morbidity and mortality in the adult animals. The striking feature was the presence in the vascular endothelium throughout the body of affected animals, of an unidentified, presumably protozoan parasite, morphologically similar to *Toxoplasma gondii*. These parasites were most numerous in animals dying at the acute stage of the

disease. A wide range of serological tests failed to reveal the cause of this condition and all transmission attempts failed. On the basis of the results of these studies the authors conclude that this is probably a hitherto undescribed condition and have named it Dalmeny disease.


## Résumé

Cet article décrit en détail l'aspect clinique et pathologique d'une épizootie caractérisée par un taux élevé de morbidité chez des bovins adultes dans un troupeau de Holstein.

L'affection était particulièrement caractérisée par la présence dans l'endothélium vasculaire d'un parasite non-identifié, vraisemblablement un protozoaire morphologiquement semblable à *Toxoplasma gondii*. Ces parasites furent trouvés en nombre beaucoup plus grand chez les animaux morts dans la phase aigüe de la maladie.

Dans le but de déterminer la cause, une grande variété d'épreuves sérologiques furent essayées sans succès. De même, tous les essais de transmission demeurèrent infructueux.

D'après les résultats obtenus dans l'étude de cette épizootie, les auteurs concluent être probablement en présence d'une affection non-décrite jusqu'à présent et l'ont nommée "Maladie de Dalmeny".



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