

# Clinical signs, daily rate of infection, physical changes of the blood and pathomorphological changes in cattle artificially infected by *Trypanosoma vivax*

Signes cliniques, taux d'infestation journaliers, modifications hématologiques et pathomorphologiques sur du bétail infesté artificiellement par *Trypanosoma vivax*

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## RESUME (\*)

L'auteur décrit l'évolution, les symptômes, les modifications hématologiques et les lésions de l'infection expérimentale du bétail par *T. vivax*.

La maladie suit une évolution subaiguë aboutissant à la mort de 6 des 8 animaux infestés.

Les modifications hématologiques montrent une anémie qui se développe dans les deux semaines précédant la mort.

Les lésions sont caractérisées par des signes de septicémie hémorragique, particulièrement sur le cœur, la rate, les ganglions lymphatiques et le foie.

## INTRODUCTION

Animal trypanosomiasis is a notifiable disease in Ghana. During the period 1960 - 1965, 943 outbreaks were reported to the Ministry of Agriculture (NYARKO, 15). The peak of the reported cases was in July and August (103 and 102 outbreaks). In the other months the frequency of the disease varies from 67 to 79 cases. NYARKO (15) considers *Trypanosoma vivax* as the most important cattle trypanosoma in Nigeria and possibly in West Africa as a whole. Differences in the pathogenicity with location are quite considerable. It is generally quite virulent in West Africa but mild in East Africa. The same conclusions were arrived at by HENNING (8), JOWETT (12), HORN-

BY (9), CURSON (2) and PARKIN (16). PARKIN (16) stated that in South Africa if animals suffering from infection with the Zululand strain of *T. vivax* were kept under good conditions and fed well the disease rarely proved fatal. On the other hand, HUDSON (10) studied in bovines an unduly severe and rapidly fatal form of *T. vivax* infection resembling the peracute disease in pigs caused by *T. simiae*. Trypanosomes were usually found in great concentrations in the blood, particularly in smears taken just before and after death. METTAM (13, 14) and STEWART (18, 19) found out that *T. vivax* was responsible for many severe outbreaks of trypanosomiasis in West Africa, and appeared to be at least as serious as *T. congolense*. The clinical and pathological picture described corresponds to the lesions during haemorrhagic septicaemia. NYARKO (15) described three forms of try-

(\*) Un résumé approfondi en français figure en conclusion de l'article.

panosomiasis in cattle in Ghana : acute, chronic and cryptic form. The severity of the disease in West Africa is described by JONES-DAVIES (11) in Nigeria when he found the strains of *T. vivax* resistant against Berenil. FIENNES (6) published the results of autopsies on cattle infected by *T. vivax* at Kiboko in Kenya, and a wide review of the literature on course and pathology of trypanosomiasis was published by FIENNES, JONES and LAWS (4).

## MATERIALS AND METHODS

The experiment was conducted at the Agricultural Research Station, Nungua, Faculty of Agriculture, University of Ghana, Legon. The Station is located 20 miles east of Accra in a gently rolling country of low elevation typical of the western Accra Plains. The average annual rainfall is 32 ins. with recorded extremes of 17 to and 53 ins., of a double-peaked monsoon regime. The major rains extend from March to July with a peak in June, the minor rains from September to November with the peak in October. The atmosphere is humid and temperatures high but both are offset by cooling off-sea-breezes. The Station does not belong to the typical tse-tse infested area and only very few cases of animals infected by trypanosomes are found. The experiment started on 26th of June, 1967.

A group of 8 cows including 3 West African Shorthorns, 4 N'Damas and 1 Sokoto Gudale were selected. All of them were culled animals over 8 years of age. They were examined four times during the week before the start of the experiment for blood parasites, and none were found. The blood picture was in normal range and the clinical examination did not reveal any pathological changes.

The animals were infected subcutaneously with 2 ml of virulent blood collected from Sokoto Gudale bullock, who was transferred to A.R.S. Nungua from Agricultural Irrigation Research Station, Kpong, Volta Region, a typical tse-tse area. The number of trypanosomes in 100 fields in the Sokoto Gudale bullock on 26th June was 67 parasites. The blood smears were sent to the Institute of Parasitology, Academy of Sciences, Praha, for classification. The trypanosomes were typified as *Trypanosoma vivax* (length 14.7 - 20.5  $\mu$ , maximum width

1.4 - 2.9  $\mu$ , nucleus 1.4 - 2.2  $\mu$ , distance of the nucleus from the anterior end 4.4 - 8.8  $\mu$ , blepharoplast located terminally or sub-terminally).

Clinical examination was daily for temperature, pulse, respiration and general clinical stage. Blood examination was performed every other day (RBC and WBC improved Neubauer counting chamber, PVC microhaematocrit, HB p. 100 MRC grey wedge photometer, ESR Wintrobe tubes, haematological indices, white cells differential counts; anticoagulant used, double exalate mixture. The number of parasites in 100 fields in thick drop was counted daily.

## RESULTS

### *Clinical signs*

The alteration of trias in artificially infected cattle started the 6th day after inoculation and remained altered for 1 or 4 days. The body temperature was mostly affected while the pulse rate and respiration did not always show acceleration above average (Gr. n° 1). During the following course of infection the trias varied within normal range. On day of death, temperature dropped deeply below normal. In two animals that survived the infection, the trias became normal one or two days after initial rise in temperature. The rise in temperature co-incided with the appearance of parasites in the blood (N'D 10, N'D 12) or appeared 1 day before (WAS 133), 2 days before (WAS 45), 4 days before (N'D 11, N'D 5, SG.62), or 1 day after the parasites could be revealed in the blood (see Tab. n° 1). The number of parasites varied considerably and did not seem to be correlated with clinical changes.

The other clinical signs of animals that died within 9 weeks after infection (WAS 45, WAS 108, WAS 133, N'D 11, N'D 5, N'D 10) : (Tab. p. 256).

The two animals (N'D 12, SG.62) that survived developed the following clinical picture : N'D 12 :

On 7th to 8th day after infection, lacrimation from both eyes, listlessness.

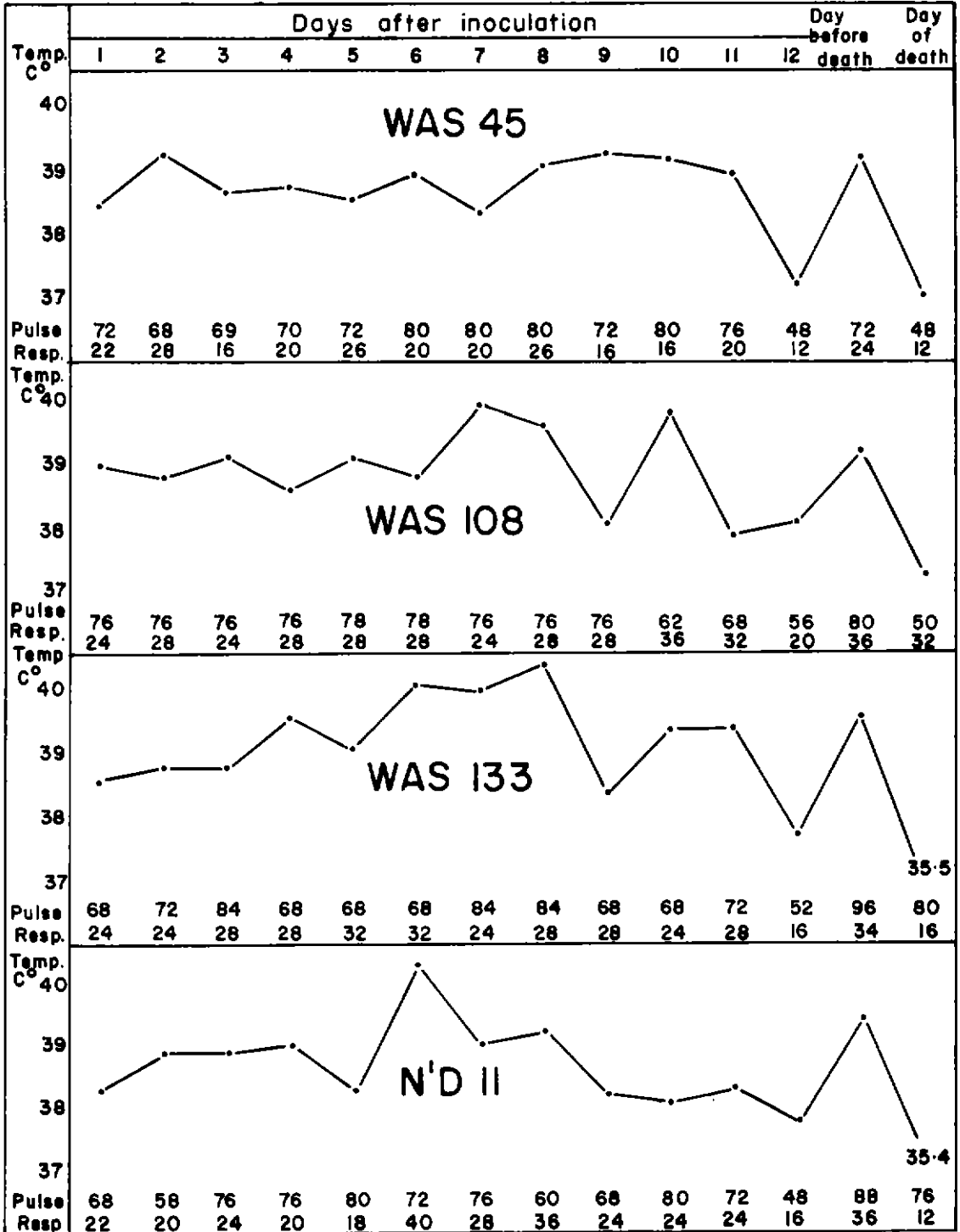
On 29th day bottle jaw started.

On 32nd day the head got swollen, scouring.

On 49th day the bottle dissolved and scouring stopped.

Temperature, pulse rate and respiration of  
artificially infected cattle

Gr. No.1.



Gr. No I. contd.

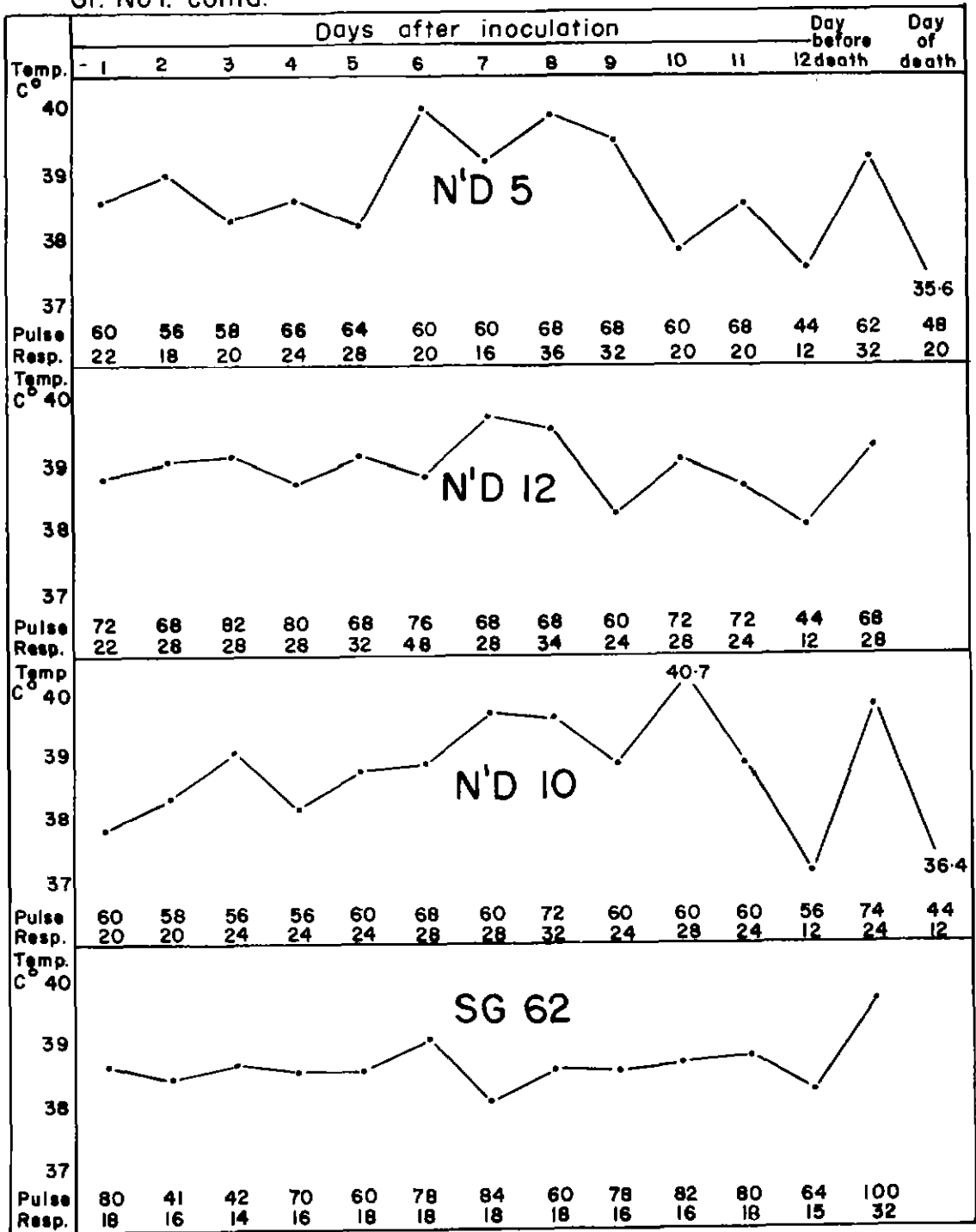


Table N° I  
The daily and weekly index of infection in 100 fields  
Nombre journalier puis hebdomadaire des trypanosomes pour 100 champs microscopiques.

N° of animal	Day after inoculation																	Weeks after inoculation					
	1-5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	4	5	6	7	8	9
WAS 45	-	-	-	-	-	749	163	90	-	-	4	173	119	43	81	10	6	1	5	-			
WAS 108	-	36	302	486	71	41	-	9	260	-	3	43	2	4	2	23	67	4	10	15	10	18	
WAS 133	-	-	133	32	24	36	169	-	-	-	-	-	-	-	-	1	-	-	-	-			
N'D 11	-	-	-	-	-	141	30	18	8	2	4	4	15	-	4	1	13	3	33	1	86	397	208
N'D 5	-	-	-	-	-	26	2	-	-	4	7	102	286	-	21	2	1	6	6	29	51	85	
N'D 12	-	-	90	226	61	5	52	2	1	-	-	-	-	1	-	-	-	-	-	-	-	-	
N'D 10	-	-	52	226	326	421	352	26	7	23	-	6	4	-	2	3	2	12	59	1	-	-	
SG 62	-	-	-	-	-	2	-	-	1	-	-	-	-	-	-	-	-	-	-	6	-	-	
N° animal	Jours après l'inoculation																	Semaines après l'inoculation					

WAS 45 - Died 37th day after inoculation

WAS 108 - " 57th " " "

WAS 133 - " 47th " " "

N'D 11 - " 58th " " "

N'D 5 - Died 53rd day after inoculation.

N'D 12 - Died after 15 months.

N'D 10 - Died 48th day after inoculation.

SG 62 - Still alive.

Clinical sign	No. of animal showing clinical sign	The day of onset of clinical sign after infection	Duration of clinical signs
Listlessness, loss of appetite, retarded rumination, bristled hair coat . . . . .	6	4 — 8	3 — 13
Profuse lacrimation . . . . .	4	16 — 39	Until death
Watery scouring . . . . .	4	26 — 42	Until death
Bottle jaw . . . . .	5	28 — 45	Until death
Blood stained faeces . . . . .	1	40	7
Loss of hair on the back and tail . . . . .	2	36 — 49	Until death
Bleeding from the mouth and nostrils . . . . .	4	38 — 54	2 — 4
Swollen head . . . . .	1	34	Until death
Sitting on hind legs unable to get up . . . . .	5	34 — 57	1 — 13
Lying on one side with the legs stretched . . . . .	6	36 — 56	Until death
Death . . . . .	6	37 — 58	

From 50th day condition improving.

Died after 15 months.

SG. 62 :

On 6th day after infection listlessness, not grazing well.

On 15th day lacrimation from both eyes, stopped after 5 days.

From 6th week condition improving and no alteration of health could be noticed.

Still alive.

The first signs of alteration of general conditions appeared on 4th, 5th, 6th, 7th and 8th day after infection and were characterized by listlessness, retarded rumination, loss of appetite, lacrimation and bristled coat. This condition continued from 3-13 days in various extent. The most characteristic clinical signs during the course of the infection were profuse lacrimation, bottle jaw, bleeding from the nostrils and watery scouring with blood in the faeces. Two or three days before death the animals were unable to get up and died in coma. The loss of weight was from 35 to 133 lbs. (Tab. n° 2).

Table N°II  
Body weight of experimental animals  
Poids des animaux d'expérience

N° of animal	Weight in weeks (1 bs)										loss of weight in lbs
	-1	1	2	3	4	5	6	7	8	9	
WAS 45	562	560	530	515	501	480	450				112
WAS 108	536	530	522	506	475	470	462	450	435		101
WAS 133	554	550	499	461	448	440	432	421			133
N'D 11	495	490	468	452	438	429	418	409	401	395	100
N'D 5	502	498	470	450	439	422	405	398	370		132
N'D 12	586	580	552	531	519	533	533	538	535	540	46
N'D 10	430	468	443	433	406	401	400	395			35
SG 62	836	830	810	798	780	775	778	780	775	785	51
N° animal	Poids chaque semaine										Perte de poids

*Values of blood picture (Tab. n° 3)*

Number of erythrocytes before infection was above the average range published (SHALM, 17). Considerable drop began three weeks before death. During the course of infection the erythrocyte number dropped from 27 to 78 p. 100 in animals that died. In two animals that survived the decrease in number was only 3 to 5 p. 100.

Haematocrit values are below the normal range except one animal (N'D 12) and two animals that survived haemoglobin content dropped remarkably as from 5th week after infection.

Mean corpuscular haemoglobin is deeply below normal range and indicates microcytosis. Its values do not change remarkably throughout the course of the disease. It has to be pointed out that microcytosis was present in the experimental animals even before infection. Similarly hypochromia indicated by low values of mean corpuscular haemoglobin has no special trend during the course of the disease and varies dependently on the haemoglobin and erythrocytes number. Mean corpuscular haemoglobin concentration is on the lower limit of average range.

Number of leucocytes before infection ex-

ceeded highly the normal range (except WAS 108). The remarkable drop in number started in 2nd week after infection and remained on the lower range for 4-8 days. In the two week before death the number of leucocytes increased again.

Erythrocytes sedimentation rate in 1 hour did not show any changes, therefore the 24 hours interval was observed. It is generally known the ESR in cattle is of very limited value (SHALM, 17), but it was noticed that in 4 animals there was an increased fall in last two weeks before death.

The differential counts of leucocytes show that there was neutrophilia in all the animals before start of the experiment. From 12th day of infection lymphocytosis appeared. The second day after infection eosinophilia started and continued to 10th day. Monocytes number was on the bottom limit of normal range or were not found at all. No basophiles were found.

In the two animals that survived no remarkable alteration of blood picture developed apart from slight drop in RBC in SG. 62 on the 16th day after infection and lasted for 4 days. Within the same period the decrease of WBC in the same animal appeared.

**RESULTS OF AUTOPSIES**

(Figures in brackets mean number of animals affected.)

- General : Numerous decubiti part. on the hip joints, tuber coxae and on the head (6). Dehydration (6). Gelatinous infiltration of subcutaneous tissue (4).
- Lymphatic glands : All enlarged and oedematous (6). L. jujunales enlarged and oedematous with medullar and cortical haemorrhages (1).
- Lungs : Hypostatic pneumonia of the left lung (1), right lung (1), both lungs (2). Emphysema interstitialis of the left lung (1), right lung (1). Hydrothorax with serous exudate (3), gelatinous exudate (1), sanguineous exudate (1).
- Heart : Hydropericard with gelatinous exudate (2), sanguineous exudate (1). Degeneration and dilatation of heart muscle (4), subepicardial (5), myocardial (4), subendocardial (6) ecchymosae.
- Alimentary tract : Asites with straw coloured exudate (4), gelatinous infiltration of omental fat (3). Catarrhal (1), haemorrhagic (3) abomasitis, haemorrhagic omasitis (1), chronic duodenitis, jejunitis, ileitis (1), haemorrhagic jejunitis (3), Ulcerative jejunitis (1), haemorrhagic typhlitis with blood stained content (3), haemorrhagic colitis (1).

TABLE N° III

Mean values of blood picture of animals that died within 9 weeks after infection  
 Valeur moyenne de l'image sanguine des animaux (morts moins de 9 semaines après l'infection)

Days/Weeks after inoculation	RBC (millions/mm <sup>3</sup> )			PCV (p.100)			Hb (g/100 ml)			WBC (1000/mm <sup>3</sup> )			ESR (mm/24h)			MCV ( μμ )		
	Mean	Max	Min	Mean	Max	Min	Mean	Max	Min	Mean	Max	Min	Mean	Max	Min	Mean	Max	Min
Days-2	11.17	13.21	7.73	31	36	26	10.36	11.10	8.88	16.26	19.90	8.40	6	15	3	30	34	25
2	11.05	12.88	8.70	30	35	25	10.06	11.10	8.58	16.89	20.15	10.10	6	12	3	27	31	25
4	10.90	13.46	7.78	31	36	26	9.57	11.54	8.14	16.98	20.30	11.05	5	8	3	29	33	22
6	9.57	11.61	7.94	29	34	22	9.76	11.84	7.40	12.25	16.00	7.55	6	10	4	31	35	28
8	9.87	12.18	7.50	28	30	21	9.20	11.10	7.40	11.19	20.15	5.55	6	9	4	29	35	25
10	9.60	11.47	6.27	27	30	19	8.63	9.77	7.40	10.42	12.20	7.80	8	16	5	29	35	27
12	9.01	12.45	6.94	27	30	21	8.76	10.36	7.40	9.81	12.45	5.45	7	10	4	29	35	27
14	9.33	11.81	6.16	27	34	21	9.27	11.10	7.25	8.57	12.30	6.95	7	15	4	30	37	24
16	9.39	10.61	7.23	27	32	19	8.63	10.51	6.81	8.77	12.35	4.30	8	20	4	28	32	26
18	9.66	11.58	6.89	28	36	22	8.76	11.40	6.96	7.77	9.00	5.95	6	10	3	30	32	26
20	9.59	10.91	6.88	27	37	20	8.51	11.10	6.22	9.02	11.35	7.40	7	12	5	28	35	23
Weeks- 4	9.62	12.69	6.90	26	34	18	8.38	9.18	6.81	11.31	13.95	7.40	8	13	5	28	32	24
5	9.03	12.35	5.58	25	33	15	7.26	9.47	5.18	12.52	18.50	7.15	9	19	5	28	33	26
6	7.39	11.63	4.82	19	25	15	6.27	8.14	5.33	11.24	18.00	6.50	11	15	6	27	31	21
7	5.56	9.35	3.80	14	23	8	5.47	7.70	3.26	12.97	16.50	12.00	13	20	6	28	34	24
8	6.25	9.54	3.00	17	24	8	5.36	6.81	3.20	14.27	14.90	13.90	12	20	7	26	29	25
9	5.87	5.87	5.87	15	15	15	5.18	5.18	5.18	13.80	13.80	13.80	14	14	14	26	26	26
	Nombre d'érythrocytes			pourcentage du culot de centrifugation par rapport au volume total.			Hémoglobine			Nombre de leucocytes			vitesse de sédimentation			volume moyen des hématies		



TABLE N°III (suite)

Mean values of blood picture of animals that died within 9 weeks after infection  
 Valeur moyenne de l'image sanguine des animaux (morts moins de 9 semaines après l'infection)

(1) Days/Weeks after inoculation	(2) MCH (u u g)			(3) MCHC (p.100)			Neutrophiles (p.100)			Lymphocytes (p.100)			Monocytes (p.100)			Eosinophiles (p.100)			
	Mean	Max	Min	Mean	Max	Min	Mean	Max	Min	Mean	Max	Min	Mean	Max	Min	Mean	Max	Min	
Days- 2	9	11	8	34	36	31	51	70	43	44	54	25	-	-	-	5	7	1	
2	9	10	8	33	34	31	42	50	32	45	55	39	1.6	5	-	11.4	14	10	
4	9	10	7	31	33	28	39	55	30	43	54	31	1.0	2	-	17.0	23	15	
6	10	11	8	33	35	29	37	50	13	53	75	45	0.5	2	-	9.5	20	2	
8	9	11	8	32	37	29	41	55	16	46	76	29	0.20	1	-	12.8	21	8	
10	9	12	7	32	39	26	46	75	20	46	75	19	1.3	4	-	6.7	15	-	
12	9	11	8	32	35	30	27	43	10	71	85	55	0.3	2	-	1.7	5	-	
14	10	12	9	34	41	26	35	40	27	61	72	55	0.5	3	-	3.5	12	-	
16	9	11	7	32	37	28	31	43	22	66	78	55	0.5	3	-	2.5	6	-	
18	9	11	7	31	34	28	33	46	20	66	79	54	0.2	1	-	0.8	1	-	
20	9	10	8	29	35	20	41	58	30	57	70	42	0.2	1	-	1.8	3	-	
Weeks- 4	9	12	7	32	38	26	37	72	15	61	84	27	0.3	1	-	1.7	4	-	
5	8	10	7	29	36	28	42	70	30	56	66	30	0.5	2	-	1.5	7	-	
6	9	11	7	33	38	26	38	72	13	60	85	27	0.5	2	-	1.5	3	-	
7	11	13	8	37	41	33	33	53	20	65	78	59	0.4	1	-	1.6	3	-	
8	9	11	7	34	40	28	31	37	22	67	75	62	0.8	1	-	1.2	2	-	
9	9	9	9	34	34	34	50	50	50	50	50	50	-	-	-	-	-	-	
(1).Jours et semaines après inoculation	(2) Quantité moyenne d'hémoglobine par g. d'hématies			(3) Concentration moyenne d'hémoglobine dans 1 g. d'hématies															

Liver	: Enlarged with rounded edges (4), fatty degeneration (4), distended gall bladder (2), thickened bile (4).
Kidneys	: Gelatinous infiltration of perirenal fat (2), Fatty degeneration (5), subcapsular and cortical haemorrhages (2), urinary bladder distended (2), petechial haemorrhages on mucous membrane of urinary bladder (2).
Reproductive system	: Endometritis of left uterine horn, cystic degeneration of ovaries (1).
Spleen	: Enlarged, soft consistency (6), congestion of superficial blood vessels (4), pinpoint haemorrhages along the edges (3).
Muscles	: Pale and oedematous (6), gelatinous infiltration of intermuscular connective tissue (3).

## DISCUSSION

The investigation presented was to find out the clinic course and physical changes of the blood and post-mortem findings in artificially infected animals by *Trypanosoma vivax* under the conditions in Ghana. The author is aware of the fact, that the number of animals used is limited but it would hardly be possible to use more animals and leave them to die. The technical difficulties of examination of greater number of animals have to be taken into consideration as well.

*Trypanosoma vivax* seems to be one of the main causative organisms of trypanosomiasis in Ghana. The investigations made in Accra Plains by the author revealed that all cases of trypanosomiasis (120 animals with 26 positive findings in the blood) were caused by *Trypanosoma vivax*. Similar investigation was undertaken in Upper Region in animals stationed in Bolgatanga slaughterhouse. In 20 selected animals showing clinical signs, 18 of them showed *Trypanosoma vivax* in very high concentration in the blood samples (VOHRADSKY, 21). Other authors (NYARKO, 15), (METTAM, 13, 14), (STEWART, 18, 19), (EDWARDS et al. 3) came to the same conclusion that *T. vivax* is responsible for most of the cases in West Africa as a whole. FOLKERS and JONES-DAVIES (7) in Northern Nigeria found the incidence of *T. vivax* in blood smears to be 35 p. 100 in local herds and 48 p. 100 in nomadic herds of cattle. The number of animals used in our experiment did not permit any conclusions on breed tolerance to trypanosome infection, but results published by CHANDLER (1) and STEWARD (20) show that N'Dama and West African

Shorthorn cattle possess practical resistance against trypanosomiasis.

The course of the disease in artificially infected cattle can be characterized as subacute except two animals (N'D 12, SG 62), which developed the chronic form of the disease. SG 62 is still alive and does not show any alteration of clinical picture. N'D 12 collapsed after 15 months and died in spite of the treatment with ethidium.

Clinical findings in our experimental animals differ from those described by NYARKO (15), who states that there are no obvious signs of the disease apart from those associated with pyrexia. According to him no oedema is usually present. HENNING (8) described the symptoms as resembling those produced by *T. congolense* but milder. METTAM (13, 14), STEWART (18, 19) found pyrexia, weakness so that the animal was unable to rise and coma appeared before death. Bottle jaw, blood-stained faeces, bleeding from the nostrils and mouth as it appeared in some of our animals, were not usually observed.

Physical changes in the blood correspond to the reported values found by other authors during *T. congolense* infection (FIENNES et al. 4), (FIENNES 5). An interesting finding is that in all of the animals except one, the erythrocytes number before infection was higher than the results usually published (SHALM 17). This was the reason why blood examination of healthy animals of local breeds of cattle (N'Dama, West African Shorthorn, Sokoto Gudale, Sanga) was carried out and the average erythrocytes number obtained was over 10 millions in all breeds (VOHRADSKY, 22). These results need further study. The anaemia characterized by decrease of erythro-

cytes number and haemoglobin content started very early at the end of the third week, and two weeks before death both values fell below 50 p. 100 (except N'Dama 5 when only the haemoglobin content decreased considerably). The two animals surviving showed the decrease of erythrocytes number and haemoglobin content from 5th and 7th week after infection. These animals have been examined monthly for 15 months after infection. Their erythrocytes number varied from 6 to 8 millions. Last examination showed 5.88 millions in SG 62 and 3.50 millions in N'D 12. These animals developed the typical chronic type of the disease.

The increase of lymphocytes may indicate an attempt of the body for a defence reaction (FIENNES et al. 4). The first reaction to the infection occurred in increased number of eosinophiles shortly after infection.

Counts of parasites in peripheral blood showed such variation that it was impossible to determine any correlation between clinical findings or physical changes of the blood and trypanosome number. FIENNES et al. (4) considers these variations as a result of weather or other changes of environment.

General condition of dead animals showed dehydration, decubiti and gelatinous infiltration of subcutaneous tissue. Lesions of internal organs resembled haemorrhagic septicaemia (HENNING, 8). Lymphatic glands were enlarged and oedematous. In some of the animals haemorrhages appeared in cortex and medulla. Haemorrhages along the side of the tongue and in the larynx as described by NYARKO (15) could not be revealed. The skeletal muscles were œdematous with gelatinous infiltration of intermuscular connective tissue.

Constant lesions could be found in the heart muscle. Heart muscle was dilated, flabby, the quantity of pericardial fluid was increased and often of sanguineous colour. Subepicardial, myocardial and subendocardial haemorrhages of different size (petechiae and ecchymosae) were constantly present. These changes in the heart muscle point to circulatory failure due possibly to the partial blockage of the smaller blood vessels (FIENNES et al. 4). Hydrothorax with straw coloured or yellowish exudate was the common feature. Changes in the lungs

varied from interstitial emphysema to hypostatic pneumonia of one half or both lungs or oedema.

Alimentary canal was constantly affected. The most frequent finding was abomasitis either catarrhalis or haemorrhagica. The intestine changes were characterized by haemorrhagic jejunitis and colitis with tendency to ulceration. In one case omassus and caecum were affected by haemorrhagic inflammation of mucous membrane. Ascites of different extent and ecchymosae of peritoneum were present. Gelatinous infiltration of omental fat was noticed.

The liver lesions were of different character, most commonly the organ was enlarged with rounded edges, fatty degenerated with subcapsular haemorrhages. The gall bladder was distended with thick yellow-green bile.

The kidneys showed no signs of necrosis as published by FIENNES et al. (4). Fatty degeneration with subcapsular and cortical haemorrhages were found most often. There was retention of urine and haemorrhagic inflammation of mucous membrane of urinary bladder.

Spleen was always enlarged with pinpoint haemorrhages along the edges, marked injection of superficial blood vessels, and very soft pulp.

Generally it can be concluded that pathomorphological changes in *Trypanosoma vivax* infected animals resemble the picture of haemorrhagic septicaemia with more marked acute lesions in artificially infected animals.

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#### RESUME APPROFONDI

Au Ghana, la trypanosomiase bovine à *T. vivax* est plus fréquente et aussi pathogène que celle à *T. congolense*. Les expériences relatées par VOHRADSKY, ont été réalisées à proximité d'Accra, dans une région apparemment dénuée de glossines où les trypanosomiasés sont très rares. Elles visaient à définir les symptômes, les modifications hématologiques et les lésions provoquées par l'infection expérimentale à *T. vivax*. Huit animaux (4 Ndamas, 3 Baoulés et 1 zébu Foulbé), infectés par injection sous-cutanée de 2 ml de sang

parasité ont été suivis pendant 9 semaines au moyen des examens suivants : examen clinique journalier, examen hématologique tous les deux jours, recherche journalière des parasites en goutte épaisse. Des autopsies soignées ont été pratiquées sur tous les animaux morts au cours de l'expérience.

### Parasitologie

Les trypanosomes ont été examinés à l'Institut de Parasitologie de Prague et identifiés comme *T. vivax*. Le comptage des parasites dans le sang périphérique a donné des résultats si variables qu'il semble impossible de définir une corrélation entre le nombre des trypanosomes et les signes cliniques ou hématologiques.

### Symptômes

La maladie eut une évolution subaiguë pour 6 animaux sur 8 et chronique pour les 2 autres dont l'un (un N'dama) mourut de causes indéterminées 15 mois après l'infection. L'autre (un zébu) était encore en vie à la fin de l'expérience.

Les premiers signes d'altération de l'état général apparurent entre 4 et 8 jours après l'infection : fièvre, inattention, rumination difficile, perte d'appétit, larmolement, poil piqué. Puis les symptômes fébriles disparurent.

Les principaux signes cliniques relevés pendant l'évolution de la maladie furent de l'amaigrissement, un larmolement profus, le signe de la bouteille, des hémorragies nasales, une diarrhée parfois hémorragique. Deux à trois jours avant la mort les animaux ne peuvent plus se lever, présentent de l'hypothermie et meurent dans le coma.

### Hématologie

Les modifications hématologiques ressemblent à celles notées dans la trypanosomiase à *T. congolense*.

*Erythrocytes* : il est intéressant de noter qu'avant le début de l'expérience, chez tous les animaux, le nombre d'érythrocytes était notablement supérieur à la normale. Les signes de l'anémie furent intenses et apparurent dès la fin de la troisième semaine, la diminution du nombre des hématies dépassant 50 p. 100 deux semaines avant la mort. La vitesse de sédimen-

tation n'a, par contre, pas été sensiblement modifiée.

### Leucocytes

Les modifications de la formule leucocytaire correspondent aux réactions de défenses de l'organisme, la première réaction étant une augmentation du nombre des éosinophiles, suivie d'une lymphocytose apparaissant à partir du 12<sup>e</sup> jour.

Chez les deux animaux survivants, on n'a pas observé d'altération remarquable de la formule sanguine sauf, chez un animal, une légère diminution passagère du nombre des hématies.

### Lésions

Les lésions n'ont rien eu de pathognomonique. Les plus fréquentes furent :

- Déshydratation et infiltration gélatineuse du tissu conjonctif sous-cutané avec plaies de décubitus.
- Muscles pâles et œdématiés.
- Ganglions hypertrophiés et œdémateux avec, chez certains animaux, des hémorragies du cortex ou de la moelle.
- Lésions du cœur constantes : muscle cardiaque dilaté et flasque, pétéchies ou ecchymoses sur le myocarde, le péricarde ou l'endocarde. Liquide péricardique augmenté et souvent hémorragique.
- Foie hypertrophié avec dégénérescence graisseuse — vésicule biliaire distendue.
- Rate toujours hypertrophiée, molle, congestionnée.
- Reins : dégénérescence graisseuse avec hémorragies subcapsulaires et corticales. Pétéchies sur la vessie — rétention urinaire.
- Poumons. Lésions variant de l'emphysème pulmonaire à la pneumonie hypostatique ou à l'œdème pulmonaire. Hydrothorax avec exsudat jaunâtre.
- Lésions constantes du tractus digestif : entérite hémorragique avec tendance à l'ulcération — Acite — ecchymoses du péritoine.

L'auteur conclut que, chez les animaux infectés par *T. vivax*, les modifications pathomorphologiques ressemblent à celles notées dans les septicémies hémorragiques avec des lésions aiguës plus marquées chez les animaux infestés expérimentalement.

## SUMMARY

**Course, clinical symptoms, physical changes of the blood and post-mortem findings of cattle artificially infected by *Trypanosoma vivax* are described**

The disease ran a subacute course with fatal end in 6 of 8 artificially infected.

The physical changes of the blood anaemia which was progressive in the last two weeks before death.

The post-mortem picture was characterized by septic haemorrhagic changes, particularly in the heart spleen, lymphatic glands and liver.

## RESUMEN

**Signos clínicos, tasas diarias de infestación, modificaciones hematológicas y patomorfológicas en ganado artificialmente infestado por *Trypanosoma vivax***

El autor describe la evolución, los síntomas, las modificaciones hematológicas y las lesiones de la infestación experimental del ganado por *T. vivax*.

La enfermedad sigue una evolución subaguda terminando en 6 muertos entre 8 animales infestados.

Las modificaciones hematológicas demuestran una anemia que se desarrolla durante las dos semanas precediendo la muerte.

Se caracterizan las lesiones por signos de pasteurelosis, particularmente en el corazón, el bazo, los ganglios linfáticos y el hígado.

## BIBLIOGRAPHIE

- CHANDLER (R. L.), « Comparative tolerance of West African N'Dama cattle to trypanosomiasis », *Ann. trop. Med. Parasit.*, 1952, **46** (2): 127-34.
- CURSON (H. H.), « Nagana in Zululand », 13-14 Rep. D.V.E. and R., 1928, 1, 309.
- EDWARDS (E. E.), JUDD (J. M.) et SQUIRE (F. A.), « Observations on trypanosomiasis in domestic animals in West Africa. I - The daily index of infection and the weekly haematological values in goats and sheep infected with *T. vivax*, *T. congolense* and *T. brucei* », *Ann. trop. Med. Parasit.*, 1956, **50** (3): 223-41.
- FIENNES (R. N. T. W.), JONES (E. R.) et LAWS (S. G.), « The course and pathology of *Trypanosoma congolense* (Brodén) disease of cattle », *J. comp. Path. Therap.*, 1946, **56** (1): 1-27.
- FIENNES (R. N. T. W.), « Haematological studies in trypanosomiasis of cattle », *Vet. Rec.*, 1954, **66** (30): 423-34.
- FIENNES (R. N. T. W.), « The results of autopsies in trypanosome infected cattle », *Brit. vet. J.*, 1953, **109**: 511.
- FOLKERS (C.) et JONES-DAVIES (W. J.), « The incidence of trypanosomes in blood smears of cattle presented for trypanosomiasis treatment in Northern Nigeria », *Bull. Epizoot. Dis. Afr.*, 1966, **14** (4): 409-421.
- HENNING (M. W.), « Animal disease in South Africa ». South Africa, Central News Agency Ltd, 1949: 545.
- HORNBY (H. E.), « Report of the Proceedings of the Fifth Pan-African Veterinary Conference », Nairobi, April, 1923.
- HUDSON (J. R.), « Acute and subacute trypanosomiasis in cattle caused by *T. vivax* », *J. comp. Path. Therap.*, 1944, **54** (2): 108-19.
- JONES-DAVIES (W. J.), « The discovery of Berenil-resistant *Trypanosoma vivax* in Northern Nigeria », *Vet. Rec.*, 1967, **80** (17): 531-32.
- JOWETT (W.), « Further note on a cattle trypanosomiasis of portuguese East Africa », *J. comp. Path. Therap.*, 1911, **24** (1): 21-40.
- METTAM (R. W. M.), « Ann. Rep. Vet. Dept. T. for 1934 », Uganda Protectorate.
- METTAM (R. W. M.), « Ann. Rep. Vet. Dept. for 1938 », Nigeria, 1939, pp. 23-25.
- NYARKO (D.), « Report on the WHO training course on trypanosomiasis in Africa », Animal Health Division, Min. Agric. Kumasi, 1966.
- PARKIN (B. S.), « Symptomatology of trypanosomiasis in domestic animals », *Onderstepoort J. vet. Res.*, 1935, **4** (2): 251-67.
- SCHALM (O. W.), « Veterinary hematology », Philadelphia, Lea and Febiger, 1961.
- STEWART (J. L.), « Treatment of trypanosomiasis by tartar emetic, Antimosan and Surfen C in the Gold Coast », *J. comp. Path. Therap.*, 1935, **48** (1): 316-18.
- STEWART (J. L.), « Reports Dept. Animal Health for years 1936, 1937 and 1938 ».
- STEWART (J. L.), « The west african shorthorn cattle. Their value to Africa as trypanosomiasis resistant animals », *Vet. Rec.*, 1951, **63** (27): 454-57.
- VOHRADSKY (F.), « Probleme der Fleischhygiene in Ghana », *Fleischwirtschaft*, 1968, **48** (10): 1350-54.
- VOHRADSKY (F.), « Annual Report, Agric. Res. Stn Nungua University of Ghana 1966/67 [Research Proj. 1968 (10)].