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Haematological Studies on West African Dwarf (WAD) Bucks Experimentally Infected with Trypanosoma Vivax and Trypanosoma Brucei and Response to Treatment with Diaminazene Aceturate

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Abstract- This study investigated the haematological changes in West African Dwarf (WAD) bucks experimentally infected with Trypanosoma vivax and Trypanosoma brucei. Each of the group is eight in number while the control experimental group had five bucks. Clinical records (weight, rectal temperature) for the animals were monitored. The haematological parameters accessed include packed cell volume (PVC) estimation of Haemoglobin (HB) White and Red Blood Cell count (WBC and RBC) mean corpuscular Haemoglobin (MCH), Mean Corpuscular Haemoglobin concentration (MCHC) and were calculated accordingly.

The PVC valves varies from the pre-infection levels of 21.4 – 23.0 and 24.0 – 1.9.9 in the 1st – 3rd for T. brucei and T. vivax respectively to infective stage 21.5 – 20.3 and 22.8 –13.5 as the anaemia progressed. The effect of the infection for T. vivax and T. brucei was acute and chronic respectively as the infection was more severe in T. vivax than T. brucei. Knowing that the WAD bucks are trypano tolerant however, the effect of the parasite on the haematological features showed that anaemia was normocytic and normochronic for most periods. The intensity of the anaemia was related to the degree of parasitemia and in case where the animals are infected adequate dietary measures and proper sanitation need to be taken to ensure productivity is not hindered.

Keywords: haemalotogical changes, parasitemia, trypanosoma, anaemia.

I. Introduction

Trypanosomiasis is an infective disease which affects domestic and game animals including man. It is caused by flagellated protozoan parasite of the genus Trypanosoma and transmitted mainly by different species of tsetse fly of the genus Glossina [9]. Trypanosoma vivax, Trypanosoma congolense and Trypanosoma brucei are the main species of trypanosome of importance in livestock, that cause Animal Africa Trypanosomaisis (AAT) [1]. Trypanosomiasis is a major constrain on livestock production in Africa and of all the livestock diseases endemic on the African continent, trypanosomisis has been regarded as the single factor which limits the number and productivity of ruminant; sheep, goat and cattle. It is known to render approximately a quarter of African arable land mass unsuitable for profitable livestock farming [18]. Reminants; cattle, goat and sheep represent an important source of animal protein in many countries of world. Supplying a good percentage of the daily meat and dairy products in cities and villages in many countries including Nigeria [22]. Apart from being a source of animal protein, their waste are also very important in agriculture [23]. Ruminants like goat and sheep are used in special ceremonies such as weddings and burial in Nigeria. However, parasitic diseases like trypanosomiasis coupled with inadequate management practices, hamper the productive husbandry of these animals [25]. In infected areas, the disease may result in severe reduction in animal productivity reflected in poor growth, low milk production and meat yields, reduced capacity for work and financial loss in terms of veterinary controls. If these infected animals are left untreated animals may die of anaemia, heart failure, and inter-current bacterial infections that take advantage of the animals weakened resistance or suppressed immune system. The economic impact of the disease trypanosomiasis on these animals has been shown to be substantial [17]. Response to infection by trypanosomiasis may be influenced by the stress of work, intercurrent disease, poor nutrition etc. [21]. Drug treatment remains the only means of intervention, there is no vaccine against trypanosomiasis and prospects of vaccine are very poor owing to the significant antigenic variation exhibited by the trypanosome [13]. There were initial suggestions that indigenous sheep and goats are more resistant than imported exotic breeds to syringed or needle passed Trypanosoma vivax as well as field challenged [8]. Various breeds of livestock have been re-cognized as having degrees of tolerance to trypanosomiasis enabling them to survive and produce in areas where

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other breed could succumb [12]. [15] reported that Trypanosoma vivax and trypanosoma congolense were the most prevalent species encountered in sheep and goat because of their grazing requirement which compels the animals to traverse different vegetation zones especially during the dry season to the Southern areas of Nigeria many of which are tsetse fly infected. Infection in these animals causes symptoms manifested by intermittent fever, anemia, pyrexia, lymphatic enlargement with hepatomegly and a progressive cachexia [5] .However, the severity of the infection in a host animal is influenced by a number of factors: virulence of the different special of trypanosoma, environment of the host, age, nutritional status, weight etc. [20]. This work was carried out to investigate the etiology of the disease trypanosomiasis and the haematogical changes in the West African Dwarf (WAD) bucks when infected with Trypanosoma vivax and trypanosome Bruce their susceptibility to the infection and response to treatment with diaminazene aceturate.

II. Materials and Methods

a) Study Area

The Study was carried out at the experimental house of the Animal science Department of Michael Okpara University of Agriculture, Umudike Abia State. The University is located at about longitude 7032'East and latitude 5029; North 129 M2 above sea level.

It has warm hound climate and temperature that ranges from above 290C in the wet season to slightly over 250C in the hot season Umudike falls within the rainforest zone of south Eastern Nigeria with a mean altitude of 123m.

b) Experimental Design

21 West African Dwarf (WAD) bucks were divided into 3 groups as follows.

Group A: 8 WAD bucks were infected with trypanosome brucei

Group B: 8 Wad Bucks were infected With trypanosome vivax

Group C: 5 WAD bucks were uninfected (control)

To infect the designated bucks in group A 4ml of blood was obtained from mice inoculated with Trypanosoma brucei and diluted with 1ml of normal saline, ml of the diluents was used to infect the WAD bucks through the jugular vein. To infect the designated bucks in group B 3ml of blood was obtained from a WAD buck inoculated with Trypanosoma vivax and diluted with Iml of normal saline, 1ml of the diluents was used to infect the WAD bucks in group B through the jugular vein. The animals were intensively maintained on Dry hay, water and concentrate adlibidum throughout the experiment. During the period of acclimatization which lasted for 21 days the animals were dewormed with levamisole, vaccinated against PPR (Peste des petil Ruminant virus) and treated with diaminazene aceturate (Berenil R) at 0.3 0.25ml to clear any possible protozoan infection, haemoparasite and trypanosome. Clinically, the rectal temperature was taken twice daily (morning and evening), respiratory rate, heart rate and body weight was recorded weekly. Other treatment were given appropriately after this period, 8 of the WAD bucks in Group A and Group B were infected into the jugular vein with 1ml of the diluents. Animals in both groups were treated with diaminazene aceturate (Berenil R) 0.30-035ml at the 8th week and 13th week respectively.

III. Sample Collection

A total of twenty one (21) West African Dwarf (WAD) bucks all makes were bled from the jugular vein after sterilizing with methylated spirit using cotton wool, Iml of Blood was collected with a 4ml vaccutainer and a disposable hypodermic syringe blood was drawn from the jugular vein into the EDTA (Ethylene diaminetertra acetic acid) vaccutainer container already prepared EDTA overnight and allowed to evaporate. These blood were thoroughly mixed to prevent clotting and lysing of Red blood cells. The samples were then transferred to the laboratory for further investigation. Samples were collected once a week between the months of June to October.

IV. Haematological Methods and Parameters Studied

Animal were examined before and during infection Packed Cell volume (PCV) was determined by micro-haematocrit method, Red and White Blood Cell (RBC and WBC) Count were estimated by the use of Neubauer-ruled haemonytometer and haemoglobun concentration (Hb) by the Acid haematin Concentration Method. Mean Corpuscular Haemoglobin (MCH), Mean Corpuscular Haemoglobin concentration (MCHC) and Mean Corpuscular Volume (MCV) were monitored weekly and Calculated according to [26]. Weight, rectal temperature, colour of mucous membrane were also monitored.

V. Results

Trypanosomes were first detected in the blood of the WAD bucks infected with T. vivax followed by the WAD bucks infected with T. brucei. The control WAD bucks remained trypanosome free throughout the period of investigation as no trypanosome was detected in their blood. As the infection progressed, the T. vivax and T. brucei showed acute and chronic form of the disease trypanosomiasis respectively.

Clinical signs: following infection of the WAD bucks with T. vivax and T. brucei, trypanosomes were detected in blood by microscopic examination of the bufty coat within the first 5th week of infection in Group B. No infection was detected in Group A. The clinical disease was characterized by marked pyrexis at an average of

390C. The temperature fluctuated daily during the period of infection, infected WAD bucks were emaciated with very pale mucous membranes anorexic with facial and sub mandibular oedema, ocular discharges and they showed signs of dullness. All animals infected showed a decreases in total body weight.

VI. Haematolagical Changes

With the onset of parasitemia, all the infected WAD bucks developed anaemia with a drop in erythrocyte (PCV, RBC, HB Values) Table 1-4. These reflected in the 5th-6th week when the animals become recumbent or reached the critical erytrocyte levels. The PCV value varied from 25-5-21.9 for the control, 25.4-19-3 for the T. brucei and 18.6-12.9 for T. vivax. The Hb value varied as follows 8.5-7.3 for the control, 8.46-6.44 for the T. brucei and 6.2-4.3 for the T. vivax (Figs 1,2,&3).

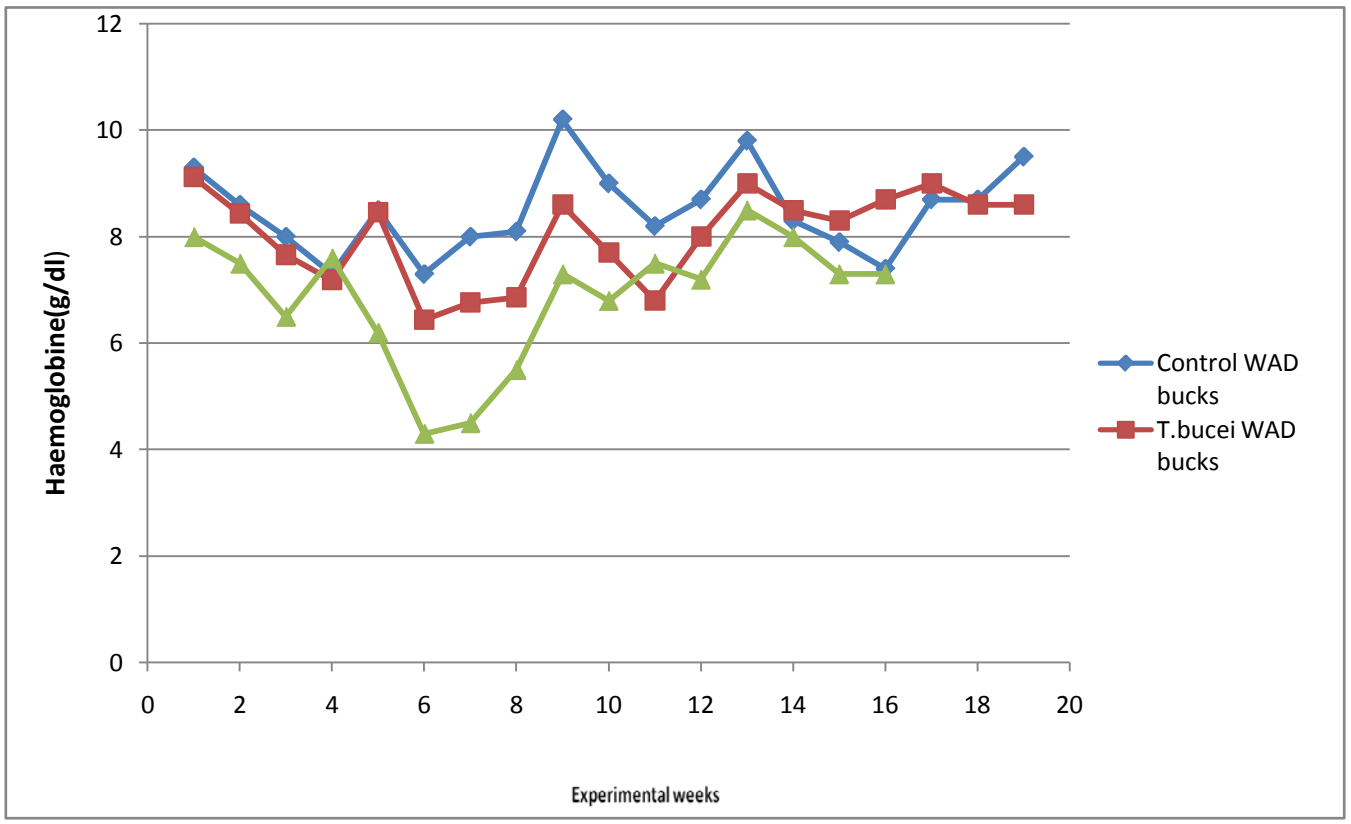

Figure1 : A Line graph showing values of Haemoglobin concentration for the period experiment

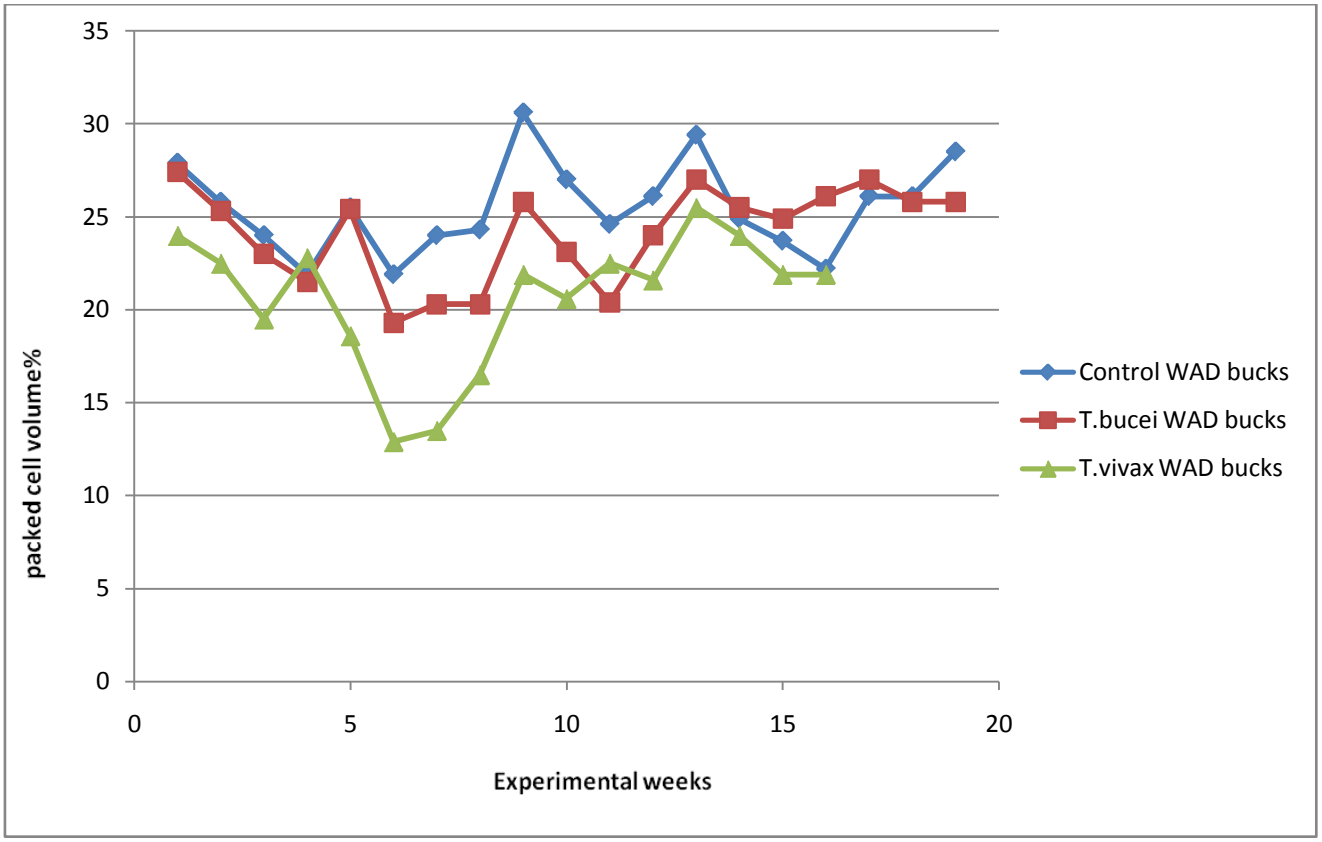

Figure 2 : A line graph showing values of packed cell volume for the period of experiment

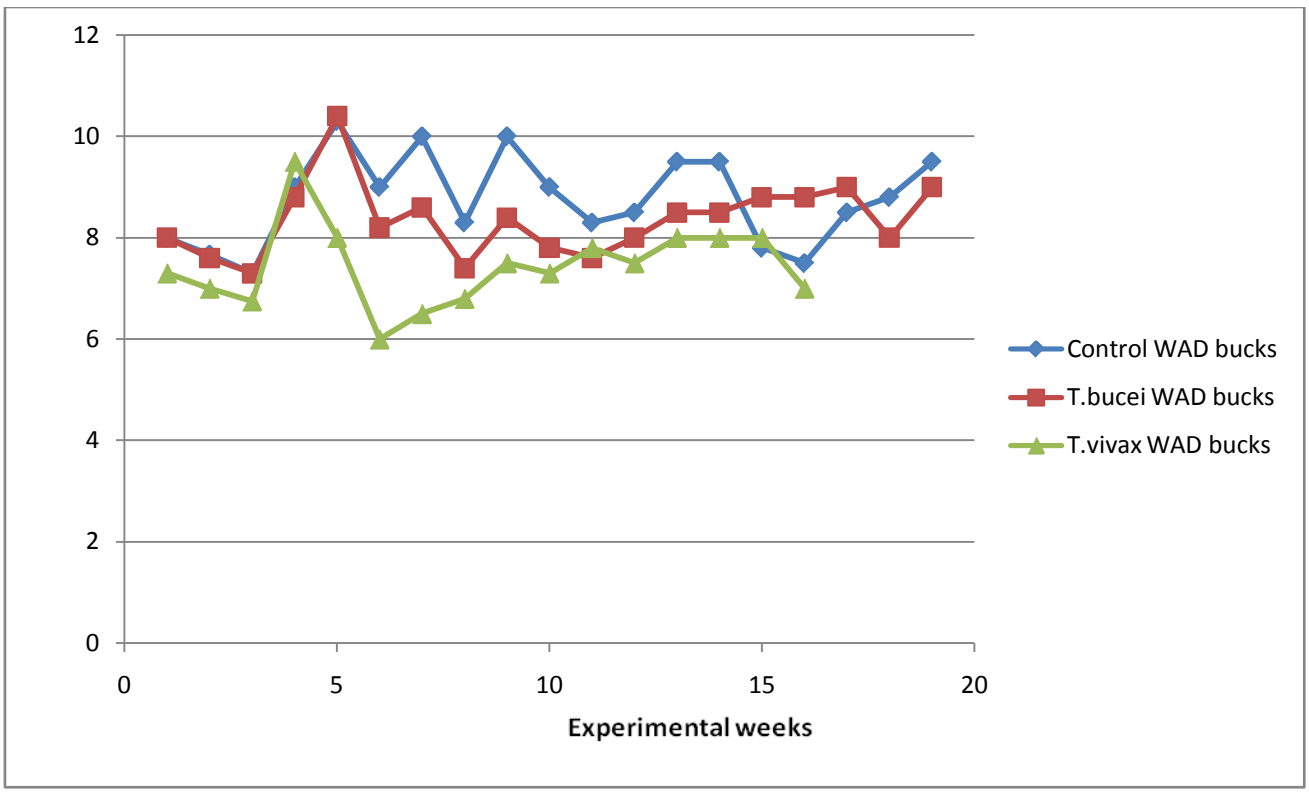

Figure 3 : A line graph showing values of White blood cell counts for the period of experiment

The anaemia developed progressively during the experiment. There were no appreciable variations in the erythrocyte values and with T. brucei but there were appreciable variation in the erythrocyte values of the WAD bucks infected with T. vivax. However, the mean MCV values of infected WAD bucks fluctuated but did not vary significantly for the normal values before infection. MCH values during infection relatively followed the pattern of MCV changes. There was significant variation in the MCHC values during the experiment, the mean total of WBC counts during the infection fluctuated but increased during the week of infection of the WAD bucks. By the end of 7th week of infection the animals that survived were treated with 0.3-0.35 mi/kg of diaminazene aceturate (berenil R) in group B, while that of group A were treated and the end of 13th week of infection and rapidly recovered. Parasite were not detectable in the blood following treatment and relapses were not encountered following an observation period of 12weeks and 6 weeks respectively for the WAD bucks in group B and A. Parasites were not encountered following an observation period of 12weeks for WAD bucks in group B and 6 weeks group A.

Table 1 : Mean values of red blood cells RBC (x106ul) and estimation of haemoglobin (Hb) for the period of experiment

Weeks	Mean values of RBC (x106ul)			Mean values of Hb g/dl		
	C	Tb	Tv	C	Tb	Tv
1	5.75	5.65	4.95	9.3	9.12	8.0
2	5.30	5.25	4.65	8.6	8.44	7.5
3	4.90	4.50	4.05	8.0	7.66	6.5
4	4.55	4.15	4.40	7.3	7.18	7.6
5	4.95	4.85	3.60	8.5	8.46	6.2
6	4.55	3.75	2.55	7.3	6.44	4.3
7	4.95	3.9	2.65	8.0	6.76	4.5
8	5.00	4.25	3.4	8.1	6.86	5.5
9	6.25	4.95	4.45	10.2	8.6	7.3
10	5.55	4.40	4.15	9.0	7.7	6.8
11	5.05	3.95	4.55	8.2	6.8	7.5
12	5.35	4.60	4.35	8.7	8.0	7.2
13	6.05	5.40	5.25	9.8	9.0	8.5
14	5.15	5.15	4.85	8.3	8.5	8.0
15	4.85	5.05	4.85	7.9	8.3	7.3
16	4.55	5.25	3.65	7.4	8.7	7.3
17	5.35	5.60	---	8.7	9.0	---
18	5.35	4.85	---	8.7	8.6	---
19	5.85	5.60	---	9.5	8.6	---

	Pre-infective phase	Infective phase	Treatment phase	
Tb -	*Trypanosoma brucei*	Week 1-3	Week 4-12	Week 13-19
Tv -	*Trypanosoma vivax*	Week 1-3	Week 4-7	Week 8-19
C -	*Control.*	------	-------	-------

Table 2 : Mean values of packed cell volume (PCV) Mean corpuscular haemoglobin (Hb) g/dl for the period of experiment

Mean values of PCV						
				Mean values of MCH		
Weeks	TC	TB	TV			
1	27.9	27.4	24.0	16.2	16.1	16.2
2	25.8	25.3	22.5	16.2	16.1	16.1
3	24.0	23.0	19.5	16.3	17.0	16.0
4	21.9	21.5	22.8	16.0	17.3	17.3
5	25.5	25.4	18.6	17.2	17.4	17.2
6	21.9	19.3	12.9	16.0	17.2	16.9
7	24.0	20.3	13.5	16.2	17.3	17.0
8	24.3	20.3	16.5	16.2	16.1	16.2
9	30.6	25.8	21.9	16.3	17.4	16.4
10	27.0	23.1	20.6	16.2	17.5	16.5
11	24.6	20.4	22.5	16.2	17.2	16.5
12	26.1	24.0	21.6	16.2	17.4	16.6
13	29.4	27.0	25.5	16.2	16.7	16.2
14	24.9	25.5	24.0	16.1	16.5	16.5
15	23.7	24.9	21.9	16.3	16.4	15.1
16	22.2	26.1	21.9	16.3	16.6	20
17	26.1	27.0	----	16.3	16.1	----
18	26.1	25.8	----	16.3	15.4	----
19	28.5	25.8	----	16.2	15.4	----
		Pre-infective phase	Infective phase	Treatment phase		
Tb - *Trypanosoma brucei*		Week 1-3	Week 4-12	Week 13-19		
Tv - *Trypanosoma vivax*		Week 1-3	Week 4-7	Week 8-19		
C - *Control.*		------	-------	-------		

Table 3 : Mean values of mean corpuscular volume (MCV) in femto litres (FL) and haemoglobin (Hb) in percentage (%) for the period of experiment

Mean values of MCV			Mean values of HB (%)			
C	Tb	Tv	C	Tb	Tv	
1	48.9	48.6	48.9	63.7	62.5	54.9
2	48.6	48.5	48.4	58.9	57.8	51.4
3	48.5	48.4	48.2	54.8	52.5	44.5
4	48.3	51.8	52.0	50.0	49.2	52.1
5	48.6	52.1	52.0	58.2	57.9	42.5
6	48.3	52.0	50.2	50.0	44.1	29.5
7	48.5	51.9	51.2	54.8	46.3	30.8
8	48.5	51.2	48.7	55.5	47.0	37.7
9	48.8	52.3	49.4	69.9	58.9	50.0
10	48.7	52.1	47.5	61.6	52.7	46.9
11	48.5	52.1	49.4	56.2	46.6	51.4
12	48.6	52.2	47.5	59.6	54.8	49.3
13	48.7	51.5	49.5	67.1	61.6	58.2
14	48.5	48.1	48.8	56.8	58.2	54.8
15	48.5	49.2	49.5	54.1	56.8	50.0
16	48.4	49.7	49.3	50.6	59.6	50.0
17	48.6	50.0		59.6	61.6	
18	48.6	49.5		59.6	58.9	
19	48.7	50.0		65.1	58.9	
		Pre-infective phase	*Infective phase*	*Treatment phase*		
Tb - *Trypanosoma brucei*		*Week 1-3*	*Week 4-12*	*Week 13-19*		
Tv - *Trypanosoma vivax*		*Week 1-3*	*Week 4-7*	*Week 8-19*		
C - *Control.*		------	-------	-------		

Table 4 : Mean values of Clinical parameters monitored

Clinical parameters	Tv	Tb	C
Weight (kg)	8.0 ± 2.0^{a}	6.0 ± 1.6^{b}	10.0 ± 2.0^{c}
Rectal temperature (°C)	39.16 ± 0.27^{a}	39.16 ± 1.0^{a}	30 ± 0.05^{b}
Respiratory rate (cpm)	40 ± 10^{b}	30 ± 10^{a}	30 ± 10^{a}
Heart rate (1pm)	90 ± 30^{a}	90 ± 20^{b}	90 ± 30^{a}

Means in the same row with different superscripts are Significantly different (P<0.05)
Tv Trypnosoma vivax
Tb Trypnosoma brucei
C control

VII. Discussion

The haemotological Values of the parameters monitored revealed that Trypnosoma vivax and Trypanosoma brucei infected WAD bucks showed acute and chronic course of trpanosomiasis respectively while values of the control animals remained within the normal levels (Tables 1-3).

There was a rapid development of anaemia in T. brucei and T. vivax infected WAD bucks with the PVC dropping as low as 27.9-23.0 and 24.0-19.5 respectively.

This was a more serious anaemia than that previously recorded by [19], he observed 0.25 to 0.30 in T. brucei infection but less severe than PVC value of 0.11 recorded in naturally T. brucei infected bucks [16]. Although clinical symptoms associated with trypanosomasis observed in this study include high rectal temperature, ocular discharge, decrease in weight and anaemia severity of the disease and more in T. vivax infected WAD bucks and more pathogenic than those of T. brucei infected bucks. This is similar to work of previous researchers [16][27][2][5] and [14]. They observed such symptoms as rectal Temperature fluctuation, pale mucous membrane, weakness, anaemia among others also infection with T. brucei had nervous system disorder. Anaemia which is a major consequence of the disease contributed more to the outcome of the infection than any other pathological entity and was characterized by depressed erythrocyte values. This result is in agreement with observation of [16] and [3]. They recorded that if the infection is left untreated could lead to death of the animal.

From the Pre-infection levels of 27.4-23.0 and 24.0-19.5 in the 4^{th} to 7^{th} and 1^{st} to 3^{rd} week for T. brucei and T. vivax respectively and as it progressed was found to be normacytic and normochronic for most periods and its intensity was related to the degree of the parasitemia. There was an increase within 4^{th}-5^{th} week in the MCH Values of infected bucks and this is correlated with an increase in the MCV values within the same period (table 4). It is noteworthy that the rise in MCH values was observed at the onset of anaemia and similar observation was made by Naylor (1971) in T. Congolese infected cattle. The increase in MCH and MCV values were observed due to increased erythropoiesis indicating that erythorid response peaks as the anaemia enrages.

The failure of the bone marrow to generate sufficient erythrocytes was partly responsible for persistent anaemia as indicated by low PCV values during the 4th-7th week (fig 3) of infection. The level of Parasitemia is concurrent with a relatively stable reduction in Hb and RBC levels during the chronic phase of infection. This is in keeping with the development of anaemia which was more pronounced during this period and also presumptive evidence of possible damage to the host cells and tissues by the invading trypanosomes [4], [7], [6].

Animals given good nutrition and rest are more likely to recover rapidly than undernourished and stressed animals. No vaccines are available against trypanosomiasis and prospect of vaccines are very poor owning to the significant antigenic variation exhibited by trypanosome [13]. Therefore a tsetse fly eradiation campaign can be conducted to help reduce the transmission of trypanosomiasis. The use of drugs or chemoprophylaxis and chemotherapy for the prevention and treatment of trypanosomiasis has also been effective [11].

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