2.4.1.6 Giemsa Staining

Some sections suspected to carry coccidia were pressed between two clean slides. These slides were left to dry for 24 hours. The following day the air-dried slides were fixed in absolute alcohol for 30 minutes and stained for 30 minutes with a freshly prepared dilution of 0.5 ml of Giemsa solution in 10.0 ml of distilled water with phosphate buffer pH 6.8.

Giemsa Stain Preparation:

Azure II eosin 3.0 g

Azure II 0.8 g

Glycerol 250 ml

Pure methanol 250 ml

In this work, improved Giemsa R66 solution was used (GURR 1979). The slides were then rinsed carefully with distilled water which was slightly acidified with 1% acetic acid. The slides were then air-dried vertically and observed under a light microscope.

2.4.1.7 Mounting

Specimens for microscopic examination were placed on a microscope slide, surrounded by a suitable mounting medium DPX and covered by a cover slip. This excluded air and dust, provided the appropriate optical conditions for examinations and protected the objective lens. A drop of DPX mountant was placed on the slide in the centre. The drop was of such a size as to fill the space between the cover slip. The edge of the cover slip was placed on the side of the mounting medium using a mounting needle or forceps. The cover-slip was left to settle.

The required objective lens was brought into the line of focus and the iris diaphragm was adjusted accordingly.

Objective	Numerical Aperture	x % of Light
x 10	0.30 x 70%	= 0.21
x 20	0.46 x 70%	= 0.32
x 40	0.70 x 70%	= 0.49
x 100	1.25 x 70%	= 0.87

The figures on the right helped in setting the Aperture iris diaphragm ring in position to control the amount of light reaching the camera. The shutter was released by pressing the exposure button on the control box and the film automatically advanced to the next frame. The film end light showed when the film was finished.

2.5 HAEMATOLOGY

Vacutainer tubes containing EDTA as an anticoagulant were used to collect 5.0 ml of blood from the goats. The blood was used to analyse the Packed Cell Volume (PCV), haemoglobin concentration (Hb), numbers of White Blood Cells (WBC) and Red Blood Cells (RBC). The analysis could only be done accurately on fresh blood. On returning from the field, the blood was put on a Denley Spiromix (Cat No. A253) to mix the blood thoroughly before measurement of the above parameters.

2.5.1 The Haemocytometer

This is a thick rectangular glass slide with two raised cross bars on which the coverslips rests. In the central area between the crossbars are two platforms, each of which is completely surrounded by a moat. Each platform is ruled into primary squares of 1 mm^2 each. Each of the four corner primary squares is subdivided into 16 secondary squares used for WBC. The centre primary square is divided into 25 secondary squares and each secondary square is divided into 16 tertiary squares (i.e. 400 tertiary squares) to be used for the RBC count.

A clean, dry slide and coverslip were prepared. The cover slip was carefully placed on the slide making sure that it adhered well showing Newtons rings. (This was done by pressing it along the crossbars to its position). Using an RBC diluting pipette and fresh anticoagulated blood, blood was drawn by gentle sucking to the 0.5 mark. Care was taken to ensure that no air bubbles were trapped in the column of blood.

Using the mouth, the red cell diluting fluid (Formal Citrate) was drawn into the pipette to the 101 mark. Both ends of the pipette were closed by the help of the plastic tube and fingers. The blood and diluting fluid were mixed by moving the pipette back and forth at right angles to its long axis for 2 minutes. Half the contents of the pipette were expelled to remove the cell free fluid from the stem. By touching the tip of the pipette to the edge of the platform of the counting chamber, the fluid was allowed to flow under the coverslips by capillary

action. Sometimes refilling the slide was necessary if air bubbles were trapped under the coverslip or if there was spillage. The pipette was cleaned immediately with distilled water for further use.

The haemocytometer was placed on the microscope and was left for 2-3 minutes to allow the cells to settle on the surface of the platform. The microscope was adjusted to focus on the squares on the platform. Cells were counted in five of the secondary squares using the four corner and centre primary square. The cells lying in the square or cutting the upper and left lines of the squares were counted. Those lying on the lower and right lines were not counted (Archenhold et al 1978).

2.5.2 The Electronic Cell Counter

The Baker Instruments Haemotology series 130 (Cat No. 500 0004 3) was used to count the RBC, WBC and Hb. This was the procedure:

2.5.2.1 Preparation of the Instrument

The waste container had to be properly stoppered before turning the unit on. The power was switched on and the unit left to warm for 5 minutes. The STBY/Flush button was pushed twice to clean the system with Haem-clean 100. The machine was flushed again twice with Haem-line 2, the diluent and Haem-Lyse 100 (3 drops/10.0 ml). The self test was run by using 10.0 ml of

Haema-line 2 and 3 drops of Haema-lyse 100. After switching on the self test button the RBC background counts were then displayed.

2.5.2.2 Running the Samples

A vacutainer taken from the spiral mixer containing blood and EDTA was opened. The tip of the dilutor dispenser was inserted into the blood specimen and the red button was pressed noting that the tube was not removed until the delivery light came on. The amount of blood collected was 3.0 ml. The tube was removed and dilutor tip was carefully wiped in a downward motion using the lint free tissues (Kimwipes). On an angle the Haema-vial was held under the dilutor tip and the red button on the dispenser was pressed. 10.0 ml of the fluid was collected. The tip of the dilutor remained above the fluid level as the sample was being dispensed down the side of the Haema-vial. The last drop from dilutor tip was obtained by touching the tip of the dilutor to the side of the Haema-vial, the Haema-vial was capped and mixed by inversion two times. This was WBC (1:250) dilution. For the RBC cell count a further dilution from the WBC sample was made using the dilutor dispenser. The mixture was collected 40ml and 10.0 ml of diluent, mixed by inversion two times giving the RBC dilution (1:62,500).

To the WBC dilution, 3 drops of Haema-lyse were added and left to stand for 2 minutes before analysing. All the Haema-vials were clearly labelled according to the goat number and type of

sample. The RBC dilution was put under the snorkel and the RBC button switched on and the results were printed on the assay sheet. Then the WBC/Hb dilution of the same goat was also put under the snorkel and the WBC/Hb button switched on. The results were printed on the assay sheet.

After taking all the readings the Haema-clean 100 was put under the snorkel and the STBY/Flush button pushed two times. The vial of Haema-standby was placed under the snorkel and the system was flushed two times. SHND/CLEAN button was pushed and the display read OP2 after shutdown was complete. The waste container was emptied and the power was switched off. The vial full of Haema-standby was left under the snorkel and the stopper on the waste container was reset.

2.5.3 The Blood Cell Count

2.5.3.1 Red Blood Cell Count

To count the cells haemocytometer was used in earlier work, and later the electronic cell counter.

2.5.3.2 White Blood Cell Count

The procedure was the same as that for RBC when using haemocytometer except that this time a white diluting pipette and the WBC diluting fluid (1% acetic acid in Turk's solution) was used to lyse the RBC.

The pipette was filled with blood to the 0.5 mark and with diluting fluid to the 11 mark. The cells were counted in the four corner primary squares. In later samples the electronic counter was used.

2.5.4 Haemoglobin Determination

The amount of haemoglobin in the blood was measured by using the Erma Optical Haemoglobinometer (Model No. 303-n.) This measures the intensity of the colour of the fluid by a colorimetric method.

The haemoglobinometer was switched on and left for 5 minutes to warm, 0.5 ml of Haemoglobin reagent was placed in a test tube used as a blank. It was placed into the sample holder of the machine and the meter was adjusted to zero with the zero adjustment knob. By pressing the "check" knob the reading showed a known standard value. Acuglobin was used as a haemoglobin standard and its value was 14.2 g/dl. If the value given was not the same as the known value, the meter was adjusted using the scale adjustment knob. The test sample was placed in the sample holder and the Hb value was read in g/dl and the percentage noted.

2.5.5 Packed Cell Volume Haematocrit

Two capillary tubes 75 mm x 1 mm were filled with blood to 2/3 by capillary action. Using a tissue paper the capillary tube was wiped off and sealed with paraplast. The tubes were placed in a slot of Kubota Microhaematocrit centrifuge (Model No. KH 12005). It was fitted with a head for carrying up to 24 capillary tubes and was automatically set to run at 10 000 rpm for 5 minutes. The sealed end of the capillary tube pointed outwards.

Two capillary tubes were filled from one sample to improve accuracy. The PCV was determined using a Microhaematocrit Reader.

2.6 BIOCHEMICAL PARAMETERS

2.6.1 Blood Sampling

Blood samples were collected weekly from the confined goat-kids. Vacutainer needles size 21G x one and half (0.80 x 38 mm) were used with 15 ml plain vacutainer tubes. The vacutainer tubes were used to collect 10.0 ml of blood for serum, total protein and albumin measurement.

The blood was collected from the jugular vein in the neck of the animal. The neck was slightly twisted to create pressure on the jugular vein for it to be superficial. Seventy per cent alcohol was rubbed onto the skin and the vacutainer needle was slowly pushed into the vein at an angle of 30%. Sufficient pressure below the needle was applied to ensure easy flow of the blood. The vacutainer needle was withheld a short distance from the tube until blood appeared at the nozzle of the needle. At this point a vacutainer was inserted until the required volume was collected. The needle was removed by releasing the pressure on it and pressing on the skin at the point of insertion (Kelly 1984). Adequate mixing was ensured by inverting the tube several times. The used needles were incinerated.

2.6.2 Treatment of Blood Samples

For serum, 10 ml of blood was collected in plain vacutainer tubes. This was left to stand at room temperature for two and half hours until the blood had clotted. The tubes were then centrifuged on a bench Hitachi Centrifuge, (Model number 05p-21) for five minutes at 2500 rpm. The serum was then separated from the clot and put into labelled test tubes and kept at -20 ^{o}C for storage.

2.6.3 Estimation of Serum Total Proteins

The frozen serum was left to thaw at room temperature and the following reagents were prepared:

2.6.3.1 Stock Biuret Solution

0.20 M of sodium hydroxide was prepared by weighing 8 g of pellets and made up to a litre with distilled water. Sodium potassium tartrate (45.0g) was dissolved in 400.0ml of 0.20M sodium hydroxide solution, 15.0 g of copper sulphate was added to the mixture and the whole solution made up to a litre by adding 0.20M sodium hydroxide (Plummer, 1978).

2.6.3.2 Biuret Test Reagent

10.0 ml Stock Biuret reagent was diluted to 50.0 ml with 0.20 M sodium hydroxide containing 0.5% (W/V) potassium iodine (Plummer, 1978).

2.6.3.3 Standard Protein Solution

0.1 g Bovine serum albumin (0.100g) kept at 4 ^{o}C (Sigma No. A6793) was dissolved in 100.0 ml of isotonic sodium chloride (1.8 g sodium chloride in 200.0ml of distilled water).

2.6.3.4 METHOD

To 3 test tubes the following reagents were added:

SOLUTION	TEST (ml)	BLANK (ml)	STANDARD (ml)
Saline	2.95	3.00	-
Serum	0.05	-	-
Albumin	-	-	3.00
Biuret Sol.	3.00	3.00	3.00

All the above tubes were placed in a water bath at 37 °C for 10 minutes. They were later removed and allowed to cool. The values were read on a Hitachi Spectophotometer (Model No. 100-20) at wavelength 550 nm.

2.6.4 ALBUMIN DETERMINATION AND PREPARATION OF REAGENTS

1.0 M Sodium citrate ($Na_3C_6H_5O_7 \cdot 2H_2O$) 29.4% (w/v) was prepared by dissolving 29.4 g of sodium citrate in 100.0 ml of distilled water. 1.0 M citric acid ($C_6H_8O_7 \cdot H_2O$ 21% w/v) was also prepared by dissolving 21.0 g in 100.0 ml distilled water. Bromocresol green was buffered by adding 17.3 ml of 1.0 M sodium citrate, 32.7 ml of 1.0 M citric acid and 6.0 ml of 0.01 M bromocresol green to 900.0 ml of distilled water. The pH of solution was adjusted to 3.8 by adding 1.0 M citric acid and the volume made up to a litre.

2.6.4.1 Standard Albumin Solution

Standard albumin solution was prepared by dissolving 0.500g of bovine serum albumin in isotonic saline which was made up to 10.0 ml. Three test tubes were set up as follows:

REAGENT	TEST (ml)	BLANK (ml)	STANDARD (ml)
Bromocresol Green	4.00	4.00	4.00
Serum	2.00	–	–
Distilled Water	–	0.20	–
Standard	–	–	0.20

The contents in each test tube were mixed and left to stand at room temperature for 5 minutes. The absorbance was read on the Hitachi Spectrophotometer (Model No. 100-20) at 640 nm.

2.6.5 Determination of Globulin Concentration

The globulin concentration was obtained by subtracting the albumin from the total protein concentration.

2.6.6 A:G RATIO

It is the ratio of the albumin over globulin concentrations. This gives an overall evaluation of protein status and a basis for some tentative and additional studies of the animals.

2.7 Statistical Analysis

The means and standard error of the mean (SEM) of values obtained for each week for the first five goat kids were calculated. Data were analysed on a Hewlett Packard Computer (Model No. 9816) and Basic Statistics 3.0 software at $P<0.05$ using Duncan's multiple range test for multiple comparison between the means and Bartlett's test of homogeneity of variance. Points that have a similar letter are not significantly different.

CHAPTER THREE

RESULTS

CHAPTER III

RESULTS

3.1 Clinical Progress of the Disease - First Set of Kids

The experiment was carried out in two groups. The first group of five kids preinfected from their mothers appeared clinically normal up to 6-8 weeks after isolation, except for goat 1 where symptoms appeared only after 14 weeks.

The following results show the progress of the disease (coccidiosis) and measurement of various parameters. The graphs show the last 8 weeks of the experiment for goats 1-5, and all the weeks for goats 6-10.

3.1.1 Temperature

The general body temperature of goats varies from 38.6 to 40.2 ^{o}C (Kelly, 1984). The average temperature of the first group was 39.4 ^{o}C though as the disease progressed it went up to 40.0 ^{o}C-43.8 ^{o}C and suddenly dropped when disease symptoms were severe and before the animal was sacrificed. This increase could be noted during the last 1-2 weeks, when the animals were developing a high fever and were lethargic. In goat 4 the highest temperature recorded was during the 6th and 7th weeks of the experiment and it dropped in the 8th week.

3.1.2 Weight

The body weight decreased in the last 2 weeks; though in most animals weight was constant for the previous 3 weeks. This lack of weight increase may have been due to loss of apetite. As can

be seen in Figs 3.5; 3.15; 3.25; 3.35; 3.45, all the animals had a noticeable decrease in weight during the last 1-2 weeks before the animals were sacrificed. This could have been due also to the loss of water when the animals had diarrhoea.

3.1.3 Other Clinical Symptoms

Between the 5th-7th week clinical symptoms began to show in all five goats 1 and 5 after 7 weeks; goats 2 and 3 after 5 weeks; goat 4 after 6 weeks. The first symptoms noticed were nasal discharge, lethargy and inappetance.

In goats 2, 3, 4 and 5 diarrhoea started between the 5th and 7th week, when other clinical symptoms began (see Tables 3.3-3.13). Diarrhoea lasted for three weeks in goat 3, for a week in goats 2, 4 and 5 and only two days in goat 1 before the animals were sacrificed. Generally there were signs of diarrhoea on the tail and around the anus (see Fig 3.1.3.1). For the first few days the appearance of the faeces were semi-solid and greyish in colour. As the disease progressed they became watery with or without blood and the colour would change from grey to green and finally to yellow. At this time the animal would be emaciated, anorexic and very weak. Goat 3 during the last stages of the disease had a swollen face and erected hair on the skin.

3.1.4 Oocysts Count

3.1.4.a Three Kids with their Mothers Before Isolation

This part of the experiment was carried out to determine whether mothers were infected and to compare the number of oocysts of kids to that of their mothers. These three mothers all had diarrhoea

Fig 3.1.3.1 Signs of diarrhoea on the tail and around the anus of a goat kid.

after the kids were weaned on 25th October, 1987 but they were treated with Dimetol 50 ml/kg weight.

Table 3.1

Date of Faecal Sampling	Mother	No of Oocysts/g	Kid	Approximate Age in Weeks	No. of Oocysts/g
					6
9/10/87	63	1775	1	13	>10
	51	1929	2	14	20846
	58	552	3	13	>10
16/10/87	63	4102	1	10	188 604
	51	1236	2	10	70 000
	58	67	3	10	16 010
25/10/87	63	1648	1	11	9 958
	51	1977	2	11	9 069
	58	298	3	11	15 384

3.1.4.b Experiment

All the five kids used for the experiment were already infected with coccidia, and goat 1 and 3 had a very high level at the beginning of the experiment. (see Table 3.3; and 3.5). The number of oocysts per g faeces varied during the course of the experiment (see Fig 3.6; 3.16; 3.26; 3.36; and 3.46). In goats 1, 2, 3 and 4 had one peak during the 3rd week.

In goats 3 and 4, the peak was not pronounced, and the level increased just before death when clinical symptoms worsened. A slight increase was observed in goats 1 and 5 also before death.

The main species of coccidia identified during the oocyst counts were E. arloingi, see Fig 3.1.4.a which was about 60%, and E. ninakohylakimovae, which was 20%. The other species could not be clearly identified from those described by Norton (1986), see Fig 3.1.4.b. It was during the decline of oocyst number that clinical symptoms of coccidiosis worsened.

3.2 Clinical Progress of the Diseases, Second Set of Kids

The second set of kids were isolated a week after the 4th animal had been sacrificed. Goat 1 was isolated together with the second set of kids, since it still looked healthy and active. It was a carrier at this stage and served as a source of coccidial oocysts. Otherwise it had no appearance of disease. These animals showed signs of clinical symptoms within 1-2 weeks after isolation.

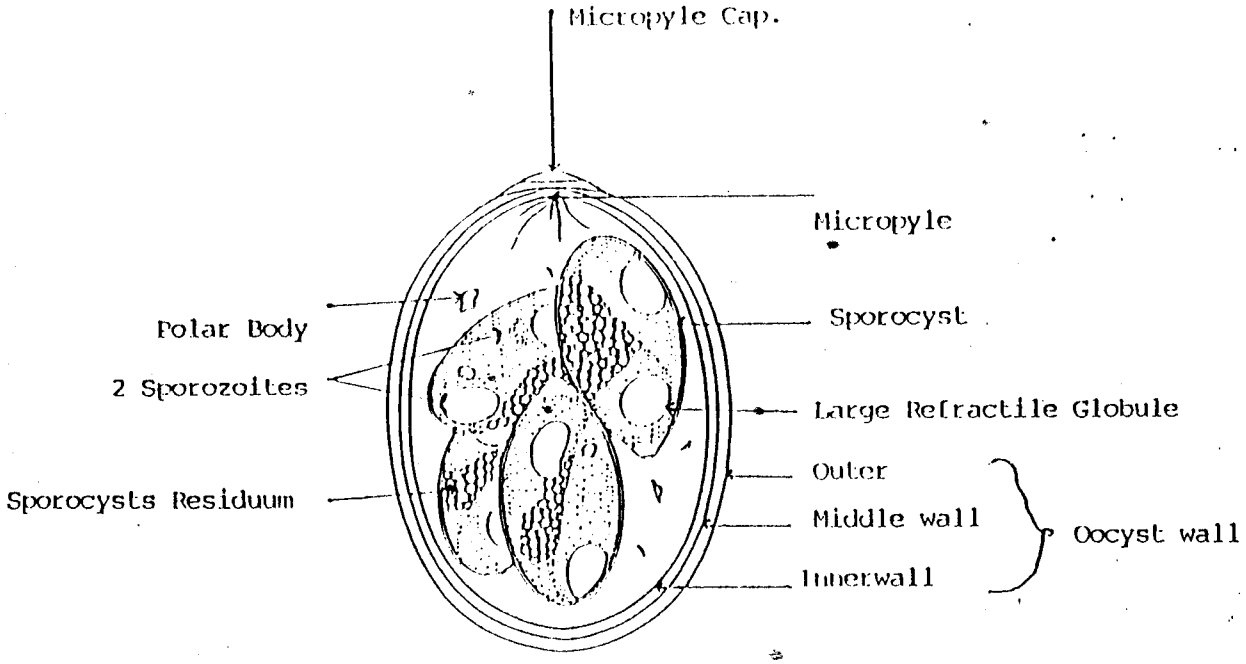
3.2.1 Temperature

The average temperature was 39.3 °C but there was a drop a day before each animal was sacrificed. (see Fig 3.54; 3.64; 3.74; 3.84; and 3.94). During the first week of the experiment they all had high temperatures and they were all treated with Alamycin (Oxytetracycline) 50 mg/ml 10 kg body weight to remove any extraneous infection. Diarrhoea stopped after two days of treatment.

FIG. 3.1.4a

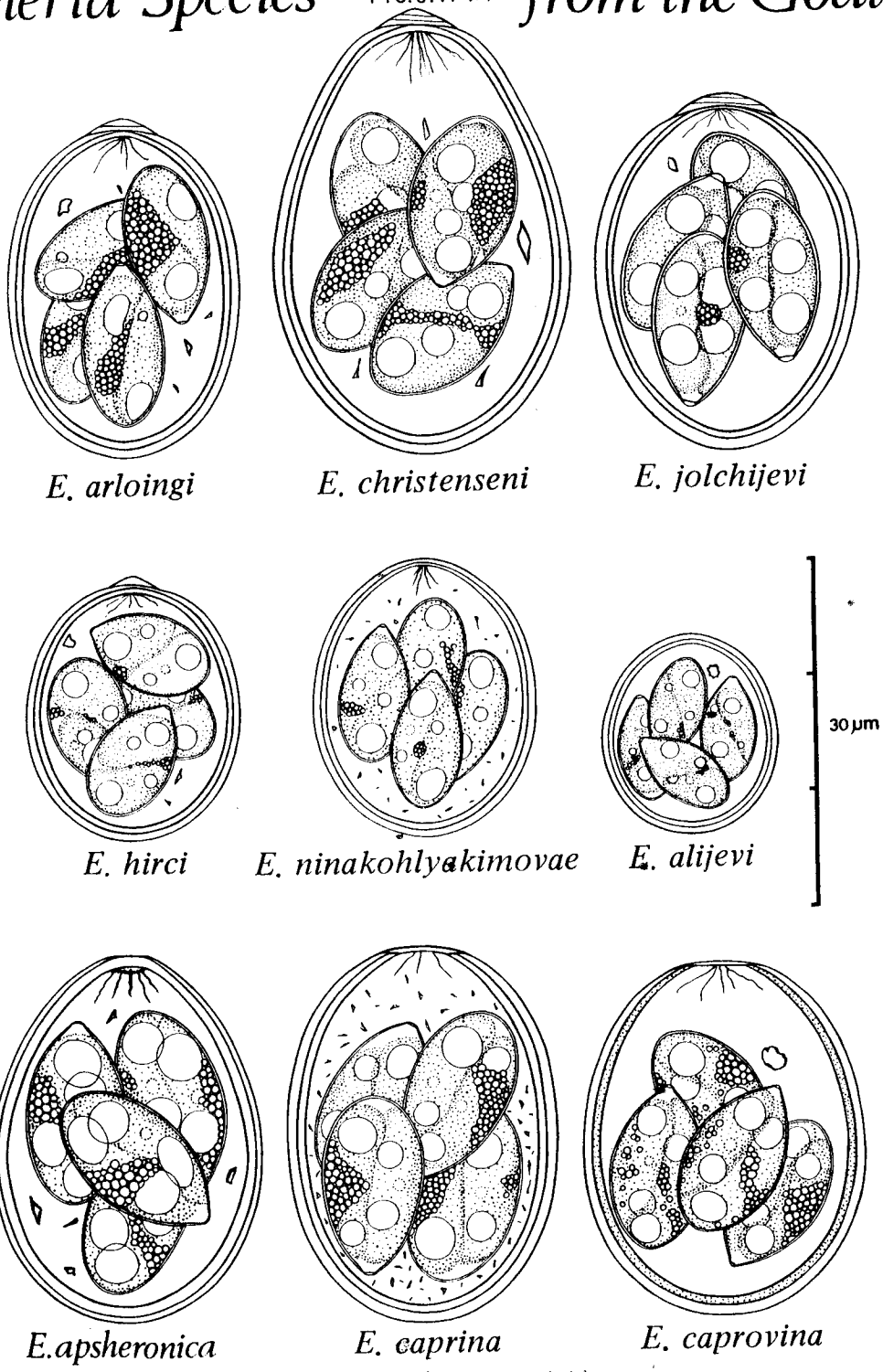
EIMERIA SPECIES FROM THE GOAT

OOCYST



E. arloingi

Eimeria Species FIG. 3.1. 4b. *from the Goat*



With courtesy of Norton (1986)

3.2.2 Weight

Their weight was increasing gradually before the animals were isolated for the experiment. From the time they were isolated there was no increase in weight, (see Figs 3.55; 3.75; 3.85; 3.95;) except for goats 6 and 7 where there was a slight increase during the second week, (see Fig 3.65). Some of the goats, particularly 9 and 10 were weaned early, and this would have put nutritional stress on the animals.

3.2.3 Other Clinical Symptoms

During the first and second weeks of the experiment, goats 6 and 7 had nasal discharge. Goat 6 had diarrhoea before it was weaned but after 3 days of treatment with alamycin it became well. Whether the diarrhoea was due to coccidia or other infection was not determined.

In all the goats, diarrhoea was noted 4-14 days after the experiment had started. Just as in the first set, the stools were at first grey and semi-solid and as the disease progressed became watery with or without blood. The colour also changed from grey to green then to yellow, sometimes with mucus. During the last two days the animals were emaciated, very weak and anorexic. The animals succumbed rapidly, this may partly be due to early weaning.

3.2.4 Oocyst Count

The species of Eimeria identified from the oocysts in this set of kids were the same as those in the first set. Goats 6 and 9 had a gradual increase, then a peak and a decline in the number of oocysts, (see Figs 3.56; and 3.86). For example, in goat 9 the number of oocysts increased from 77 000 to 400,000 [illegible]

gram faecal sample in the first week. During the second week on the day the animal died the number decreased to 140 400 oocysts per gram, (see Fig 3.86). In goat 8 the number decreased from 202 000 to 88 000 in the first week and later increased in the second week to 146 000, (see Fig 3.76).

3.2.5 Amprolium Treatment

Goats 3, 5, 6, 7 and 10 were all treated with amprolium (55 mg/kg) just a day before the animals were sacrificed.

3.3 Haematology

3.3.1 Red Blood Cell Count - (Figs 3.7; 3.17, 3.27; 3.37; 3.47)

The normal value of numbers of erythrocytes of a healthy goat is reported as being between $9-19 \times 10^6$ /µl with the average being 13.7×10^6 /µl (Kelly, 1984). The values of the first set of goats were considerably higher than the average normal value quoted for European goats (see Table 3.2). Before the goats got clinically sick, values up to 38.2×10^6 /µl were observed (see Fig 3.7) though this decreased as the disease progressed.

With the second set of goats it was difficult to give a critical account since only two or three readings were obtained before the animals were sacrificed. The average RBC count on five goats 6; 7; 8; 9; 10; is given on table 3.2. The last two values 13.48×10^6 /µl and 13.09×10^6 /µl in goats 9 and 10 respectively were similar to those given by Kelly (1984) and Lovelace et al (1988). In general the RBC values on all the goats were quite variable from week to week.

3.3.2 White Blood Cell Count - (Figs 3.8; 3.18; 3.28; 3.38; 3.48; 3.58; 3.68; 3.78; 3.88; 3.98)

The mean WBC values obtained during the period of the study ranged from 8.47-20.34 x 10^3 /µl for the first set of goats (1, 2, 3, 4 and 5). The average values for the second set ranged from 8.90-16.13 x 10^3 /µl. These results obtained from the experimental goat kids were slightly higher than reported figures of 9.00 x 10^3 /µl quoted by Kelly (1984). Castro et al (1977) gave a mean value of kids of less than 1 year as 11.8 x 10^3 /µl for the Pygmy Goats studied at Oregon (USA). Some goats showed an increase of WBC and this may have been due to the body reaction against coccidia. The histopathological studies showed many WBC around giant schizonts, (Fig 3.8.1) or even occupying the whole infected villus, (Fig 3.3.1.1). Goat 3 which was about 5 months old had the highest mean value of 20.34 x 10^3 /µl and goat 5 had the lowest mean value of 8.47 x 10^3 /µl. Vaidya et al (1970) in a study on Indian goats of the mixed Gujarat breed reflected mean values as 14.80 ± 3.60 x 10^3 /µl. The Pygmy Goats studied by Castro et al (1977) showed that older goats had a higher average value of WBC (16.7 x 10^3 /µl) than kids of 1-2 years old.

3.3.3 Haemoglobin Concentration - (Figs 3.10; 3.20; 3.30; 3.40; 3.50; 3.60; 3.70; 3.80; 3.90; 4.00)

The average concentration of haemoglobin of the ten goat kids studied was 12.02 g/dl, the highest figure recorded was 14.30 g/dl of goat 1 which was the oldest of all the goats. The lowest figure of goat 9 had a concentration of 8.50 g/dl.

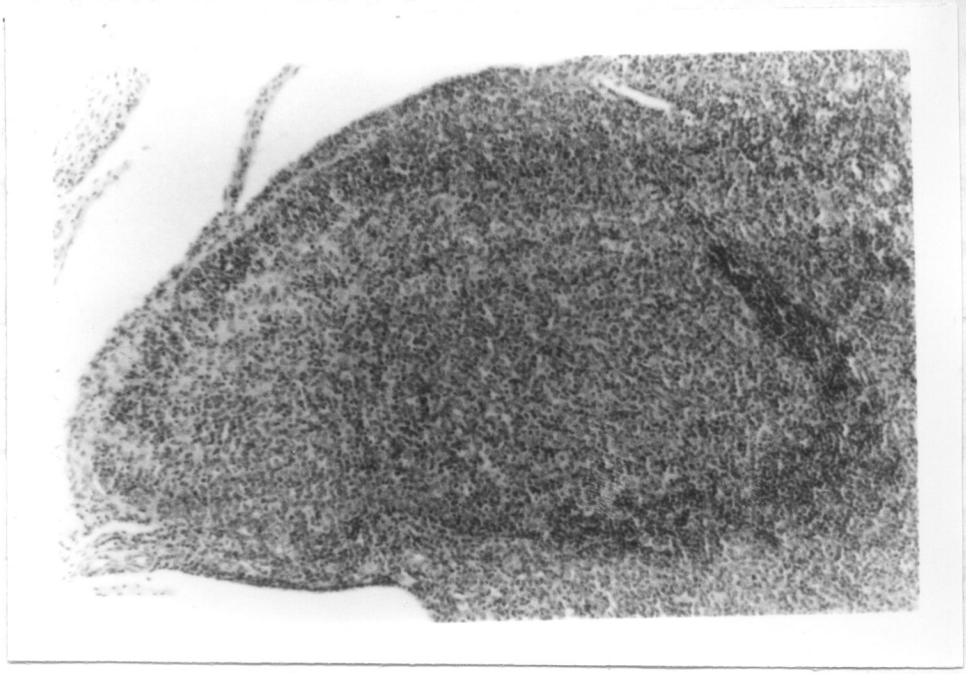


Fig 3.3.1.1 WBC occupying the infected villus. (x 100 mg)

These values fall within the range given by Kelly (1984), the normal values being 8.00-15.50 g/dl and the mean value being 11.00 g/dl. The goats from Luangwa District had a lower value 11.20 g/dl, (Lovelace et al 1988). Young kids normally have lower values compared to the adults. This was indicated by Castro et al (1977) during their study on Pygmy goats in Oregon and Lovelace et al (1988) on Luangwa goats. The mean value of goats less than a year was given as 10.90 g/dl and that of an adult goat as 11.30 g/dl. Haemoglobin levels stayed around the same levels in most goats, only goat 1, which was older, showing a marked drop, and goats 4 and 5 showing a slight decline (see Figs 3.10; 3.40; and 3.50). This could be due to iron deficiency and loss of RBC due to diarrhoea.

3.3.4 Packed Cell Volume (PCV)% - (Figs 3.9; 3.19; 3.29; 3.39; 3.49; 3.59; 3.69; 3.79; 3.89; 3.99)

It was reported by Schalm et al (1975) that the goat has the lowest PCV of common domestic animals. These goats studied showed PCV range of between 22.00-38.00%. Mukherjee and Bhattocharya (1952) of India followed eight male goats each month for a year and the PCV varied from 27.71-37.74%. The European goats values given by Kelly (1984) were from 21.00-39.00% with 30.00% as the mean value. The ten goat kids studied here showed a range of between 22.25-35.03%. This compares with the results given by Lovelace et al (1988) of a mean of 32%.

Goats 1 and 2 of the first set showed an increase in PCV during the 1st week but then, all showed a decrease. Goat 1 had an increase just before death. In the second set of goats, in goat 6 there was a gradual increase during the 1st week then a drop in the 2nd week while in goats 8 and 9 PCV continued to rise until the animal was sacrificed; goats 7 and 10 had a continuous decrease of PCV.

The lowest reading of 14% was recorded in goat 4 during the last week and half before sacrifice, (see Fig 3.19).

3.4 Serum Biochemical Parameters

Blood serum samples of the ten goat kids were analysed to establish how some biochemical reference values were influenced by coccidia as the disease progressed.

3.4.1 Total Serum Protein - (Figs 3.11; 3.21; 3.31; 3.41; 3.51; 3.61; 3.71; 3.81; 3.91;

The average values for total serum protein varied between 5.54-7.18 g/100 ml for the ten kids. Comparing these results with Pygmy goats studied by Castro (1977), these gave a mean value for 1 year old goats as 6.70 g/100 ml and older goats as 7.30 g/100 ml. The range varied from 5.58-8.64 g/100 ml. Davies and Sims (1985) carried out a study in Wales on blood biochemistry and haematalogy in domestic goats and gave mean values of serum total protein as 7.39 g/100 ml and that of a young goat as 6.90 g/100 ml. Kelly (1984) gave 6.60-7.50 g/100 ml as the normal values

with a mean value of 7.10 g/100 ml for European goats. These values correlate well with the results of the first five goats obtained from our experiment. Goats 2, 3 and 5 had peaks during the 4th, 3rd and 1st weeks respectively (see Figs 3.21, 3.31 and 3.51). During the 6th week there was a drop in goats 2, 3, 4 and 5 when the symptoms of the disease were diagnosed, and the level rose again slightly a week before the animals were sacrificed. For goat 1 there was increase in the level of total protein from the 2nd-4th weeks and it then levelled off, until the animal was sacrificed, this pattern could have been due to its age since it was the oldest goat of the ten.

With the second group there was a decrease of total protein in goats 7, 8 and 9 over the two weeks but goat 6 had a peak during the 1st week of the experiment and then a decline during the last week. In goat 10 there was an increase of the protein during the last week before the animal was sacrificed.

3.4.2 Albumin - (Figs 3.12; 3.22; 3.32; 3.42; 3.52; 3.62; 3.72; 3.82; 3.92; 3.102)

From the results the albumin readings varied from one animal to another. The mean values of the ten goat kids ranged from 3.21-5.00 g/100 ml. The first group had mean values ranging from 4.04-4.90 g/100 ml. The second group ranged from 3.21-5.00 g/100 ml. The values between the 1st group and the 2nd group were not very different. The results are high, when compared with results given by Kelly (1984) where albumin concentration was between 2.60-3.90 g/100 ml, and those given by Davies and Sims (1985)

3.19-4.93 g/100 ml and Castro et al (1977) 3.60 g/100 ml for goats which were one year old and 2.90 g/100 ml for goats 4-6 years old. However, other studies on Zambian goats (Lovelace et al 1988) give a mean ranging from 4.6 - 5.2 g/100 ml for a year long study, which is similar to ours. The graphs show an increase of albumin especially during the last week in goats 1, 3, 4 and 5 with goat 2 staying constant. In the second group the decrease started during the second week of the experiment in goats 6, 7 and 8 until the animals were sacrificed. In goats 9 and 10 there was a continuous decrease until the animals were sacrificed. The general trend between the graphs of serum total protein and serum albumin were similar with several goats showing a general decrease as the disease progressed.

3.4.3 Serum Globulins - (Figs 3.13; 3.23; 3.33; 3.43; 3.53; 3.63; 3.73; 3.83; 3.93; 3.103

The mean values of serum globulins of the 1st group of goat kids ranged from 1.51-2.46 g/100 ml. These results are lower compared to the values of the second group which ranged from 1.49-3.26 g/100 ml. Both sets of values are lower than those obtained from Pygmy goats studied by Castro et al (1977) with a mean of 4.50 g/100 ml. Davies and Sims (1985) results reflected an average range from Welsh goats as 4.67 g/100ml, a young goat having a value of 3.20 g/100ml and adult goat 3.40 g/100ml while Kelly (1984) gives the values ranging from 2.70-4.10 g/100ml

and in Lovelace et al (1988) the values of 2.3-3.6 g/100 ml were more similar to the kids on experiment.

The graphs on fig 3.23; 3.43; and 3.53 of the 1st group of goats indicated a higher concentration of serum globulin at the beginning of the experiment for goats 2, 3, 4 and 5, while goat 1 started with a low level of serum globulins but later increased gradually during the experiment. During the 6th week of the experiment the level of serum globulins began to rise in all goats. This is the period when the early signs of coccidiosis began to show. It can be seen that the values of serum albumin declined in goat 1, 3, and 4 during the 6th week whereas the values of serum globulins rise during the same period.

In the second group of goat kids, in two goats (Fig 3.63 and 3.103) there was a gradual increase in the concentration of serum globulins. Goats 7 and 8 had a drop in the 1st week but later the concentration was on an increase until the animal was sacrificed. In goats 6, 7, 8 and 10, as the globulin values increased, the albumin values decreased after the 1st week of the experiment. With goat 9 there was a decline in both albumin and globulin.

FIG. 3.4

GOAT 1

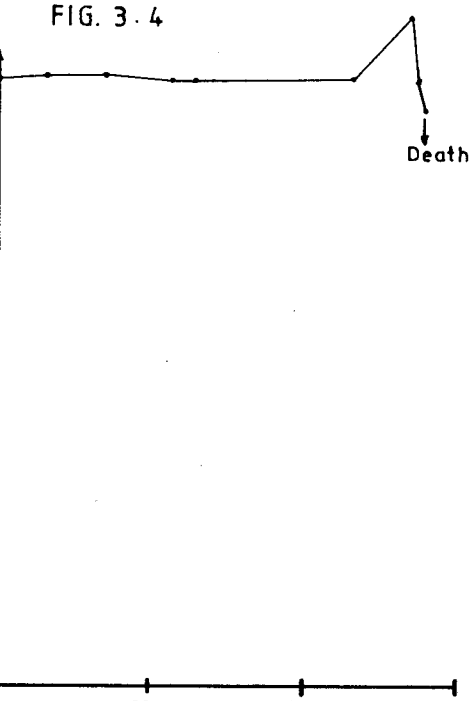


FIG. 3.5

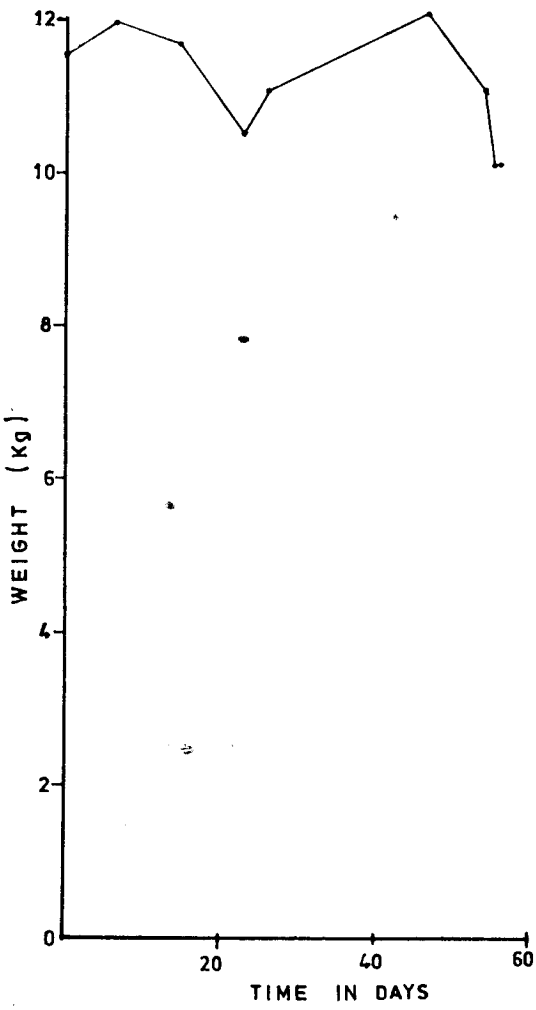
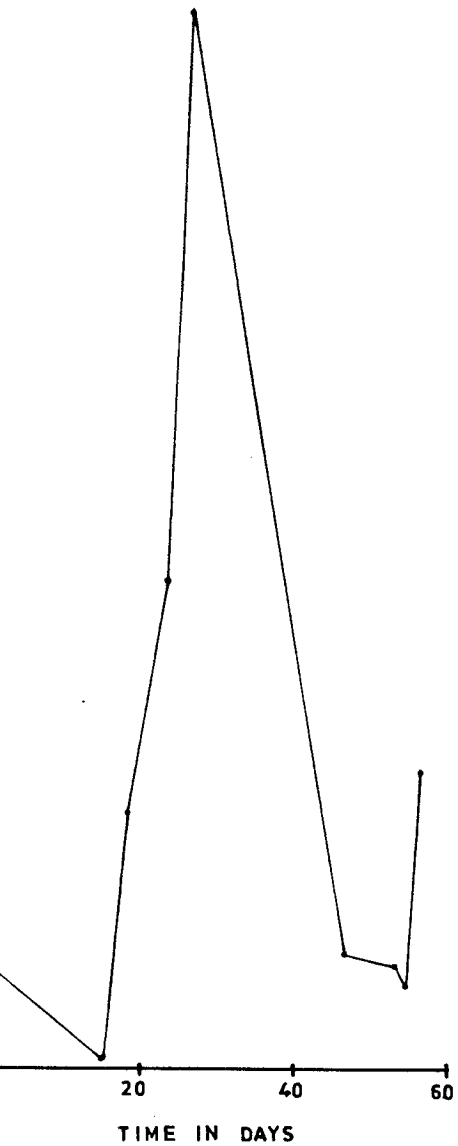


FIG. 3.6



GOAT 2

FIG. 3.14

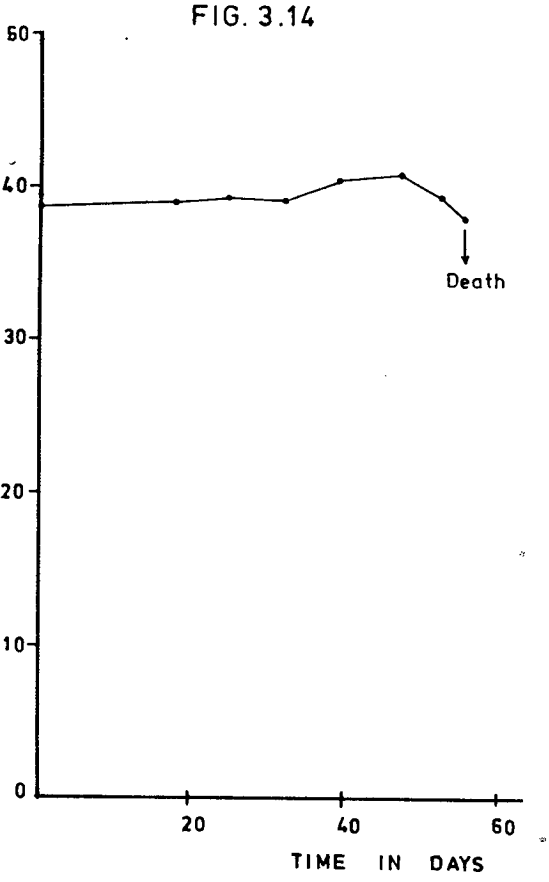


FIG. 3.15

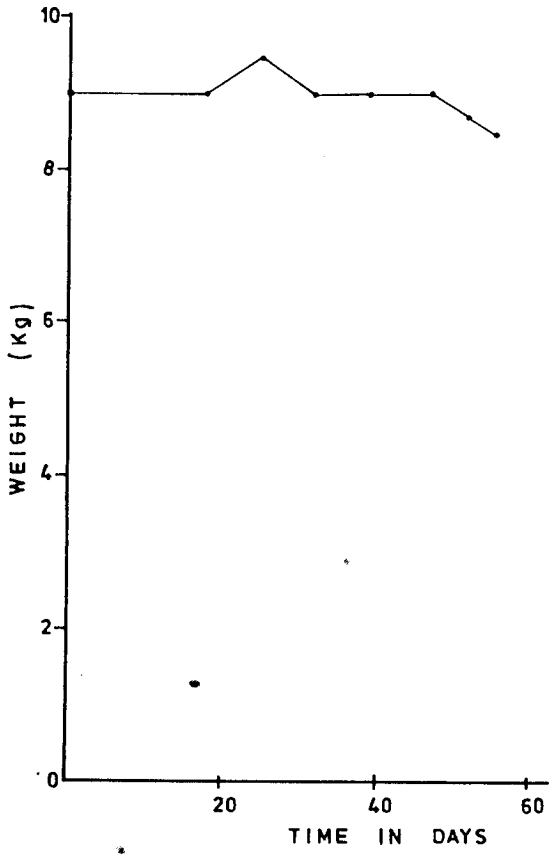
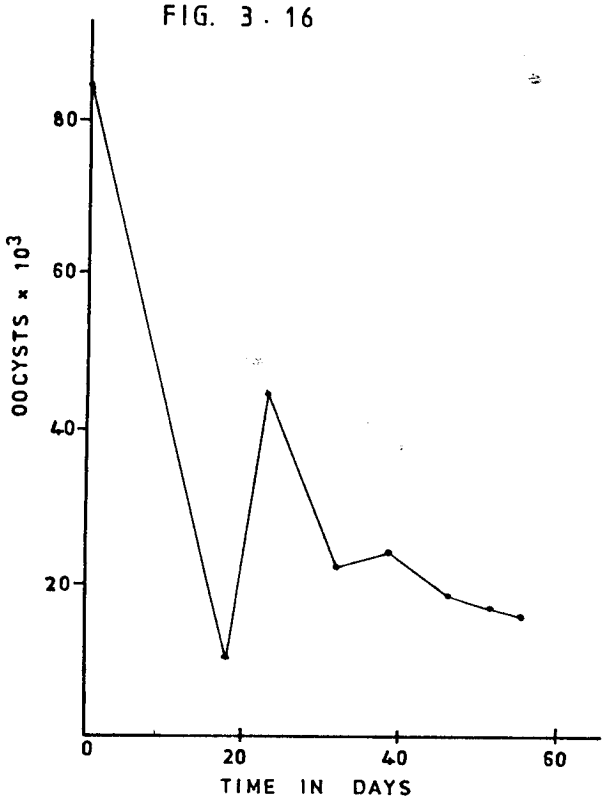
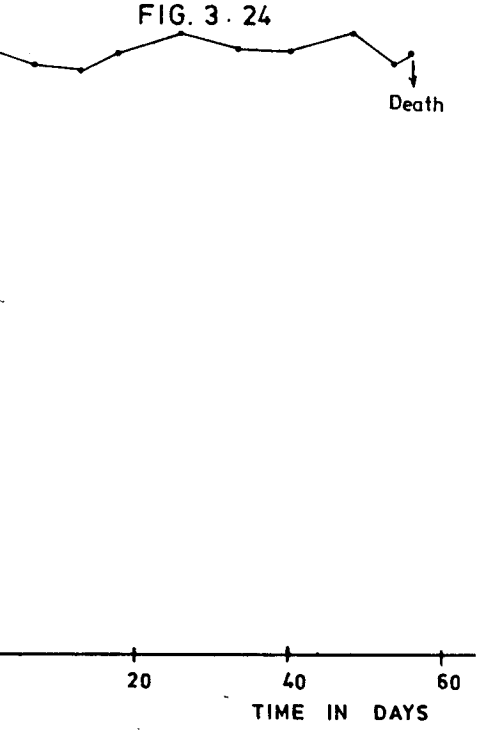


FIG. 3.16



GOAT 3

FIG. 3.24



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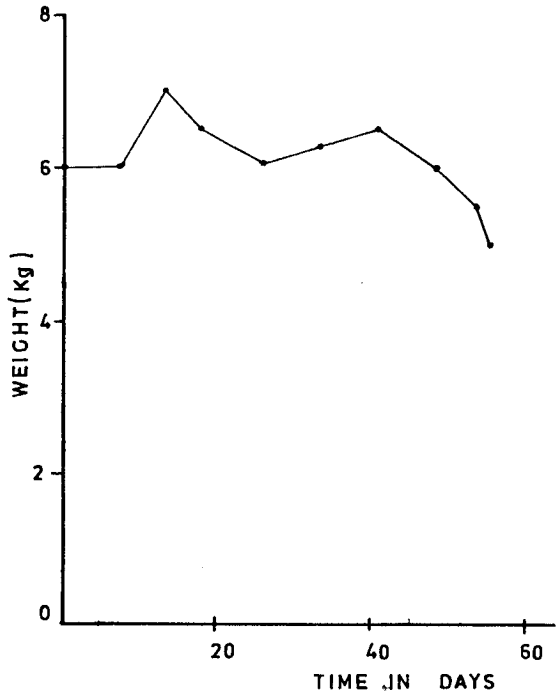
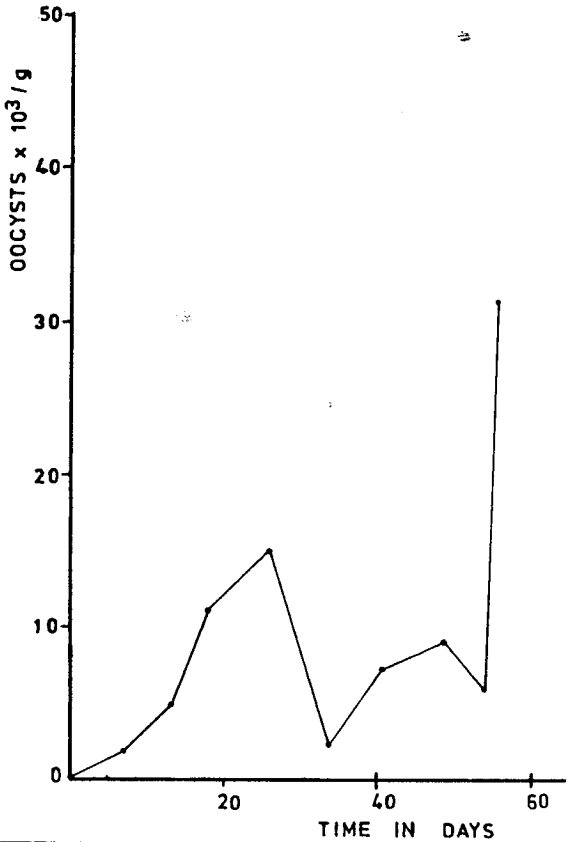


FIG. 3.26



GOAT 4

FIG. 3.34

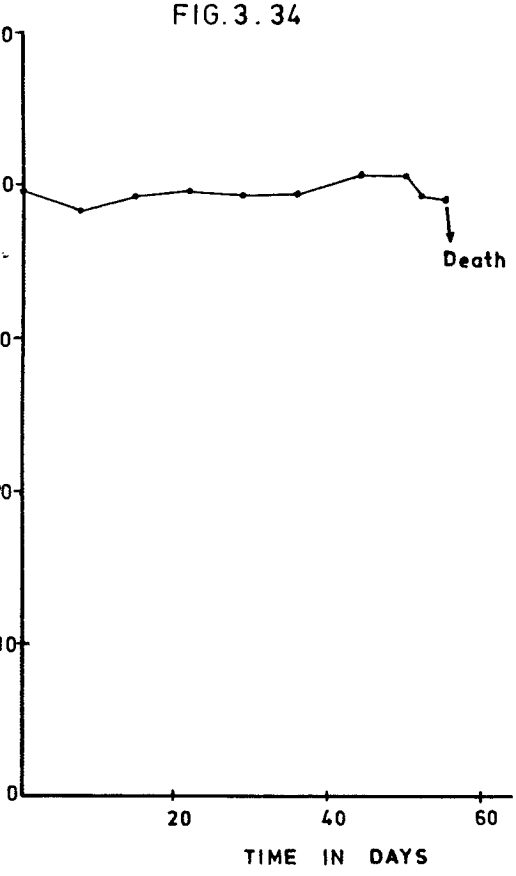


FIG. 3.35

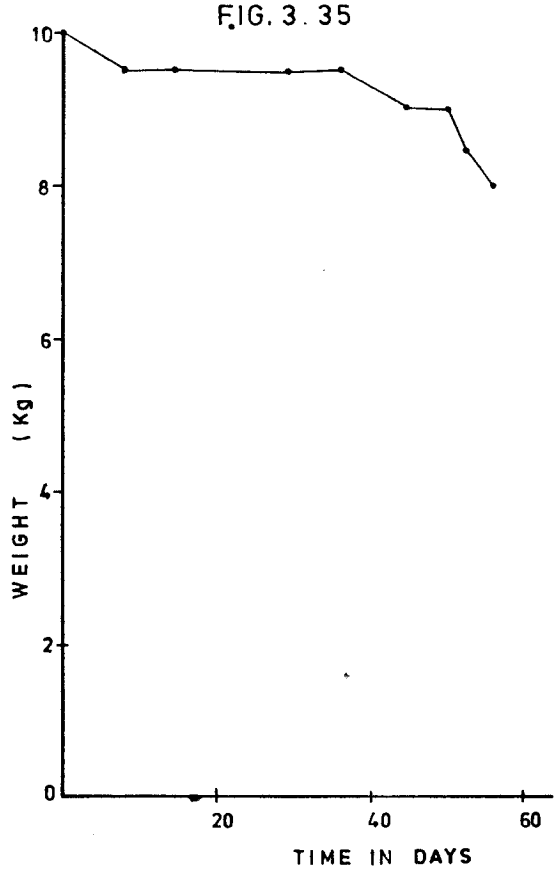


FIG. 3.36

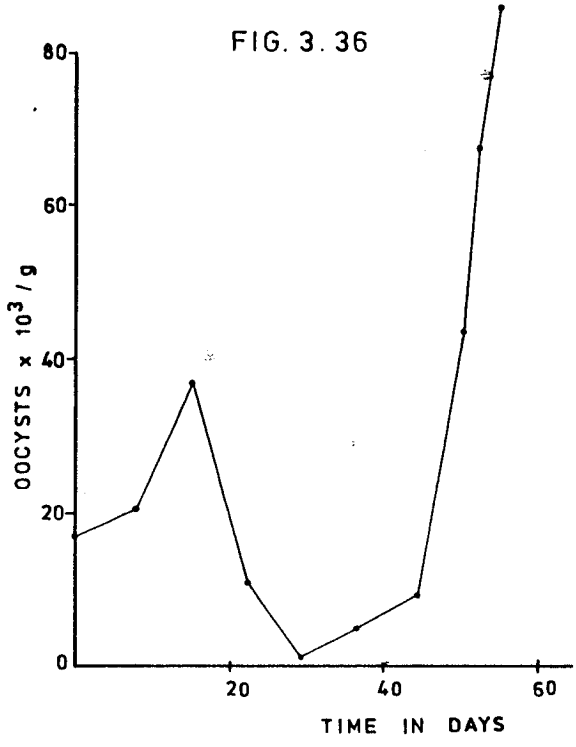


FIG. 3.44

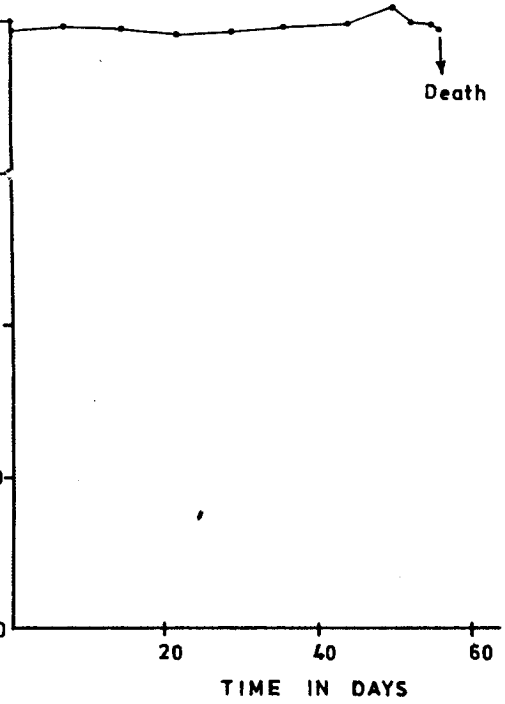


FIG. 3.45

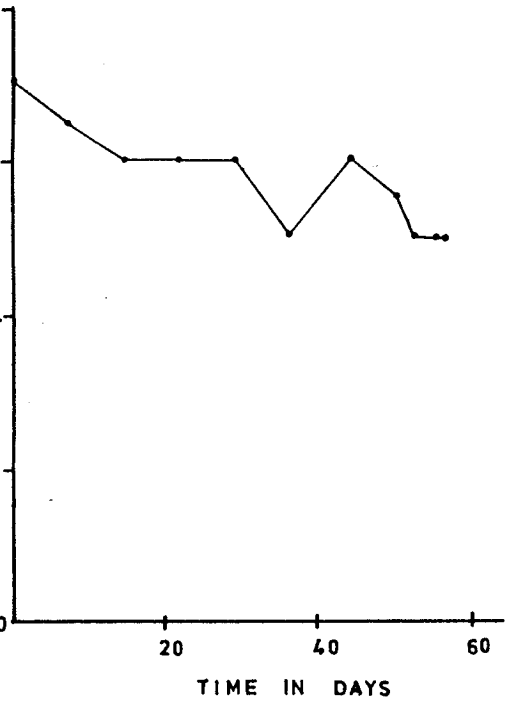
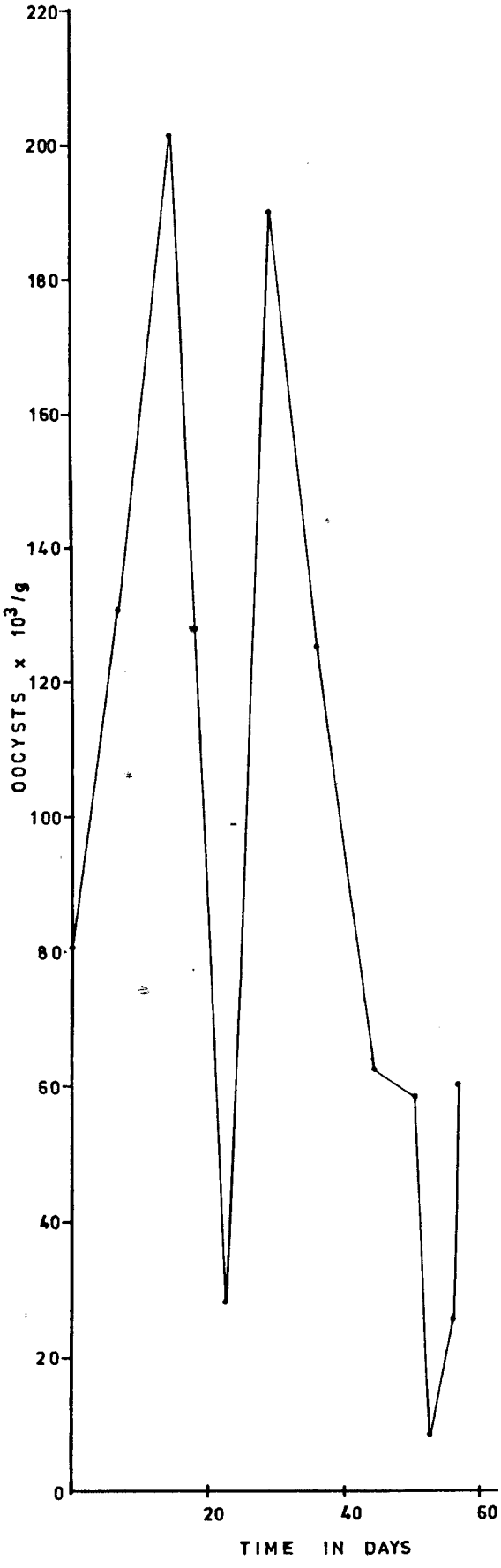


FIG. 3.46



GOAT 6

FIG. 3.54

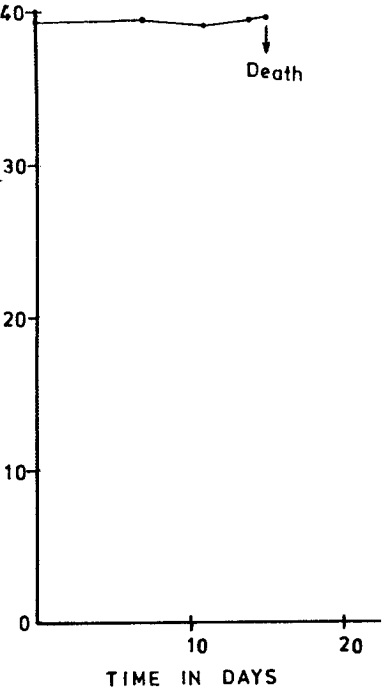


FIG. 3.55

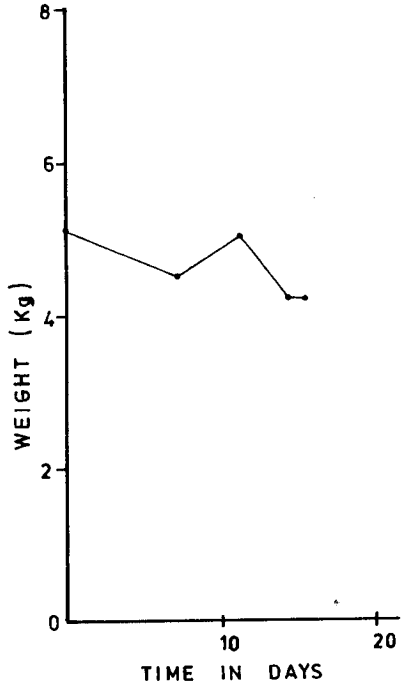
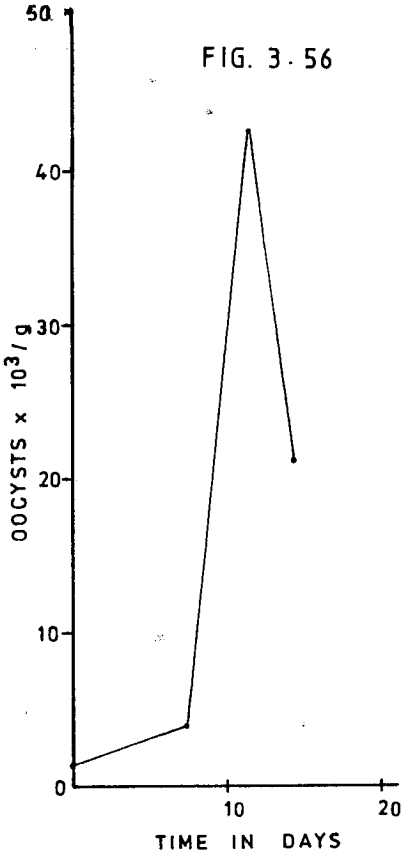


FIG. 3.56



GOAT 7

FIG. 3.64

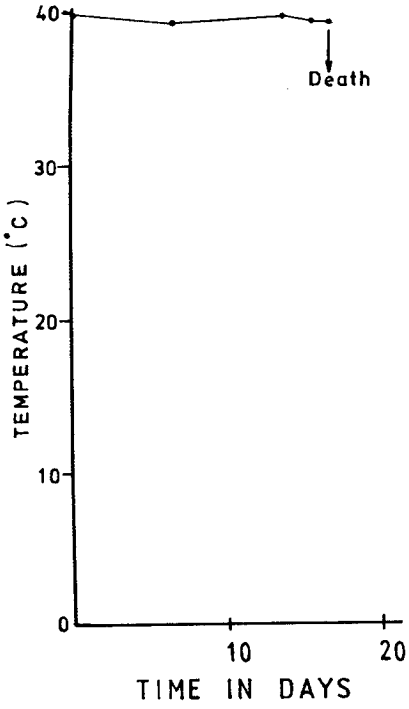
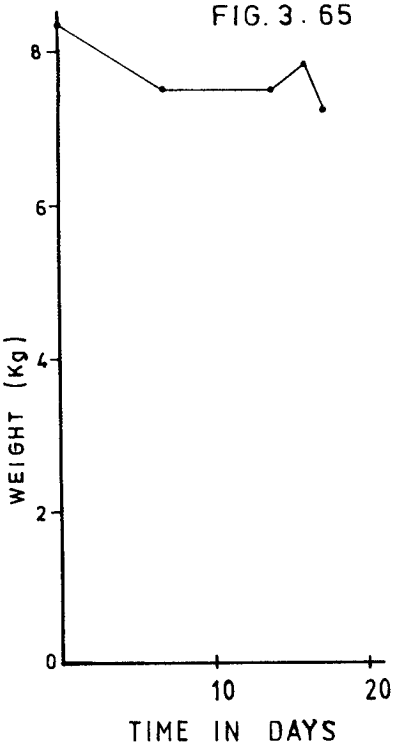


FIG. 3.65



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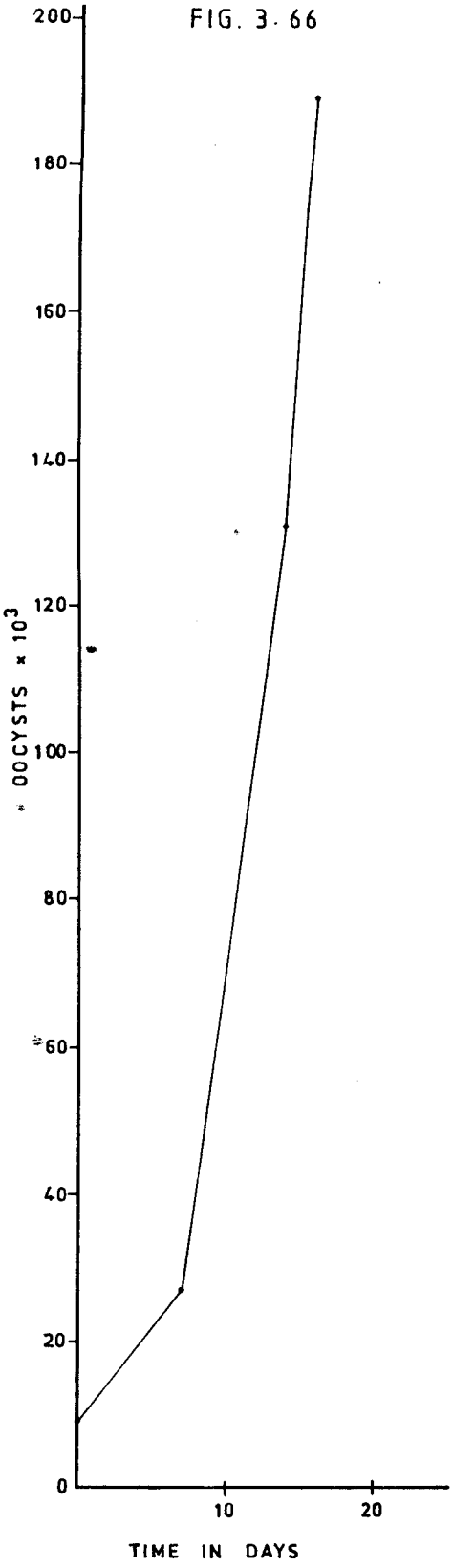


GOAT 8

FIG. 3.74

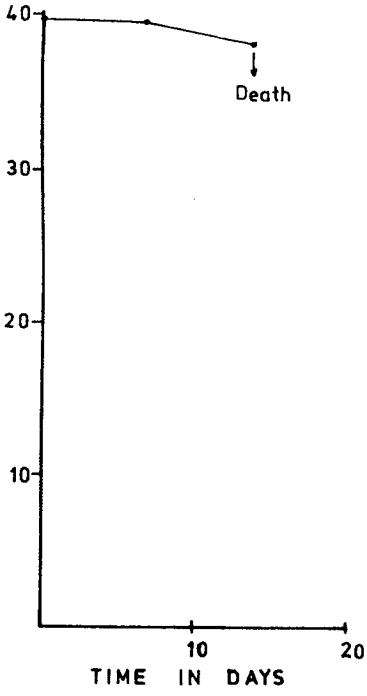
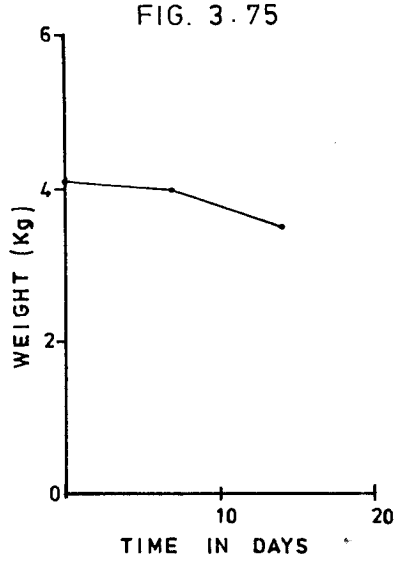


FIG. 3.75



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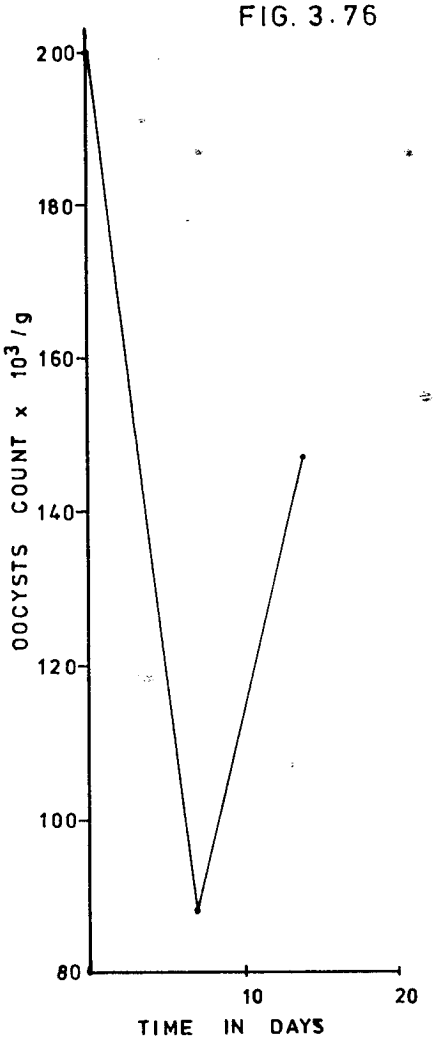


FIG. 3.84

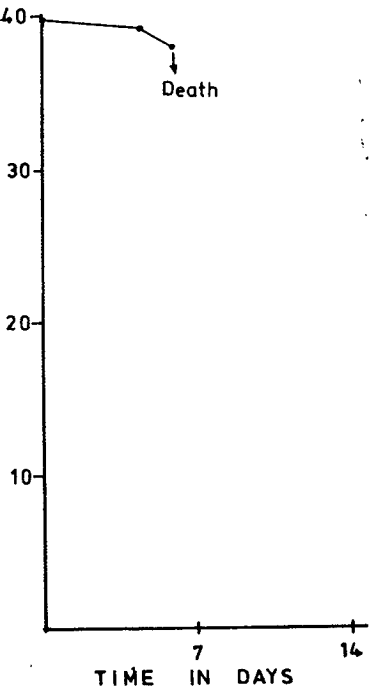
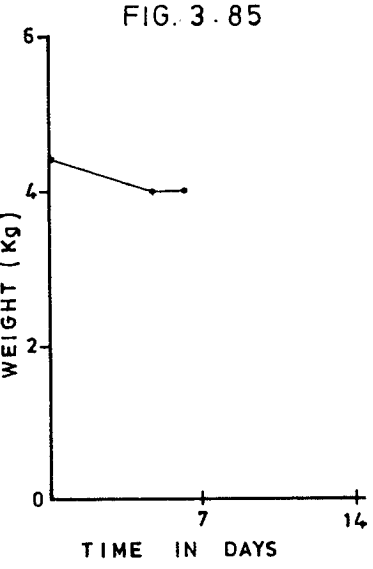


FIG. 3.85



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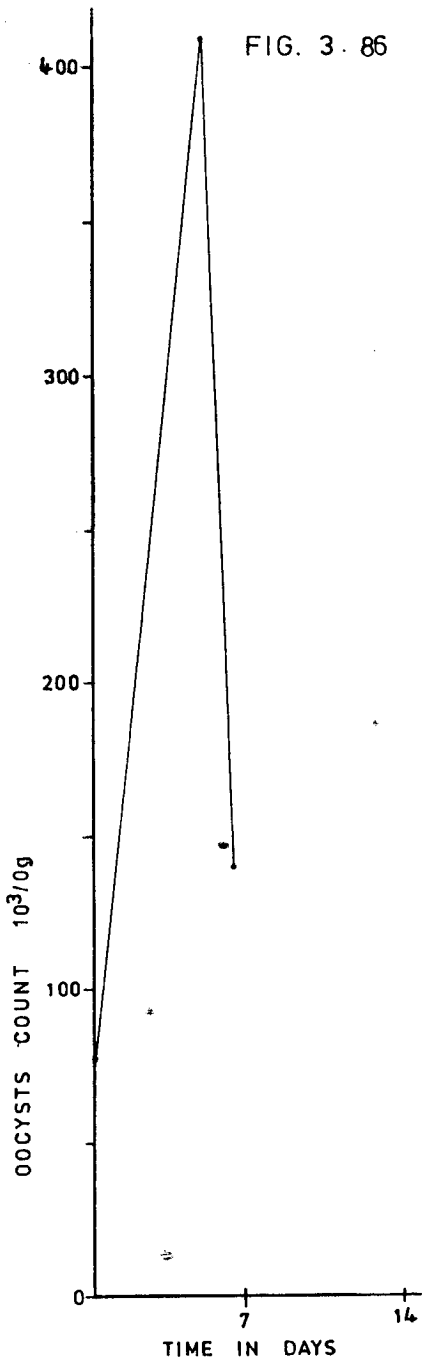


FIG. 3.94

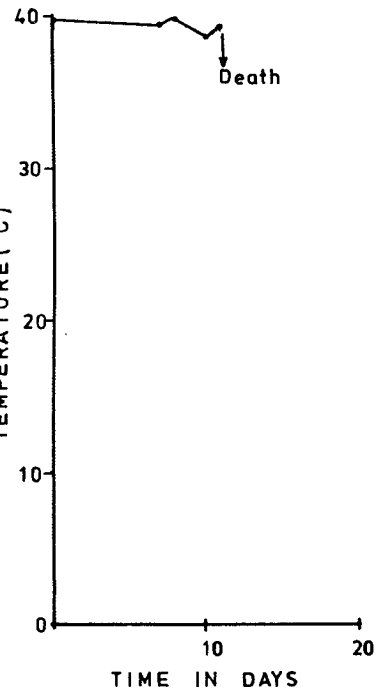


FIG. 3.95

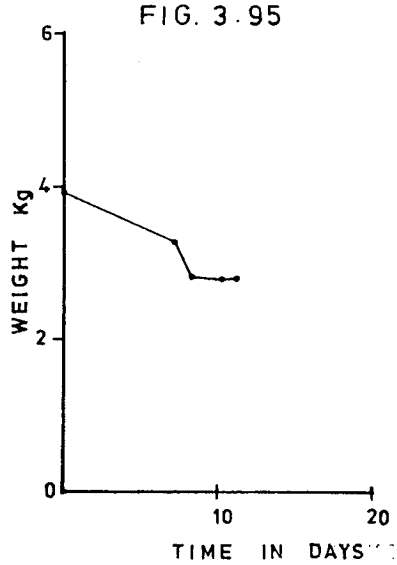
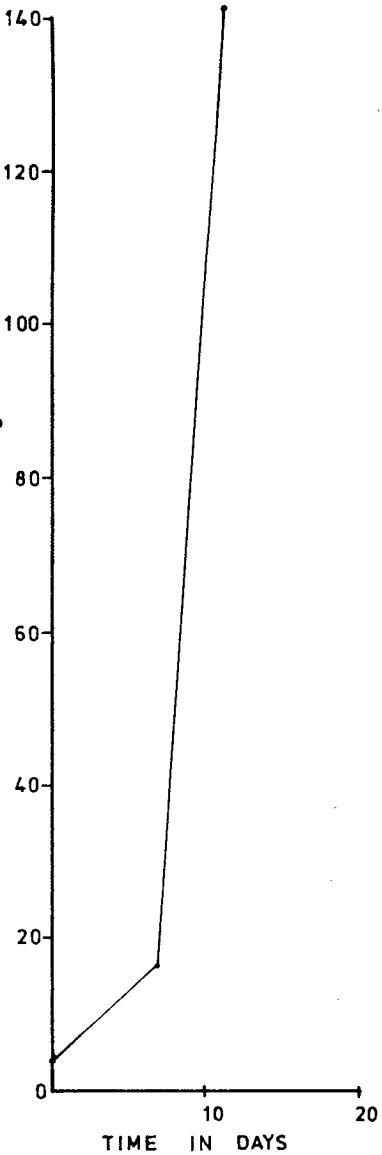


FIG. 3.96



GOAT 1

FIG. 3.7

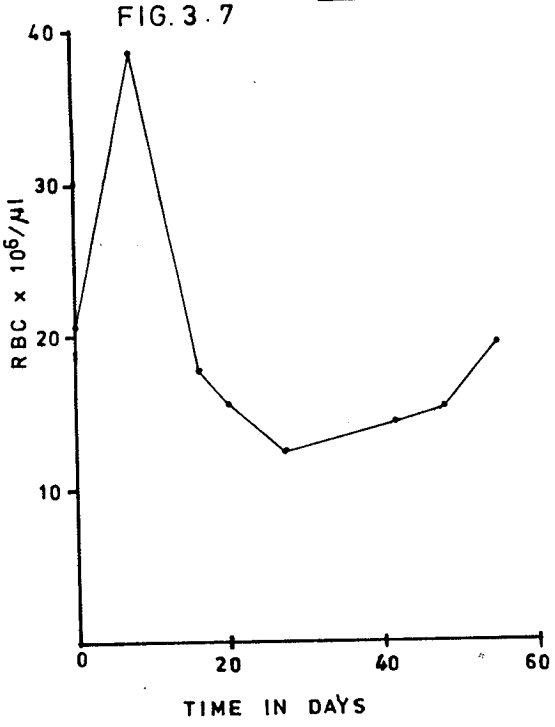


FIG. 3.8

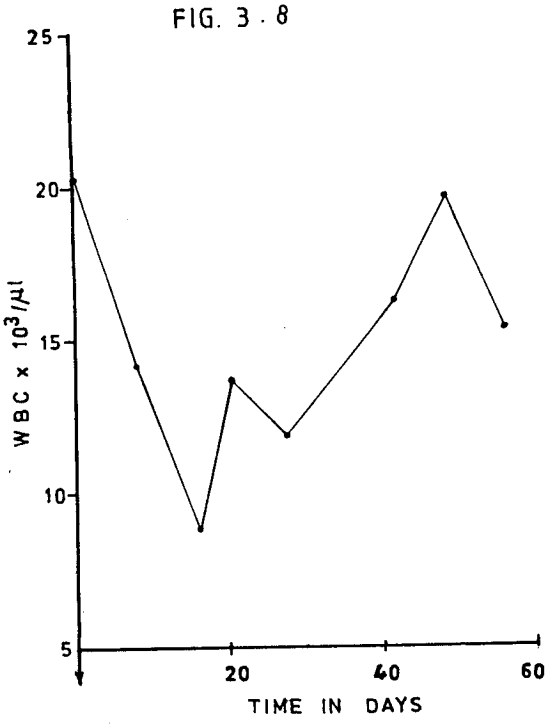


FIG. 3.9

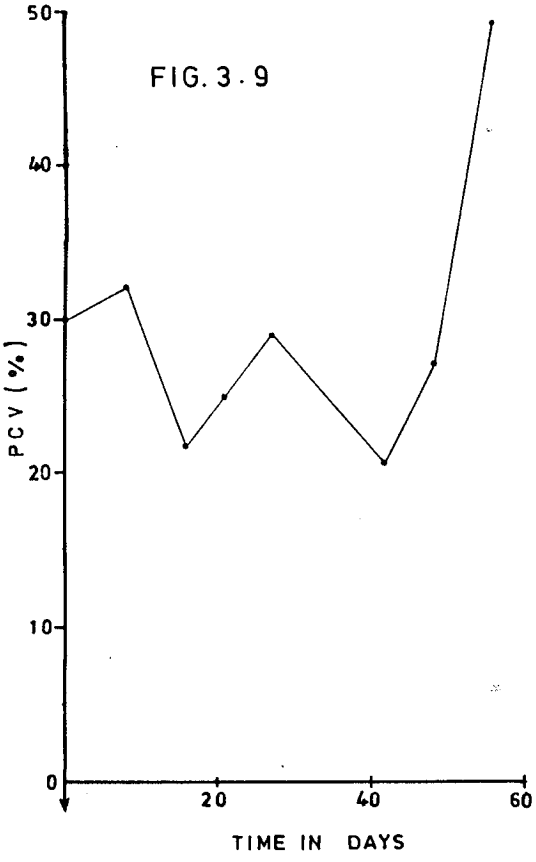
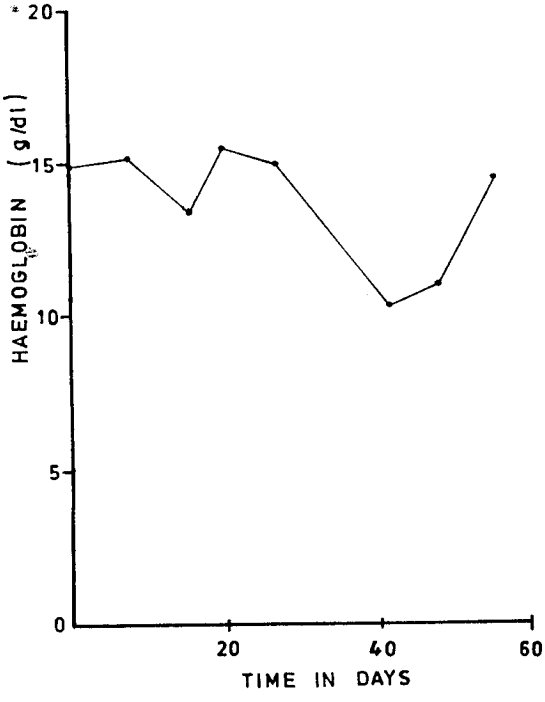


FIG. 3.10



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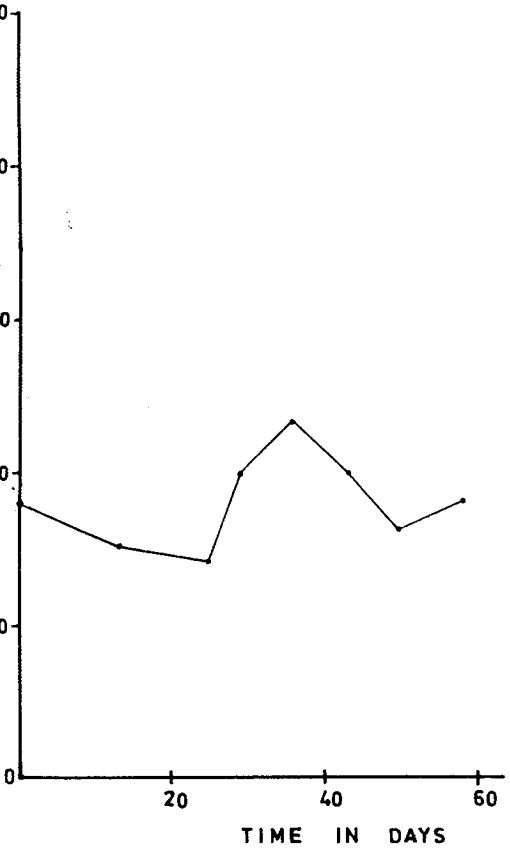

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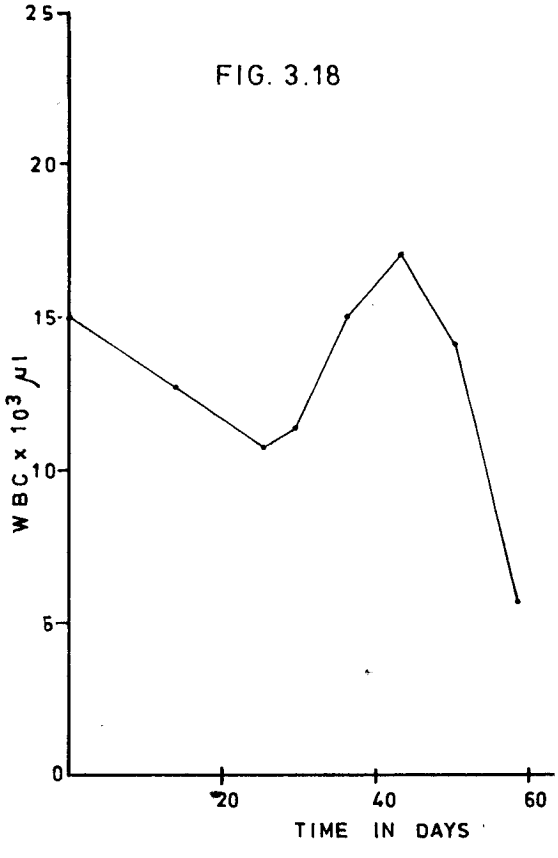

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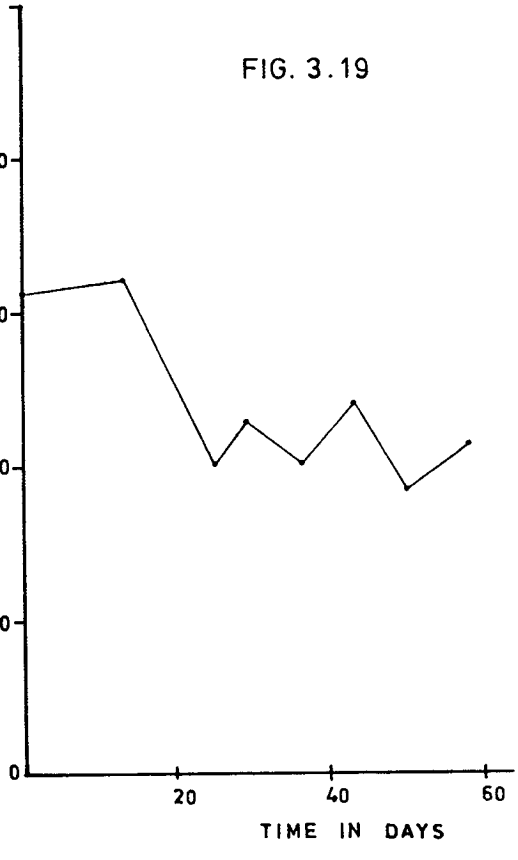
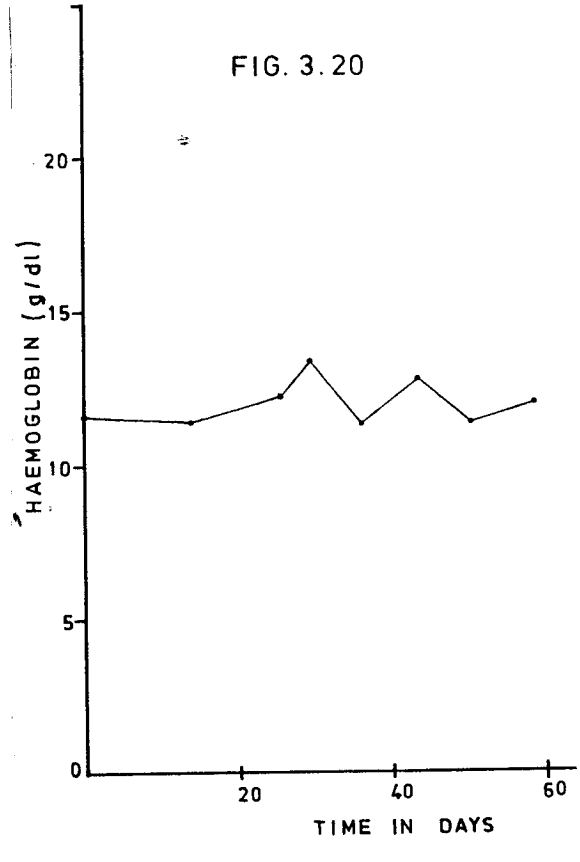


FIG. 3.20



GOAT 3

FIG. 3.27

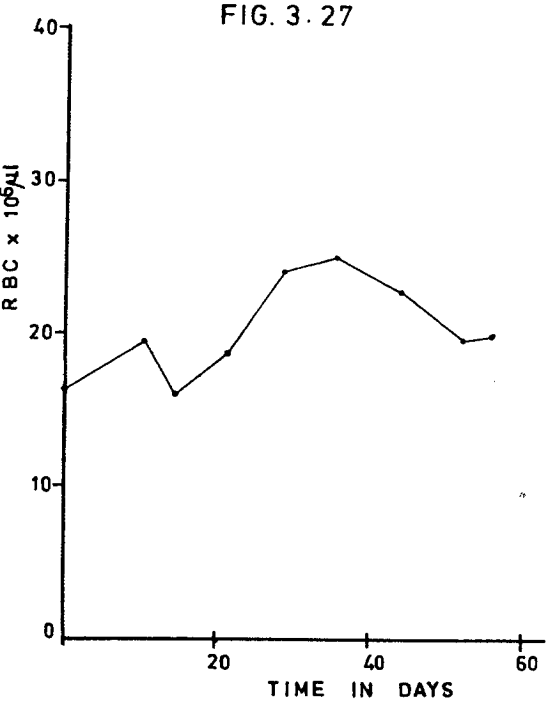


FIG. 3.28

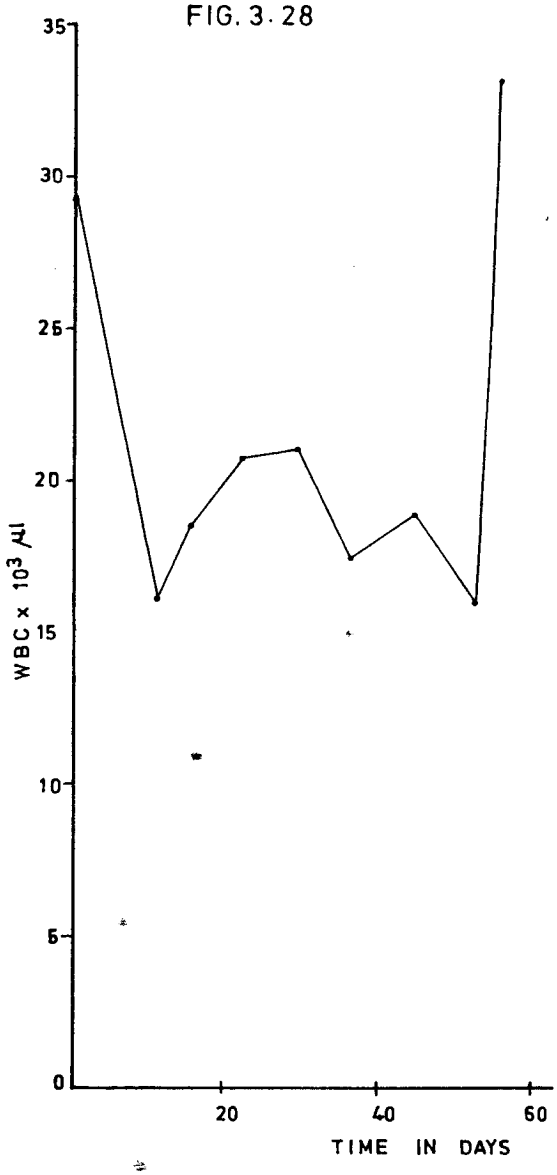


FIG. 3.29

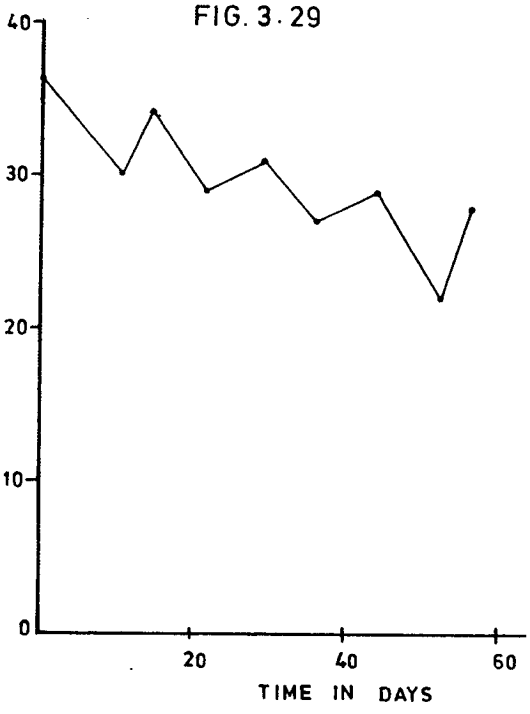
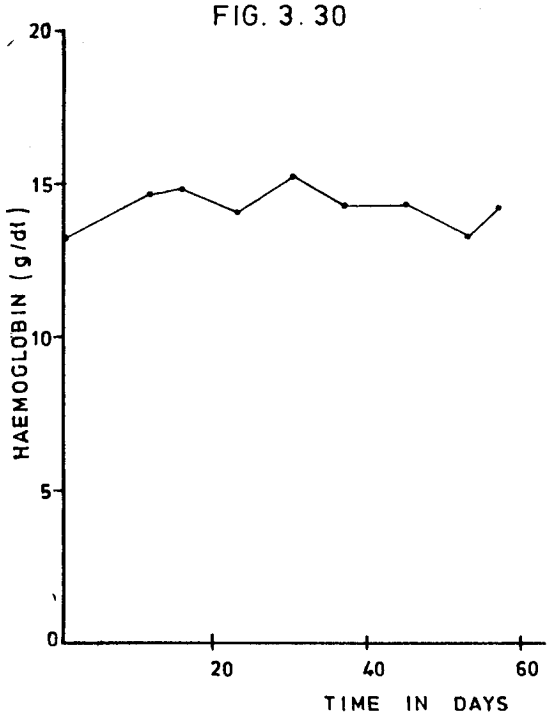


FIG. 3.30



GOAT 4

FIG. 3.37

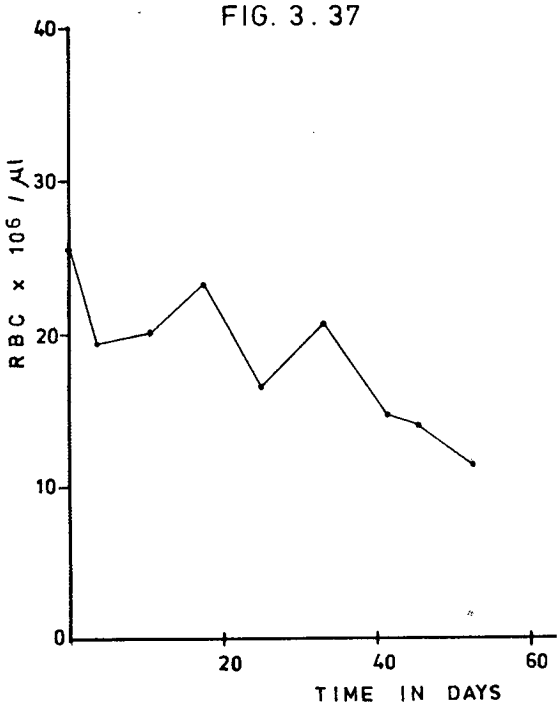


FIG. 3.38

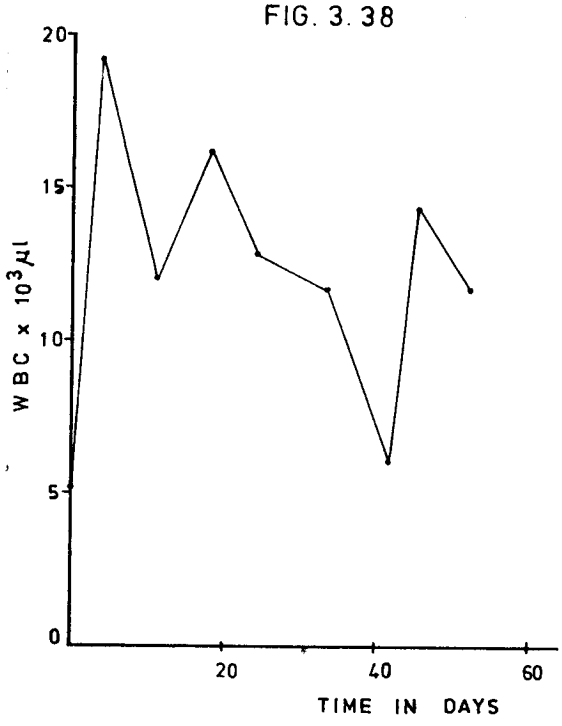


FIG. 3.39

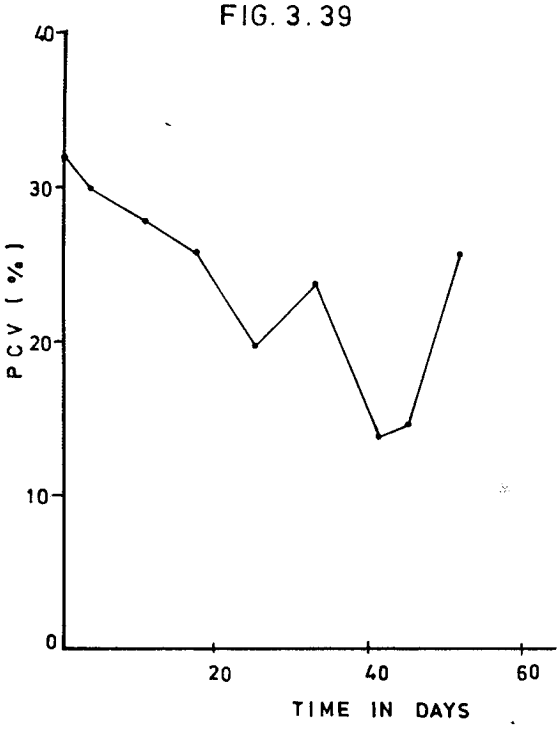
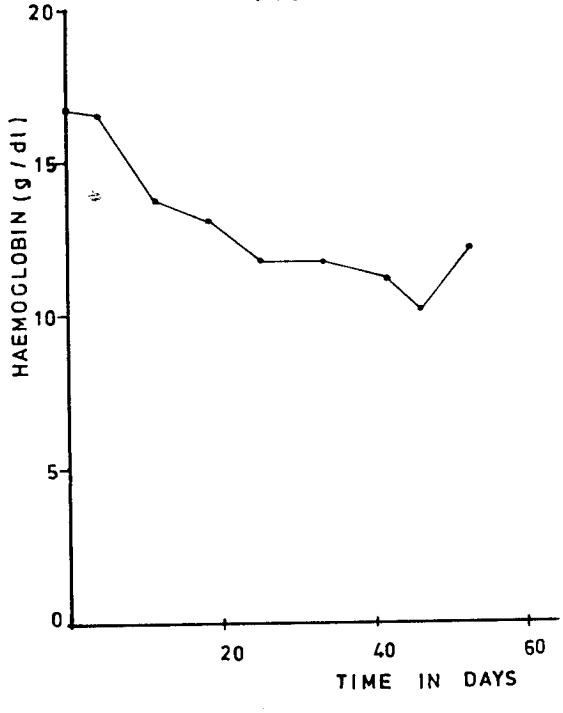


FIG. 3.40



GOAT 5

FIG. 3.47

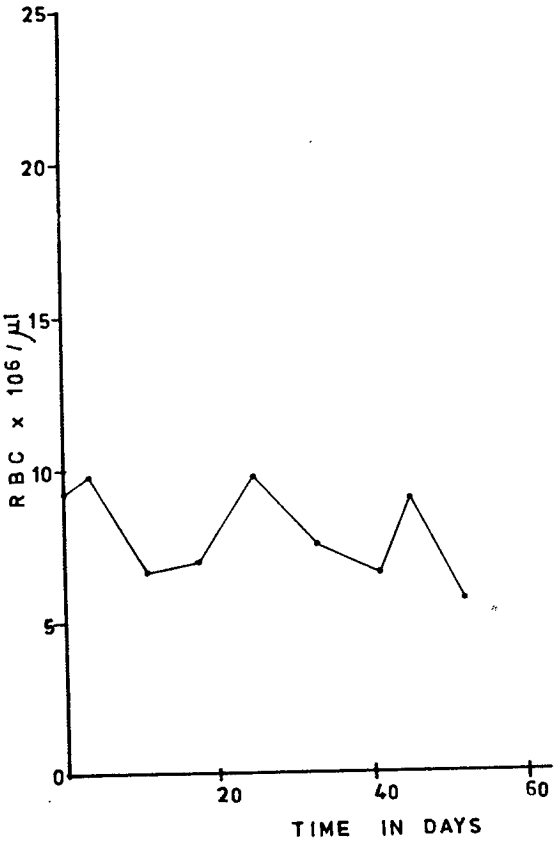


FIG. 3.48

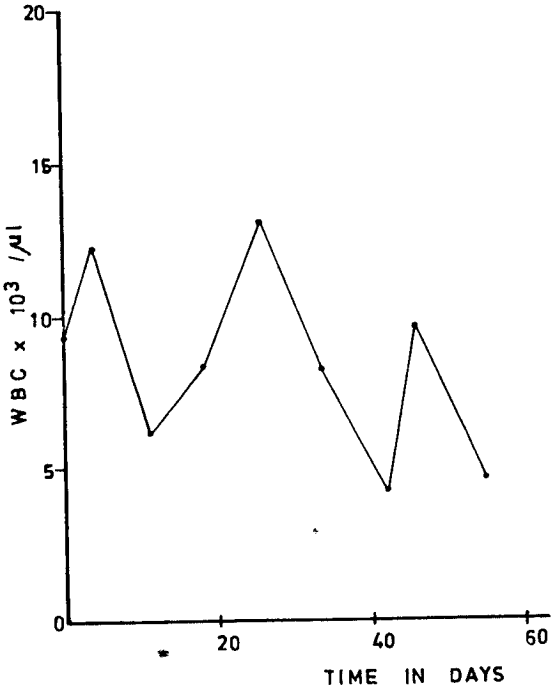


FIG. 3.49

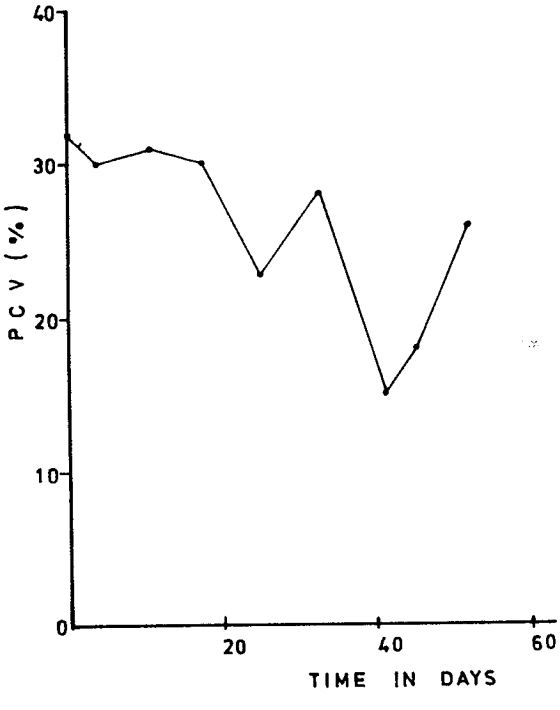


FIG. 3.50

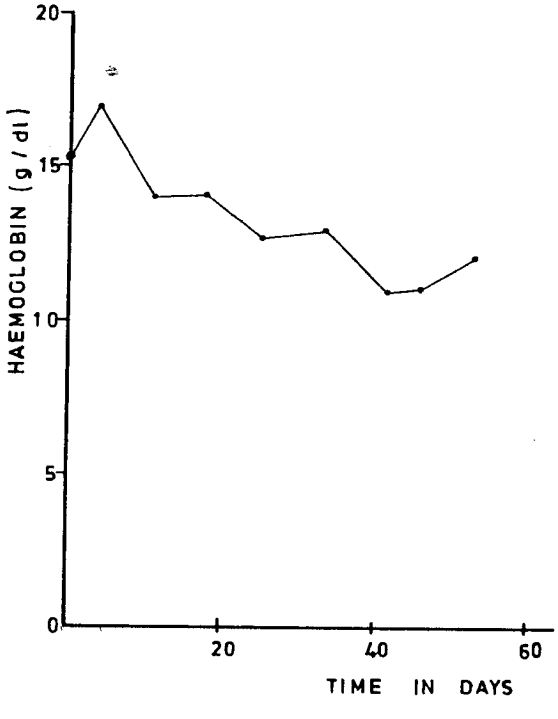


FIG. 3.57

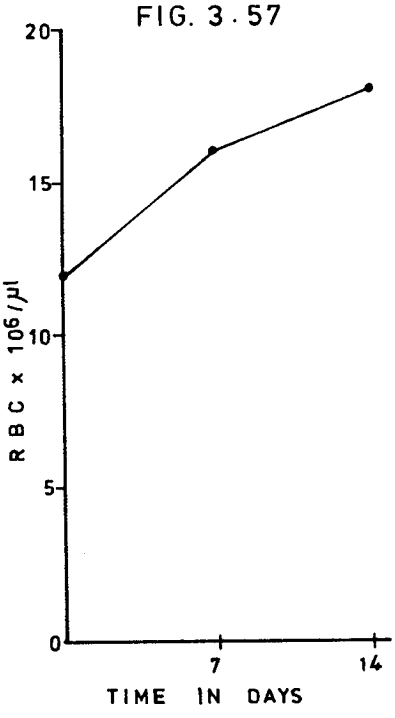


FIG. 3.58

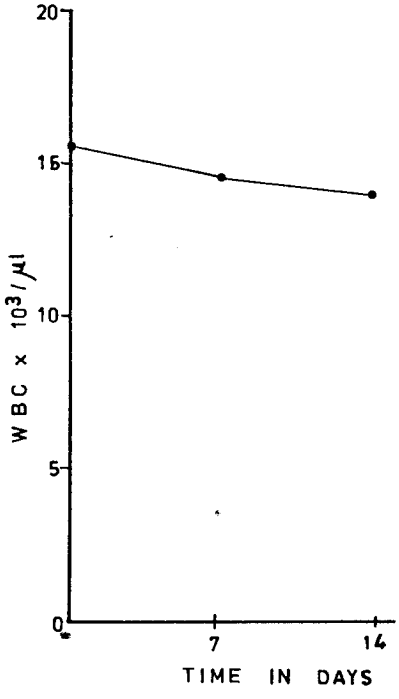


FIG. 3.59

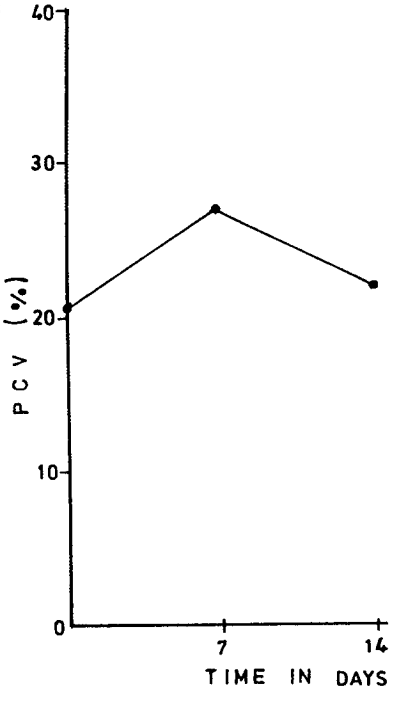


FIG. 3.60

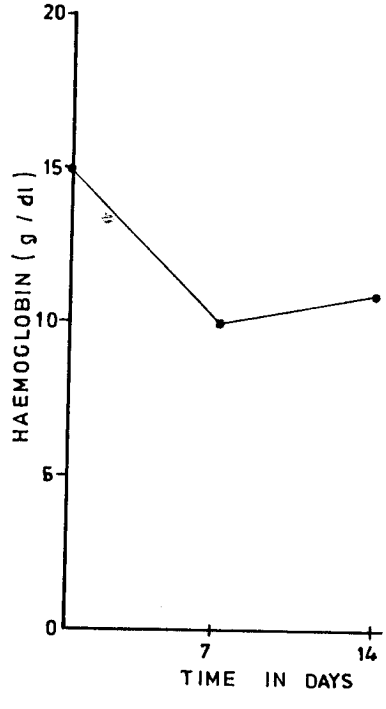


FIG. 3.67

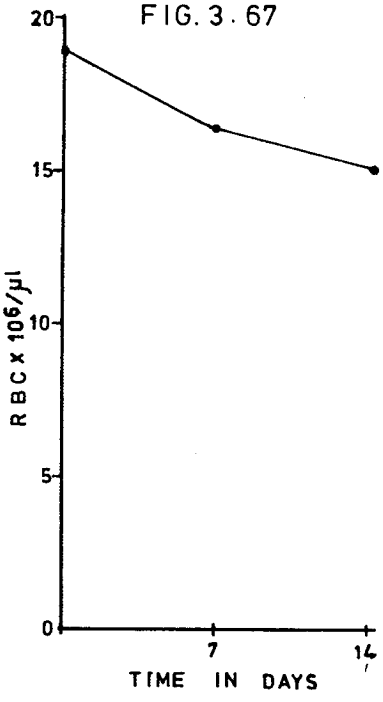
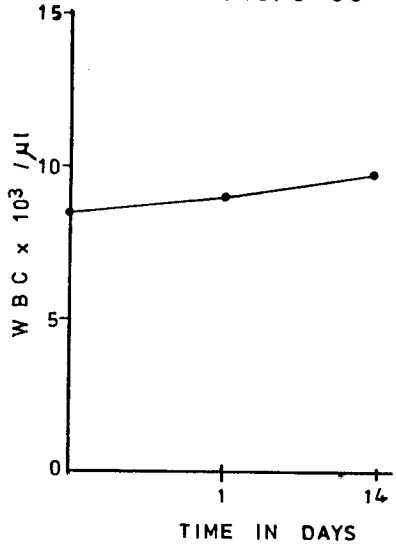


FIG. 3.68



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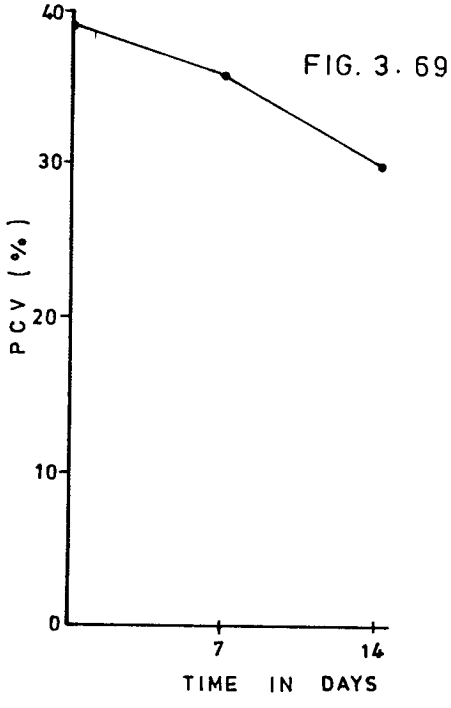

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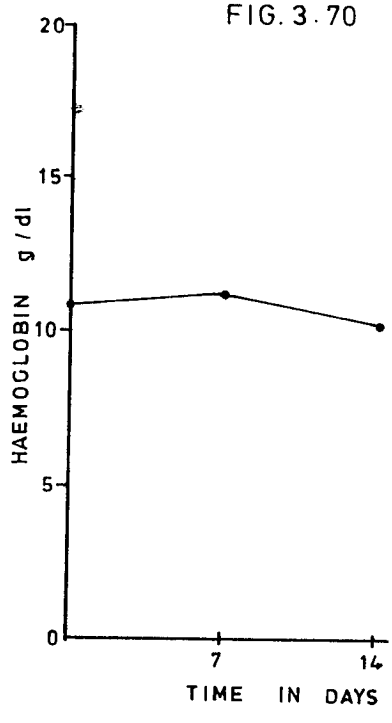
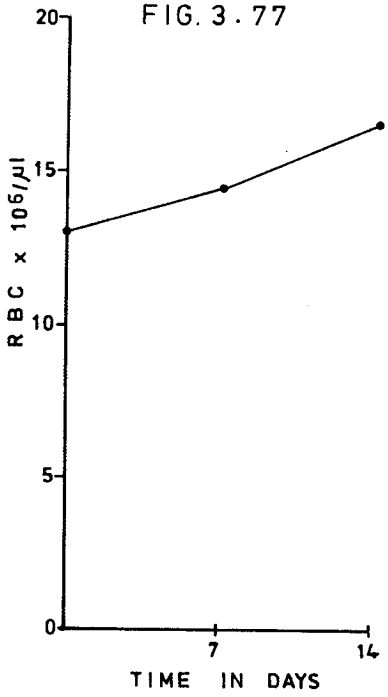


GOAT 8

FIG. 3.77



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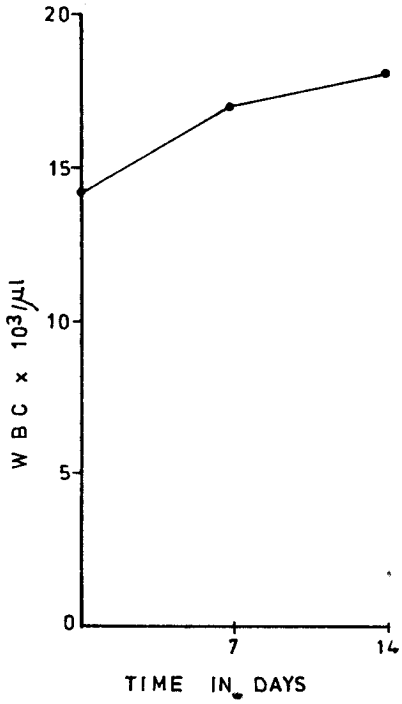

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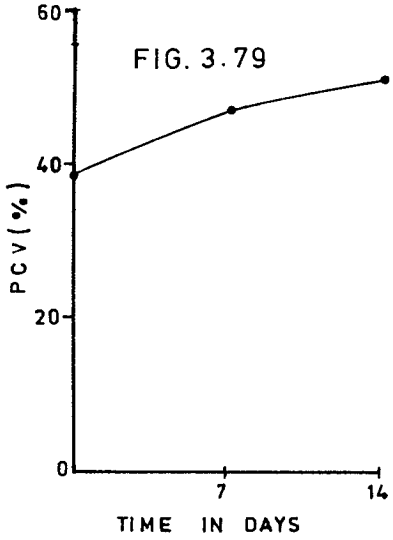
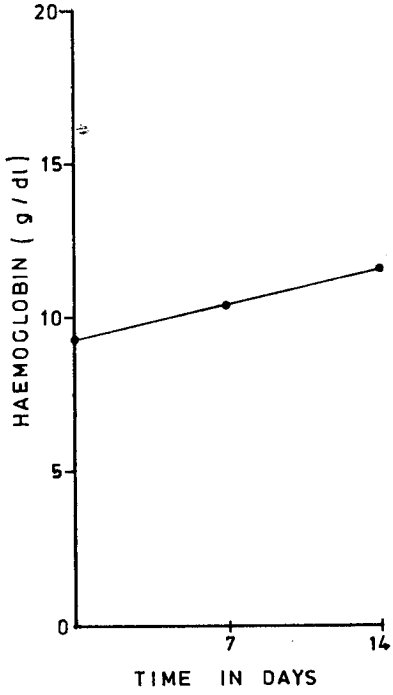
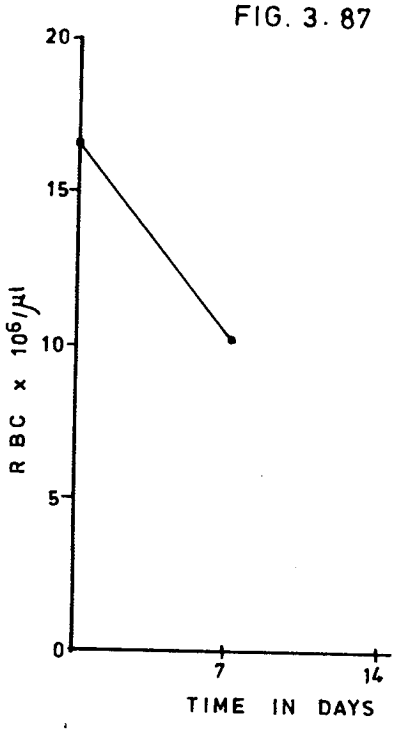


FIG. 3.80



GOAT 9

FIG. 3.87



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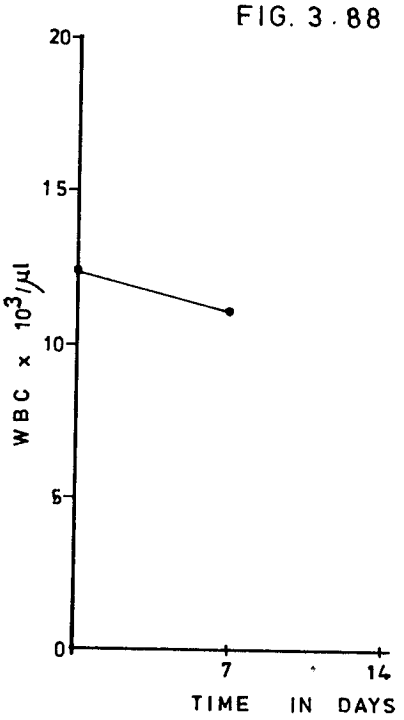

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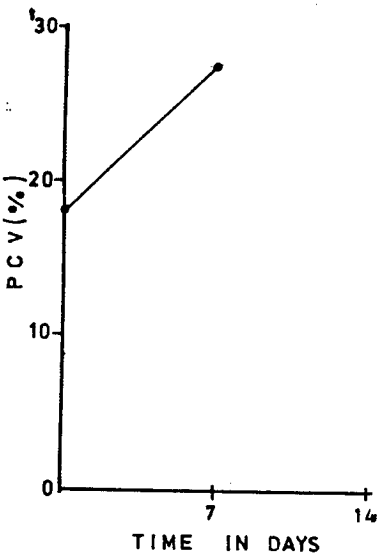
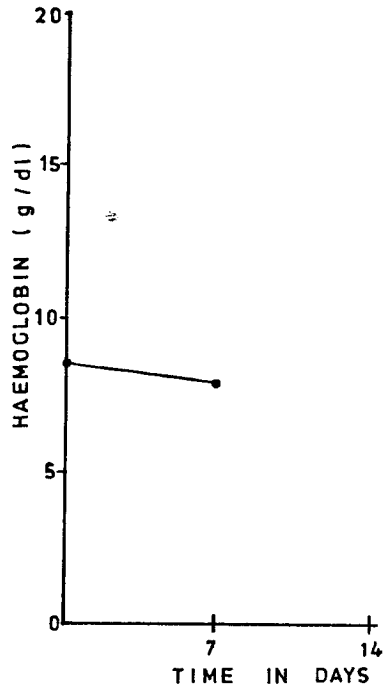


FIG. 3.90



GOAT 10

FIG. 3.97

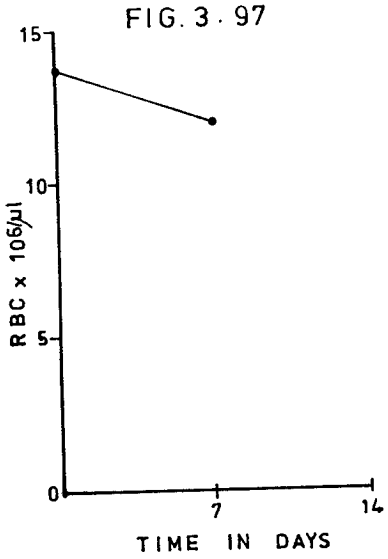


FIG. 3.98

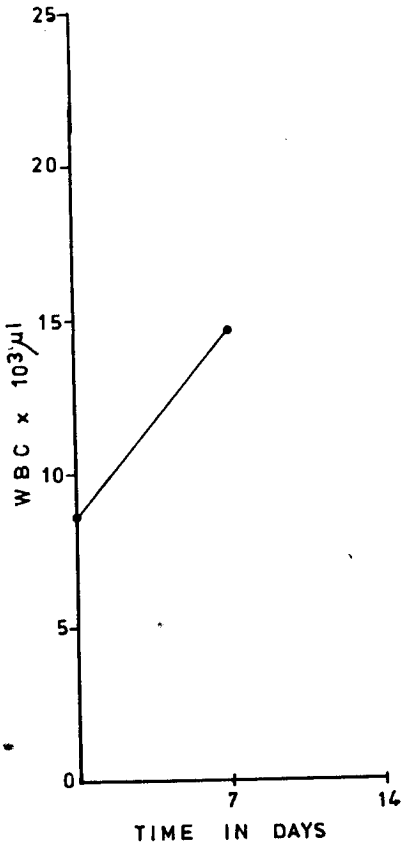


FIG. 3.99

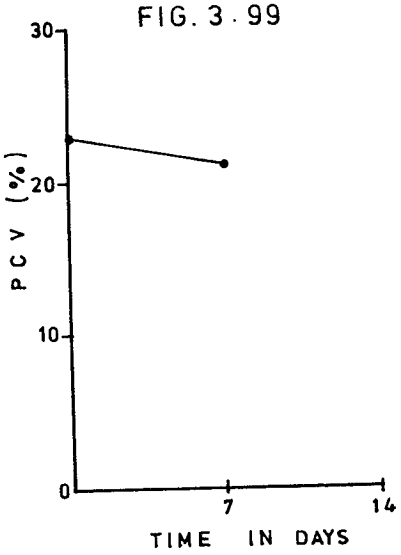
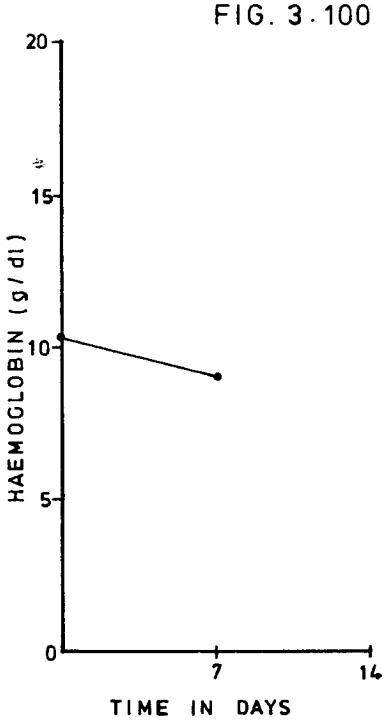


FIG. 3.100



GOAT 1

FIG. 3.11

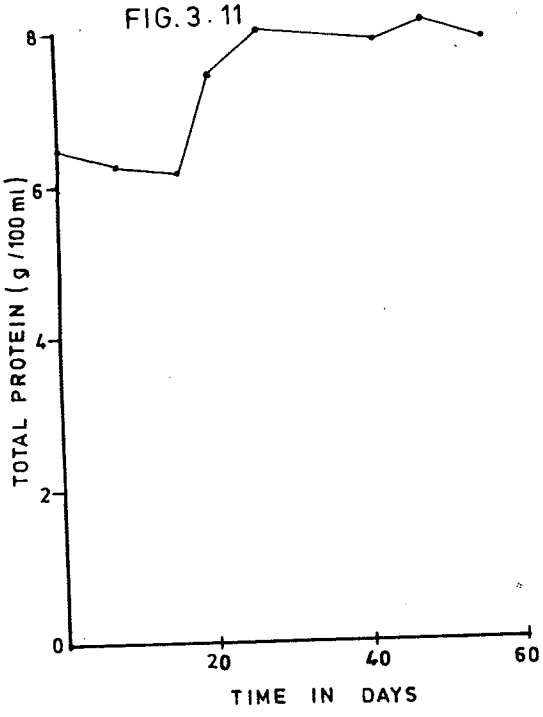


FIG. 3.12

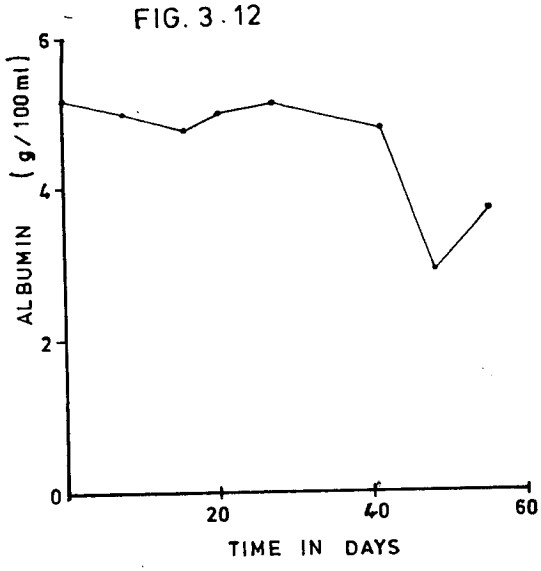


FIG. 3.13

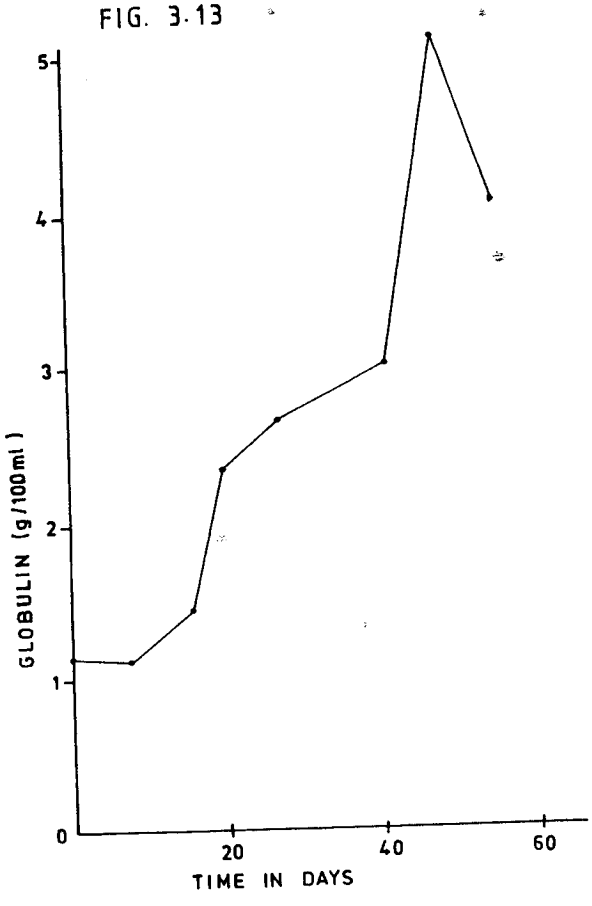


FIG. 3.21

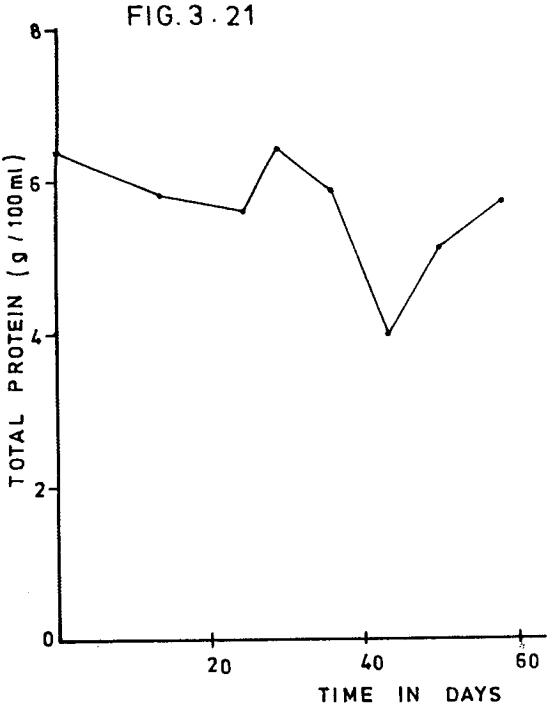


FIG. 3.22

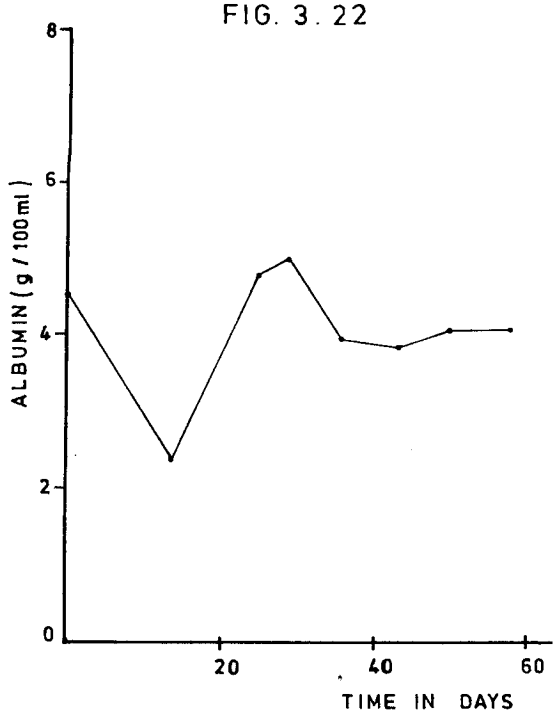
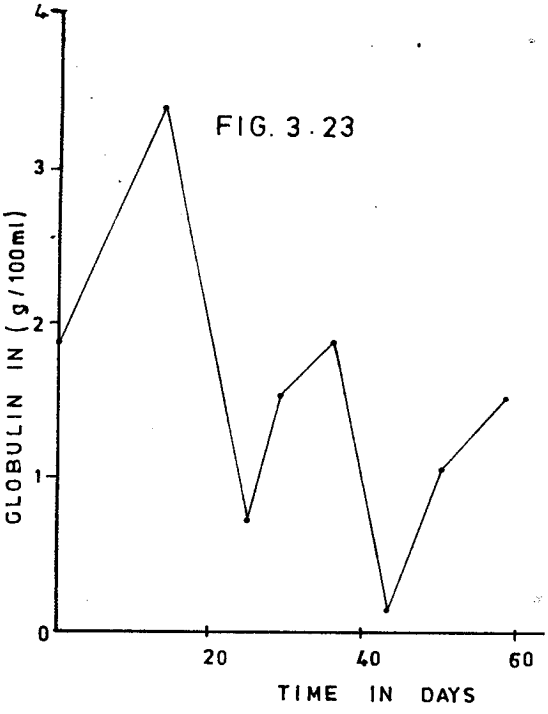


FIG. 3.23



GOAT 3

FIG. 3. 31

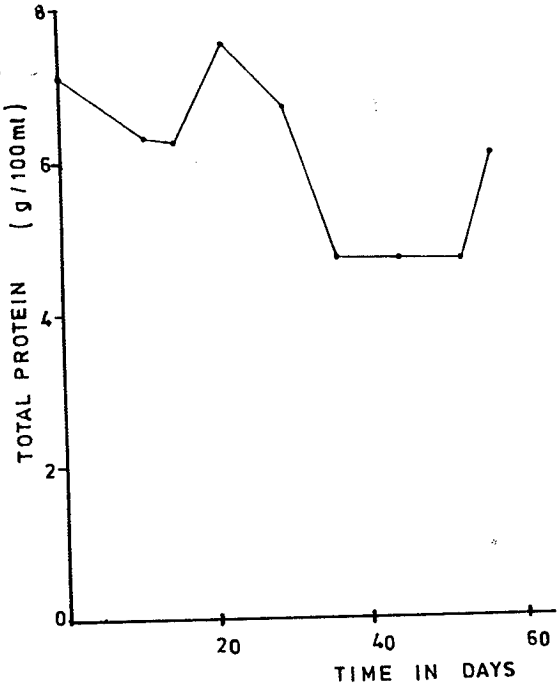


FIG. 3. 32

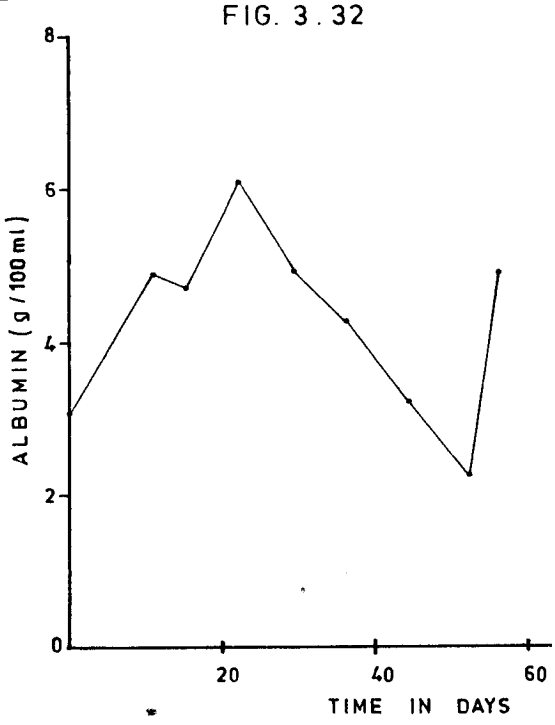
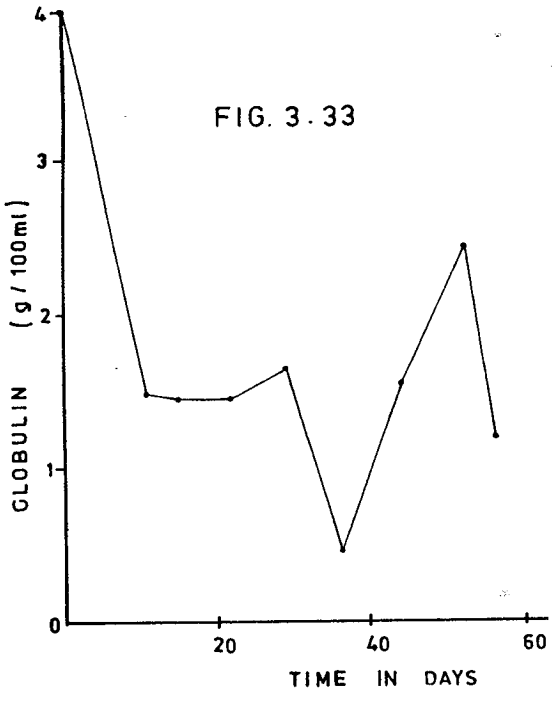


FIG. 3. 33



GOAT 4

FIG. 3.41

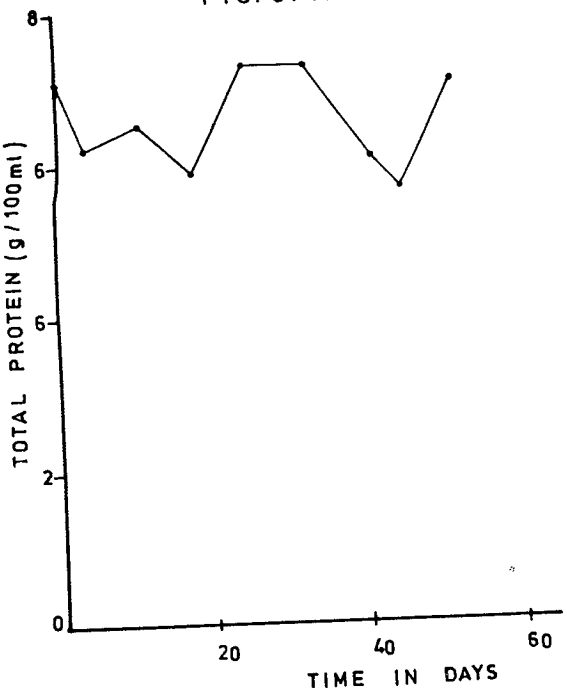


FIG. 3.42

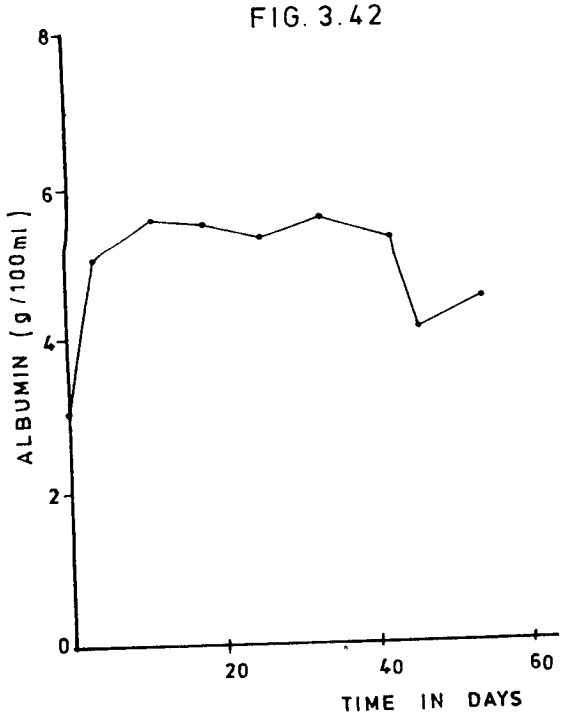
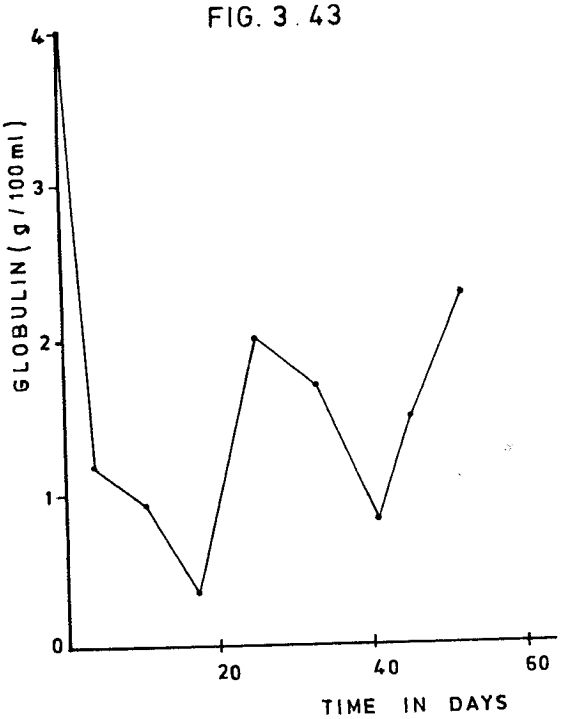


FIG. 3.43



GOAT 5

FIG. 3.51

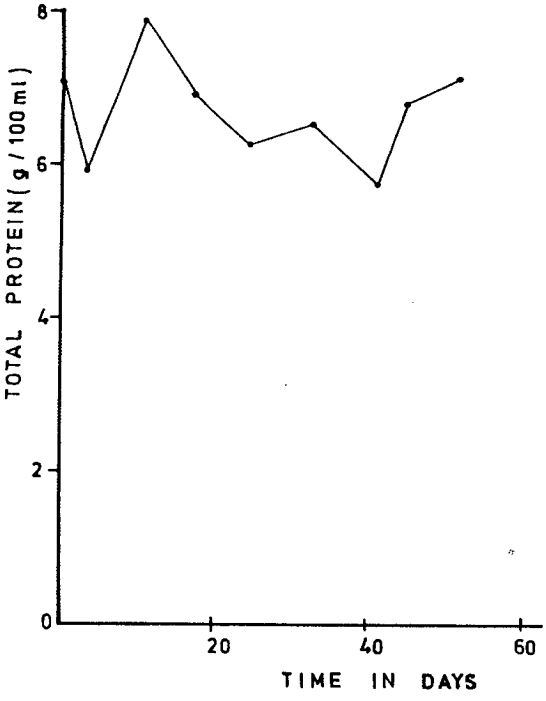


FIG. 3. 52

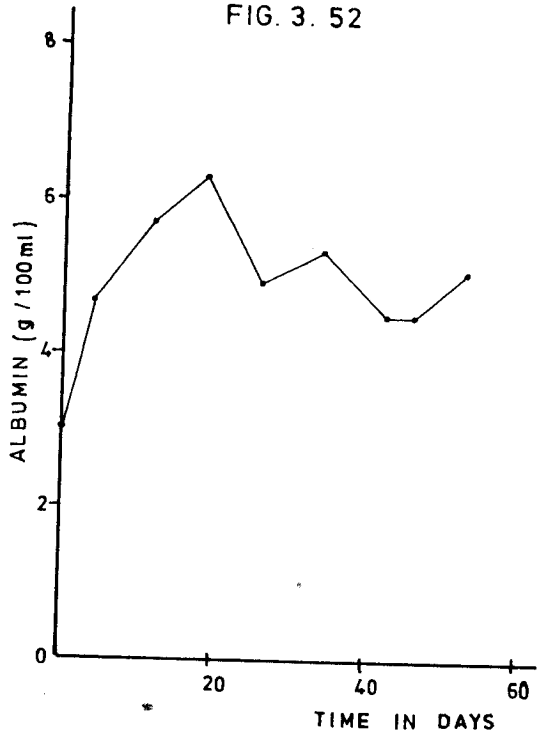
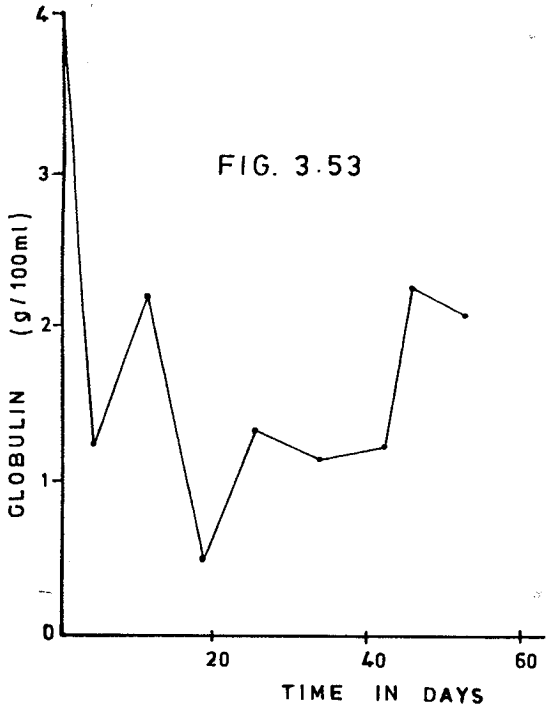


FIG. 3.53



GOAT 6

FIG. 3.61

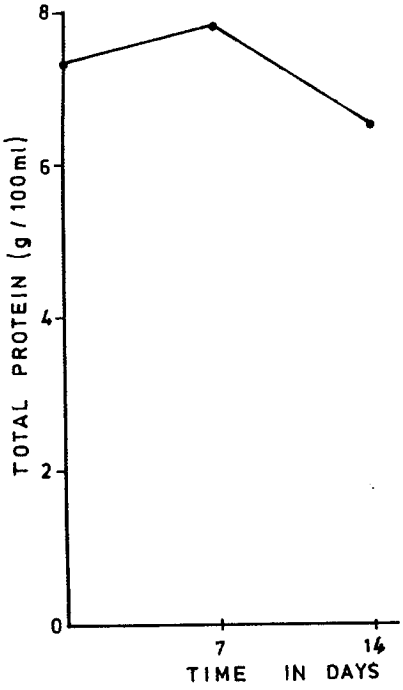
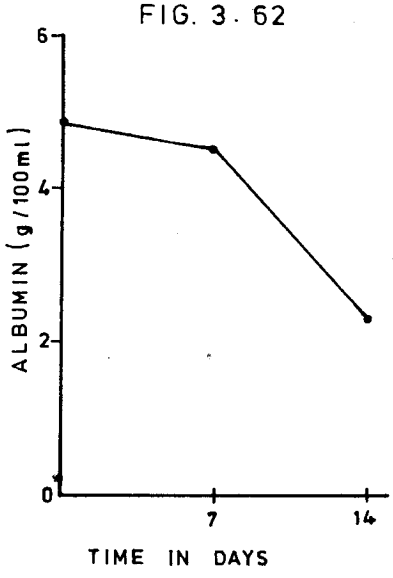


FIG. 3.62



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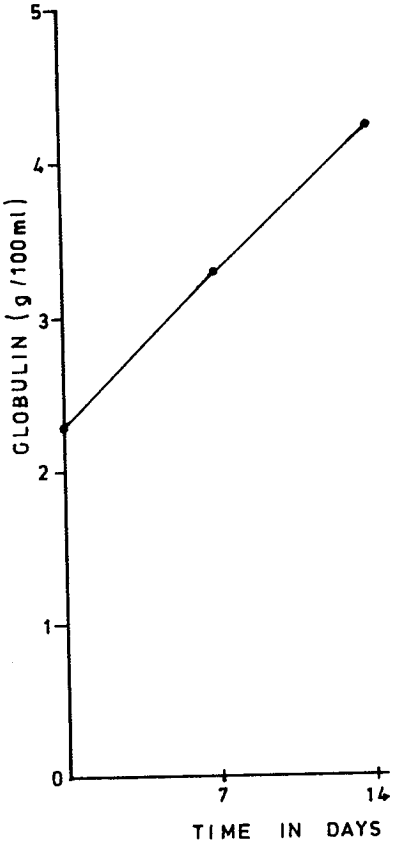


FIG. 3.71

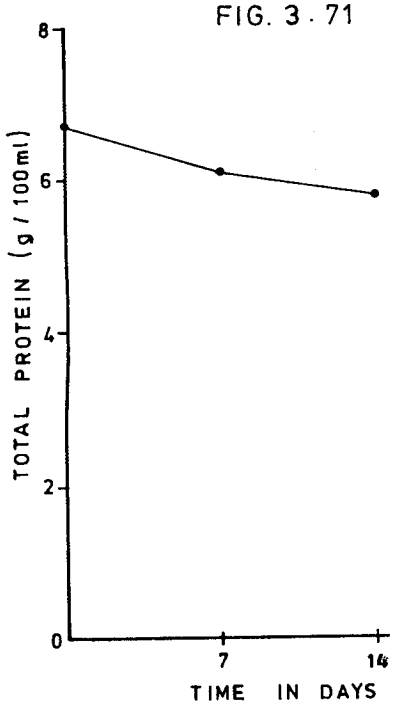


FIG. 3.72

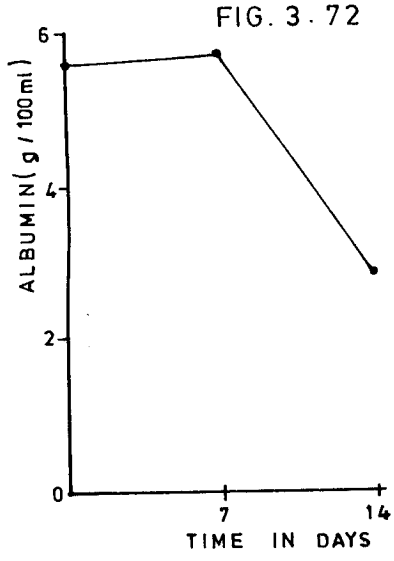


FIG. 3.73

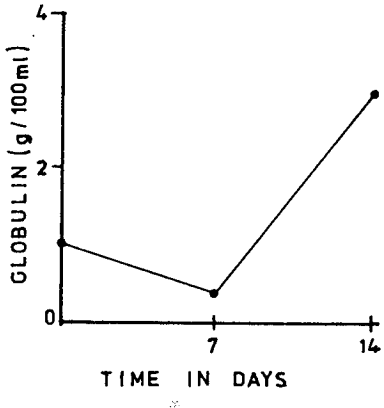


FIG. 3.81

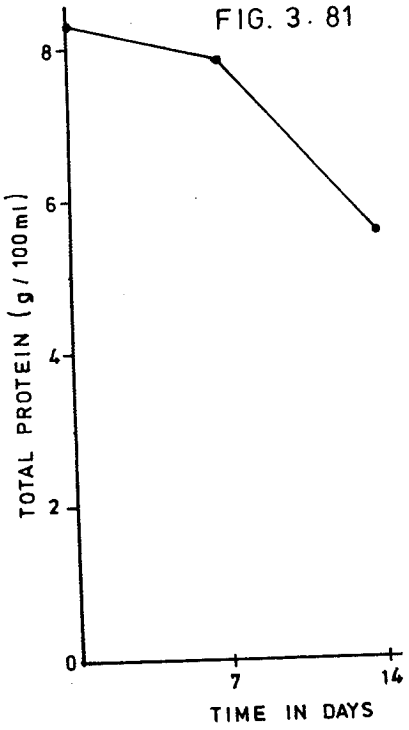


FIG. 3.82

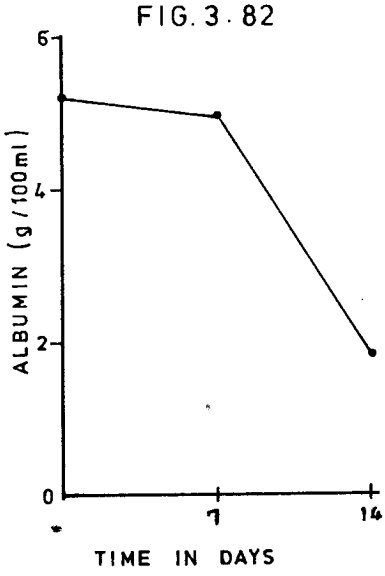


FIG. 3.83

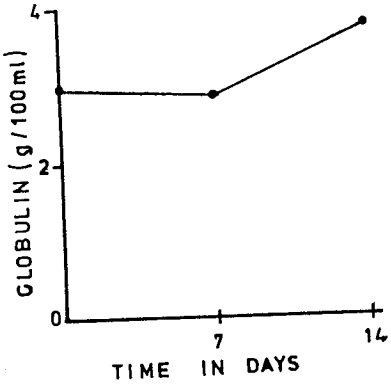


FIG. 3.91

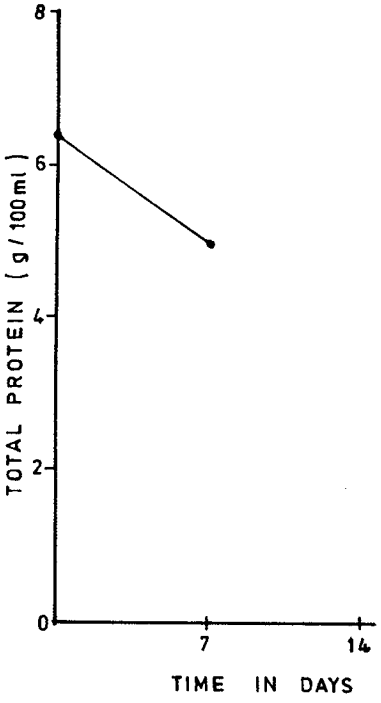


FIG. 3.92

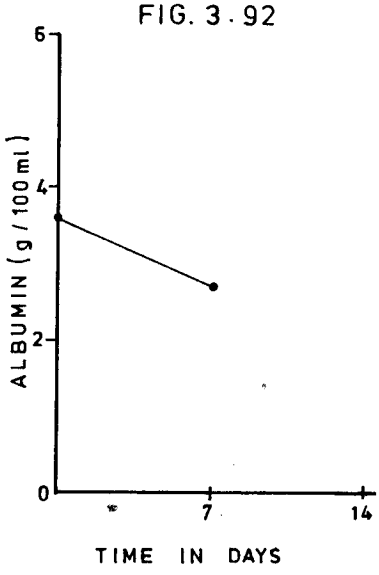


FIG. 3.93

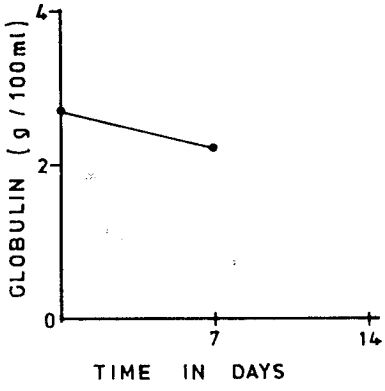


FIG. 3.101

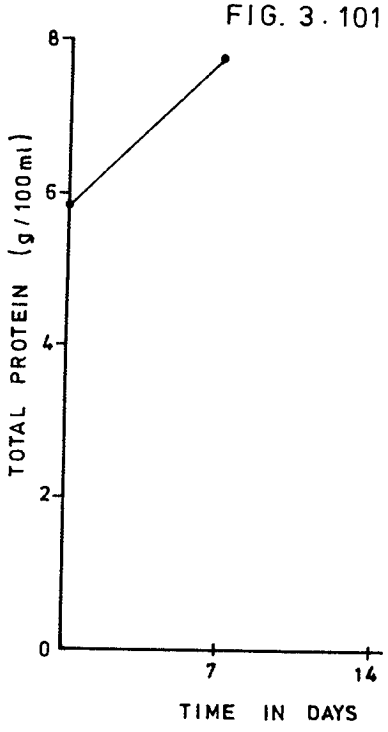


FIG. 3.102

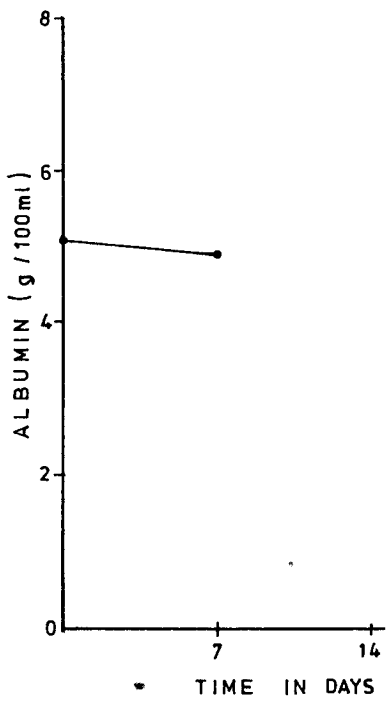


FIG. 3.103

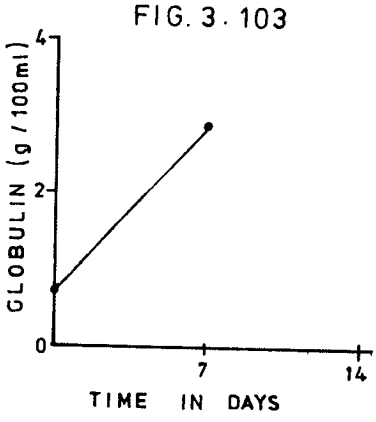


Table 3.2

Results Showing the Mean of the Parameters measured during the Experiment

Goat No.	Hb Conc. g/dl	PCV %	WBC x 10^3 /ul	RBC x 10^6 /ul	Total Protein g/100 ml	Albumin g/100 ml	Globulin g/100 ml	A/G Ratio	No. of Weeks of Experiment
1	14.30	32.20	16.02	19.50	6.80	4.33	2.46	2.23	14
2	11.98	23.71	13.12	17.69	5.54	4.04	1.51	6.09	8
3	14.16	30.10	20.34	19.78	6.00	4.35	1.67	3.95	8
4	13.12	23.89	11.35	18.49	6.56	4.86	1.70	4.58	8
5	13.41	25.89	8.47	16.08	6.69	4.90	1.79	3.98	8
6	12.27	24.20	14.63	15.93	7.18	3.94	3.24	1.97	2
7	11.87	35.03	8.90	16.86	6.20	4.71	1.49	6.80	2
8	10.73	25.50	16.13	14.99	7.29	4.03	3.26	1.30	2
9	8.50	23.00	11.75	13.48	5.70	3.21	2.49	1.28	1
10	9.85	22.25	11.73	13.09	6.87	5.00	1.87	4.14	1
				Mean Values for Healthy Goats (Kelly, 1984)					
	11.00	30.00	9.00	13.70	7.10	6.45	3.40	3.25	2.26
				Luangwa goats mean Values (Lovelace et al, 1988)					
	11.20	32.00	13.50	13.60	7.80	4.84	4.80	3.00	1.60

3.4.4 Albumin:Globulin (A/G) Ratio

The mean values of the A/G ratio of the ten kids ranged from 1.28-6.80. The first group had higher values than the second group. The mean values of the second group varied between 1.28-6.80 and in the first group the mean values ranged between 1.51-6.09. The lower results could be due to the rapid decline in the second group with the development of the disease, leading to increased production of serum immuno-globulins and the decline of serum albumin and total protein due to the loss of proteins and poor appetite. The second group were under nutritional stress due to early weaning.

3.5 Life Cycle of Eimeria in Zambian Goats

The appearance of different stages of the life cycle was studied using sections from the 10 goats (and earlier samples) and representative microphotographs are shown.

In Fig 3.5.1 a microgamete can be seen actually inside a macrogametocyte, with about six microgametes around the edge. One could suggest that fertilization is taking place. Many microgametes have been released from the adjacent microgametocytes and microgametes are seen scattered around. Zygotes are easily identified in Fig 3.5.2 with a prominent nucleus in the centre and plastic granules forming a wall around it. The size of zygotes is not uniform and the shape varies from oval to spherical.

The mature oocyst is seen in Fig 3.5.3 in the lumen of the intestine ready to be passed out together with faeces. Once outside, sporulation begins, and one is able to count

four sporocysts and in Fig 3.5.4 a sporozoite is clearly seen. Sporulation time varies from 4-48 hours depending on the season when the oocysts were deposited. On a hot day after an hour or two in sucrose during the floatation method, it was observed when counting that some of the oocysts had already sporulated. The walls were quite distinct. Unfortunately since we were using a light microscope, we were unable to locate the next stage of a sporozoite infecting an epithelial cell.

The size and location of the giant schizont which develops from the sporozoite was not always the same. Some occupied the serosa (Fig 3.5.5), others the lumen of the villi (Fig 3.5.6). In Fig 3.5.7 some mature merozoites can be seen inside the schizont bundled together like sticks, the wall though faint is still intact. This was observed at 40x magnification. Fig 3.5.8 shows mature merozoites and the wall is not visible at the 100x magnification. The nuclei in merozoites can be seen as deep purple spots centrally positioned. Due to lack of an electron microscope, we were unable to see the subsequent stage of a first generation merozoite penetrating the epithelial cells. Though one could suggest that a merozoite can be seen in Fig 4.5.3a (ii) which may be about to enter an epithelial cell.

The secondary schizonts (Fig 3.5.9a) are smaller and there is a space between the merozoites and the wall. The merozoites in Fig 3.5.9 b and c are larger and fewer compared to those in the giant schizont in Fig 3.5.8. It is possible to count the number of merozoites in Fig 3.5.9c and the nuclei are quite visible. Fig

3.5.10 indicates a second generation merozoite inside an epithelial cell which will form a progamont. Progamonts can be seen clearly in Fig 3.5.11a.

In Fig 3.5.11b, some of the progamonts have started to differentiate into macro- and microgametocytes. Figs 3.5.12 a and b show different developing stages of macrogametocytes, zygote with a nucleus and immature oocyst with a distinct wall around it. Figs 3.5.13 a and b show an unusual section from the small intestine with only microgametocytes present in the villi. Figs 3.5.14 a and b show different developing stages of microgametocytes.

Figs 3.5.15 a and b show a coccidial polyp with crypt-like epithelium showing synchronised division of coccidia progamonts and their host cells. This was similar to that reported by Gregory (1987).

3.6 Effects of Amprolium on Oocysts

The histological results from goats treated with amprolium one day before sacrifice showed no difference from the untreated goats except in wet smears showng oocysts. Wet smears made from intestine of amprolium treated goats showed oocysts disintegrating. In Figs 3.6a and 3.6b the oocysts have no walls. Fig 3.6a shows two different sets of oocysts some without the wall, and in others the contents are still seen but the wall has been disrupted in various places and the micropyle cap is disjoined.

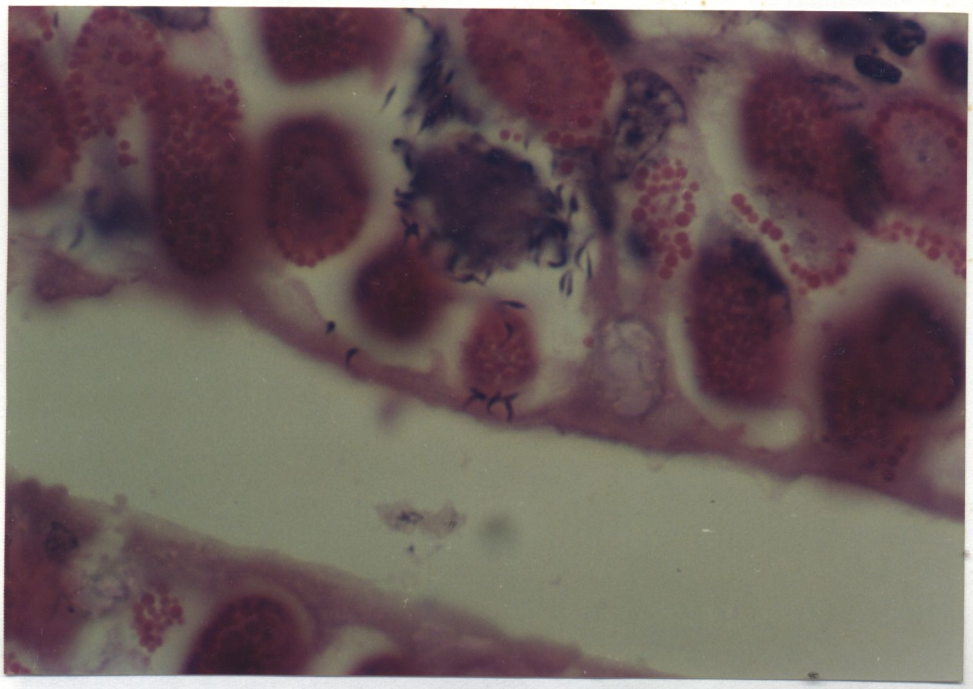


Fig 3.5.1 Microgamete darkly stained inside a macrogamete. This may indicate fertilisation. Six microgametocytes are seen on the peripheral of the macrogametocvte. (x 40 mg)

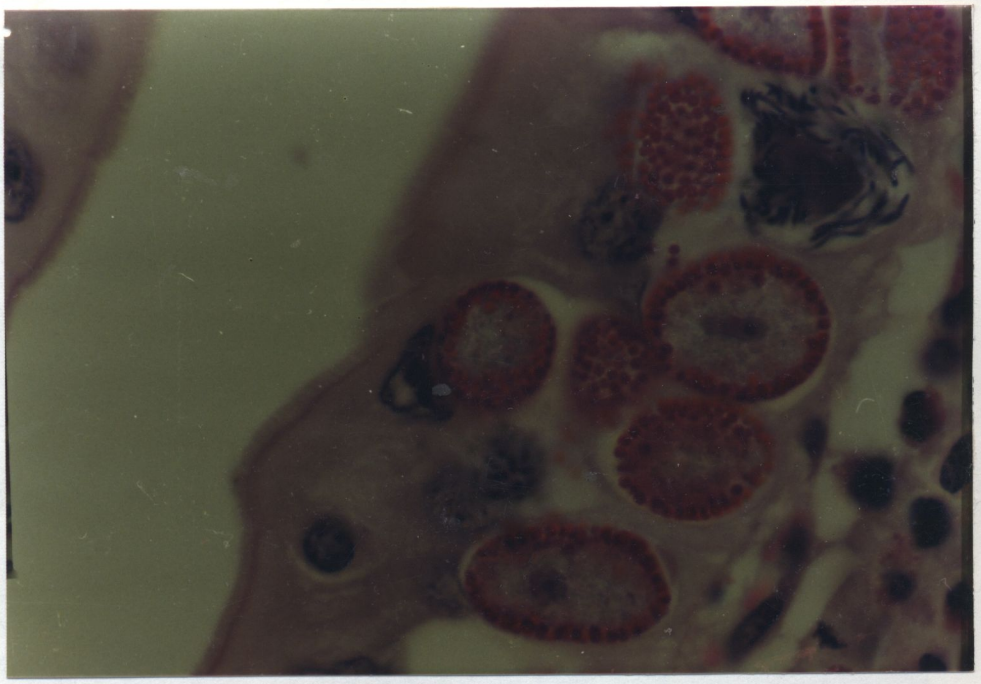


Fig 3.5.2. Zygotes with purple stained nuclei in the centre. The walls are forming around the zygotes, with pink stained plastic granules. (x 40 mg)

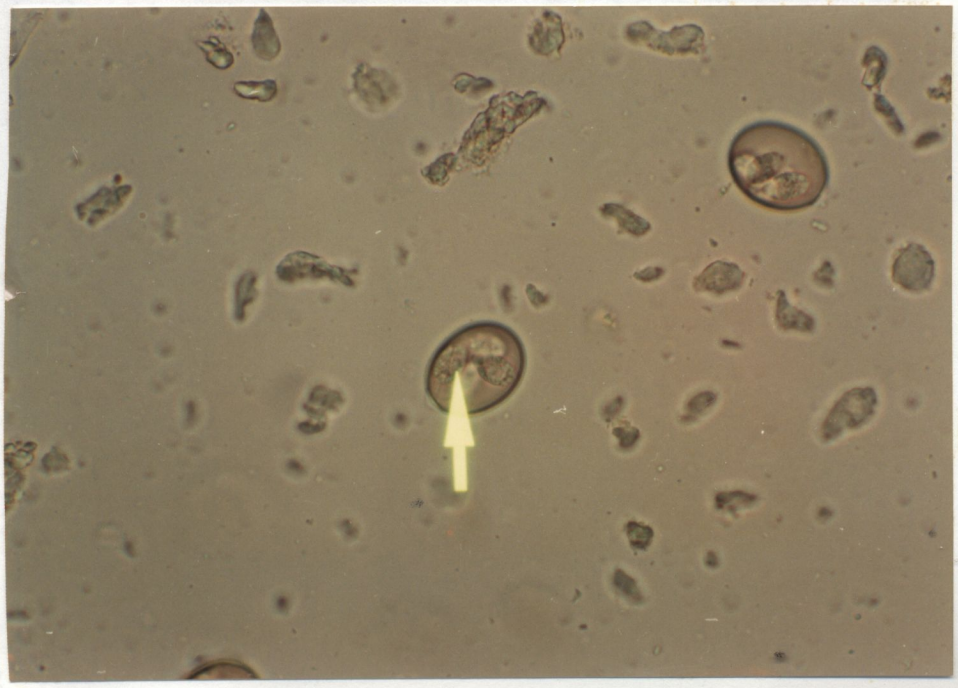


Fig 3.5.3 Mature oocyst with four sporocysts. (x 40 mg)

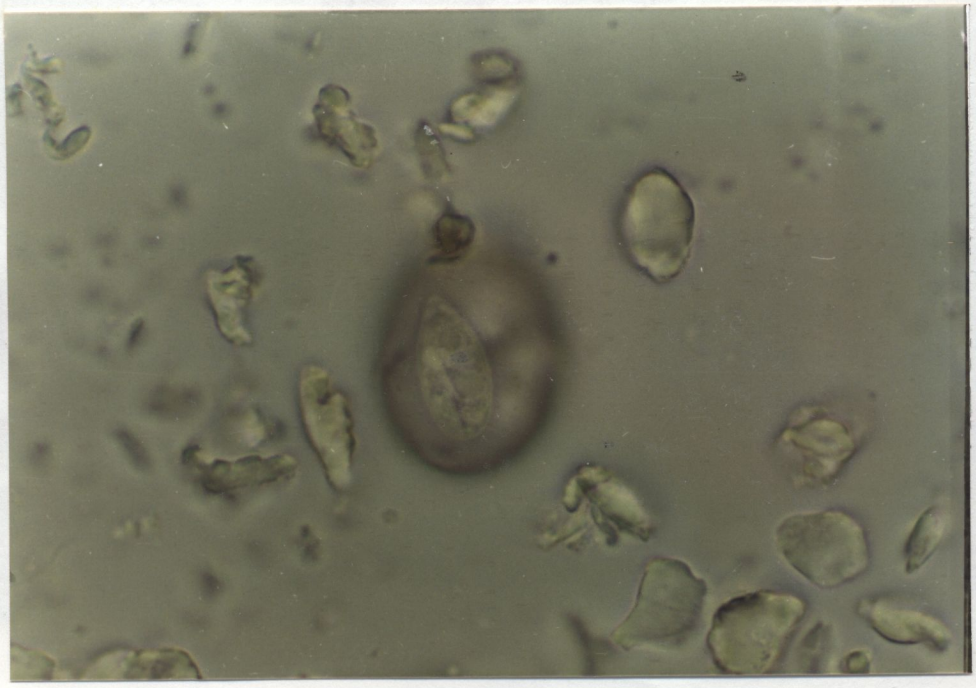


Fig 3.5.4 Mature oocyst showing a sporocyst with two sporozoites. (x 100 mg)

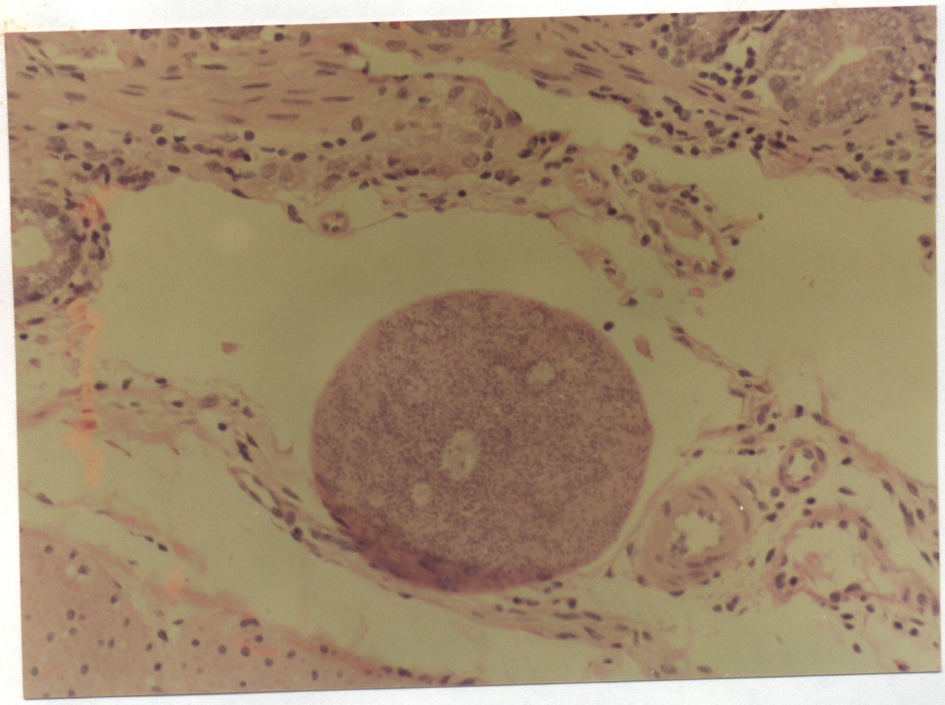


Fig 3.5.5. Giant schizont seen in the serosa. (x 20 mg)

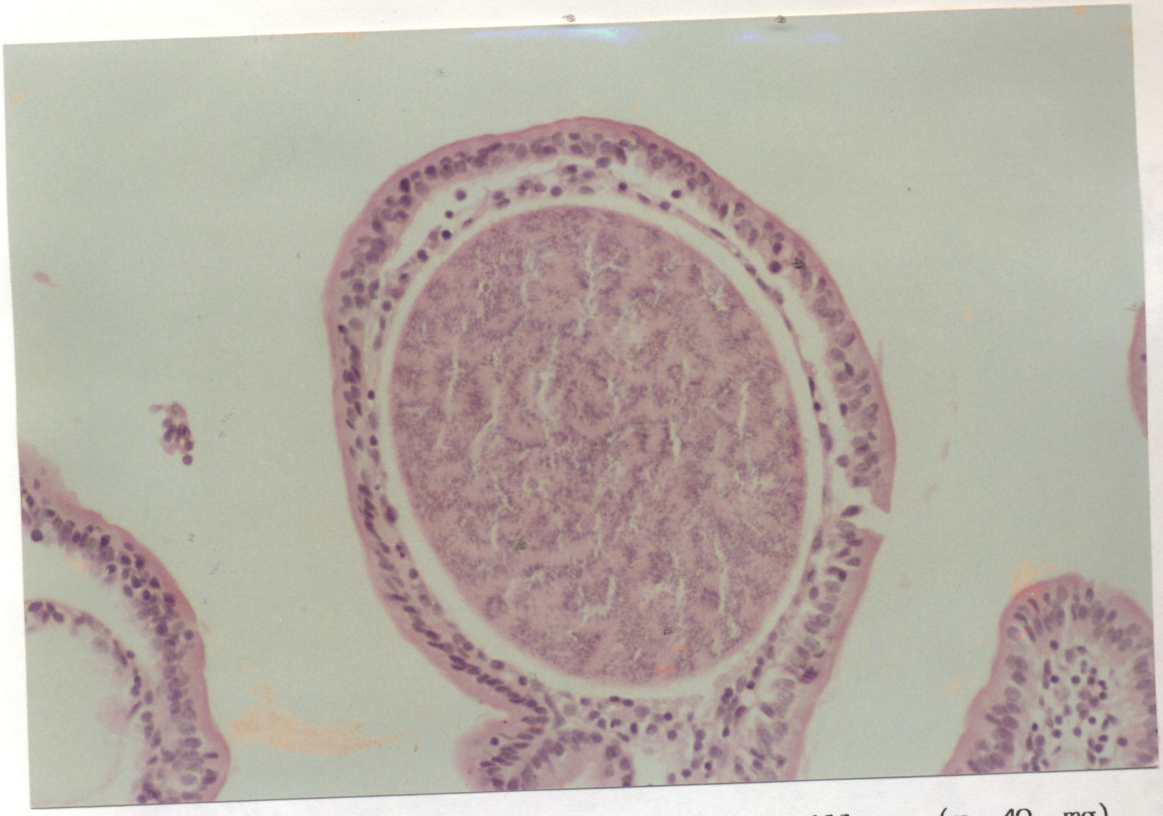


Fig 3.5.6 Giant schizont in the lumen of a villus. (x 40 mg)

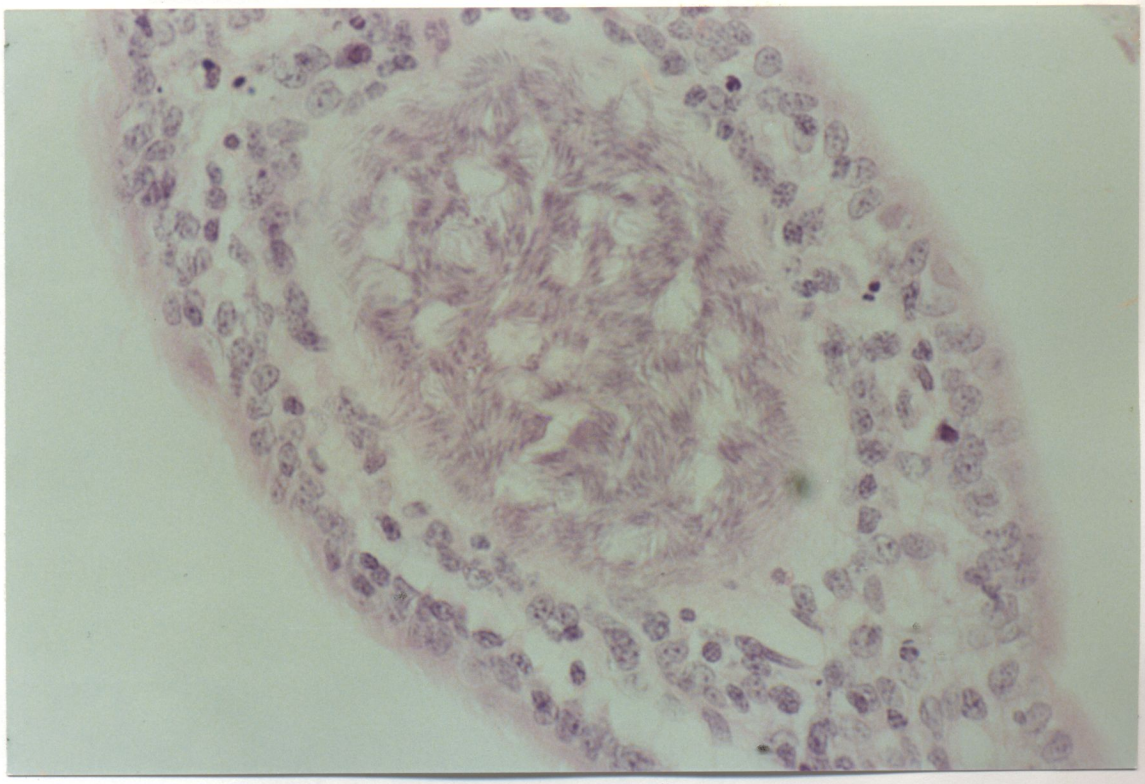


Fig 3.5.7 Mature giant schizont with merozoites arranged like bundles of sticks. (x 40 mg)

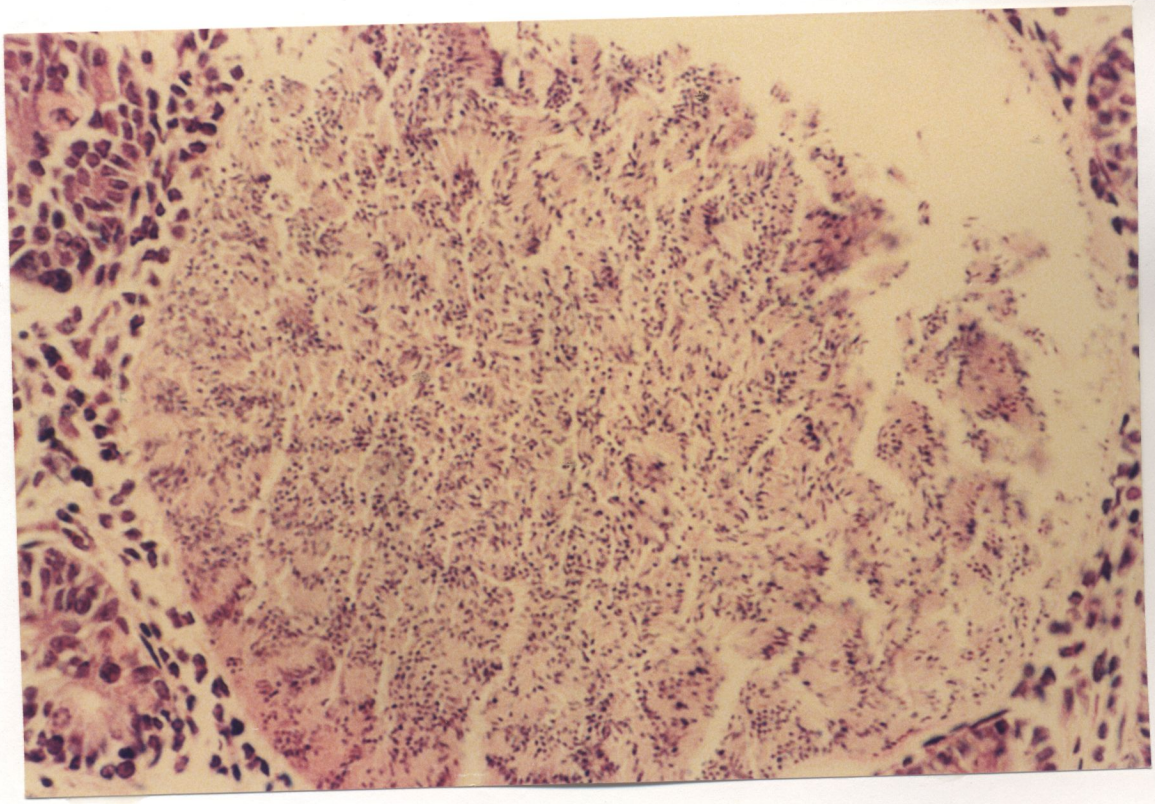


Fig 3.5.8 Mature schizont showing merozoites with black stained nuclei. (x 100 mg)

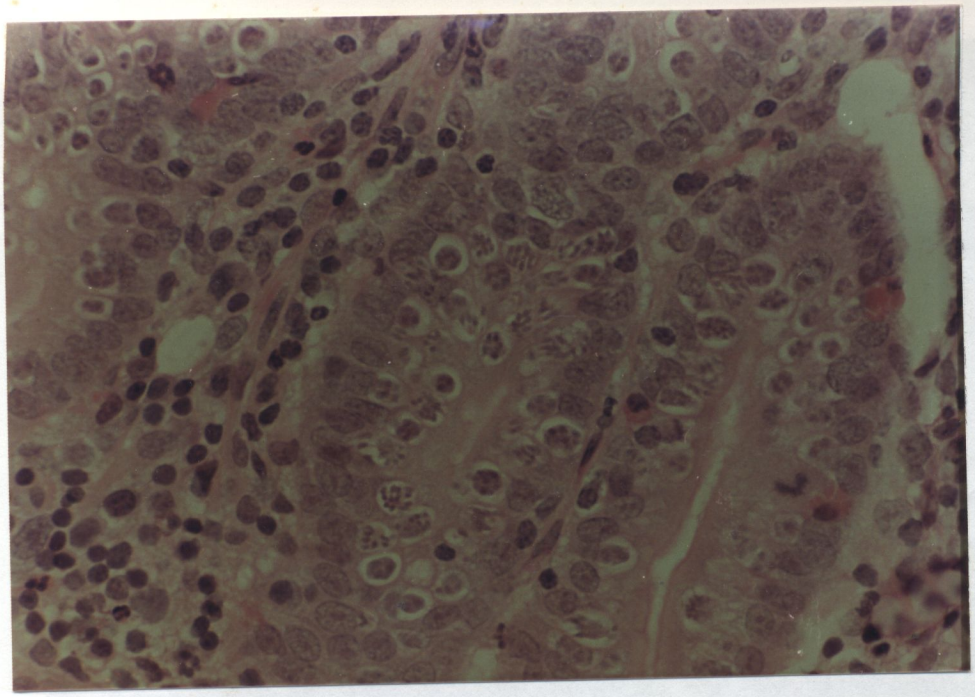


Fig 3.5.9a Small schizonts with mature merozoites note the size of the schizonts and the fewer number of merozoites. (x 20 mg)

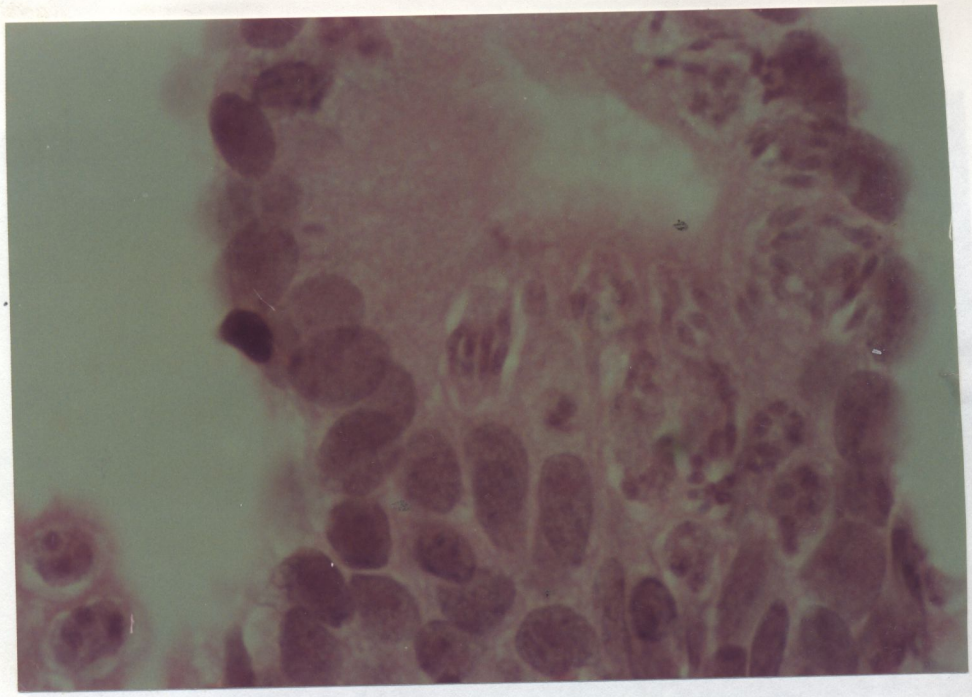


Fig 3.5.9b Small schizonts. (x 40 mg)

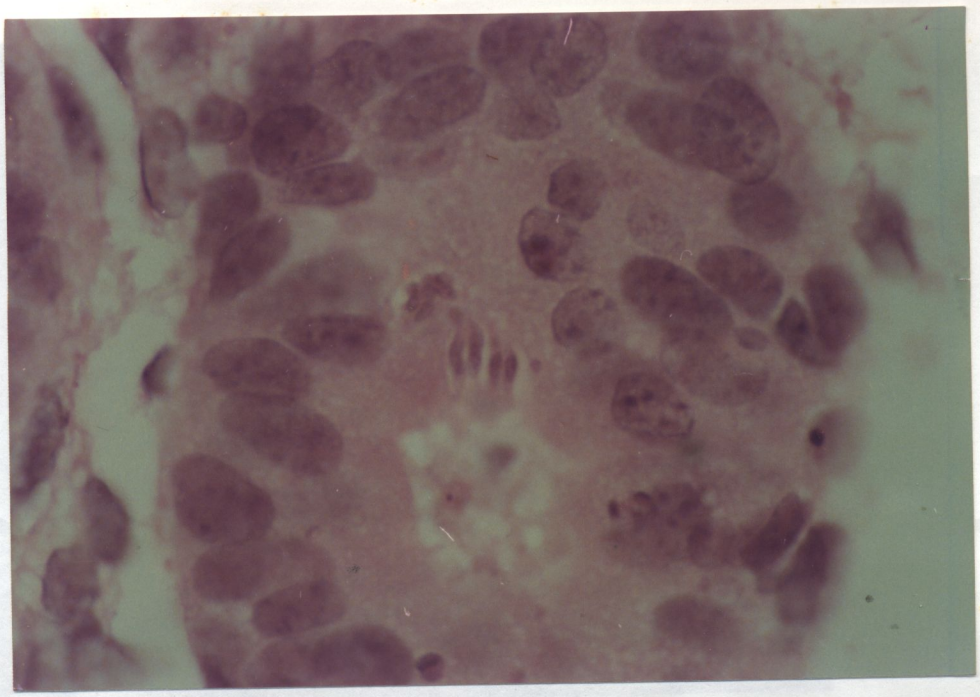


Fig 3.5.9c Four merozoites seen in a small schizont. (x 100 mg)

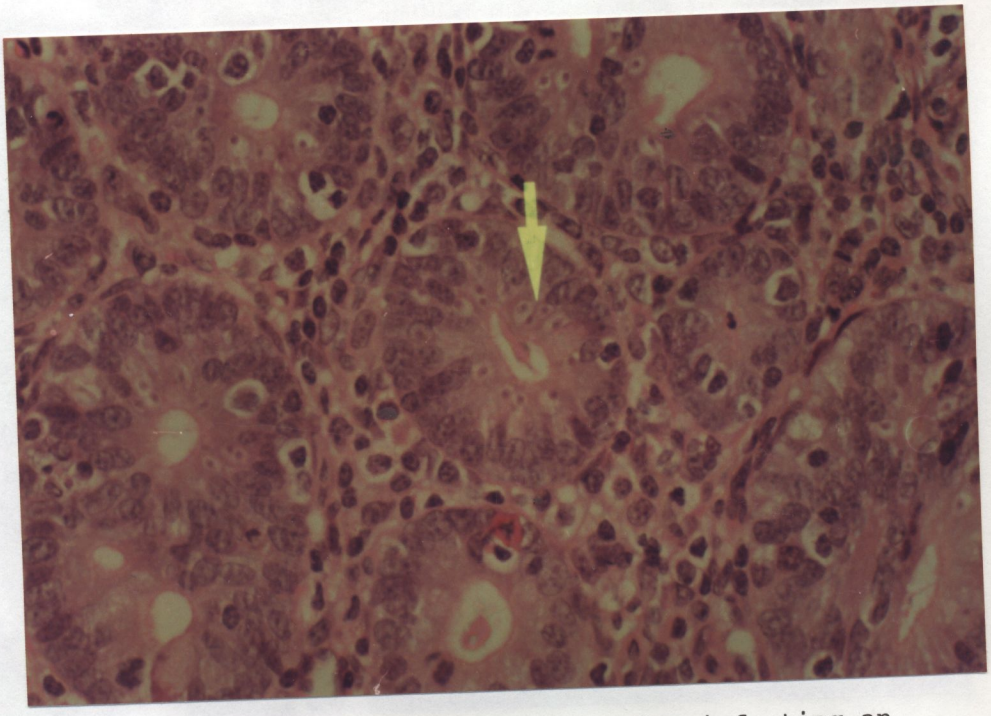


Fig 3.5.10 Arrow indicating a merozoite infecting an epithelial cell. (x 20 mg)

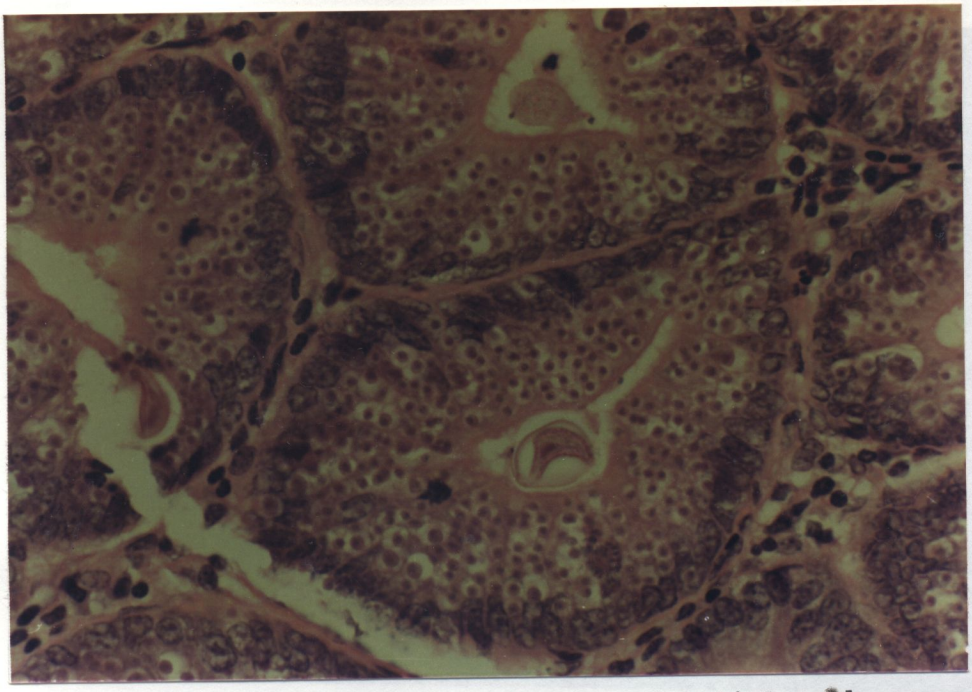


Fig 3.5.11a Progamonts seen in the epithelial cells and cysts in the lumen of the villi. (x 40 mg)

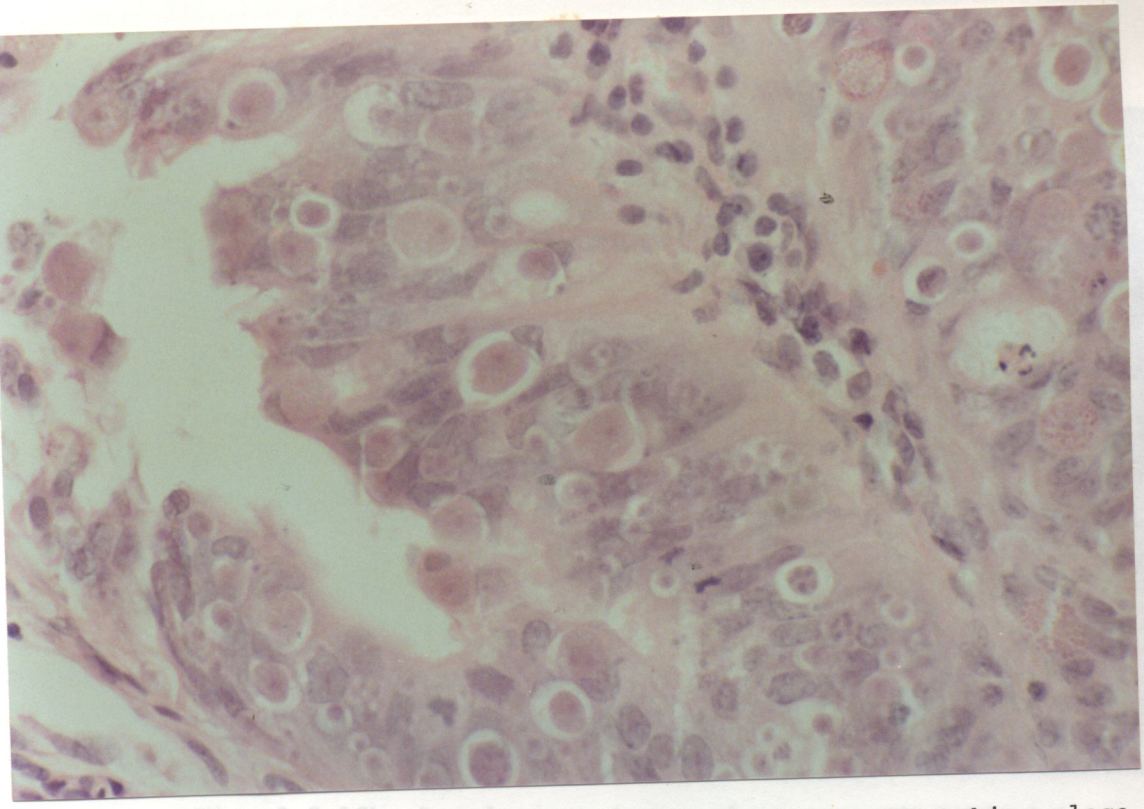


Fig 3.5.11b Differentiation of progamonts taking place (x 40 mg)

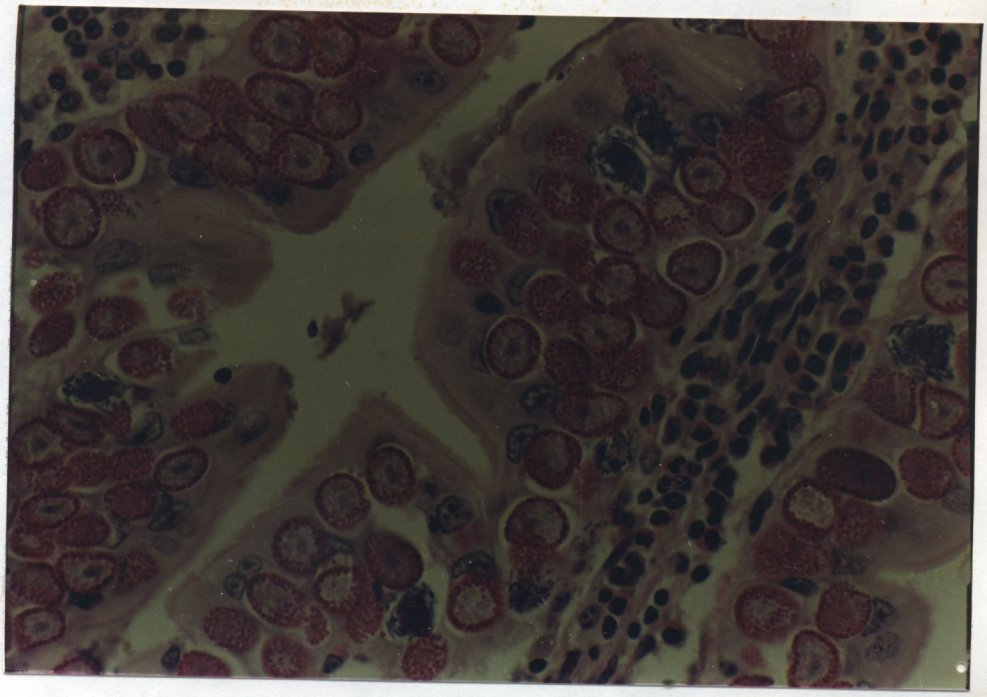


Fig 3.5.12a Developing stages of macrogametocytes to zygotes with walls forming from plastic granules. (x 20 mg)

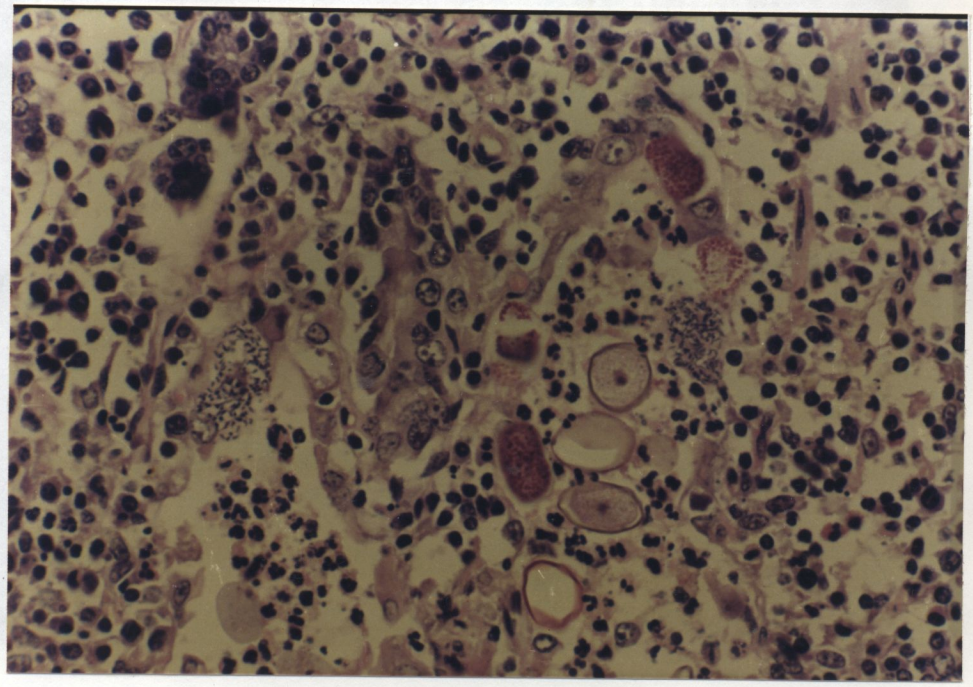


Fig 3.5.12b Developing stages of macrogametocytes to zygote and immature oocysts with distinct walls around them. (x 20 mg)

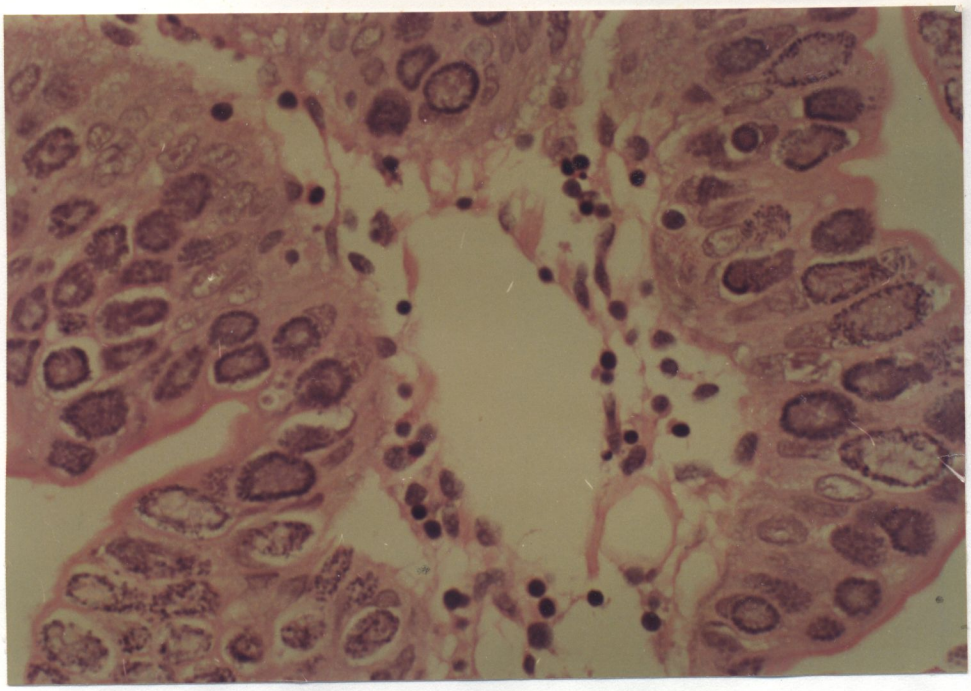


Fig 3.5.13a Villus showing microgametocytes only in different stages of development. (x 20 mg)

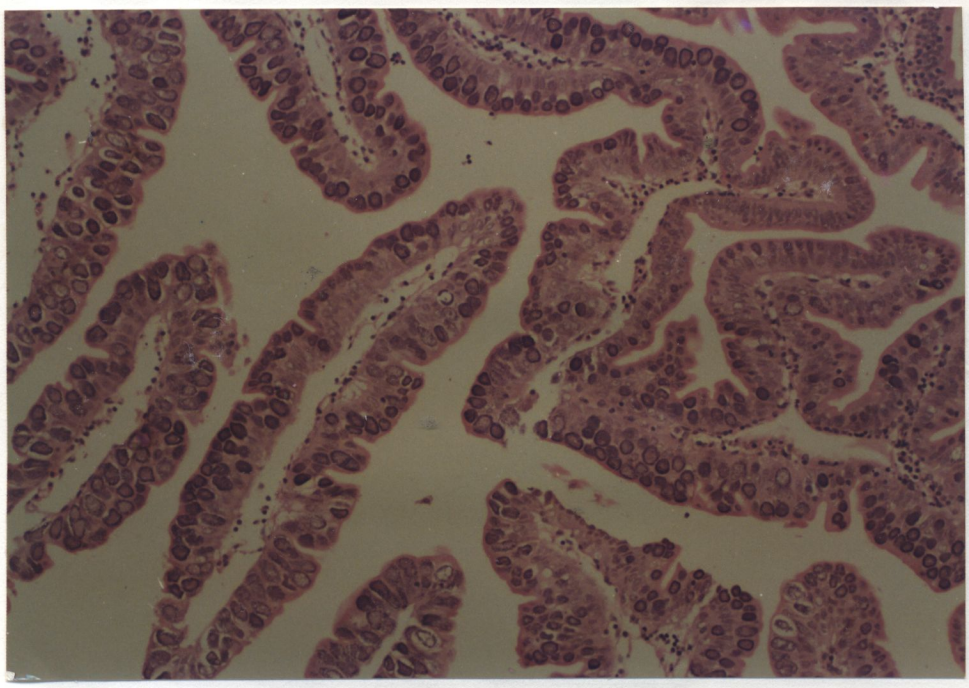


Fig 3.5.13b Villi showing microgametocytes only. (x 10 mg)

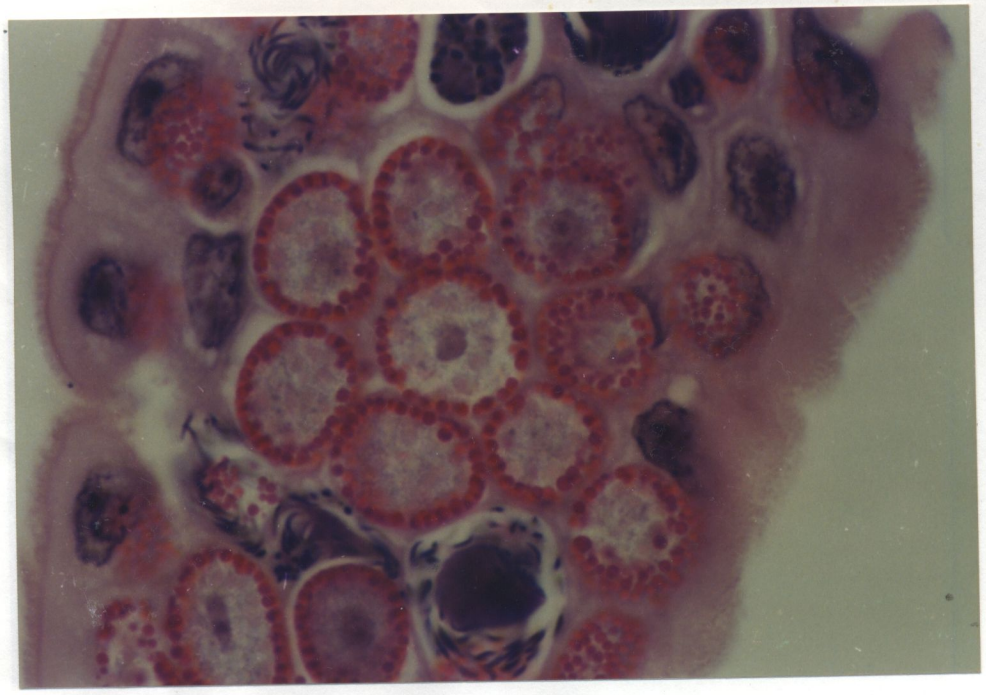


Fig 3.5.14a Developing stages of microgametocytes darkly stained with a purple nuclei. (x 40 mg)

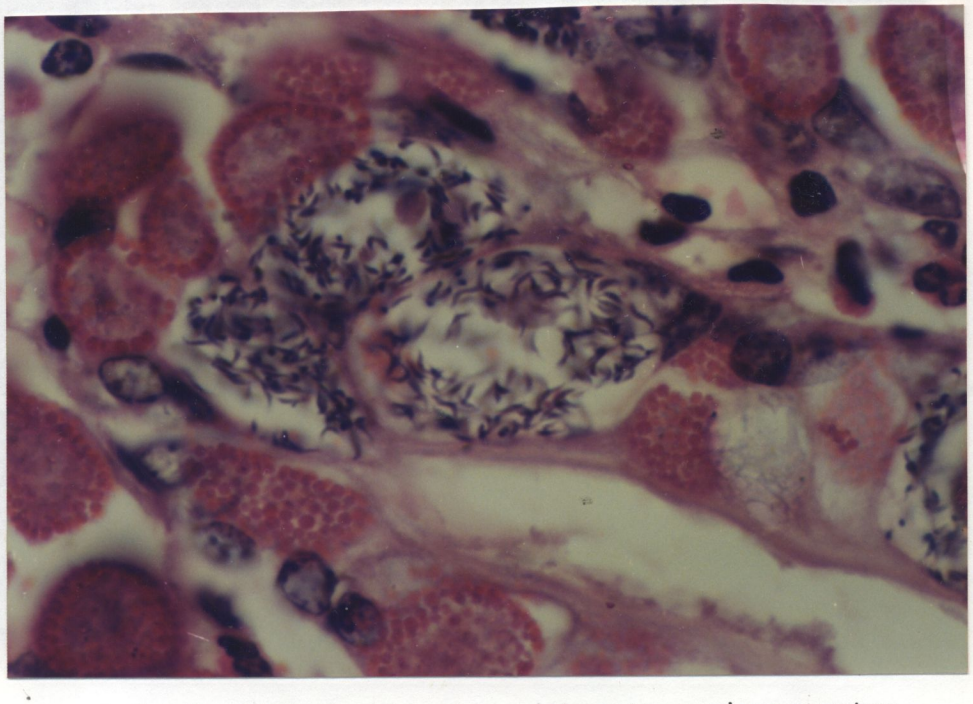


Fig 3.5.14b Microgametocytes with mature microgametes. (x 40 mg)

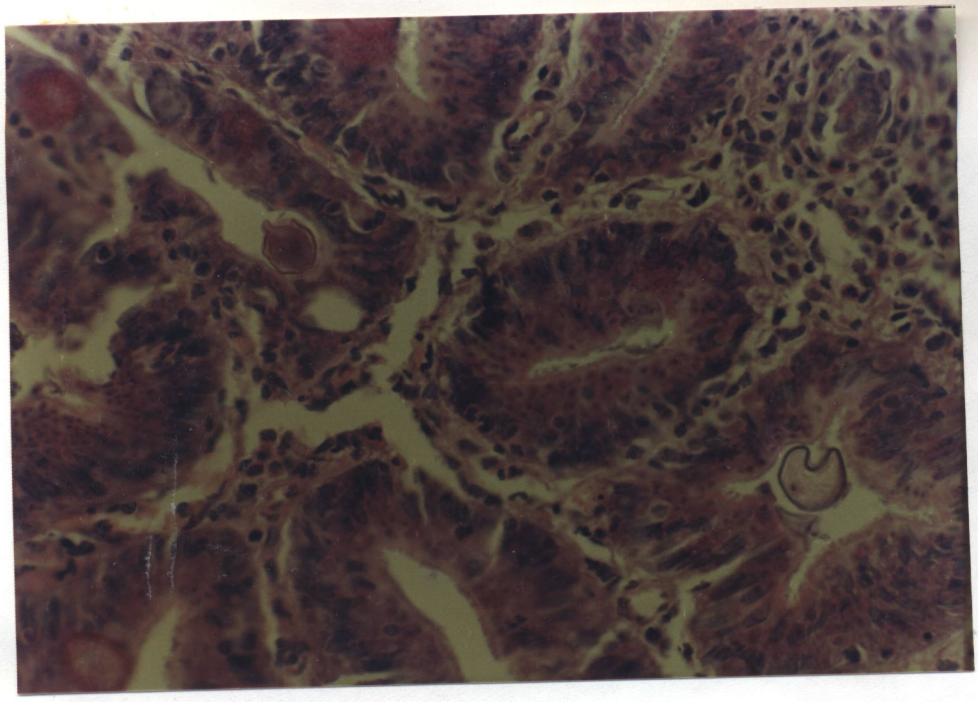


Fig 3.5.15a Synchronised division of coccidia with their host cells. (x 20 mg)

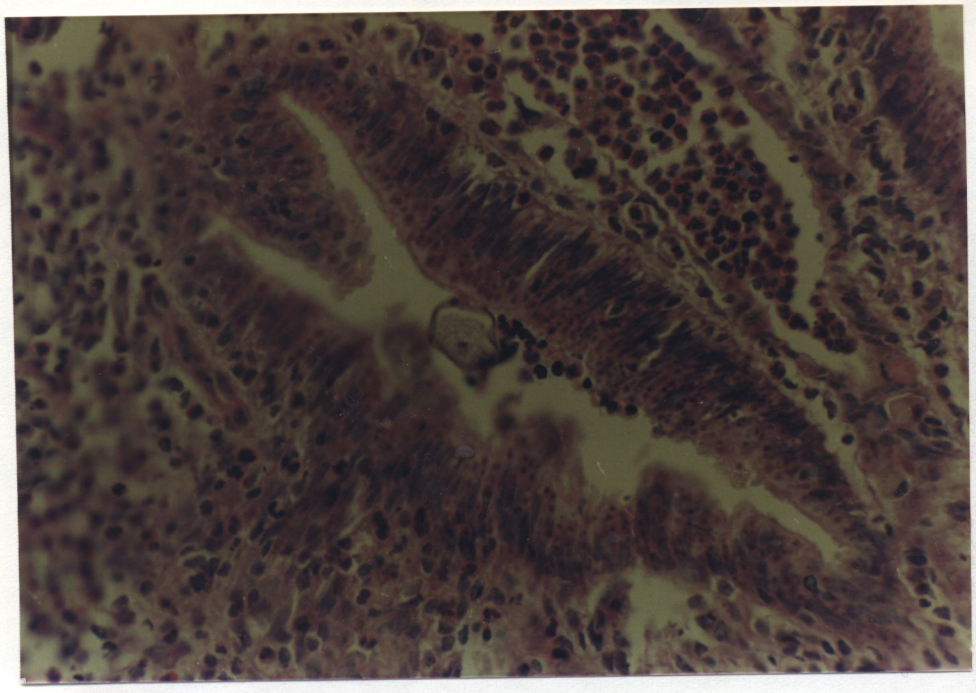


Fig 3.5.15b Synchronised division of coccidia with their host cells. (x 40 mg)

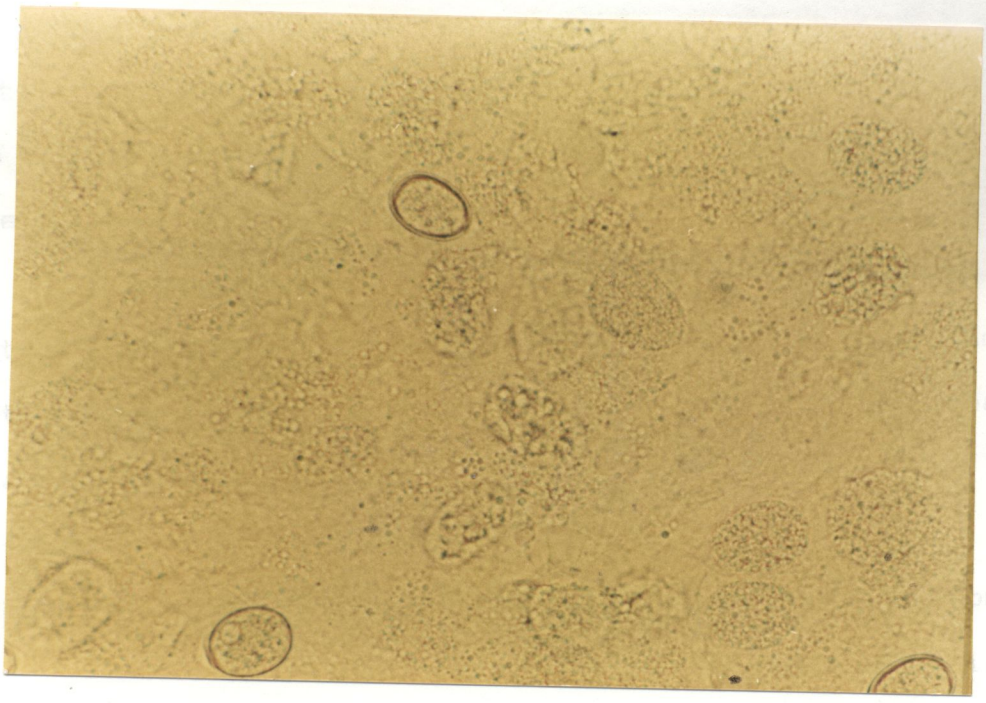


Fig 3.6a Oocysts without visible wall. (x 40 mg)

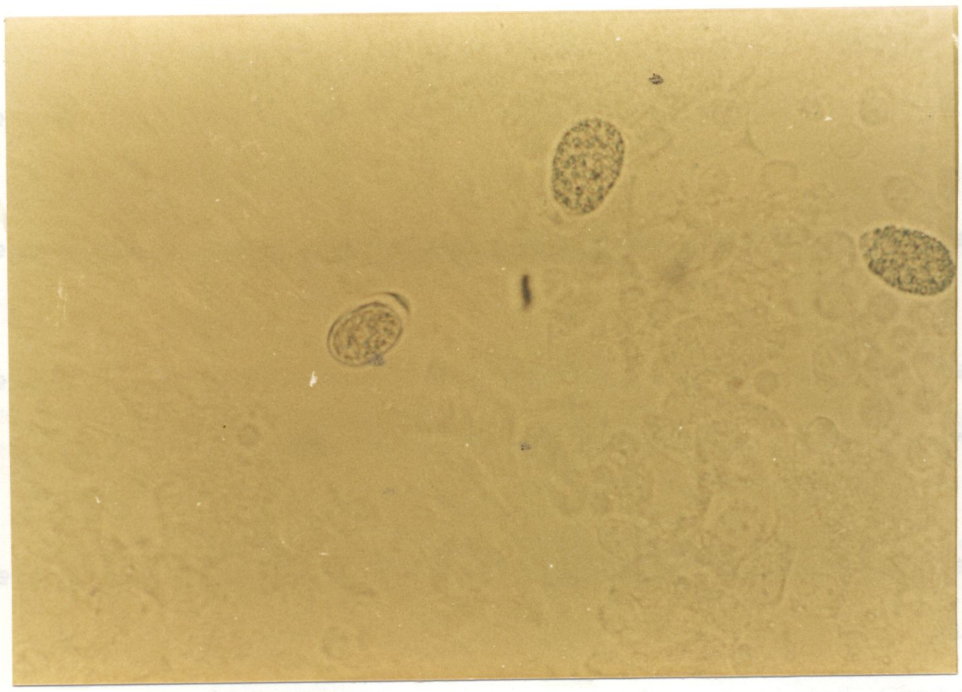


Fig 3.6b Disruption of oocysts walls, microphyle cap disjoined and oocyst contents seen without a wall around them. (x 40 mg)

3.7 Intestinal Lesions Observed through the Stereo Microscope

A stereo microscope was used during the preliminary investigations and the intestinal lesions observed were divided into five categories. The shape and state of the villi was also recorded. The general appearance of the villi were that they were smooth and flat due to the damaged epithelial tissue, or rough and protruding. Some were tinted with blood, others appeared white or cream coloured.

Different types of Lesions.

Category A: Haemorrhagic Lesions - These were lesions with a lot of blood on the surface of the epithelial tissue. During post-mortem, a hand lens was used to identify them. However, during histopathological examinations these did not show significant infection, except a few gametocytes, with a stream of red blood cells and a few white blood cells, see Fig 3.7.1.

Category B: Foldings - These appeared as contour ridges on the surface, Fig 3.7.2a.

Category C: Rosettes - The villi protruded from the surface in the form of a rose, Fig 3.7.2 b.

Category D: Nodules - A small round swelling on the infected tissue which appears white or stained with blood, Fig 3.7.3.

Category E: White Spots - These were visible as small clear white dots through the serosa on the outside of the intestine. The majority of these contained the small schizonts and were found mainly in the jejunum and ileum.

In most of the lesions except for haemorrhagic lesion, it was observed that coccidia of different species was present, as identified from oocysts present on the tissue studied.

3.8 Degenerating Schizonts

Cellular response was very significant in all the goats especially in reaction to the giant schizonts. There were different forms of degeneration as seen in Figs 3.8.1; 3.8.2; 3.8.3; 3.8.4. Sometimes the contents were seen clumped together as in Fig 3.8.1. Red or white patches appeared inside the giant schizont, Fig 3.8.2 and 3.8.3 respectively. Cilia-like structures could be seen on the peripheral edge of the giant schizont (see Fig 3.8.2). In other cases a mass of red spots could be seen, (Fig 3.8.4). White blood cells surrounded almost all of the degenerating schizonts. Some of the WBC were seen inside the schizonts as in Fig 3.8.5a, and a degenerating schizont with no wall but with many WBC inside it is shown in Figs 3.8.5 b and c. There was no degeneration observed in any of the small schizonts, or in the gamonts.

3.9 Results of the Survey of Three Zambian Farms

After analysing the faecal samples from the two commercial farms and from a village, it was observed that all of the goats studied were infected with coccidia. The levels were higher during the rainy season than in the dry season (Tables 3.9.1; 3.9.2; and 3.9.3). Fig 3.9.4 shows the mean oocyst count on the three farms.

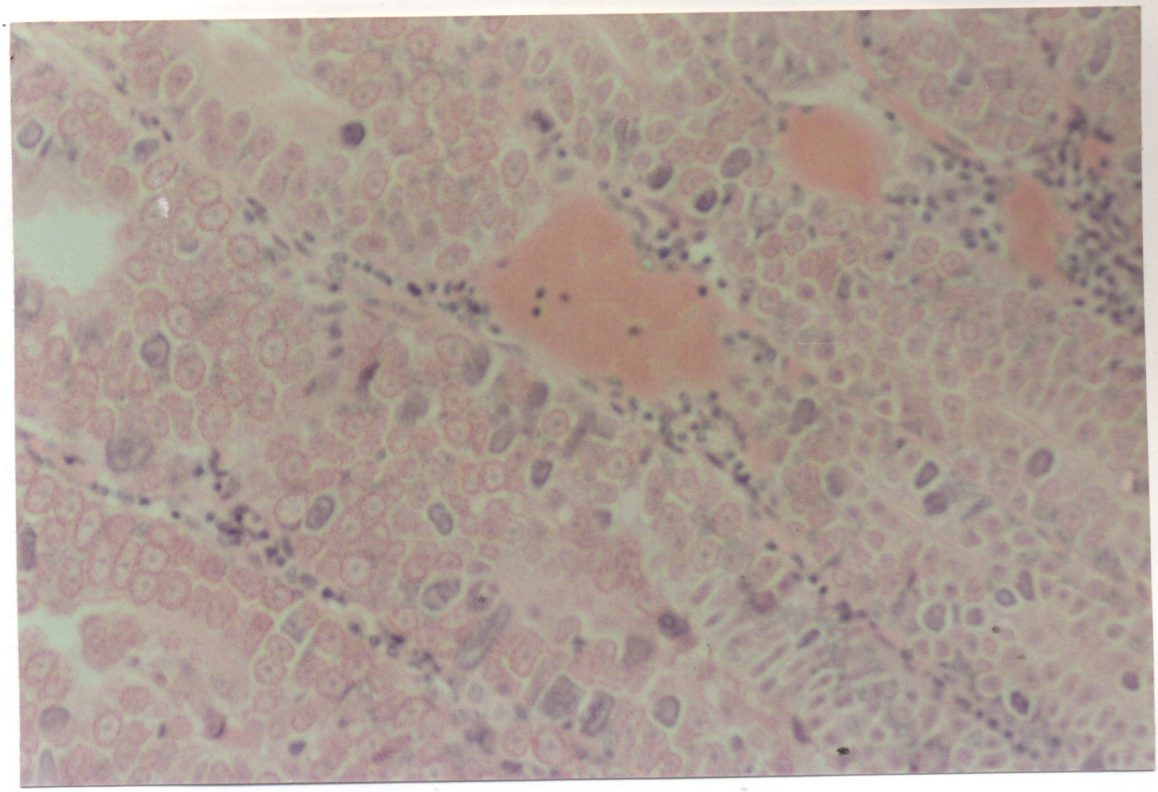


Fig 3.7.1 Haemorrhagic lesions showing many RBC and few WBC, surround the lesions are both macro and microgametocytes. (x 20 mg)

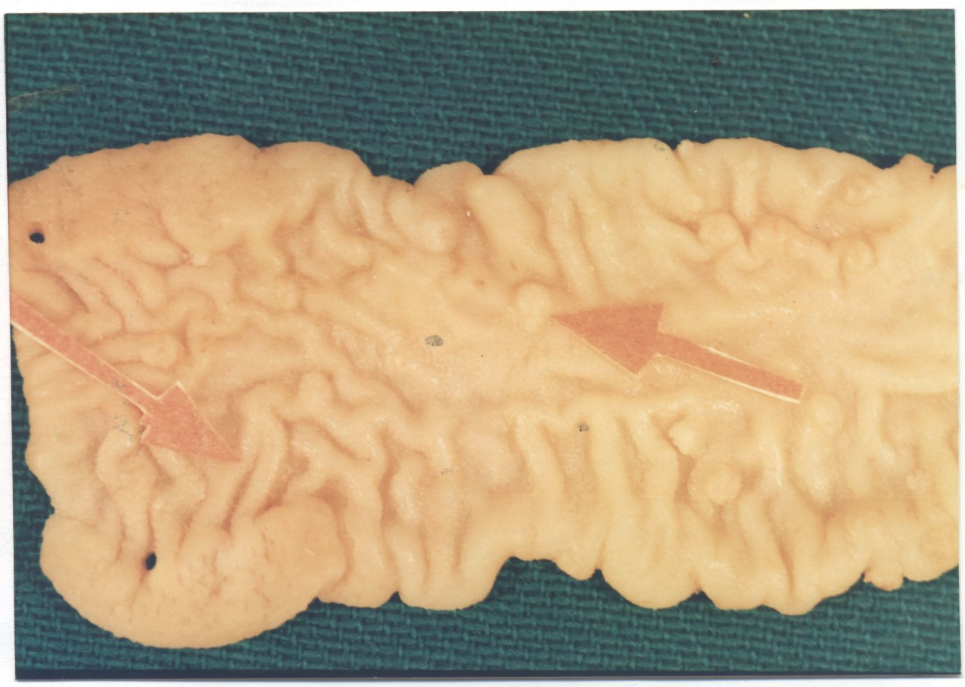


Fig 3.7.2a Folding type of lesions and a few immature rosettes

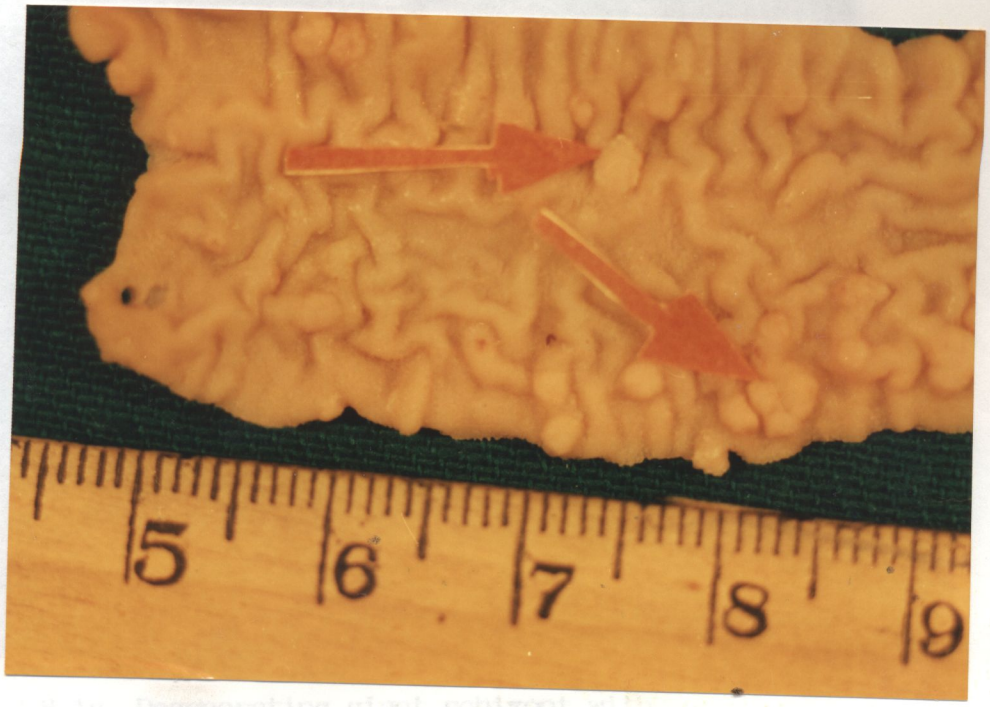


Fig 3.7.2b Arrow showing the small intestinal lesion (rosettes)

Fig 3.7.3 Nodule type of lesion

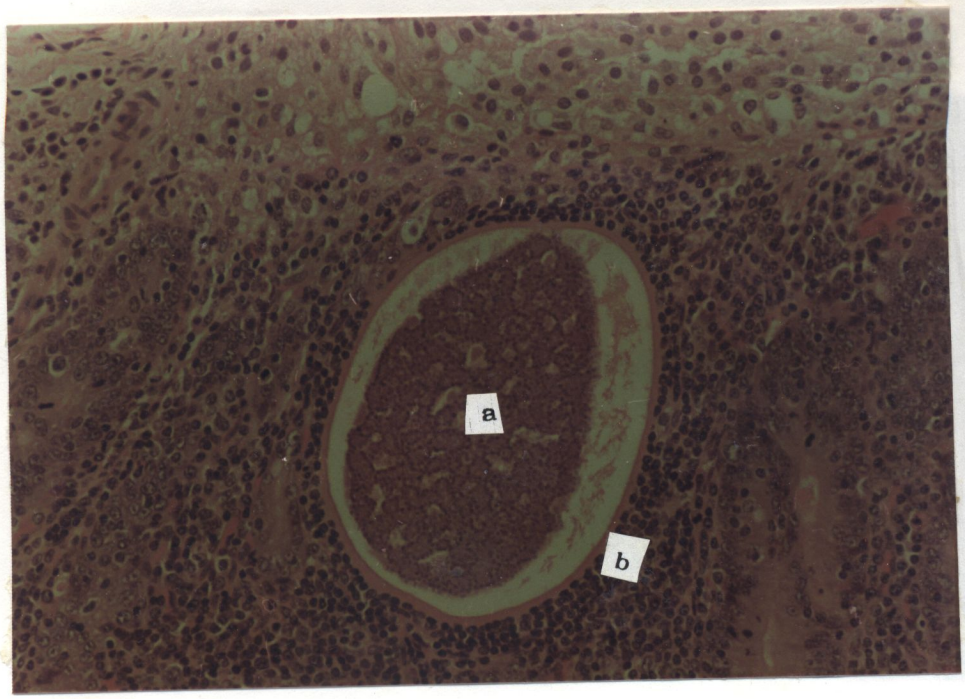


Fig 3.8.1a Degenerating giant schizont with contents clumped together.

b WBC surround the giant schizont. (x 40 mg)

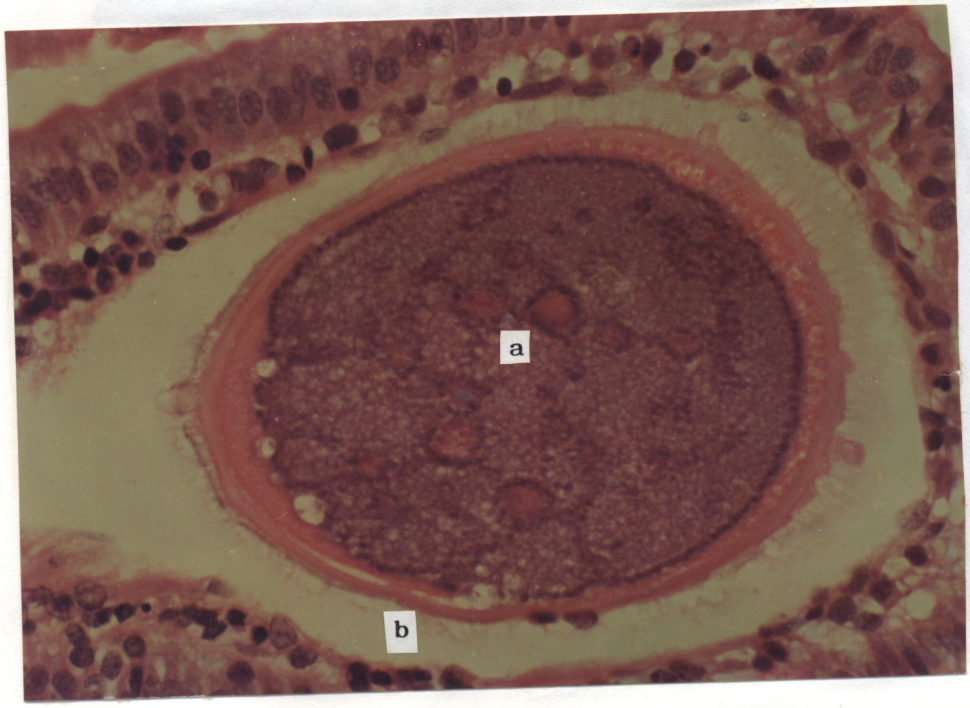


Fig 3.8.2a Red patches inside a degenerating giant schizont.

b Cilia like structures on the peripheral edge of the giant schizont. (x 100 mg)

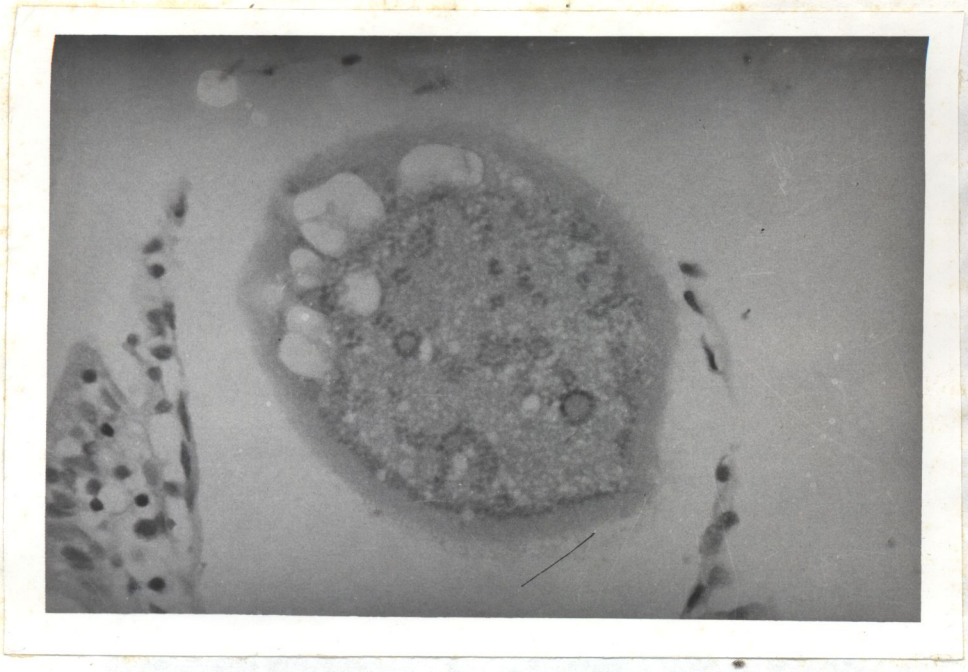


Fig 3.8.3 White patches on the degenerating giant schizont. (x 40 mg)

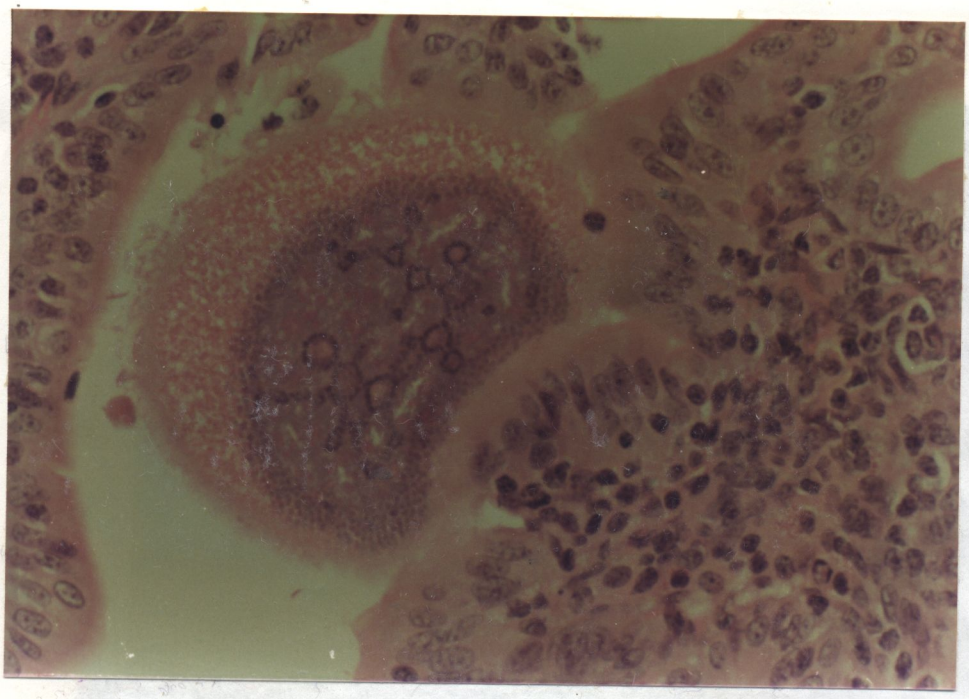


Fig 3.8.4 Red spots on the edge of the degenerating giant schizont. (x 20 mg)

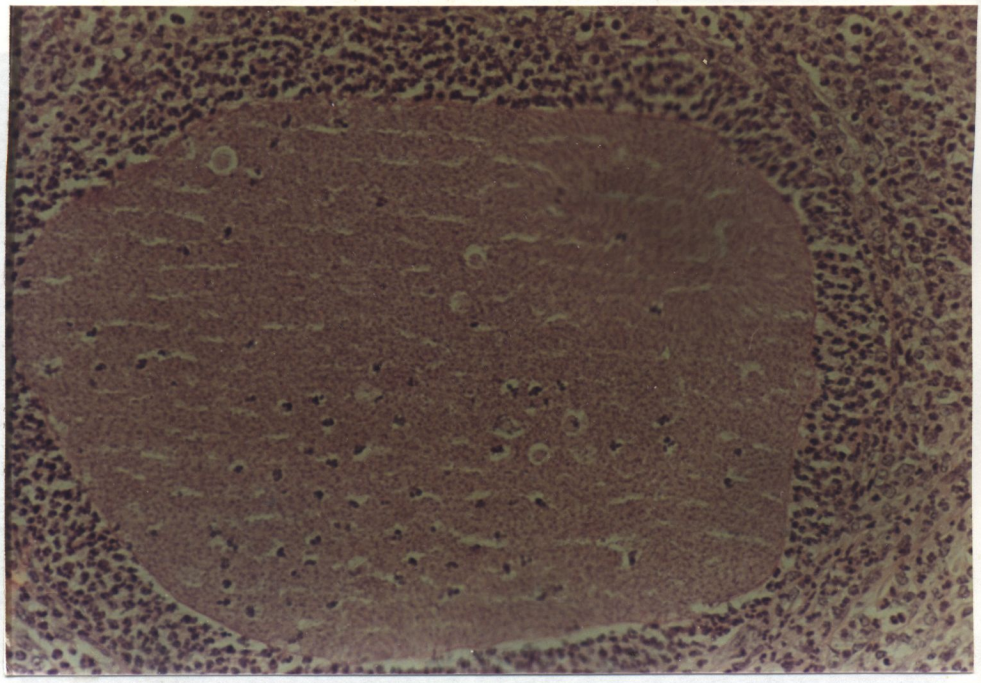


Fig 3.8.5a WBC inside and around degenerating giant schizont (x 100 mg)

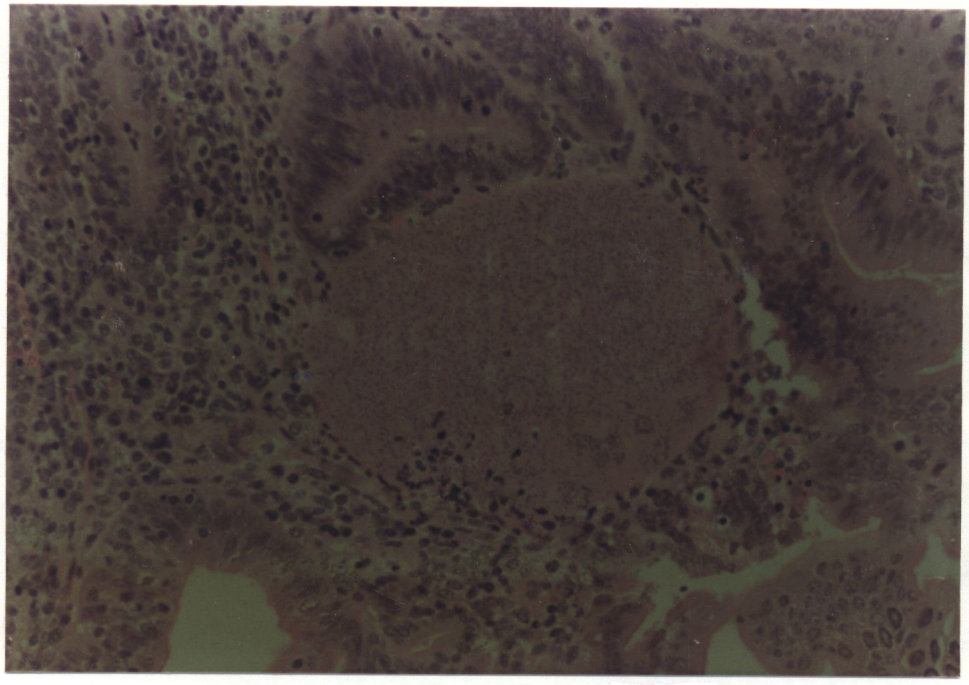


Fig 3.8.5b WBC inside the degenerating giant schizont and the wall of the schizont is not distinct. (x 40 mg)

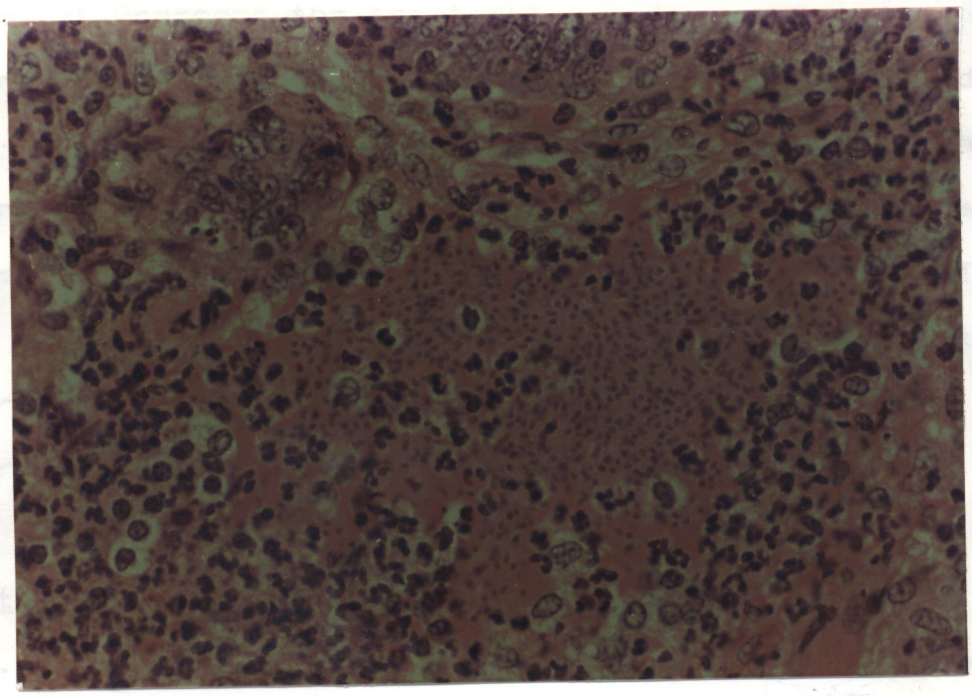


Fig 3.8.5c WBC inside and around degenerating giant schizont. (x 40 mg)

On the two commercial farms goats were being treated with Amprolium, at Grasmere the treatment was done regularly and at Dan's Farm, as need arose, that is when the animals showed any signs of the disease. From our results the treatment appeared however not to eradicate the infection. In the village samples the oocyst levels were higher than the other two farms. This could be due to the fact that these animals were not treated at all. Normally in the village when an animal fell sick it is slaughtered.

3.10 Statistics Analysis - First Set

In Fig 3.104 there was no significant difference in the means of temperature during weeks 0,1,2,3,4,5,8 and that of 6,7; 4,5,0 and that of weeks 6 and 7. There was a significant difference between weeks 1,2,3,8 and weeks 6 and 7.

There were no significant differences on the means of PCV (Fig 3.110), between weeks 1-7; 2-6; 1,2,3,4 and 7; 0,1 and 7. The difference was evident between means of week 0, and of weeks 5 and 6 (PCV 0.05). There was a decline during the 8th week.

The means of haemoglobin indicated significant statistical differences during weeks 1 and 7; 0,2,4 and that of 7; 1 and that of 2,3,4,5,6 and 8. The highest haemoglobin concentration was during week 1 and lowest during week 7. There were no significant differences in the rest of haemoglobin means. Serum albumin (fig 3.112) results showed a significant difference between means of weeks 0 and that of 1,3,4,5; 0 and that of 2; 7 and that of 1,3,4,5; $P< 0.05$. The highest concentration peak is evident

during week 3. There were no significant differences in the rest of the serum albumin means

In Fig 3.113 indicates the means of serum globulin. There was no significant differences in the means during weeks 1-6; 1,2,4,5,6,7 and 8; 0,7 and 8 P> 0.05. Globulin was significantly different during the zero and third week. Globulin concentration was higher during zero week and lower during the third week.

In Fig 3.114 there were no significant differences in the means of A/G ratio during weeks 0,1,2,4,5,7 and that of 8; 3,6 and that of 1,2,4,5,7 and 8. There was significant differences during weeks 0 and that of 3 and 6.

The ANOVA did not show any significant variations on the means of weight, oocysts, WBC, RBC, and Total Protein P> 0.050. (Figs 3.105, 3.106, 3.107, 3.108, 3.112). The data for the second group were too few for meaningful statistically analysis.

The statistical analysis carried out was useful for confirmation of our observations. The temperature measurement indicated statistical significant differences between weeks 6,7 and weeks 1,2,3 and 8. It was during week six that symptoms of coccidiosis were seen. In the last week all the animals had a drop in temperature just before death, hence the means were significantly declined during the 8th week. Although the differences were not statistically significant in oocysts it is interesting to note that the mean during the last week of the study was numerically higher than any other period during the course of the study.

Also from the statistical analysis, one can suggest that other probable symptoms include an increase in PCV, Hb, total protein and albumin and a A/G Ratio and globulin especially during the last wo weeks before the animals were sacrificed. This could be due to the loss of water when the animals had diarrhoea and hence creating haemo-concentration.

Table 3.3

GOAT 1

Date of Birth	Sex	Date of Weaning	Date of Isolation	Age Weeks	Date of Examination	Plotted Days	Weight Kg	Temperature 'C	Oocyst Count $00/g \times 10^3$	Clinical Observations
12/07/87	M	25/10/87	17/10/87	12	09/10/87		8.50	39.40	$00>10^6$	Healthy, active and eating well
				13	16/10/87		9.0	39.4	$00>10^6$	" " "
				14	26/10/87		9.50	39.5	20008	Same as above
				15	06/11/87		10.00	39.9	9958	"
				16	13/11/87		12.00	39.6	8601	"
				17	20/11/87		11.00	39.0	20794	"
				18	25/11/87		11.00	38.4	80808	"
				20	02/12/87		10.00	37.8	30910	"
				21	09/12/87		11.00	39.4	27509	"
				22	16/12/87	0	11.42	39.2	13305	"
				23	23/12/87	7	11.85	39.7	1466	"
				24	03/01/88	15	11.60	39.5	32646	Nasal discharge, active and eating well.
				25	08/01/88	23	10.50	39.3	62466	Active, healthy and eating well.
					11/01/88	26	11.00	39.3	136232	"
				28	01/02/88	47	12.00	39.6	14118	Looked weak and ill.
				29	08/02/88	54	11.00	43.8	12791	Bloody water stool.
					09/02/88	55	10.00	39.6	11212	Not eating, dysentry, mucus and bloody stool.
			Sacrificed		10/02/88	56	10.00	37.5	39512	

POST MORTEM OBSERVATIONS

Duodenum: Foldings and white spots through the entire length, few rosettes tinted with blood.
Jejunum: Many white spots and haemorrhagic lesions, few foldings.
Ileum: Mainly foldings tinted with blood and white spots.

HISTOPATHOLOGICAL OBSERVATIONS

Foldings (B): The entire tissue was covered with white blood cells, few degenerating giant schizonts, no gamonts or small schizonts.
Rosettes (C): Few progamonts and developing gamonts but many white blood cells. Lots of red blood cells in haemorrhagic lesions.
Nodules (D): Few giant schizonts and developing gamonts. In some areas only microgametocytes. Many white blood cells.
White Spots (E): Villi full of white blood cells, degenerating giant schizonts surrounded by the white blood cells. Very few developing gamonts.

GOAT 1

Date of Examination	Plotted Days	Hb g/dl	PCV %	WBC x 10 ^{3} ul	RBC x 10 ^{6} ul	Total Protein g/100 ml	Albumin g/100 ml	Globulin g/100 ml	A/G Ratio
28/10/87		14.60	44	13.1	13.17	8.00	4.50	1.50	3.1
09/11/87		15.00	42	13.15	21.7	6.97	3.01	3.96	1.00
20/11/87		13.80	38	9.35	24.51	8.11	5.30	3.01	1.84
24/11/87		12.00	38	15.85	13.7	8.00	4.50	3.50	1.14
01/12/87		15.50	35	21.00	17.40	7.00	4.20	2.71	1.49
08/12/87		13.00	42	21.15	13.51	8.51	5.41	3.10	3.10
15/12/87	0	14.76	39	20.00	21.34	8.07	5.12	2.15	1.45
22/12/87	9	15.00	32	14.05	23.13	8.11	4.20	1.10	1.45
31/12/87	18	12.20	31.5	9.05	17.33	8.09	4.64	1.44	3.10
04/01/88	20	15.00	35	10.05	15.29	7.00	4.30	2.04	2.10
11/01/88	27	14.80	29	11.05	12.49	7.81	5.12	2.69	1.90
25/01/88	41	10.20	20.6	16.10	14.14	7.64	4.64	3.00	1.55
01/02/88	48	10.80	27.0	13.5	15.12	7.91	2.81	5.10	0.55
08/02/88	55	14.40	48.0	15.2	13.64	7.78	3.70	4.05	0.92

Table 3.4

GOAT 2

Date of Birth	Sex	Weaned	Date of Isolation	Age Weeks	Date of Examination	Plotted Days	Weight Kg	Temperature 'C	Oocyst Count $00/g \times 10^3$	Clinical Observations
05/07/87	M	25/10/87	27/10/87	13	09/10/87		6.90	39.0	4168	Active, healthy, eating well.
				14	16/10/87		7.00	39.3	70000	Same as above
				15	23/10/87		7.30	39.5	1018	"
				16	30/10/87		8.00	38.8	1132	
				17	05/11/87		7.50	39.1	9069	"
				18	13/11/87	0	9.00	38.8	84626	"
				19	24/11/87	18	9.00	39.0	11150	"
				20	01/12/87	25	9.50	39.2	45404	Active, eating well, nasal discharge.
				21	08/12/87	32	9.00	39.1	22073	"
				22	15/12/87	39	9.00	40.2	24791	Looked weak though eating well.
				23	23/12/87	47	9.00	40.8	18701	Had diarrhoea, running nose.
					26/12/87		-	-	-	Got worse, not eating well, diarrhoea.
					28/12/87	52	8.70	39.4	17816	Bloody greyish-yellow diarrhoea with lots of mucus, failing to walk or eat.
			Died	24	31/12/87	55	8.50	38.00	16931	Found dead.

POST MORTEM OBSERVATIONS

The tissue had degenerated so much that it was not possible to obtain information from it.

GOAT 2

Date of Examination	Plotted Days	Hb g/dl	PCV %	WBC x 10 ^{3} ul	RBC x 10 ^{6} ul	Total Protein g/100 ml	Albumin g/100 ml	Globulin g/100 ml	A/G Ratio
26/10/87	0	11.50	31	15.10	17.43	6.20	4.41	1.83	2.34
09/11/87	14	11.10	32	12.50	14.61	5.75	2.88	2.87	0.91
20/11/87	25	12.30	30	10.50	10.39	5.47	3.76	1.71	2.19
25/11/87	30	10.40	28	11.10	15.45	6.03	4.52	1.51	2.99
11/12/87	46	11.80	30	14.25	22.30	5.80	4.20	1.60	2.62
28/12/87	63	10.30	24	13.35	13.73	5.84	5.30	0.54	27.13
15/12/87	50	11.60	19.6	10.50	15.31	5.10	4.24	0.86	4.93
29/12/87	58	10.00	21.8	10.05	17.76	5.62	4.10	1.52	2.7

Table 3.5

GOAT 9

Date of Birth	Sex	Date of Weaning	Date of Isolation	Age Weeks	Date of Examination	Plotted Days	Weight Kg	Temperature °C	Oocyst Count 9 OO/g x 10	Clinical Observations
11/07/87	F	25/10/87	27/10/87	13	3/10/87		5.40	39.40	00>10	Active, healthy and eating well
				14	16/10/87		5.40	39.50	00>10	Same as above
				15	23/10/87		5.60	38.7	16010	"
					30/10/87		5.50	39.4	24609	"
				16	6/11/87		5.50	39.2	153846	"
				17	13/11/87	0	6.00	38.7	879	"
				18	20/11/87	7	6.00	38.3	2283	"
				19	26/11/87	13	7.00	38.6	5148	"
				20	1/12/87	18	6.50	39.5	11698	"
				21	9/12/87	26	6.00	40.2	15125	"
				22	16/12/87	33	6.30	39.9	2919	Diarrhoea started, very active, eating well
				23	23/12/87	40	6.50	39.8	7983	Greyish diarrhoea, active, eating well
				24	31/12/87	48	6.00	40.5	9310	Greyish yellow diarrhoea, active, encrusted nose, eating well.
					5/01/88	53	5.50	38.70	6786	Yellowish diarrhoea, weak, pilo-erection on the entire body, encrusted nose, not eating well.
					6/01/88		-	-	-	Pilo-erection, swollen face, yellowish bloody diarrhoea, very weak, rarely eating.
			Died	25	7/01/88	55	5.00	39.40	316168	Treated with Amprolium.

POST MORTEM OBSERVATIONS

Duodenum: Foldings, rosettes and nodules scattered on the entire length of the tissue.
Jejunum: All the different types of lesions were found along the entire length. These were so crowded together that one had to use a hand lens to

GOAT 3

Date of Examination	Plotted Days	Hb g/dl	PCV %	WBC x 10^3 ul	RBC x 10^6 ul	Total Protein g/100 ml	Albumin g/100 ml	Globulin g/100 ml	A/G Ratio
26/10/87		13.2	35	14.65	16.93	6.06	4.88	1.18	4.13
09/11/87	0	13.3	36	28.90	16.19	7.08	3.09	3.99	0.77
20/11/87	11	14.7	30	16.00	19.39	6.37	4.88	1.49	3.27
24/11/87	15	14.9	34	18.45	16.06	6.23	4.76	1.47	3.24
[illegible]	22	14.15	29	20.50	13.59	7.48	6.07	1.41	4.30
[illegible]	29	15.3	31	20.70	23.33	6.63	4.99	1.64	3.04
[illegible]	36	14.3	37	17.25	24.75	4.72	4.29	0.43	9.98
[illegible]	44	14.3	29	18.65	22.78	4.72	3.21	1.51	2.13
01/12/87	52	13.3	22	15.80	19.41	4.89	2.29	2.40	0.95
04/01/88	56	14.2	28	22.70	19.75	6.06	4.88	1.19	4.14

Table 3.6

GOAT 4

Date of Birth	Sex	Date of Weaning	Date of Isolation	Age Weeks	Date of Examination	Plotted Days	Weight Kg	Temperature °C	Oocyst Count $\frac{3}{00/g \times 10}$	Clinical Observations
					13/11/87		10.3			
27/06/87	M	03/09/87	11/11/87	20	17/11/87	0	10.00	39.70	17644	Active, healthy and eating well.
				21	25/11/87	8	8.50	38.10	20160	Same as above
				22	29/11/87	12*	9.50	39.20	37082	"
				23	03/12/87	12	10.00	39.50	11859	"
				24	16/12/87	29	8.50	39.10	1922	"
				25	23/12/87	36	9.50	39.20	5998	"
				26	31/12/87	44	9.00	40.20	3812	"
					03/01/88	47	-	-	-	Active, nasal discharge and eating well.
					06/01/88	52	9.0	40.2	43718	Signs of diarrhoea on the tail, weak, still eating, nasal discharge.
				27	08/01/88	*55	8.50	39.20	67584	Yellowish bloody diarrhoea, not eating well, very weak. Pilo-erection, eye and nasal discharges.
			Sacrificed		11/01/88		8.00	39.00	95996	Watery stool with lots of bloody discharge, not eating and very weak.

POST MORTEM OBSERVATIONS

Duodenum: Foldings and Haemorrhagic lesions throughout the entire length of the duodenum.
Jejunum: Mainly nodules with diameter ranging between 1-2 mm, Haemorrhagic lesions were plenty.
Ileum: Few rosettes and white spots.

HISTOPATHOLOGICAL OBSERVATIONS

Haemorrhagic (A): Very few gametocytes but many white blood cells all over the tissue.
Foldings (B): Same as above except in this case a few degenerating schizonts with white blood cells around them.
Nodules (D): Same as above.
White Spots (E): No gamonts, few degenerating giant schizonts surrounded by white blood cells. Lots of white blood cells all over the tissue.

GOAT 4

Date of Examination	Plotted Days	Hb g/dl	PCV %	WBC x 10^3 ul	RBC x 10^6 ul	Total Protein g/100 ml	Albumin g/100 ml	Globulin g/100 ml	A/G Ratio
20/11/87	0	16.7	32	5.15	25.52	7.09	3.05	4.03	0.76
24/11/87	4	16.6	30	19.15	19.35	6.20	5.00	1.20	4.17
01/12/87	11	13.3	28	12.00	6.56	5.59	0.97	5.76	
09/12/87	19	13.4	28	16.15	20.19	5.87	5.48	0.39	14.05
15/12/87	25	11.92	30	12.95	16.56	7.29	5.24	2.04	2.57
23/12/87	33	11.80	24	11.75	20.75	7.31	5.59	1.72	3.25
31/12/87	41	11.20	14	7.90	14.75	6.08	5.24	0.84	6.24
04/01/88	45	10.25	12	5.70	14.43	5.66	4.18	1.50	2.77
11/01/88	52	12.20	26	11.40	11.72	7.01	4.41	2.60	1.70

Table 3.1

GOAT 5

Date of Birth	Sex	Weaned	Date of Isolation	Age Weeks	Date of Examination	Plotted Days	Weight Kg	Temperature °C	Oocyst Count /g x 10^3	Clinical Observations
					13/11/87					
29/06/87	F	02/08/87	11/11/87	20	17/11/87	3	7.00	39.2	30172	Active, healthy and eating well.
				21	24/11/87	10	8.50	39.5	154598	Same as above.
				22	02/12/87	18	9.00	39.4	208188	"
				23	09/12/87	25	9.00	39.3	23088	"
				24	16/12/87	32	8.00	39.2	138604	"
				25	23/12/87	39	5.00	39.4	125832	"
				26	31/12/87	47	8.00	39.6	80360	"
					04/01/88		-	-	-	Diarrhoea started.
					06/01/88	50	5.50	40.2	59871	Greyish watery stool without blood, pilo-erection. still eating and active.
				27	08/01/88	52	5.00	39.8	10746	Greyish, mucus watery stool, not eating well and not active.
					11/01/88	55	5.00	39.7	26390	Yellowish bloody diarrhoea, not eating and weak. Given Amprolium.
			Sacrificed		12/01/88	56	5.00	39.3	61998	The condition of the animal was critical.

POST MORTEM OBSERVATIONS

Duodenum: Foldings and haemorrhagic lesions throughout the entire length of the duodenum.
Jejunum: Nodules and few rosettes.
Ileum: Very few white spots.

HISTOPATHOLOGICAL OBSERVATIONS

Foldings (B): A few degenerating giant schizonts, the whole tissue covered with white blood cells, no gametocytes seen.
Nodules (D) Same as above.
White Spots (E): Few degenerating giant schizonts with white blood cells covering the entire tissue.

GOAT 5

Date of Examination	Plotted Days	Hb g/dl	PCV %	WBC x 10^3 ul	RBC x 10^6 ul	Total Protein g/100 ml	Albumin g/100 ml	Globulin g/100 ml	A/G Ratio
20/11/87	0	15.20	32	9.45	18.47	7.10	3.09	4.01	0.77
24/11/87	4	17.00	30	12.35	19.67	5.92	4.64	1.28	3.62
01/12/87	11	14.10	31	6.25	13.56	7.92	5.71	2.21	2.58
08/12/87	18	14.20	30	8.30	14.17	6.82	6.31	0.51	12.37
15/12/87	25	12.80	28	13.00	19.69	6.32	4.99	1.33	3.75
22/12/87	32	13.00	25	8.20	15.06	6.51	5.36	1.15	4.66
31/12/87	41	11.00	15	4.35	13.45	5.75	4.52	1.23	3.67
04/01/88	45	11.20	18	9.75	18.35	6.79	4.52	2.27	1.99
11/01/88	52	12.20	26	4.55	11.82	7.08	5.00	2.08	2.40

Table 3.8

GOAT 6

Date of Birth	Sex	Date of Weaning	Date of Isolation	Age Weeks	Date of Examination	Plotted Days	Weight Kg	Temperature °C	Oocyst Count OO/g x 10³	Clinical Observations
20/9/87	F	11/1/88	12/1/88	16	22/1/88			39.7		After weaning it had high temperature and diarrhoea, was treated with alamycin
				16.5	25/1/88	6	3.10	39.3	1504	
				17	29/1/88	7	-	-	-	Diarrhoea abated, was very active and eating well.
				19	1/2/88	11	4.50	39.4	4120	Looked well and active
				18.5	5/2/88		5.00	39.0	42710	Signs of diarrhoea on the tail and anus, nasal discharge, still eating
				19	8/2/88	14	4.20	39.2	20195	Weak, greyish diarrhoea with mucus, nasal discharge, not eating well, treated with amprolium
			Sacrificed		9/2/88	15	4.20	39.1	-	Very weak and emaciated, diarrhoea. Greyish green diarrhoea with blood, very weak.

POST MORTEM OBSERVATIONS

Duodenum: Many white spots on surface
Jejunum: Few scattered foldings and many haemorrhagic lesions, distal end had many white spots, very few nodules and haemorrhagic lesions.
Ileum: Few white spots, a lot of foldings at the distal end.

HISTOPATHOLOGICAL OBSERVATIONS

Foldings(S): Many white blood cells all over the tissue especially the villi. A few degenerating giant schizonts, no gametocytes, progamonts or small schizonts were seen.
Rosettes (S): Many white blood cells scattered. Very few gametocytes, among those, microgametocytes were less than macrogametocytes. Very few degenerating giant schizonts.
Nodules (S): Same as in Rosettes
White Spots (E): Same as in Rosettes

Table 3.9

GOAT 7

Date of Birth	Sex	Date of Weaning	Date of Isolation	Age Weeks	Date of Examination	Plotted Days	Weight Kg	Temperature 'C	Oocyst Count $00/g \times 10^3$	Clinical Observations
5/11/87	M	11/1/88	18/1/88	11	22/1/88		-	39.3	-	Active, eating well, treated with alamycin as a prophylaxis against bacteria since it had high temperature.
					25/1/88	0	8.30	39.9	948	Diarrhoea abated-active and eating well
				12	27/1/88		-	-	-	Active, eating well, serious nasal discharge
				12.5	1/2/88	7	7.50	39.2	26468	Active, eating well, nasal discharge
				13	8/2/88	14	7.50	39.7	131040	Active, eating well, nasal discharge.
					10/2/88	16	7.80	39.5	189543	Active, eating well, pilo-erection, bloody stool not very watery. Treated with Amprolium.
			Sacrificed		11/2/88	17	7.20	39.30	1002240	Weak, eating well, nasal discharge. Bloody watery stool.

POST MORTEM OBSERVATIONS

Duodenum: Many nodules throughout the entire length, the diameter ranged between 1-2 mm.
Jejunum: Nodules with a tint of blood, at the distal end were few white spots and scattered rosettes. Size of nodules ranged between 1-4 mm.
Ileum: Nodules with a diameter ranging between 1-4 mm. Few foldings and white spots.

HISTOPATHOLOGICAL OBSERVATIONS

Foldings (B): Very few giant schizonts but lots of white blood cells. In many villi there were no other cells to be distinguished except for white blood cells. Did not see any gametocytes or small schizonts.
Rosettes (C): Many white blood cells all over the tissue but not as many as those of above. Very few degenerating giant schizonts.
Nodules (D): Complete life cycle showing all the developing and mature stages. Mature giant schizonts in the loop of villi. Many progamonts with two or three in one cell. Some degenerating giant schizonts.
White Spots (E): Many mature small schizonts, progamonts, developing and mature giant schizonts. Developing and mature gametocytes, some oocysts in the lumen.

Table 3.10

GOAT 8

Date of Birth	Sex	Date of Weaning	Date of Isolation	Age Weeks	Date of Examination	Plotted Days	Weight Kg	Temperature 'C	Oocyst Count $OO/g \times 10^3$	Clinical Observations
11/11/87	F	11/01/88	18/01/88	10.5	22/01/88		-	-	-	Active, healthy, treated with Alamycin
				11	25/01/88	0	4.10	39.9	201312	Active, eating well
				12	01/02/88	7	4.00	39.5	88020	Active, eating well.
				12.5	05/02/88	11	-	-	-	Signs of diarrhoea on the tail and anus. Active, eating well and nasal discharge.
					06/02/88	12	-	-	-	Very weak, not eating well, greenish-grey diarrhoea with no blood, emaciated.
		Sacrificed		13	08/02/88	14	3.50	38.0	146808	Very weak, unable to walk or feed, greenish diarrhoea no blood.

POST MORTEM OBSERVATIONS

Duodenum: Foldings and nodules of diameter ranging between 2-3 mm.
Jejunum: Rosettes with diameter ranging between 2-5 mm, a few nodules tinted with blood. Towards the distal end nodules crowded together.
Ileum: Very few nodules scattered.

HISTOPATHOLOGICAL OBSERVATIONS

Foldings (B): Few developing giant schizonts
Rosettes (C): Many degenerating giant schizonts, macro and micro-gametocytes developing and mature ones were found in many villi crypts. Progamonts and oocyst patches were many.
Nodules (D): Possible fertilisation, giant schizonts seen in serosa. Both macro and micro-gametocytes developing and mature ones were found in many villi crypts, also progamonts and oocyst patches. Degenerating giant schizonts and very clear small schizonts with released merozoites. Could identify life cycle of the parasite
White Spots (E): Degenerating giant schizonts (red patches inside) few progamonts and macro and micro-gametocytes.

GOAT 6

Date of Examination	Plotted Days	Hb g/dl	PCV %	WBC x 10 ^{3} ul	RBC x 10 ^{6} ul	Total Protein g/100 ml	Albumin g/100 ml	Globulin g/100 ml	A/G Ratio
25/01/98	0	15.00	20.6	15.20	13.04	7.26	4.99	2.27	2.19
01/02/98	7	10.30	22.00	14.19	18.30	7.95	4.52	3.22	1.39
08/02/98	14	11.50	30.00	14.40	17.44	8.42	2.30	4.12	0.55

GOAT 7

Date of Examination	Plotted Days	Hb g/dl	PCV %	WBC x 10 ^{3} ul	RBC x 10 ^{6} ul	Total Protein g/100 ml	Albumin g/100 ml	Globulin g/100 ml	A/G Ratio
25/01/98	0	13.00	30.00	9.50	19.29	6.67	5.59	1.08	5.18
01/02/98	7	12.40	36.00	9.00	16.26	6.11	5.71	0.40	14.27
08/02/98	14	11.2	30.10	9.80	15.04	5.92	2.22	2.98	0.95

GOAT 8

Date of Examination	Plotted Days	Hb g/dl	PCV %	WBC x 10 ^{3} ul	RBC x 10 ^{6} ul	Total Protein g/100 ml	Albumin g/100 ml	Globulin g/100 ml	A/G Ratio
25/01/98	0	9.00	18.00	14.00	10.18	9.90	5.84	3.09	1.71
01/02/98	7	10.60	27.50	12.00	14.62	7.90	4.99	2.91	1.71
08/02/98	14	11.70	31.00	13.10	16.30	5.99	1.85	3.91	0.49

Table 3.11

GOAT 9

Date of Birth	Sex	Date of Weaning	Date of Isolation	Age Weeks	Date of Examination	Plotted Days	Weight Kg	Temperature 'C	Oocyst Count 10^3/g	Clinical Observations
7/12/87	M	11/01/88	18/01/88	6.5	22/01/88		4.30	39.9	-	Active, eating well, treated with alamycin.
				7	25/01/88	0	4.40	39.9	77736	Active, eating well
					27/01/88		-	-	-	Active, pale mucus membranes, enlarged lymph nodes.
					29/01/88		-	-	-	Weak, eating and diarrhoea.
				8	01/02/88	7	4.00	39.1	400840	Weak, not eating well, greenish-grey diarrhoea no blood, nasal discharge.
					02/02/88	8	-	-	-	Very weak, not eating well, greenish-grey diarrhoea, nasal discharge.
			Sacrificed		03/02/88	9	4.00	38.0	140408	Extremely weak, failing to eat, greenish-grey diarrhoea, nasal discharge.

POST MORTEM OBSERVATIONS

Many white spots on the duodenum and ileum. Spot size about 0.5 mm in diameter.
Ileum: Large fat deposits, few nodules about 3 mm diameter, few scattered rosettes 3 mm in diameter and foldings at the posterior end of ileum.
Jejunum: Many nodules with diameter ranging between 1-2 mm.

HISTOPATHOLOGICAL OBSERVATIONS

Foldings (B): Gametocytes, degenerating giant schizonts, second generation schizonts, bacteria. In certain sections of the villi there were only microgametocytes, lots of progamonts. Could identify life cycle of the parasite.
Rosettes (C): Mainly microgametocytes with a few areas showing both macro and micro-gametocytes. Many filamentous bacteria on edge of tissue.
Nodules (D): Different types of degenerating giant schizonts observed, gametocytes both micro and macro with oocysts in the lumen.
White Spots (E): Mainly microgametocytes. Mature giant Schizonts and some degenerating schizonts with white patches inside them. Lots of filamentous bacteria.

Table 3.12

GOAT 10

Date of Birth	Sex	Date of Weaning	Date of isolation	Age Weeks	Date of Examination	Plotted Days	Weight Kg	Temperature 'C	Oocyst Count $00/g \times 10^3$	Clinical Observations
16/12/87	M	11/01/88	18/01/88	5.5	22/01/88		-	39.9	-	Active, eating well and treated with alamycin.
				6	25/01/88	0	3.98	39.5	4038	Active, healthy and eating well.
				7	01/02/88	7	3.30	39.3	16978	Active, healthy eating well though looking weak.
					02/02/88	8	2.80	39.7	-	Active, eating well, nasal discharge, signs of diarrhoea on tail and anus.
					03/02/88		-	-	-	Weak, eating slowly, nasal discharge, greenish-grey diarrhoea.
					04/02/88	10	2.80	38.4	-	Very weak, not eating much, nasal discharge, emaciated, watery stool. Treated with Amprolium.
		Sacrificed		7.5	05/02/88	11	2.80	39.0	141040	Watery stool with lots of mucus, very weak, not eating, emaciated.

POST MORTEM OBSERVATIONS

The gut had undigested green matter.
Duodenum: Lots of foldings and isolated nodules.
Jejunum: Lots of white spots, tiny nodules between 1.5 - 2 mm in diameter with a tint of blood on them.
Ileum: Foldings

HISTOPATHOLOGICAL OBSERVATIONS

Foldings (B): Lots of gametocytes, degenerating giant schizonts, second generation schizonts, bacteria. In certain sections of the villi there were only microgametocytes, lots of progamonts. Could identify life cycle of the parasite.
Rosettes (C): Mainly microgametocytes with a few areas showing both macro and micro gametocytes. Many filamentous bacteria on edge of tissue.
Nodules (D): Different types of degenerating giant schizonts observed, gametocytes both micro and macro with oocysts in the lumen.
White Spots (E): Mainly microgametocytes. Mature giant schizonts and some degenerating ones (with white patches inside them) Lots of filamentous bacteria.

GOAT 9

Date of Examination	Plotted Days	Hb g/dl	PCV %	WBC x 10^3 ul	RBC x 10^6 ul	Total Protein g/100 ml	Albumin g/100 ml	Globulin g/100 ml	A/G Ratio
25/01/88	0	8.9	28.00	12.20	16.74	8.41	3.69	3.72	1.35
01/02/88	7	9.1	19.50	11.20	10.22	4.99	2.74	2.26	1.21

GOAT 10

Date of Examination	Plotted Days	Hb g/dl	PCV %	WBC x 10^3 ul	RBC x 10^6 ul	Total Protein g/100 ml	Albumin g/100 ml	Globulin g/100 ml	A/G Ratio
25/01/88	0	10.4	23.00	8.66	13.88	5.89	5.12	0.77	6.65
01/02/88	7	9.3	21.5	14.80	12.30	7.85	4.88	2.97	1.64

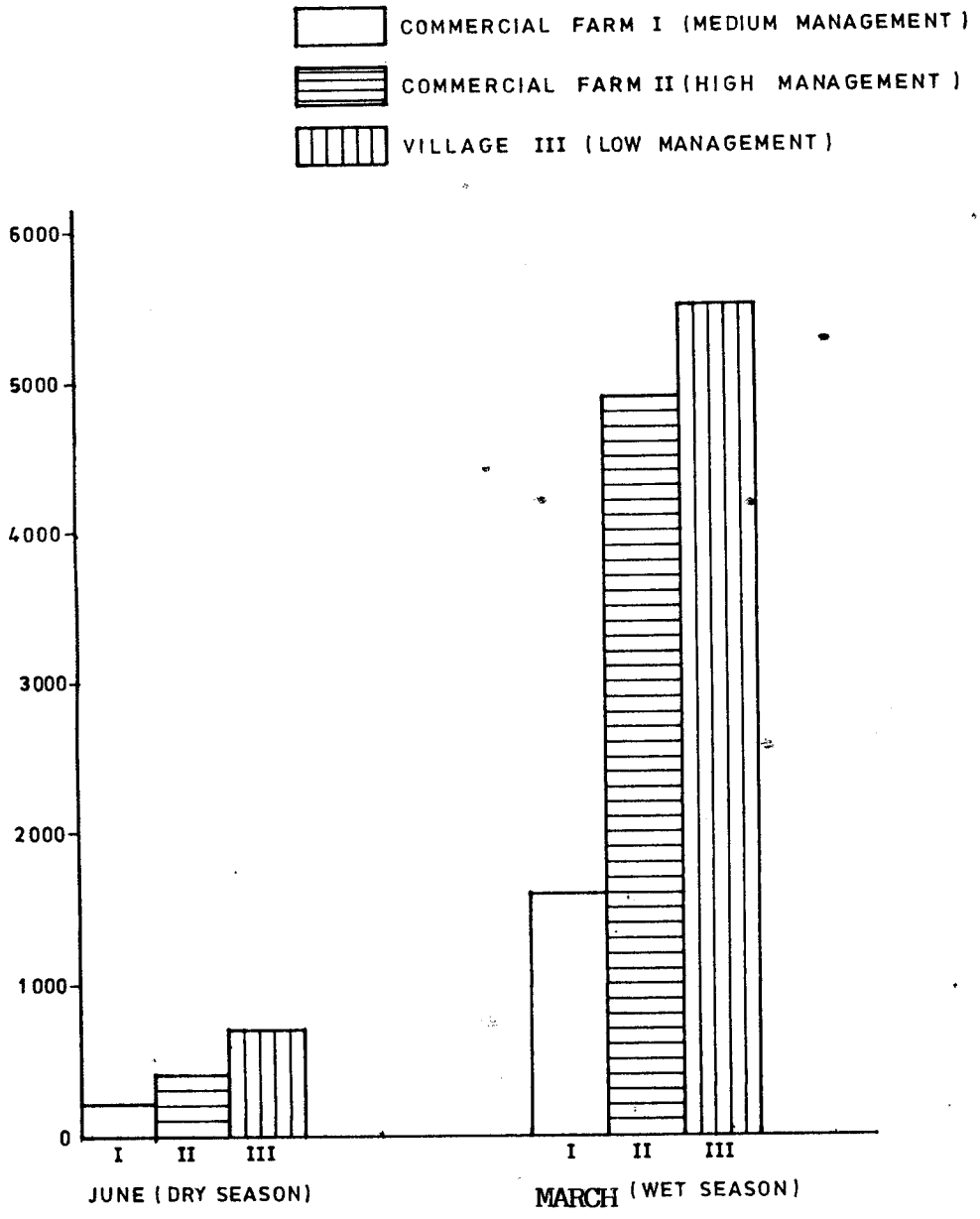
Table 3.9.1

GRASMERE FARM - (HIGH MANAGEMENT)

OOCYST COUNT/G FAECAL SAMPLE

GOAT NO.	JUNE 1988	MARCH 1989
003	396	1886
005	2	994
006	328	590
009	184	13 608
011	90	14 526
012	123	199
013	1	2374
015	2	1462
017	64	1357
018	19	153
019	40	263
020	2073	12 118
021	277	1596
022	356	1169
024	1890	5494
108	1322	2952
109	534	13 930
110	25	14 526
117	658	4583
118	1329	4243
Mean Values	396	4901

FIG. 3.9.4: MEAN OOCYST COUNT o/g ON THREE DIFFERENT FARMS



CHAPTER FOUR

DISCUSSION

FIG. 3.106

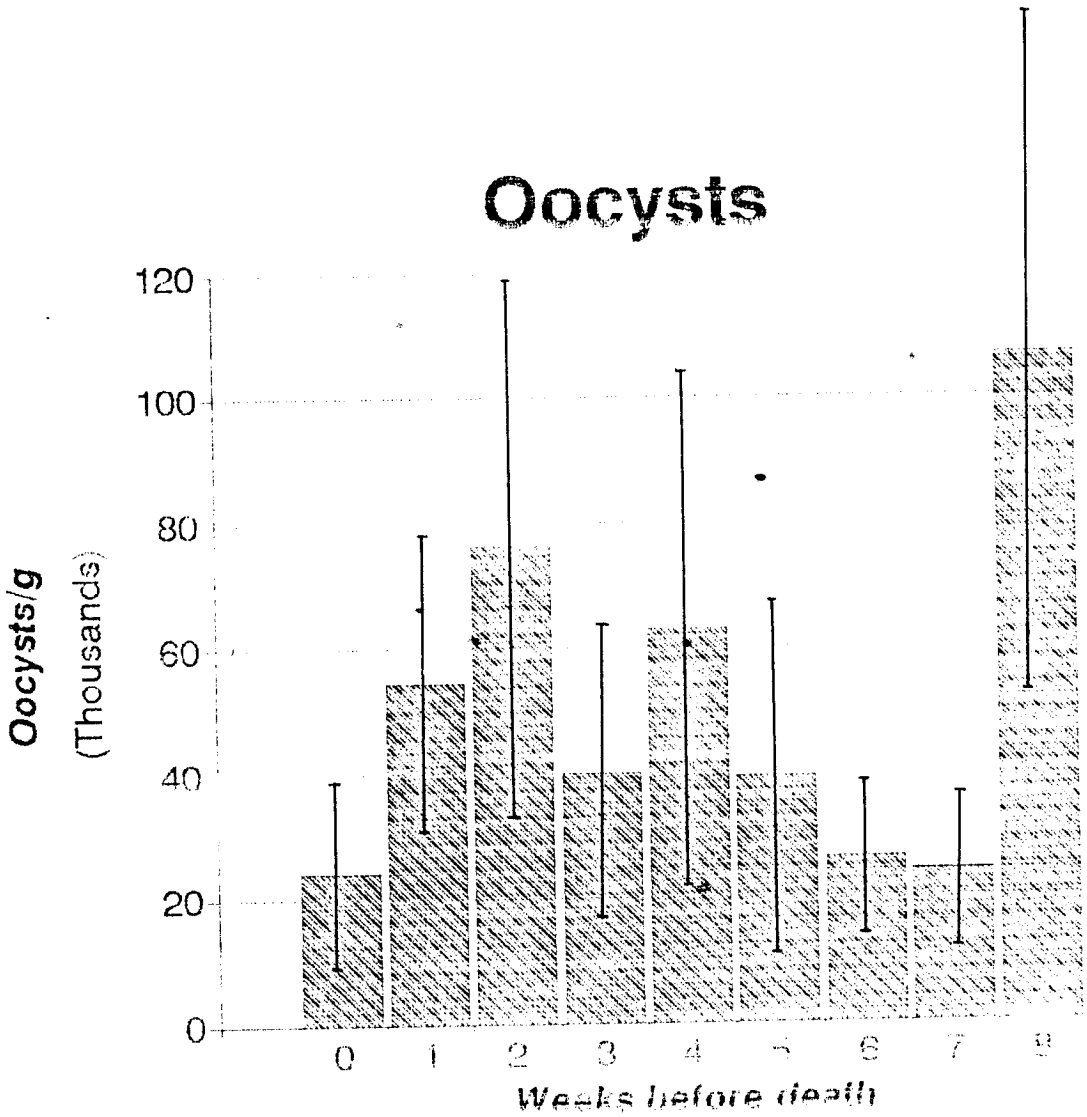

Oocysts
Oocysts/g
(Thousands)
120
100
80
60
40
20
0
0
1
2
3
4
5
6
7
8
Weeks before death

FIG. 3·105

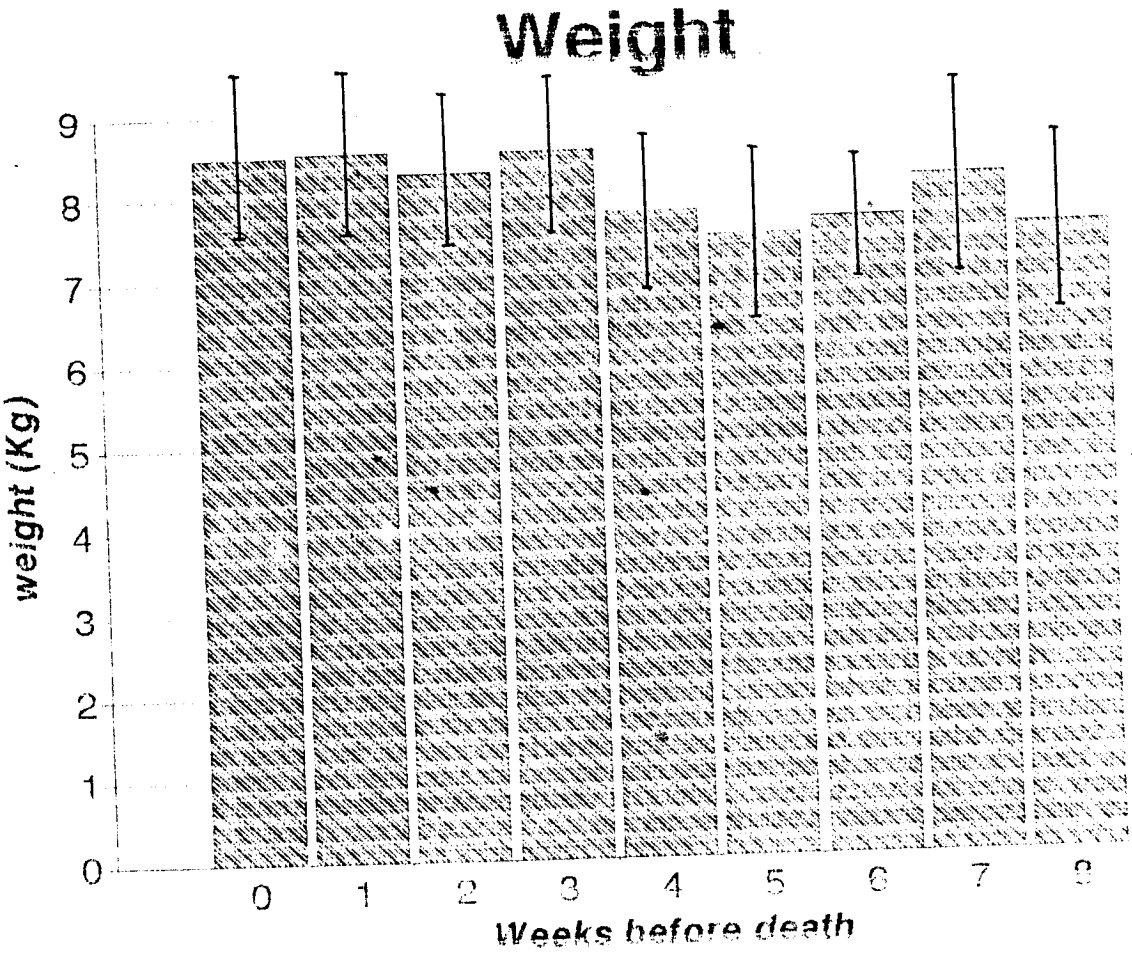


FIG. 3.111

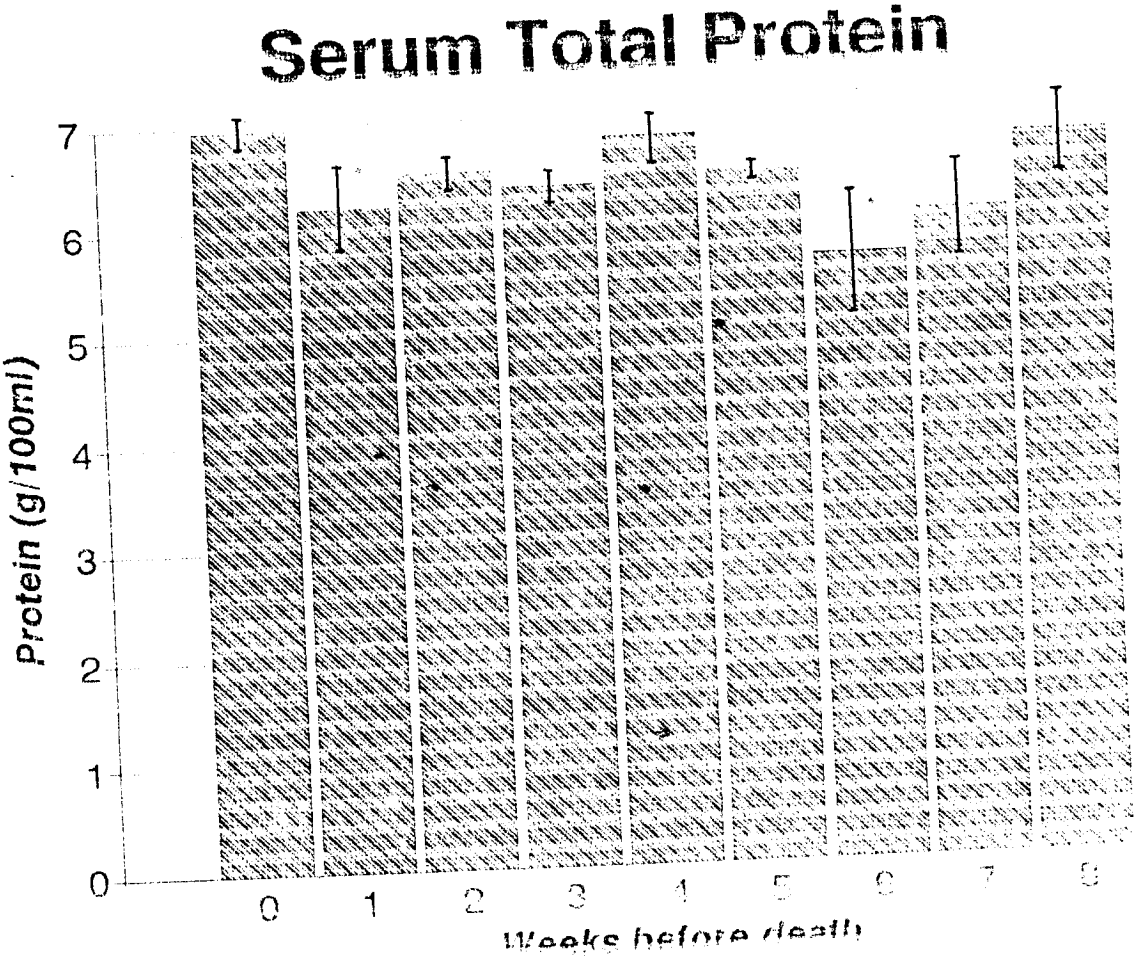

Serum Total Protein
Protein (g/100ml)
0
1
2
3
4
5
6
7
0
1
2
3
4
5
6
7
8
Weeks before death

FIG. 3-112

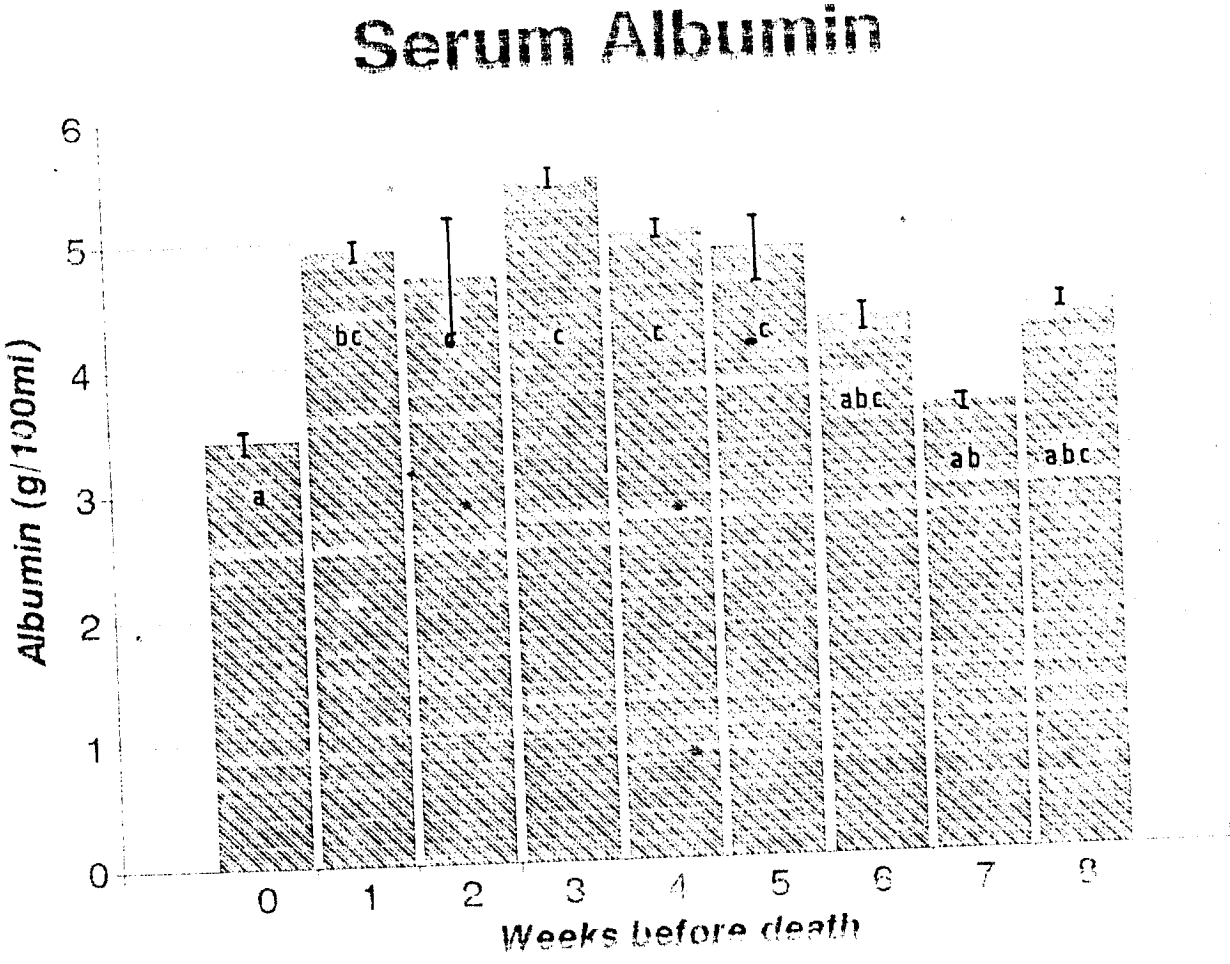

Serum Albumin
Albumin (g/100ml)
6
5
4
3
2
1
0
a
bc
c
c
c
c
abc
ab
abc
0
1
2
3
4
5
6
7
8
Weeks before death

FIG. 3.113

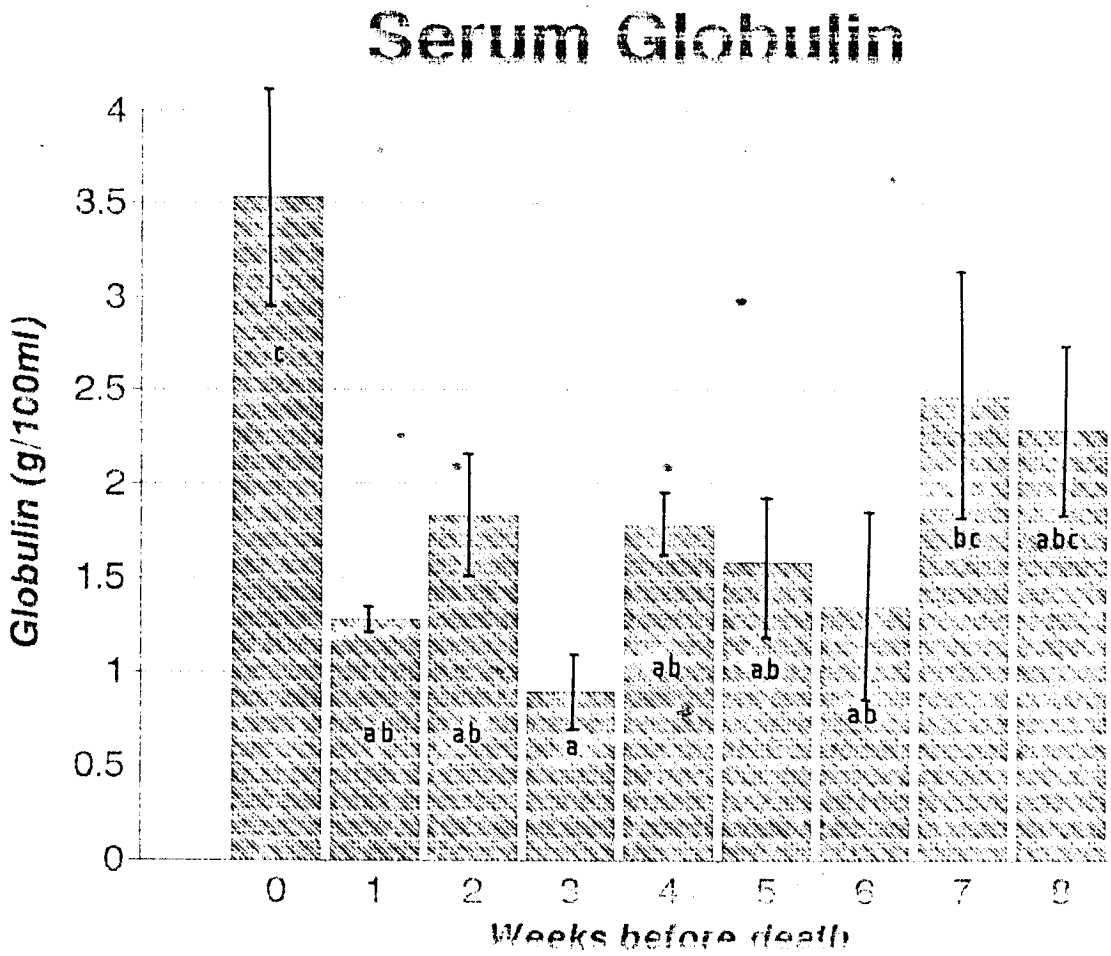

Serum Globulin
Globulin (g/100ml)
4
3.5
3
2.5
2
1.5
1
0.5
0
c
ab
ab
a
ab
ab
ab
bc
abc
0
1
2
3
4
5
6
7
8
Weeks before death

FIG. 3.114

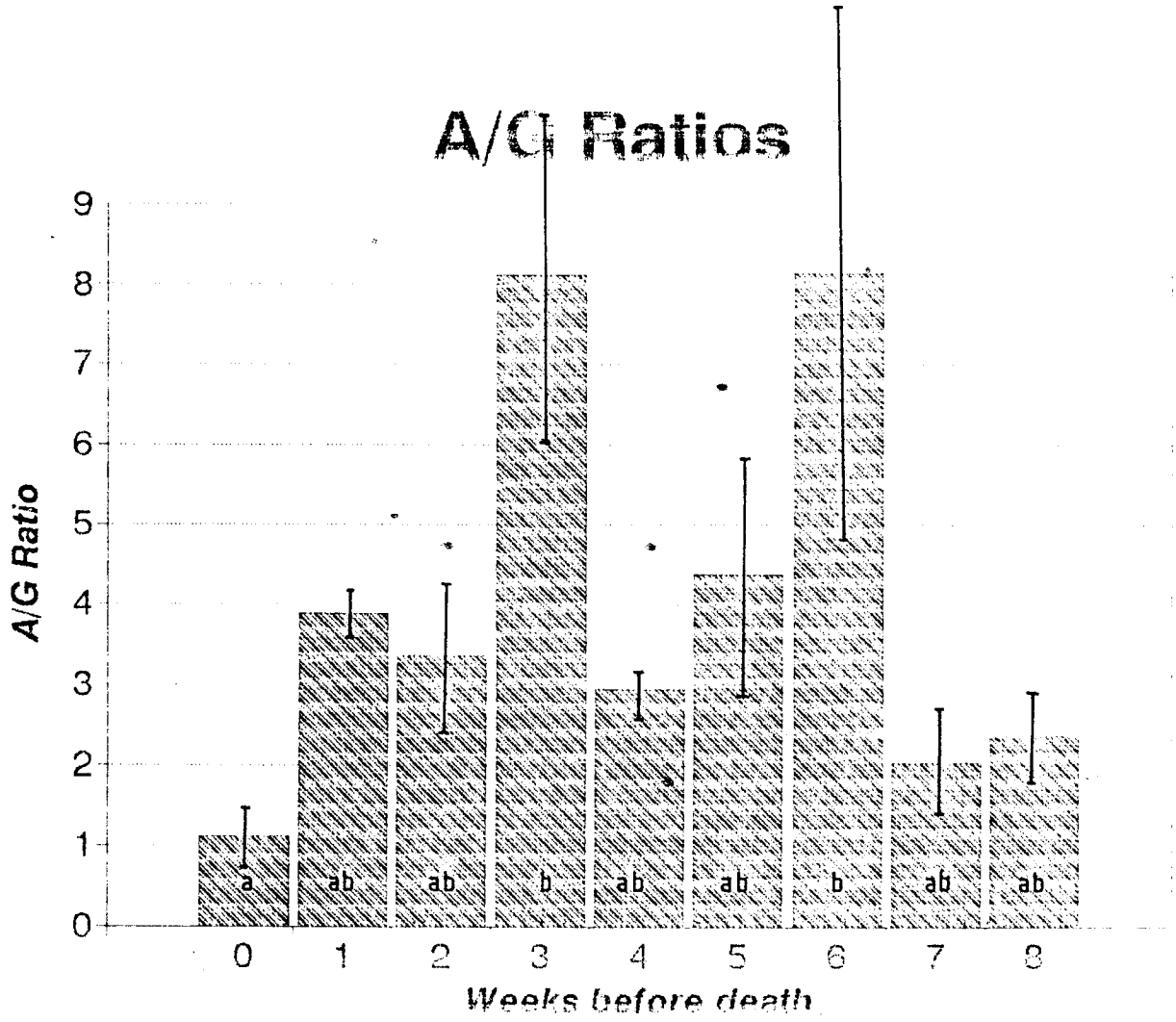

A/G Ratios
A/G Ratio
0
1
2
3
4
5
6
7
8
9
a
ab
ab
b
ab
ab
b
ab
ab
0
1
2
3
4
5
6
7
8
Weeks before death

FIG. 3.108

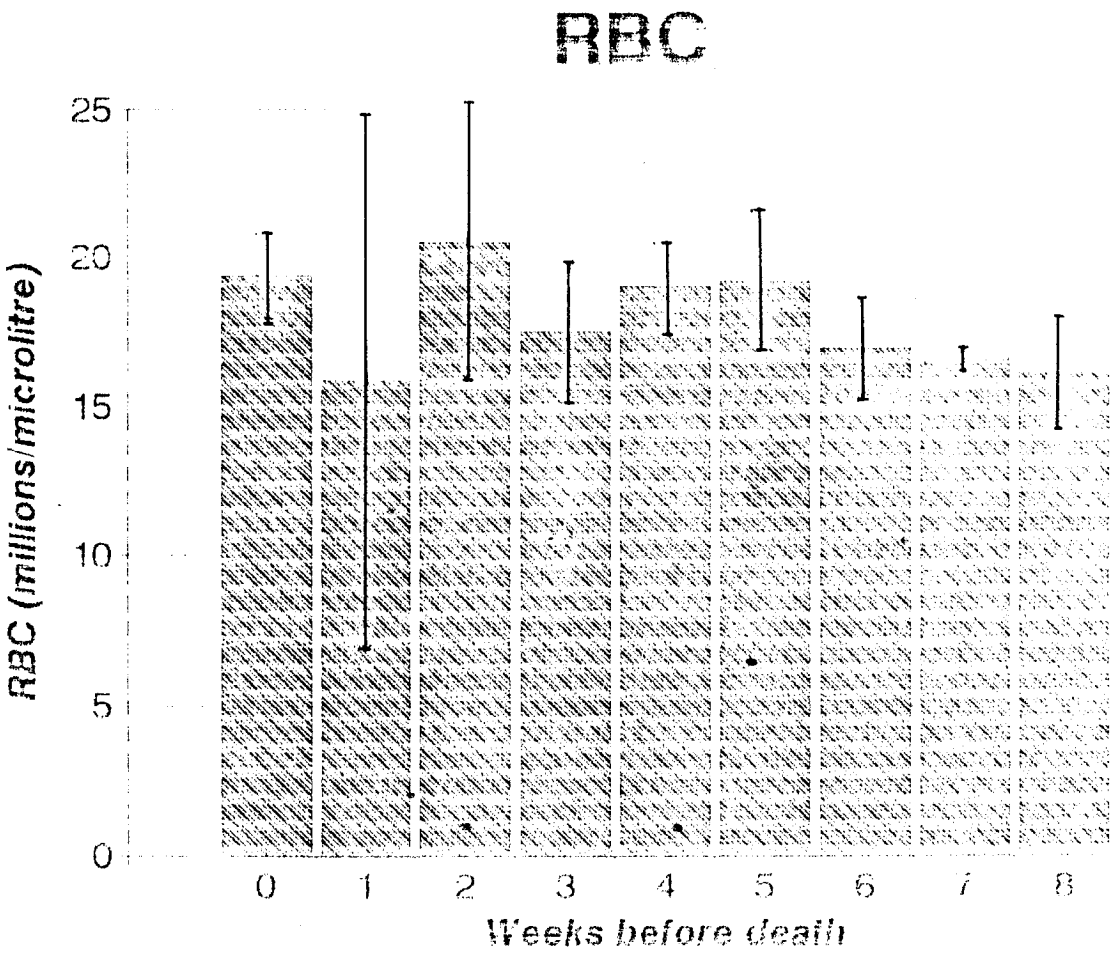

RBC
25
20
15
10
5
0
RBC (millions/microlitre)
0
1
2
3
4
5
6
7
8
Weeks before death

FIG. 3·107

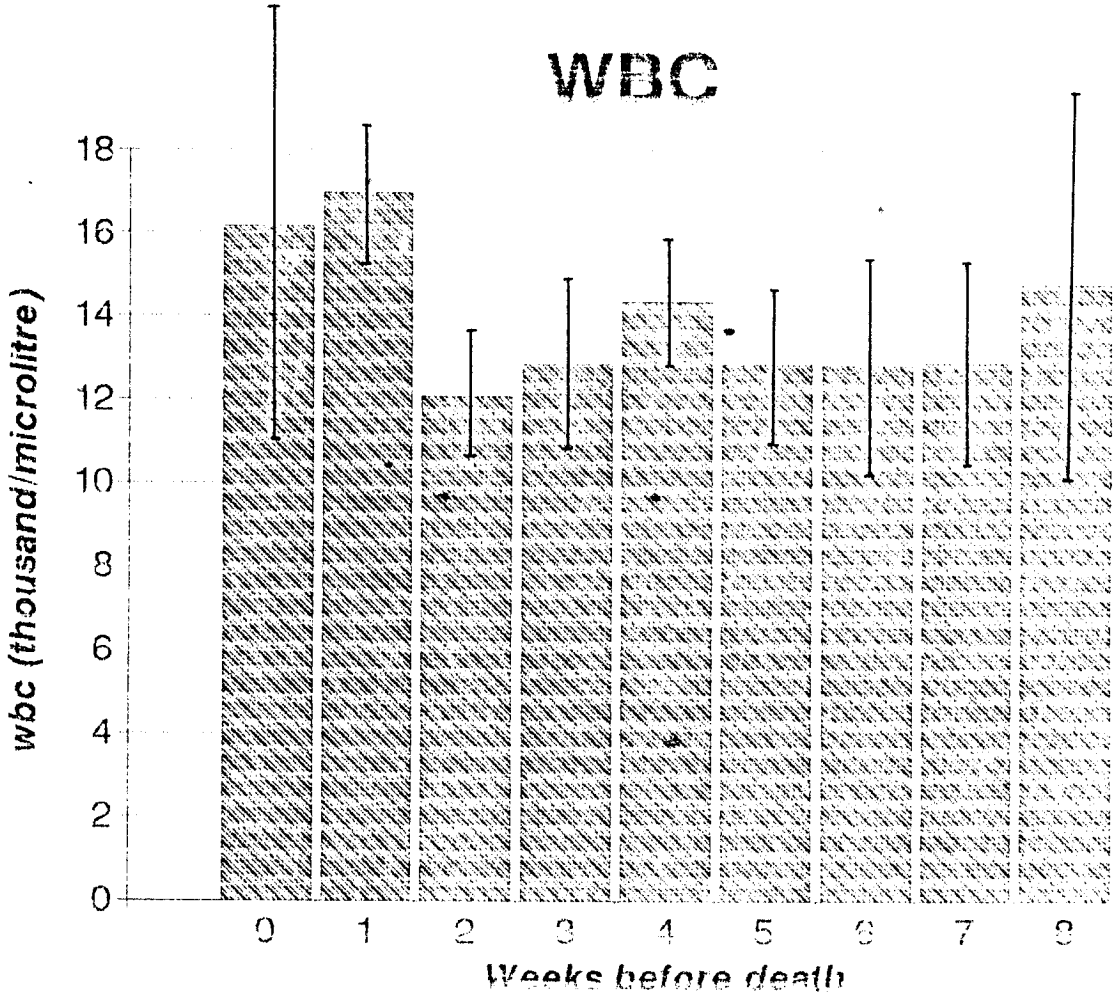

WBC
wbc (thousand/microlitre)
0
2
4
6
8
10
12
14
16
18
0
1
2
3
4
5
6
7
8
Weeks before death

FIG. 3.110

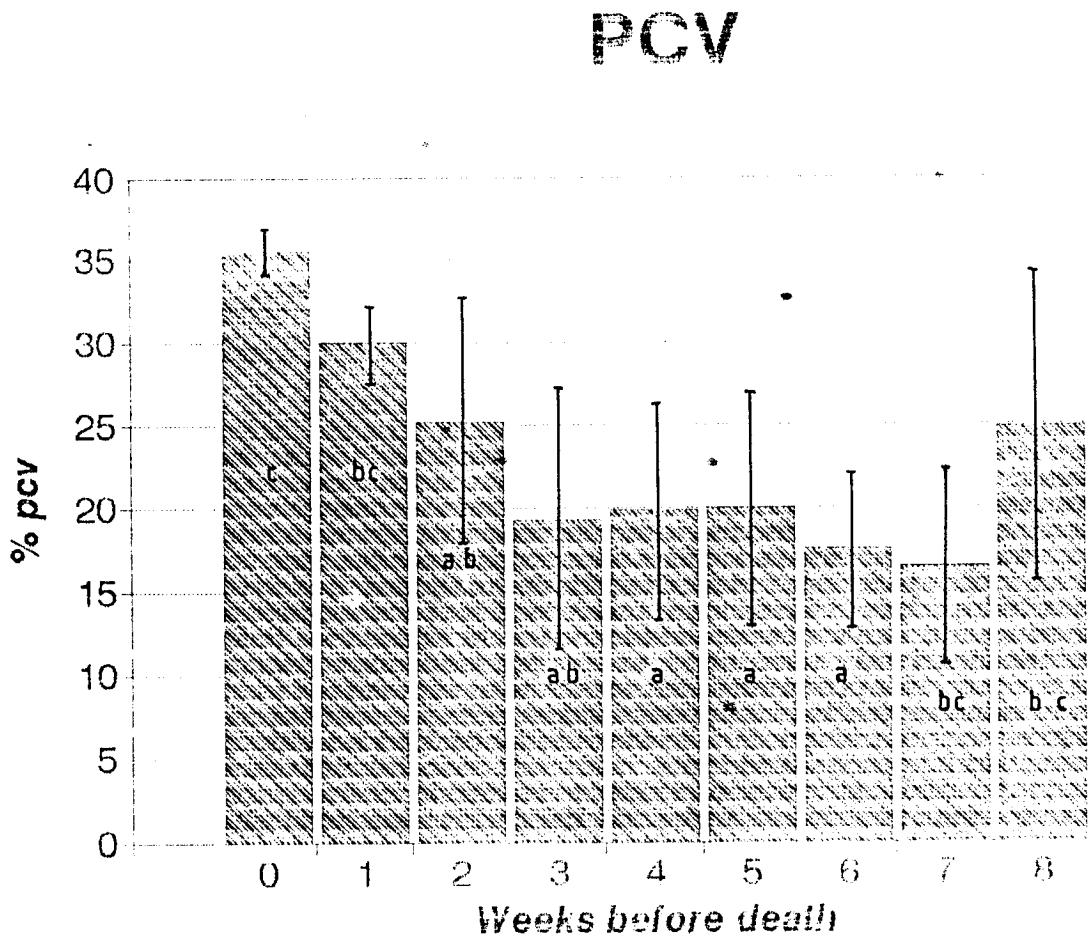
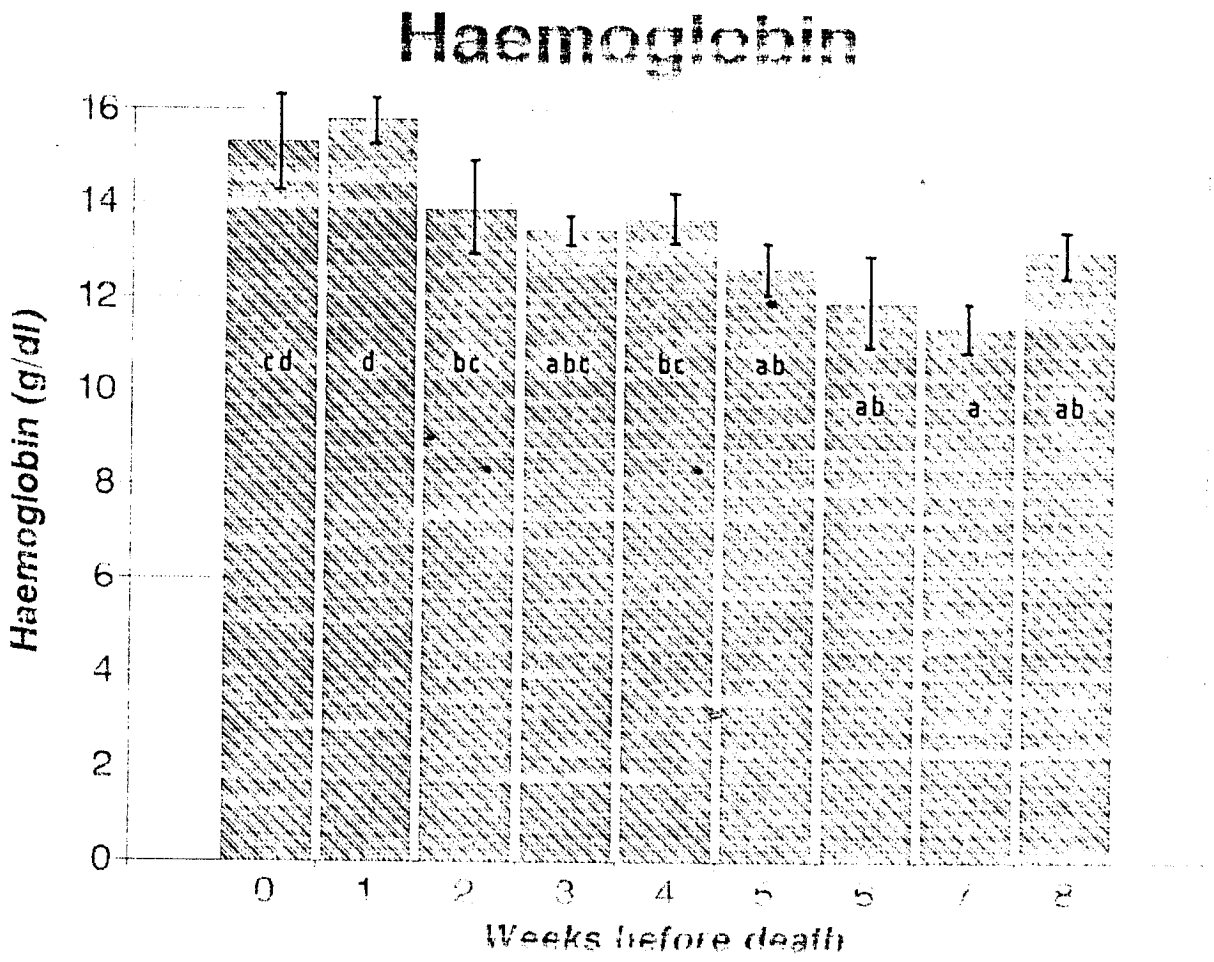

PCV
40
35
30
25
20
15
10
5
0
% pcv
c
bc
a b
a b
a
a
a
bc
b c
0
1
2
3
4
5
6
7
8
Weeks before death

FIG. 3.109


Haemoglobin
Haemoglobin (g/dl)
16
14
12
10
8
6
4
2
0
cd
d
bc
abc
bc
ab
ab
a
ab
0
1
2
3
4
5
6
7
8
Weeks before death

CHAPTER IV

DISCUSSION

4.1 CLINICAL SYMPTOMS

The clinical symptoms observed were concluded to be the results of a natural infection of coccidia. All the animals looked very weak and unable to feed well. Some goats had febrile periods during the course of study, for example goat 2 and all those in the second group. The fever observed could have been associated with other infectious agents like bacteria and virus besides coccidia.

Pellet formation of stool was lost as the disease progressed, resulting in diarrhoea. The colour of the faeces during the first few days was grey to greyish-green. This is similar to that observed by Gregory et al (1987)* who described it as due to mucofibrious exudate. As the animal's condition worsened the colour of the diarrhoea changed to yellow and was watery, sometimes with blood. This was a common symptom associated with coccidiosis.

The weight of all the kids decreased as the disease worsened. This occurred during the last 1-2 weeks of the experiment in the first group, and after a week in the second group. This could have been due to diarrhoea and undernourishment as the animals

were not feeding well during the later stages of coccidiosis. Panyari (1988) reported that the Anglonubian goats he studied suffered greater retardation in growth post-infection by coccidia.

The temperature of the goats was fairly constant except in the later stages of the disease when the animals became feverish. This was followed by a drop in temperature as the disease progressed, just prior to the animal's death . The average temperature of a healthy goat is about 39.4 °C (Lloyd 1981). Goat 2 on the day it developed diarrhoea had a temperature of 40.8 °C and seven days later it was found dead. In goat 1 when the temperature was at its peak 43.8 °C, the level of WBC and globulins was also rising. Kelly (1984) suggested that antibody production is increased when the body temperature is elevated. Animals in a state of dehydration are prone to hyperthermia because of the reduction of heat loss by evaporation of tissue fluids. In the case of the kids studied the mean temperature was not very high.

The second group of goats had very high temperatures after they were weaned and isolated from their mothers. Goat 6 had diarrhoea but they were all immediately treated with Alamycin Since the goats were all less than 10 kg body weight the recommended dose was 1 ml/dose orally once. The rise in temperature and diarrhoea could have been due to sudden weaning and isolation into an area previously infected with large doses of oocysts. The early age of weaning of goats 8 (9.5 wks), 9 (5.5 wks) and 10 (4.5 wks) may have put a nutritional stress on these kids to add to the stress of the disease of coccidiosis.

The drop in temperature prior to death, noted in all animalscould be associated with emaciation due to malnutrition and circulatory collapse (Kelly 1984).

If one compares the numbers of oocysts shed by the mothers to those shed by kids, it indicates that the kids shed more oocysts than did their mothers. Both groups showed mixed infections. These results are similar to those studied by Valenzuela et al (1988) and Pout (1973) who studied naturally infected sheep in Southern Chile and England respectively and who observed that sheep older than one year passed fewer oocysts than the lambs. The lambs showed a rapid increase in oocyst count at an early age and then a decline after the animals were 9 months old. During the experiment, the room in which the animals were confined was cleaned only once a week and primary infection was from the does but the successive infection came from the kids themselves. This latter infection proved to be fatal since all goats fell critically ill and were sacrificed. The high mortality could have been due to the animals ingesting more pathogenic species which survived better in the prevailing environment as the temperature of the room was not too high and was well ventilated. Since it was during the rainy season, fairly high humidity occurred on certain days. These factors were very conducive to sporulation.

As the number of oocysts declined, clinical symptoms of the disease became apparent. A large output of oocysts is not necessarily correlated to extent of visible coccidiosis.

Gregory and Norton (1986) reported that healthy animals may pass out a million oocysts per gram of faeces whereas in a kid dying of coccidiosis the count may be less than 10,000 oo/g. They further explain that the high counts are due to non-pathogenic species which could mask the significant number of E. arloingi and E. ninakohlyakimovae which are pathogenic in goats, (Ajayi and Todd 1977). One could suggest that infection may be determined by two factors, the age at which ingestion took place and the size of infective dose. It is not clear whether a clinical case of coccidiosis arises due to the number ingested or to the number of pathogenic species ingested. Hammond et al (1967) described that different coccidia species had different prepatent periods e.g. E. ninakohlyakimovae had a prepatent period of 11 days and Pout (1969) reported that of E. arloingi may be as long as 26 days. Ajayi and Todd (1977) reported that the prepatent period varied from 13 to 18 days for E. arloingi, E. crandallis and E. ninakohlyakimovae.

Different species of Eimeria were observed during this study and identification was attempted. E. arloingi constituted about 60%, E. ninakohlyakimovae about 20% and the remaining 20% was comprised of other species. Alonge (1977) after oocyst counts in Nigerian dwarf goats reported that E. arloingi constituted 32% and is believed to be the most pathogenic species in goats, followed by E. ninakohlyakimovae 16% and E. crandalis 7%. Gregory and Norton (1986) observed that E. arloingi was the commonest species of coccidia and it caused recognisable papillomatous lesions in the small intestine. They further mentioned that E. ninakohlyakimovae and E. caprina were more

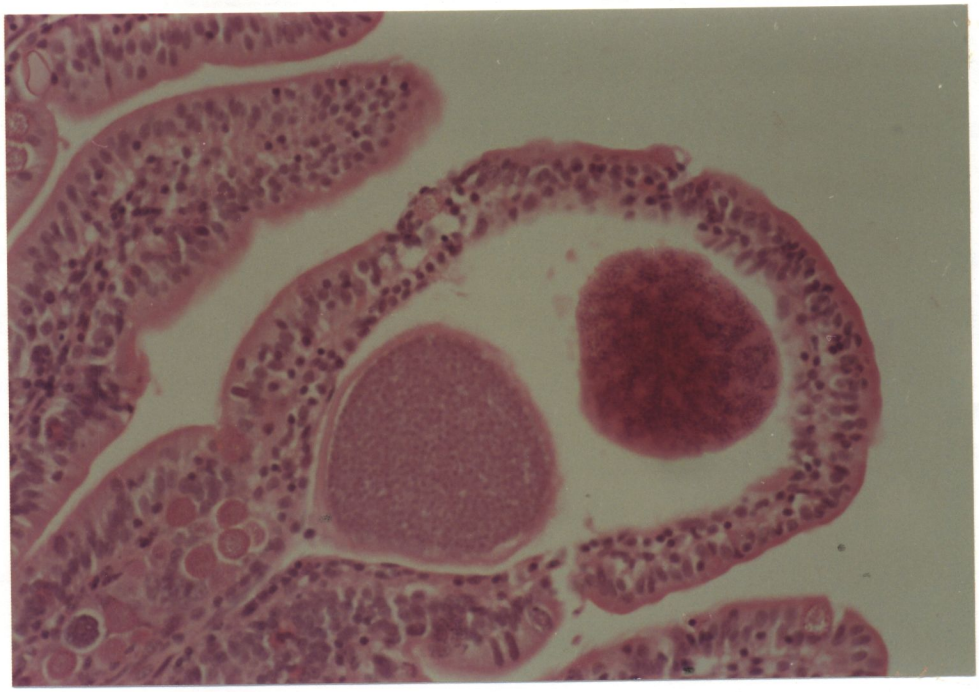


Fig 4.5.2.1 Two giant schizonts occupying the lumen of a villus. (x 40 mg)

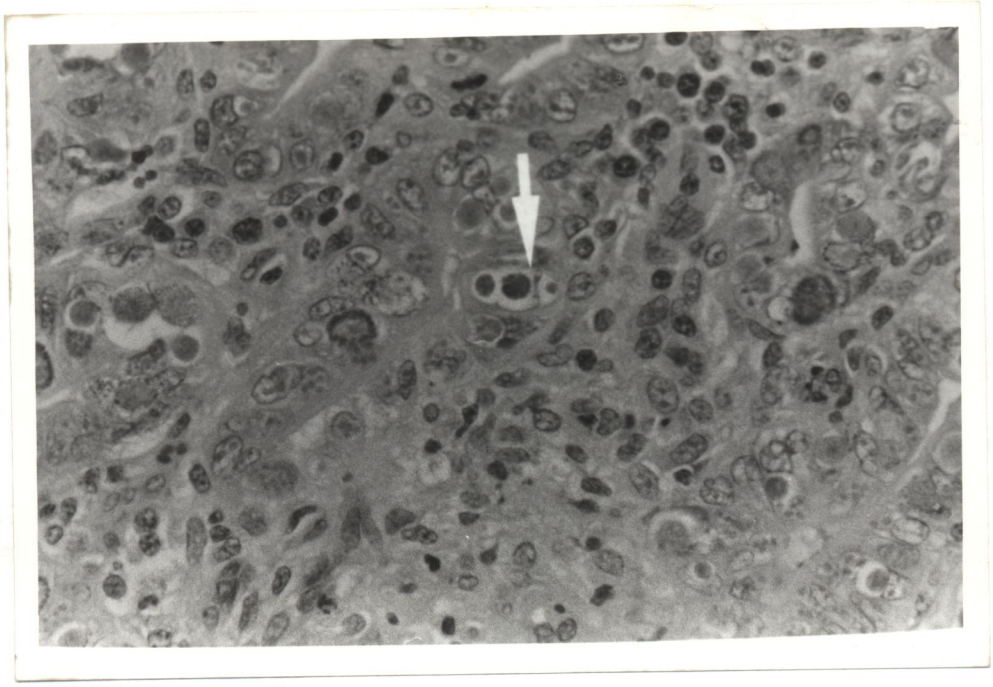


Fig 4.5.2.2 Three progamonts occupying one epithelial cell. (x 20 mg)

destructive because they could cause widespread denudation of the mucosa in the upper and lower large intestine. Opoku-Pare and Chineme (1979) reported that E. arloingi seemed to be the most common species in a mixed infection, i.e. 58% in a group of 55 Sokoto Red and Kano Brown cross goats. Next prevalent was E. ninakohlyakimovae and E. intricata. Since E. intricata is generally considered non-pathogenic, the lesions observed in our study could have been caused by E. arloingi and E. ninakohlyakimovae.

4.2. Haematology

The knowledge of haematological values is important in the diagnosis of many diseases in domestic animals. Work on the various haematological constituents in the blood of goats is scarce. In our study, the loss of blood during the period when some of the animals had diarrhoea had little effect on the total erythrocyte count values. It has been noted by Kelly (1984) that very young animals have lower values of PCV, RBC and Hb than adolescent and young adult animals of the same species. He also reported that animals that have been accustomed to being handled for blood sampling have lower values of the above parameters than animals not so regularly sampled. Dietary iron requirement needed for haemoglobin formation is small because the body of the animal is able to conserve the iron resulting from haemoglobin destruction.

Leukocytes are less numerous than erythrocytes in circulatory blood. Swenson (1977) reported that there are approximately

1,300 erythrocytes to every leukocyte in the blood stream of goats. The increase of WBC towards the end of the experiment in some of the goats was due probably to the body defence mechanism triggered by the presence of coccidia and bacteria. Vaidya et al (1970) reported that on the average, young goats have a higher count than older goats. There were a number of neutrophils and eosinophils at the site of infection especially around the giant schizonts. (see Fig 3.8.1b).

4.3 Biochemistry Parameters

Biochemical analysis of serum helps in confirming diagnosis, prognosis and response to treatment in many diseases.

4.3.1 Total Serum Protein

There was a general decrease of total serum protein when clinical symptoms began to show, i.e. around the 6th week in 1st group, and the 2nd week in 2nd group. These results may have been due to an excessive loss of protein, decreased protein synthesis by the liver, or starvation, since most animals had loss of appetite as the disease worsened. With the second group there was a sharp decline in total protein except in goat 10 where there was a slight increase. Schalm et al 1975 reported that heavy infestation of parasites can affect the animal by reducing the water and food consumption which results in emaciation. They further stressed that excessive water loss in faeces can also reduce the amount of protein resorbed from the gut since the animal is unable to resorb either water or protein.

4.3.2 Serum Albumin

The level of albumin, being the most abundant of the plasma proteins, affects the total protein concentration. From the graphs given the decrease in albumin also paralled a decrease of total protein in most of the animals.

Hypoalbuminaemia occurs in starved animals and in gastrointestinal diseases causing malabsorption (Kelly, 1984). The general trend in all the goats studied was a decline of albumin as the disease progressed and during the development of diarrhoea.

4.3.3 Serum Globulins

Globulin concentration rarely shows a decrease because a reduction of one globulin is compensated by an increase in another (Kelly, 1984). Most of the serum globulins are immunoglobulins which the body synthesises whenever there is an infection. There was a higher concentration of globulins at the beginning of the experiment, possibly because all goats had been exposed to coccidia from the faeces of their mothers. The second group of animals had a very high temperature and fever, and goat 6 had diarrhoea, which could have contributed to a high concentration of globulins.

The immunological response to coccidia in goats is not well understood, however, Rose (1984) reports that infection of chickens with E. tenella results in an increase in the

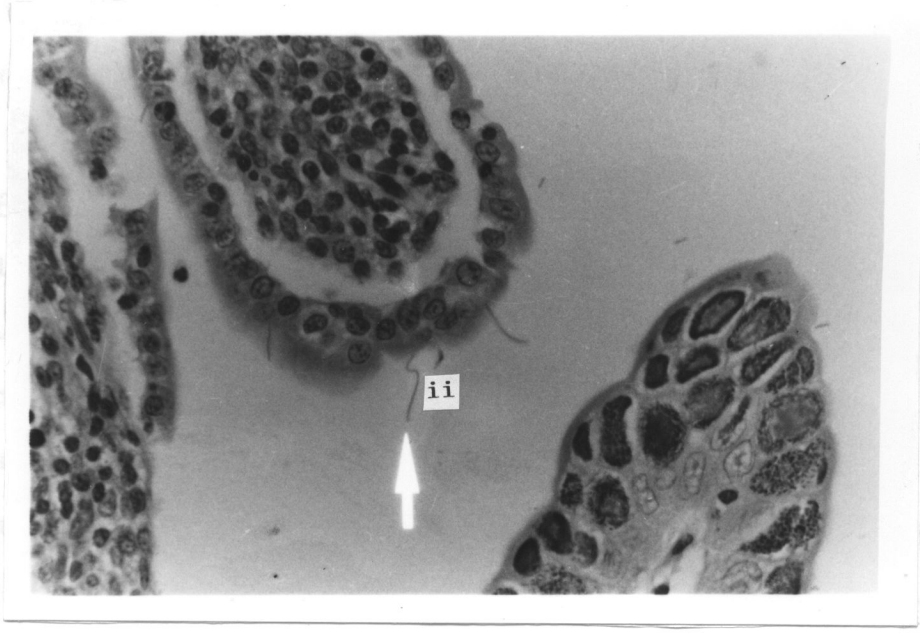


Fig 4.5.3a (i) Arrow showing filamentous bacteria. On the left a villus with microgametocytes only. (x 40 mg)

(ii) A merozoite penetrating an epithelial cell.

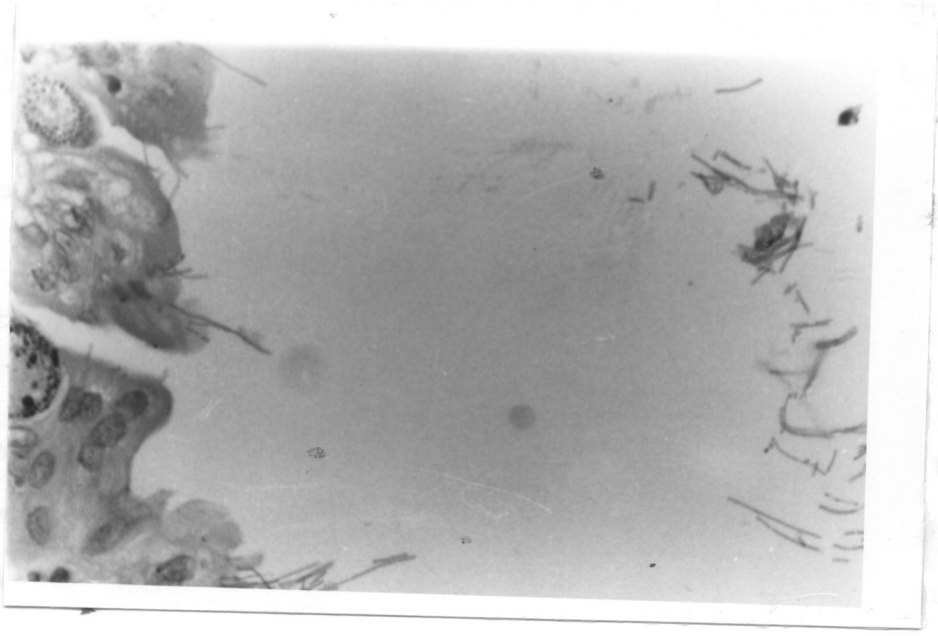


Fig 4.5.3b Filamentous bacteria on the villi and on the right of the villi. (x 40 mg)

concentration of IgA in caecal contents and in the numbers of IgA containing cells in the mucosa.

4.3.4 A/G Ratio

Many disease patterns are reflected by an alteration in the relative proportions of the two main groups of plasma proteins. In conditions of severe loss of albumin there will be a marked increase in globulins and the A/G ratio becomes less and is referred to as the inversion of A/G ratio (Kelly, 1984). From the results we may suggest that the A/G ratio ranged between 1.28 - 6.80 which is higher than the 'normal' range given by Kelly (1984) 0.63 - 3.90. However, as the albumin decreased the globulins increased in several cases. This change was noted during the 6th week in the first group and 1st week in the second group, which was the period when the animals showed symptoms of coccidiosis.

The statistical analysis carried out was useful for confirmation of the observations. The temperature measurements indicated statistical significant differences between week 6 and week 8. It was during week 6 that symptoms of coccidiosis were seen, hence one can suggest that high temperature is one of the symptoms. In the last week all the animals had a slight drop in temperature just before death. Also from this statistical analysis, one can confirm that other probable symptoms include drops in PCV, decrease in total protein, albumin and globulin.

4.4 Administration of Amprolium

There were no clear differences in the biochemistry, haematology or histology of goats treated with amprolium and those not treated except for the oocysts observed on wet smears (see Figs 3.6a and 3.6b). *The results do not give a clear indication whether the degeneration is due only to Amprolium. The lack of difference could be due to the short period between when the* animals were treated and when sacrificed (24 hours). The animals were too ill for any significant change to be seen in the animals in a short time. Thus this experiment did not clearly demonstrate at what stage of the life cycle Amprolium had its inhibitory effect. Ruff and Reid (1977) recommended 3-5 days of treatment with Amprolium are necessary and Roberson (1988) recommends 14 days for treatment of moderate or severe outbreaks of coccidiosis.

Amprolium is a thiamine antagonist and Roberson (1988) reported that it acts chiefly on the first generation schizonts, preventing differentiation of the merozoites. He further suggested that it also suppresses to some degree the sexual stages and sporozoites. Jeffers (1989) reported that the principal activity of most anticoccidial drugs is directed against the asexual haploid stages and can therefore very efficiently eliminate the more sensitive individuals in the population. The results obtained in this study could not confirm these observations, since degeneration of schizonts occurred in all animals both treated and not treated.

The mode of action of many anticoccidial drugs depends on the product, and the Eimerian species. E. tenella in chickens is resistant to sulfaquinoxaline and sulfamethazine, (Ruff and Reid, 1977). Amprolium is not very effective against E. maxima and E. mivati in chickens (Roberson, 1988). Some drugs act on different stages of the same species of Eimeria.

There is still a lot to be studied on the biochemistry of coccidia to understand the exact mode of action of the anticoccidial drugs. In the case of Amprolium future work should examine at which stage of the life cycle of coccidia it intervenes most effectively.

4.5 Life Cycle of Coccidia

4.5.1 Exogenous Stages

The life cycle observed in the 10 goat kids was similar to that described by Gregory and Norton (1986), Lima (1981) and Anon (1988) in goats. Various authors have described similar life cycles for coccidia in other animals. Pout (1976) describes the life cycle of coccidia in sheep and mentions that all species of coccidia have a similar life cycle with minor differences. Gregory et al (1987) also describes a similar life cycle in British sheep. Reid (1977) describes the life cycle of E. tenella in chickens, and Vetterling (1986) the endogenous cycle of the swine coccidium E. debliecki.

One difference which may be noted in these life cycles is in the number of schizont generations. In Zambian goat kids, two generations were observed and this is consistent with many other observations. Ruff and Reid (1977) reported that the number of asexual generations of coccidian parasites is usually a genetic property of the parasite, though the host may play a role in determining the number of schizogonic generations. Infection was natural hence there were mixed infections of coccidian parasites in this experiment. This made it difficult to successfully identify the species from the oocysts collected or from the histological results. The two main species identified were E. arloingi which was in abundance and E. ninakohlyakimovae. Alonge (1977) and Lima (1981) observed that past workers had not mentioned which species of Eimeria were present in their naturally infected animals and these observations may have involved mixed infections. Opoku-Pare and Chineme (1979) mentioned that the variation in the location of the plaques in the intestinal mucosa might be due to the different coccidian species involved. This was not apparent in our results, as lesions involved contained more than one species.

It was also noted that some oocysts, especially those small in size, sporulated between 2-3 hours after collection and floating in sucrose. This could have been due to the hot wet season in which the experiment was carried out. One would have assumed sucrose reduced the oxygen tension hence delaying sporulation.

Alonge (1977) reported that the majority of coccidial species in goats sporulate between 18-48 hours. Opoku-Pare and Chineme (1979) also reported that E. arloingi and E. ninakohlyakimovae sporulate at 24 and 48 hours respectively. The bigger the oocyst the longer it takes to sporulate. E. intricata took 72 hours to sporulate. Favourable conditions like adequate temperature, moisture, oxygen tension are excellent conditions for oocysts to sporulate to infective stages. It is also known that oocysts never sporulate inside the intestine of the host because of the prevailing anaerobic conditions, (Dürr and Pellerdy 1969).

4.5.2 Endogenous Cycle of Coccidia

It has been reported by many workers that Eimerian species are host specific and site-specific. It was not possible to come to this conclusion since we were unable to isolate one species in one infected area. Two or three species of Eimeria could be found in one locality which may mean that coccidia species may co-exist. This made it difficult to identify species from their schizonts or gamonts or merozoites.

There were, however, different sizes of giant schizonts, and even their location was not the same. Giant schizonts can be identified as pin-point white spots on the intestine (Gregory et al 1987), (Michael and Probert 1970). The giant schizont found within the lacteals of the villi resembled those described by Michael and Probert (1970) and Sing and Pande (1967 a, b) all these were attributed to E. arloingi. Giant schizonts could

also be seen in the serosa. Whether one should look at this phenomenon as a protective measure taken by the parasite since the immune system (Cellular Immune Response) was mainly in and around the villi is not known. The other reason could be that the mucosal tissue was too damaged and hence could not be an ideal site for further development of coccidia. Fig 4.5.2.1 shows two giant schizonts occupying the lumen of a villus. The small schizonts were observed to be adjacent to the gametocytic stages of the Eimerian species. The size, number of merozoites which ranged between 6-8 and location agreed with the report of Levine et al (1962).

Progamonts as first described by Gregory et al (1987), were similar to those we found in the goats studied. Two or three could be found occupying one epithelial cell (Fig 4.5.2.2). These could later develop into either all macrogametocytes or microgametocytes or some of each kind. Vetterling (1966) reported that as many as 5 parasites were found in a single epithelial cell. Lima (1981) also reported that gamonts were in the epithelial cells of the villi and crypts of the jejunum and ileum. The gamonts lay below the host cell nuclei and infections occurred with two or more in the same host cell. Fernando (1983), Michael and Probert (1970) Yvore et al (1980), Gregory et al (1987) all suggested that gametocytic stages are the most pathogenic. This could be mainly due to their large number.

From our study it was noted that in almost all the animals there was a section of villus showing microgametocytes only, (Fig 3.5.13). This could be due to immune response of the animal as the disease progressed. It could be suggested that the body produced some defensive mechanism which destroyed the macrogametocyte progamonts only leaving microgametocytes to develop into maturity. This may explain the phenomenon of why fewer oocysts are shed when the animal is critically sick. Since this is only a speculation more research work is needed to look into this.

Microgametes were released in large numbers and stained deeply with Mayer's Haemotoxylin. Some of them were found surrounding the macrogametocytes. On one of the photographs (Fig 3.5.1) there is a microgamete inside the macrogametocyte, whether one should suggest that fertilisation is taking place is a speculation. Vetterling (1966) reported that microgametes were seen in the clear vacuole surrounding the macrogamete suggesting that fertilisation had occurred earlier in the development of the macrogamete. The nucleus of the microgamete was seen inside the cytoplasm of the macrogamete.

In general the development of both asexual and sexual stages within the villus epithelia cells of the small intestine is often accompanied by villus atrophy. This change in the villus structure to blunt invagination of the villus and flat mucosa was reported by many other workers, Michael and Probert (1970), Gregory et al (1987), Pout (1967 and 1974), Fernando (1983).

There were no other parasites observed during the experiment neither during the clinical observations nor during the histopathological examinations. Goats are generally more susceptible to infection with nematodes than other of the gastro-intestinal parasites, (Lovelace et al, 1988 and Lloyd 1987).

The presence of filamentous bacteria was worth noting, Fig 4.5.3a (i) and (ii). These organisms were present as secondary infection since the tissue of the villus was badly damaged. Fernando (1983) reported that the environment within the intestine affects the pathogenicity of coccidial parasites, for example, the bacterial flora and nutritional status of the host. She was able to conclude that E. tenella is able to cause disease in both free and confined chicks and that the bacterial flora tends to increase the pathogenic effects of the parasite. This was also indicated from the work done by Visco and Burns (1972a). Dykstra and Reid (1978) reported that E. tenella stimulated an eight-fold increase in the numbers of the bacterium Clostridium perfringens in the caeca of monoflora chickens. This increased the pathological effects of this bacteria in sick birds.

Arakawa et al (1981), who were studying the simultaneous infections of coccidia and Salmonella e.g. S. typhimurium and E. tenella, discovered that if the two above species were given at the same time there was no effect on the number of Salmonella in the caeca. When Salmonella were given for five consecutive

days after inoculation with coccidia the number of Salmonella recovered seven, ten or fourteen days later increased in the caeca and liver, (Lafont et al 1983). Kim et al (1985) confirmed that the damage to the caecal mucosa was greater in chickens with simultaneous infections. Baba et al (1985) suggested that the increased number of Salmonella in the caeca is associated with decreased concentrations of volatile fatty acids. Our studies also showed goats with simultaneous infections of coccidia and bacteria, Fig 3.5.3a (ii).

Lantier et al (1981) however observed that, parasitism by E. falciformis increases the resistance of the mouse to a subcutaneous inoculation with Salmonella abortus ovis, though this effect is only observed if the coccidia are administered before or along with Salmonella inoculation. This therefore suggests that parasitic (coccidia) contamination can reduce bacterial development but in most cases it favours it. This may be due to pH, intestinal motility, local lesions or haemorrhage (Yvore, 1989).

Interactions between helminths and coccidia were not observed in the goats which we studied, though this is common in pasture farms or in stables where contamination by helminths occurs through litter, pasture or mothers milk (Yvore and Esnault, 1986). Stewart et al (1980) analysed the effects of E. nieschulzi and Trichinella spiralis in the rat inoculated simultaneously 9 days after inoculation by coccidia. Their observation was a faster elimination of the worm population. They proposed that this elimination could be due to non-specific

inflammatory phenomena produced by the coccidia that make the intestinal micro-environment unfavourable for the helminth. This could be also due to changes in the morphology of the villi and crypts caused by the development of coccidia. Catchpole and Harris (1989) observed that a single dose of E. crandallis, and E. ovinoidalis with 30,000 infective larvae of Namatodirus battus given to 3-5 week old lambs caused only transient diarrhoea and had no effect on growth. Then the lambs were infected first with coccidia and two weeks later with N. battus. The lambs suffered severe diarrhoea, weight loss and some died.

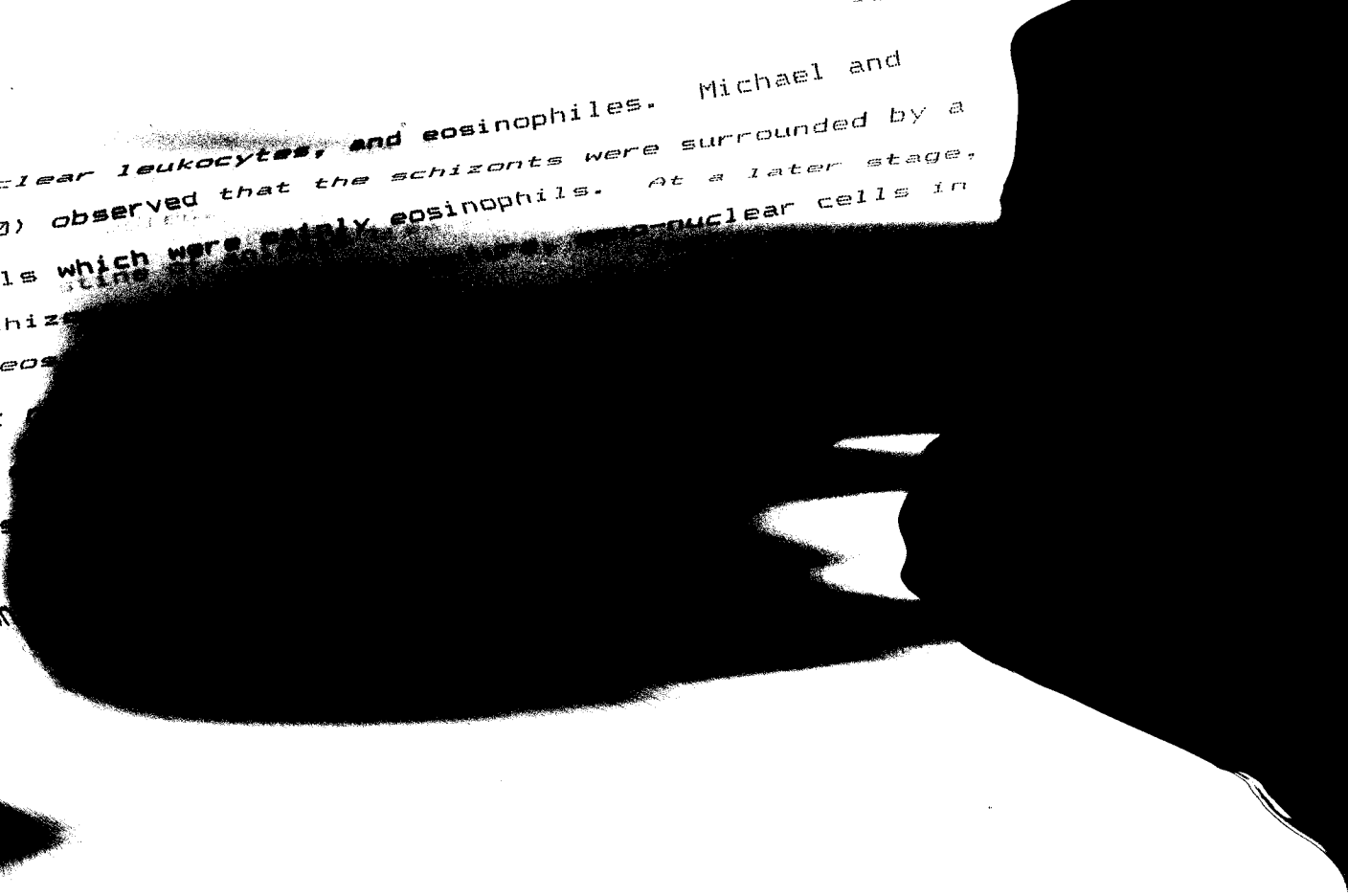
Luangwa goats (Zambia) studied by Lovelace et al (1988) showed a variety of nematode eggs. 100% of the 20 goats tested had Haemonchus and Strongyloides, 80 and 95% had Oesophagostum and Trichostrongylus respectively. It is likely that these would be occurring alongside a low level of chronic coccidial infection under village conditions.

4.6 Immune Response

Histopathological findings suggested that Cell-Mediated Immunity (CMI) was very significant in all the goats especially in reaction to giant schizonts. Britten and Hughes (1986) reviewed that Cell-Mediated Immunity has shown to be an important mechanism of protective host response against many intracellular infections. Macrophages and granulocytes can express natural cytotoxicity (Herberman et al 1986). Rose (1986) suggested that immunity to reinfection in chickens by coccidia is dependent

upon cell mediated immunity. Jeurissen et al (1989) reported that a single infection confers immunity to reinfection with the same species. Both humoral and cellular immune responses are involved in this protective immunity. Rose et al (1979) reported on the reaction of non-lymphoid cell populations such as macrophages and granulocytes. Bekhti et al (1989) suggested that the relative contribution of the different effectors of the immune response may vary according to the invading species and according to the previous status of the host. Lillehoj (1986) studied local T-Cell-Mediated responses and reported that, after the initial infection with coccidia a long-lasting species-specific immunity develops, characterised by strong antigen-specific lymphocyte responsiveness in vitro. This suggests that cell-mediated immunity plays a predominant role in the host protective immune response.

From our results, (Figs 3.3.2.1 and 3.8.3) CMI was likely to have been occurring in the villi, around and inside the giant schizonts. Many of the leucocytes were eosinophiles and other granulocytes which could not easily be identified. Augustine and Danforth (1989) reported that the specific antigens that produce immunity have not been quite identified. They suggested that the specific antigens are associated with the schizogonic stages of the parasites' life cycle. Rose (1973) also noted the same response. Lima (1981) reported that there was a focal infiltration of lymphocytes and plasma cells around the gamonts and oocysts. The cellular reaction in the lamina propria and sub-mucosa consisted of lymphocytes, macrophages, plasma cells,



Components of the humoral immune response from serum and intestinal fluid have been studied (Juerissen et al 1989). Augustine and Danforth (1989) observed that Monoclonal antibodies that reacted with antigens located on the surface and in refractile bodies of avian Eimeria sporozoites, showed to have significantly reduced cellular invasion and intracellular development of these parasites. Crane et al (1986) reported that sporozoites were lysed during an in vitro incubation with antibodies and complement. Davis and Porter (1979) ascribed the mechanism of the immunity to sporozoite-specific secretory IgA in the lumen of the caeca in chickens.

There were different forms of schizont degeneration observed in the goats studied, but whether this was only due to cellular immunity or to both humoral and cellular immunity still remains to be proved. In some cases one found the contents clumped together, or patches inside the schizonts appeared whitish or reddish. Filamentous structures could be seen on the periphery

polymorphonuclear leukocytes, and eosinophiles. Michael and Probert (1970) observed that the schizonts were surrounded by a layer of cells which were mainly eosinophils. At a later stage, when such schizonts were about to rupture, mono-nuclear cells in addition to eosinophils were found. Bekhti and Pery (1989) observed that neutrophils played a central role in host defences. In our study cellular infiltration was mainly around giant schizonts. There was no clear cellular response to the small schizonts or gametocytes.

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of the giant schizonts. There was no degeneration observed in gamonts in goats studied. Degenerating oocysts were only found free in the intestine of animals treated with Amprolium.

The mechanisms responsible for protective immunity against coccidiosis are still not understood especially in goats where both humoral and cellular immune response have not been studied in detail.

4.7 Improvement in Management Practices

Since goats are more resistant to trypanosomiasis and are able to survive in areas which cannot support cattle, they are increasingly becoming an alternative source of protein in many third world countries. Natural disasters like drought, bush fires, diseases and parasitism in the animals has brought constraints on the production of domestic animals especially cattle and small ruminants. Revaluation of overvalued exchange rates which previously encouraged imports and discouraged exports means that internal production is becoming increasingly important.

Coccidiosis is one of the major diseases found in young goats in commercial farms. This is due to intensive management practices. In rural areas coccidiosis also occurs and poor hygiene conditions could be the cause. Gregory (1989) and Fayer (1989) also reported that crowding around water and feeding areas might concentrate oocysts, and also climatic stress may render the animal to be more susceptible to infection.

Healthy adult animals serve as carriers for coccidiosis and will shed oocysts but rarely develop clinical signs of infection unless severely stressed (Fayer 1989). Confinement can be recommended only if areas are cleaned daily and bedding changed regularly, since the number of infectious oocysts in the environment is a result of the number shed per host. Feed troughs should be raised and fitted with an elevated step to reduce infection on the food from the faeces and the animal can adopt a browsing position. Treatment of goats against helminths and coccidia is recommended to those farmers who can afford it.

There is now a greater emphasis on prevention of illnesses which can affect feed efficiency and growth (Fayer, 1989) and thus production. To ensure better returns from goats, education about this disease is required for both commercial and peasant farmers. It is desirable to control parasitic diseases by preventing reinfection. In the case of coccidia the animal is susceptible to infection throughout their lives, though resistance develops with age.

The following are measures which might improve animal production: education of farmers in grass conservation, flexible veterinary services to reduce mortality, improvement of the efficiency of the extension services, increase in investment in livestock research and improvement of the marketing procedures.

The peasant farmer, in addition to the above, can be encouraged to improve husbandry practices by providing better nutrition during the dry season, by preserving the abundant fodder available during wet season for use during dry season. In-breeding and early conception should be avoided by separating the sexes and early castration of males not selected for reproduction could be carried out.

Coccidiosis is clearly a serious disease in Zambian goats and research is still underway. Considering our results obtained from the two Zambian Commercial Farms and a village, coccidia are still a very important problem in goat management. The economic impact of Eimeria spp is not fully understood as the parasite was often found to occur with other parasitic species in goats. The effect of each may be cumulative.

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