A STUDY OF STRONGYLIASIS IN HORSES IN THE EASTER BUSH AREA OF MIDLOTHIAN

BY

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CONTENTS

SUMMARY

A number of horses at three different equine units in the Easter Bush area were monitored using routine helminthological methods between November 1976 and August 1977.

Trichonema spp. were found to be the predominant strongyles in these horses during this period. It is suggested that this predominance was due to the exceptional environmental conditions prevailing in 1976 and the anthelmintic regime adopted during that year.

A seasonal rise in egg output during the late spring and summer and a corresponding increase in pasture larval contamination was also shown.

The effects of Trichonema spp. and in particular those due to the larval stages of these helminths on the haematological values and plasma protein levels was also studied. The results supported the findings of most previous workers and emphasised the importance of the estimation of peripheral eosinophil counts and of β globulin levels in the diagnosis of prepatent strongyliasis.

INTRODUCTION

Nematodes belonging to the family Strongylidea are found in equines throughout the world, though geographical variations occur with individual species. Not all the forty-odd species have been found in any one horse, but most equines harbour a number of them.

In this dissertation there is described a study of the epidemiology of strongyliasis in horses in the Easter Bush area of Midlothian. Several parameters of this complex have been studied and the data collected and analysed in an attempt to verify known information and to relate this to conditions in this area.

To obtain a representative picture of this condition in the Easter Bush vicinity, three different equine units were chosen for closer study. These represented different managerial situations whilst being subjected to similar climatic variations.

As the project proceeded, numerous problems were encountered which tended to explain the sparcity of work in the field of equine strongyle epidemiology. Nevertheless as many significant conclusions as possible have been drawn.

REVIEW OF THE LITERATURE

The literature contains a surprising lack of epidemiological work concerning strongyliasis in the horse. A great deal of work though, has been done on individual aspects of this condition and in particular on the migration paths of the large strongyles (Duncan, 1973; Poynter, 1958; Olt, 1932). Even now the vexing problem of the exact path taken by Strongylus vulgaris is not satisfactorily resolved.

Soulsby (1965) lists the principle strongyles found in the horse (see Table 1) and suggests that most equines contain at least 15 or so species, but points out that a clinical situation arises only when sufficient numbers of adult worms damage the intestinal mucosa or lesions produced by the migrating larva upset the horses' functions.

Poynter (1969) gives a good account of the growth of the knowledge of horse nematodes since 430 B.C. and declares that Russell, (1948) gave the first lucid account of strongyle numbers and species distribution. This work was carried out with thoroughbred foals and established the varying prepatent periods of the major species by routine faecal examination and larval culture. She produced the original key for infective larva differentiation. The lack of pathogenicity of even large numbers of Strongyloides westeri was pointed out and the presence of strongyle eggs in the faeces of young foals was shown to be due to coprophagia by the foal, of the dam's faeces, rather than to prenatal infection.

From 1930 to 1950 much work was done on the migration paths of the large strongyles in particular by Olt, (1932), Wetzel and Enigk (1938) and Enigk (1950). Poynter (1950) suggested a migration in the anterior mesenteric artery in the case of S. vulgaris, backing up the hypothesis of Enigk and Wetzel (1938).

TABLE 1

NEMATODES OF THE LARGE INTESTINE OF EQUINES
(Reproduced from Soulsby, 1965)

ORDER STRONGYLOIDEA

FAMILY STRONGYLIDAE

SUBFAMILY STRONGYLINAE

GENUS STRONGYLUS
Strongylus (Delafondia) vulgaris
Strongylus equinus
Strongylus (Alfortia) edentatus
Strongylus asini (Donkey, East Africa)

GENUS TRIODONTOPHORUS
Triodontophorus serratus
Triodontophorus tenuicollis
Triodontophorus minor
Triodontophorus brevicauda
Triodontophorus intermedius

GENUS OESOPHAGODONTUS
Oesophagodontus robustus

GENUS CRATEROSTOMUM
Craterostomum acuticaudatum
Craterostomum mucronatum
Craterostomum tenuicauda

GENUS CABALLONEMA
Caballonema longicapsulatum

SUBFAMILY TRICHONEMINAE

GENUS TRICHONEMA (syn. Cylicostomum)
Trichonema (Cylicostomum) tetracanthum (Principally in donkeys)
Trichonema (") labratum
Trichonema (") ornatum
Trichonema (") labiatum
Trichonema (") labiatum VAR. digitatum
Trichonema (") coronatum
Trichonema (") sagittatum
Trichonema (Cylicocercus) alveatum (Principally zebra)
Trichonema (") catinatum
Trichonema (") pateratum
Trichonema (") mettami
Trichonema (") goldi (Zebra)
Trichonema (Cylicocyclus) radiatum
Trichonema (") triramosum (Zebra)
Trichonema (") elongatum (Principally donkeys)
Trichonema (") elongatum VAR. kotláni
Trichonema (") insigne

TABLE 1 (continued)

Trichonema (Cylicocyclus) adersi (Donkey, zebra)
Trichonema (") nassutum
Trichonema (") nassutum VAR. parvum
Trichonema (") leptostomum
Trichonema (") auriculatum (Principally donkeys)
Trichonema (Cylicostephanus) calicatum
Trichonema (") minutum
Trichonema (") longibursatum
Trichonema (") hybridum
Trichonema (") poculatum
Trichonema (") asymetricum
Trichonema (Cylicodontophorus) bicoronatum
Trichonema (") euproctus
Trichonema (") ultrajectinum
Trichonema (Cylicobrachytus) brevispiculatum
Trichonema (") prionodes
Trichonema (Cylicotoichus) montgomeryi (Zebra)

GENUS POTERIOSTOMUM
Poteriostomum imparidentatum
Poteriostomum ratzii

GENUS GYALOCEPHALUS
Gyalocephalus capitatus

In 1952 Todd in a survey at a Kentucky stud farm, showed an increase in numbers of eggs passed in the faeces after parturition and suggested the presence of a post parturient rise in the number of eggs in the faeces, as seen in sheep and pigs. This work has subsequently been disputed by other workers in the field, Poynter (1958), and Duncan (1973).

Starting in 1952 Poynter conducted the first large scale survey of the incidence of equine nematodes in the United Kingdom culminating in his PhD thesis (1958). He found seasonal fluctuations in the number of eggs passed by the horse (Poynter, 1954) with faecal strongyle egg counts rising from March/April reaching maximum levels between July and September. Larval cultures showed a species distribution of 90% Trichonema spp. 5% S. vulgaris and 4% S. edentatus. He asked the question whether the increase in faecal egg counts was due to an increase in the number of adults or numbers of eggs produced by the same number of adults. Post mortem examinations carried out in January, February and March showed successively increasing adult numbers and a similar reduction in late autumn, thus confirming that it was fluctuations in adult numbers that gave the seasonal faecal egg count distribution.

Poynter's PhD thesis (1958) is worth detailed consideration. In chapter one an account of the prevalence of intestinal nematodes in horses is given, from over 16,000 faecal samples from 3,250 horses. This work was further verified by post mortem examination. Conclusions from the survey were that there exists in Great Britain in horses a wide range of nematodes and this is extended in Soulsby (1965) (Table 1).

Chapter three is a repeat of Russell's work (1948) showing the earliest appearance of strongyle eggs in horse faeces indicating the

presence of adults of each species in the intestinal lumen. The prepatent periods this established closely agreed with Russell (1948) except for Strongyloides westeri, S. vulgaris and S. edentatus in which Poynter showed shorter prepatent periods than previous workers (Table 2).

He agreed with Russell (1948) that no prenatal infection occured since cultures of strongyle eggs found in faeces below six weeks of age showed a mixed flora of species similar to that in the dam, whilst after six weeks pure cultures of Trichonema spp. were only seen, leading later to a mixed infection. He further showed the appearance of Strongyloides westeri from seven weeks onwards, disappearing at 14 weeks on average.

In chapter eight Poynter showed the effects of parasitism on growth rate, bodily condition and the red blood cell (R.B.C.) picture. A 30% reduction in the rate of growth of parasitised animals compared to controls was described and the blood picture showed a fall in R.B.C. numbers, packed cell volume (P.C.V.) and haemoglobin (Hb) values. An increase in R.B.C. values in May in both controls and parasitised groups was attributable to an increase in the nutritional status at that time, whilst mean cell volume (M.C.V.) and mean corpuscular haemoglobin (M.C.Hb.) remained constant in both groups. The anaemia was due to a decrease in the numbers of circulating erythrocytes and the anaemia could be described as normochromic and normocytic. Ascarids were not responsible for this anaemia. The anaemia was dyshaemopoetic in origin and could be linked to nutritional factors, the rise in spring of the R.B.C. values in parasitised animals occuring when an increase in nutrition counteracted the effects of the parasites.

TABLE 2

GENUS	EARLIEST APPEARANCE WKS.	AVERAGE WKS.	OTHER WORKERS AVERAGE RESULTS WKS.
TRICHONEMA	9	15	RUSSELL (1948) 15 WETZEL (1942) 10
POTERIOSTOMUM	22	23	RUSSELL (1948) 20
GYALOCEPHALUS	-	30	RUSSELL (1948) 28
STRONGYLUS VULGARIS	-	20	WETZEL (1942) 27
TRIDONTOPHORUS	-	18	RUSSELL (1948) 18
STRONGYLUS EDENTATUS	-	28	RUSSELL (1948) 50-55

(from Poynter 1958) Table showing prepatent periods established of major strongyle species of the horse.

Finally Poynter considered the migration path of S. vulgaris larvae and suggests that they leave the left side of the heart in the oxygenated blood, passing to the alimentary tract by way of the intestinal vessels.

In 1963 Schotman analysed changes in the protein patterns of serum from ponies and horses by paper electrophoresis and suggested that the increase in β globulins seen in cases of strongyliasis in the horse was due to mobilisation of lipids of the body resulting from reduced absorption of food ingested. He showed two types of changes associated with β globulin rises.

(a) Albumin - decreased
$\alpha\gamma$ - normal or slightly increased
β - increased

(b) Albumin - decreased
$\alpha\beta\gamma$ - all increased

The former was associated with anaemia and was due to the presence of large numbers of adult strongyles in the intestinal lumen, while the latter changes indicated the presence of a verminous aneurysm.

In 1964 Mathieson conducted at Easter Bush a study of strongyliasis of horses in South East Scotland and showed the species distribution and lesions associated with migrating, inhibited and adult strongyles. His work verified the findings of Poynter (1958).

Round (1968a) discussed the diagnosis of helminthiasis in the horse and used a variety of methods including faecal egg counts and larval culture, haematological examination, serum protein analysis and weight and growth parameters. He pointed out that in the acutely ill or heavily parasitised animal there may be a reduction in egg count rendering this unsuitable as a single method of diagnosis. He found obvious anaemia in young parasitised animals but not often in

older severely affected animals and related the eosinophilia seen in affected animals to larval migration. He also showed a leucocytosis in affected animals and considered loss of weight to be an important criterian. Finally he considered the increase in total serum proteins, which was due to an increase in α_2 and β globulin fractions, to be highly significant in diagnosing this condition.

In a further paper Round (1968b) examined the course of naturally acquired infections of horses given regular anthelmintic treatment and showed a correlation between routine treatment with thiabendazole and successive peaks of infection as measured by his multiple parameters.

Most workers in the field of equine strongyliasis were present at the 2nd International Conference on Equine Infectious Diseases in Paris (1969) and although the perennial problem of the migratory route of S. vulgaris was discussed, no final conclusions were reached. Many useful papers were presented. Enigk (1969) discussed the development of prepatent periods of three species of equine strongyles and established again that prenatal infection did not occur.

Round (1969) now related the development of strongyles in the horse and associated serum protein changes, with particular reference to S. vulgaris. He described an increase in β globulins after the second week of infection producing a diphosic curve with peaks at 6 and 18 weeks and a return to normal by 36 weeks. The highest levels occured at 18-20 weeks, just before patency with a fall to normal after patency. On reinfection a mixed result was obtained with some animals showing significant rises during prepatency. With the small strongyles a slightly different picture was seen with only some animals showing the rise on first infection but on reinfection all showed a

rise in β globulin levels, with a maximum at 4-5 weeks post infection. There seemed to be no correlation with absolute numbers of parasites present but rises could be definitely attributable to the presence of larval stages.

Drudge & Lyons (1969) discussed the chemotherapy of migrating larva and confirmed that high levels of thiabendazole are partially effective but that no really effective drug was available.

Poynter (1969) reiterated many points from his previous work (Poynter, 1958). On the use of egg size as a means of species differentiation he stated that apart from Parascaris equorum and Strongyloides westeri, egg differentiation is impracticable, this agreeing with Cameron (1951) and Russell (1948) but disagreeing with Lepage (1956) and Yamashita, Mori & Kobayashi (1953). The latter used this method in a survey in Japan but did not verify their work with larval cultures.

From 1971 onwards Ogbourne published several papers on aspects of the free-living and pre-infective larvae of horse strongylids. He suggested that for early speciation second stage larvae are most easily recognised from their morphological characteristics after 1-2 days in culture. He gives a detailed account of these but it is apparent that a fair degree of expertise is required before these can be profitably used.

Observations on the free-living stages of Strongylids of the horse by Ogbourne (1972) showed the effects of climate on development.

Egg development and hatching is temperature dependant and will commence above 7°C. Since these conditions prevail in Britain at times during the winter, development to the first stage larval form will often occur, but few such larvae survive, most dying soon after

hatching between October to March, as they are very susceptible to alternate thawing and freezing. In faeces deposited in March there is a high mortality of pre-infective larvae but from April to October large numbers develop to the infective stage. During these months temperatures govern the speed of development but adequate moisture is also essential. Dry conditions delay development although this will recommence when a contaminated faecal pat is adequately moistened. Rapid drying results in a much higher larval mortality. It would appear that except in extreme summers faeces would not remain dry long enough or have dried quickly enough for the yield of infective larvae to be greatly reduced. Accordingly, in a normal British summer, horse faeces act as a reservoir of infection from which the larvae are intermittently released.

In 1975 Ogbourne carried out a series of post mortem examinations and demonstrated a seasonal variation in the number of fourth and fifth stage larvae of S. vulgaris in the anterior mesenteric artery during the year, the highest numbers occuring in December and very few in May, June and July. This suggests that they may be overwintering in the host at that site, a migration to the intestines in April producing adult strongyles giving pasture contamination with eggs in June and July, so allowing reinfection for the coming winter.

Duncan (1973) published the most detailed epidemiological survey carried out to date in the United Kingdom of horses infected with S. vulgaris. He conducted this work at the University of Glasgow and the work examined several important factors of the problem.

He established that the route of migration of S. vulgaris involved a penetration phase in the intestines, with a rapid passage to the anterior mesenteric artery by three weeks. Development proceeds

here for 3-4 months and then movement back to the wall of the large intestines occurs to form a parasitic nodule in the mucosa with release of an immature adult at about six months post infection.

In considering the pathogenesis Duncan discussed the effects caused by the migrating larva and the possible damage produced by large numbers of adults in the lumen of the large intestines. He divided the clinical syndrome into several aspects.

(a) A pyrexia as prepatency occurs in the foal during the first 15 days post infection.

(b) A rise in white blood cell (W.B.C.) numbers which is both early and sharp and remains high throughout the prepatent period. This is further broken down into a decrease in lymphocytes and an increase in neutrophils with a varying but definite increase in eosinophils throughout.

(c) An increase in albumin/globulin ratio, primarily of the β globulin fraction, and of total serum proteins, around the 15th week, thus supporting the work of Round (1968c).

Radio-isotopic trace techniques with R.B.C. and plasma proteins showed an increase in albumin catabolism and a shortened R.B.C. life, thus disagreeing with Poynter's (1958) suggestion that the anaemia is dyshaemopoetic in origin.

From the epidemiological studies Duncan was able to show that infection to the foal comes from both mares contaminating the pasture and overwintering of larvae on the pasture.

He further demonstrated that overwintered larvae have all died out by June and so if foals are not turned out until this time and the mares are routinely treated to reduce pasture contamination the risk of infection to the foal is considerably reduced.

In 1976 Ooms, Oyaert, Muylle, Vanden, Hende & Decraemere discussed electrophoresis as an aid to diagnosis of helminthiasis, especially of verminous aneurysms in the horse. They agreed with Duncan (1973) that pyrexia occured as larvae penetrated the intestinal wall but also showed a second peak on re-entry six months later and felt that during the interim period the temperature remained slightly above normal. They also found a leucocytosis corresponding to the degree of severity of infection and further that faecal egg counts were not reliable as severely infected animals showed reduced levels (c.f. Round 1968a). They were of the opinion that the combination of these parameters was not enough for a good differential diagnosis and that the additional use of electrophoresis was necessary. They agreed with the previous workers (Round, 1958, Duncan, 1973) that an increase in α and β globulins occured together with hypoalbuminaemia and an increase in total serum proteins. They felt further that the more severe the infection the more pronounced the changes and that the α globulin increase was dependant upon the duration of the infection.

Recent work has been focused on the effects of small strongyles on the horse, particularly those belonging to the Trichonematidae. Inhibited development is a common phenomenon in nematodes (Michel, 1974) and the simultaneous development to maturity of numerous individuals following prolonged inhibition is an important feature of the gastro-intestinal nematodes of sheep, cattle and pigs (Armour, 1970, Gibbs, 1973). Ogbourne (1975) showed that the Trichonematidae exhibited this phenomenon, that large numbers of inhibited larvae are found in the submucosa of the large intestines during the winter and early spring and that these move 'en masse' into the lumen to complete development with rapid contamination of the pasture in spring and

summer.

Inhibition may be induced by prolonged exposure to unfavourable weather and development to adults of an inhibited population will recommence on removal of an existing adult burden by chemotherapy.

Chiejina & Mason (1977) reported a case of intermittant diarrhoea in a horse probably caused by successive waves of inhibited small strongyle larvae invading the gut lumen after repeated anthelmintic treatment.

Duncan, McBeath, Best & Preston, (1977) recently showed that fenbendazole has high efficacy against inhibited fourth stage larva, being 93% effective against Trichonema larvae at a dose rate of 30 mg./kg. and 83% effective against S. vulgaris larvae at a dose rate of 60 mg./kg. Similar results were attained against S. edentatus. Hence at high dose rates the drug is effective on both migrating and inhibited larvae, a capacity which no other drug has, to date.

As this review demonstrates, although a great deal of work has been conducted on equine strongyliasis, there has been little of a true epidemiological nature with animals infected with a single species. Undoubtably the high cost of equines as experimental animals is the root cause of this lack.

MATERIALS AND METHODS

Samples Collected	Principle Techniques Employed
1. Faeces	Faecal egg counts. Larval culture and differentiation.
2. Pasture Grass	Pasture larval count.
3. Unclotted Blood	Red blood cell count. White blood cell count. Packed cell volume. Haemoglobin estimation. Mean cell volume. White blood cell differentiation.
4. Serum	Total serum protein content. Albumin content. $\alpha\ \beta\ \gamma$ globulin content.
5. Post Mortem Material	Total large intestinal adult worm burden and strongyle species differentiation.

Faecal egg counts were carried out by a modification of the McMaster Egg Counting technique (Ministry of Agriculture, Fisheries and Food 1971). If the count was under 200 eggs per gram a more sensitive centrifugal flotation technique was adopted in which the eggs present in 0.8 gram of faeces were recovered and counted. In calculating the numbers of eggs per gram of faeces from the flotation procedure no correction factor was included to allow for incomplete recovery of the eggs.

Larval cultures were carried out using 5 grams of an equine faecal sample showing more than 400 e.p.g. by the modified McMaster technique. The faeces were held at 27°C for 8 days and the developing larvae differentiated using the key given in Soulsby (1965). In each case 100 larvae were identified and a percentage distribution of the species recorded.

Pasture larval counts were performed using a modification of the technique described by the Ministry of Agriculture, Fisheries and

Food (1971). An approximate 300 gram sample of grass was collected from sites throughout the pasture, concentrating on the areas in the field most often frequented by the horses. The sample washings were passed through sieves of pore size 150 µm. and 38 µm. and the material retained in the 38 µm. pore sieve used for larval extraction by a differential flotation technique using zinc sulphate solution of specific gravity 1.1. The grass was then oven-dried and the number of larvae per kilogram of dried herbage calculated.

Blood for examination was collected from the jugular vein using evacuated tubes (Becton Dickinson and Company, Rutherford, New Jersey) with ethylene diamose tetra acetic acid as an anticoagulant. Red blood cell counts, white blood cell counts, packed cell volumes, haemoglobin estimations and mean cell volumes were obtained using a Coulter Counter (Coulter Electronics Ltd., Dunstable, Beds., England) following the instructions given in the makers manual (Coulter Electronics Ltd., 1970).

Differential white blood cell counts were made using thin blood smears stained in 1:10 Giemsa, by the technique described by Dacie & Lewis (1975).

Total serum protein contents were evaluated using a biuret method (Henry, Sobel & Berkman, 1957). Albumin, alpha, beta and gamma globulin ratios were assessed by zone electrophoresis on cellulose acetate strips using Millipore* apparatus, the electrophoretograms being screened using a Phoroscope* densitometer. The details of the method are given in Millipore Biomedica (1976).

The absolute concentrations of serum albumin, alpha, beta and gamma globulins in g./100ml. were then calculated from the total serum

* Millipore Biomedica, Acton, Mass., U.S.A.

protein contents and percentages of each of those fractions.

A post mortem worm count was carried out following the technique described by The Ministry of Agriculture, Fisheries and Food (1971). Only the colonic and caecal contents were examined. Speciation of the strongyle worms recovered was carried out using the keys given by Lepage (1956) and Soulsby (1965). Meteorological data was obtained from the Easter Bush Weather Station.

Description of the Equine Units

Unit A This was the breeding unit of the Animal Health Farm, Royal (Dick) Veterinary Field Station, Easter Bush, Roslin. The animals studied (Table 3) were housed in the winter and turned out in spring onto pastures not grazed by horses for the previous four years. The mares were individually boxed and then put onto similarly clean pastures, separate from other horses on the farm. The animals were brought inside weekly, which facilitated routine sample collection including material for haematological examination. Routine anthelmintic treatment was carried out (Table 4) and the mares were treated twice before foaling.

Unit B This was at Gourlaw Farm, 1½ miles east of Roslin. The animals studied (Table 5) were retained outside for most of the year, only being housed in extremely cold weather. The mares were stabled during foaling. Sample collection was limited to faeces and pasture grass, but samples were routinely available (Tables 13 & 15). The horses were on pastures grazed by horses for at least one year prior to the study period, and the mares and foals were turned out onto similarly contaminated pastures. Anthelmintic treatment was haphazard (Table 6) and based largely on the routine results of this study.

Unit C This was at Mount Lothian, 1½ miles south east of Howgate, Midlothian. The animals studied (Table 7) were at pasture during the study period and sample collection was only sporadic and limited to faeces and pasture grass. The horses were kept on extensive, previously little grazed pastures. Anthelmintic usage was occasional (Table 8) and based on the faecal egg counts of this study.

RESULTS

TABLES 3-8, 18 and 36

GRAPHS 1-42

TABLE 3

DESCRIPTION OF HORSES STUDIED AT UNIT A

IDENTIFICATION	SEX	AGE (APPROX.) YRS.	DATE BORN (1977 FOALS)
00	F	7	
01	F	12	
04	F		
08	F	11	
10	F	11	
18	F	9	
Y01	F	2	
Y11	M	2	
F10 (76)	F	1	
F11 (76)	F	1	
F18 (76)	F	1	
F00 (77)	F		29.4.77
F01 (77)			27.4.77
F04 (77)	F		19.6.77
F08 (77)	M		29.4.77
F10 (77)	M		20.7.77
F18 (77)	F		15.4.77
ALBERT	M	3	

00, 01, 08, 18, F00 (77), F01 (77), F08 (77), F18 (77)
Turned out to pasture together 2nd week May 1977

Y01, Y11, F10 (76), F11 (76), F18 (76), ALBERT
Turned out to pasture together 2nd week May 1977

04, F04 (77) Turned out with mares and foals 1st July 1977

10, F10 (77) Turned out with mares and foals 15th August 1977

TABLE 4

ANTHELMINTIC USE AT UNIT A

DATE	HORSE IDENTIFICATION	DRUG USED
24.9.76	10, 18, 11, Y11	Mebendazole*
8.10.76	Y01, Y11	"
8.10.76	10, 11, 18	"
22.10.76	10, 11, 18	"
5.11.76	10, 11, 18	Pyrantel embonate**
5.11.76	Y01, Y11	Mebendazole
18.11.76	00, 01, 08, 18	"
19.11.76	Y01, Y11, 10	"
17.12.76	01	Fenbendazole***
20.12.76	Y01, Y11	"
9.3.77	Y01, Y11	"
18.3.77	18	Mebendazole
23.3.77	00, 01, 08	Fenbendazole
30.3.77	18	"
12.4.77	00, 01, 08	"
14.4.77	Y01, Y11	"
2.5.77	04	Mebendazole
9.5.77	00, 01, 08, 18	"
17.5.77	04	Fenbendazole
27.6.77	F04	"
14.7.77	10	Mebendazole
27.7.77	10	Fenbendazole

* Telmin - (Crown Chemical Co. Ltd., Lamberhurst, Kent)
** Strongid P - (Pfizer Ltd., Sandwich, Kent)
*** Panacur - (Hoechst Pharmaceuticals, Hounslow, Middlesex)

TABLE 5

DESCRIPTION OF HORSES STUDIED AT UNIT B

IDENTIFICATION	SEX	AGE APPROX. (YRS.)	DATE BORN (1977 FOALS)
Lollipop	F	2	
Blue	F	1	
Cressie	F	2	
Feztival	F	1	
Cadger	M	2	
Mais Belle	F	7	
Greta	F	14	
Val	F	13	
Towpath	F	7	
Cloudie	F	14	
Velvet	F	3	
Prince	M	2	
Nimbus	M	5	
Story	M	3	
Saturday	F	1	
Towpath Foal			31/5/77
Cloudie Foal			30/4/77
Greta Foal			22/4/77
Val Foal			21/5/77
Mais Belle Foal			1/6/77

TABLE 6

ANTHELMINTIC USE AT UNIT B

Date	Horse Identification	Drug Used
20/2/77	Prince	Thiabendazole*
	Nimbus	"
5/3/77	Story	Dichlorvos**
	Velvet	"
	Nimbus	"
	Prince	"
	Towpath	"
	Greta	"
	Cadger	"
6/3/77	Val	Dichlorvos
	Cloudie	"
	Mais Belle	"
	Cressie	"
	Lollipop	"
4/4/77	Festival	Dichlorvos
	Blue	"
20/4/77	Cloudie	Dichlorvos
23/4/77	Strike	Pyrantel Embonate
1/7/77	Val	Dichlorvos
3/7/77	Cloudie	Dichlorvos
	Greta	"
17/7/77	Velvet	Dichlorvos
29/7/77	Mais Belle	Fenbendazole

* Equizole (Merck, Sharp & Dohme Ltd., London)

** Frisk (Thomas Pettifer and Co. Ltd., Guildford, Surrey)

TABLE 7

DESCRIPTION OF HORSES STUDIED AT UNIT C

IDENTIFICATION	AGE	SEX
Dusky	5 yrs.	M
Moruisq	3 yrs.	F
Rhum	2 yrs.	F

TABLE 8

ANTHELMINTIC TREATMENT AT UNIT C

DATE	HORSE IDENTIFICATION	DRUG USED
11/2/77	Dusky	Mebendazole
	Rhum	"
	Moruisq	"
30/4/77	Moruisq	Mebendazole
	Rhum	"
16/5/77	Dusky	Mebendazole
	Rhum	"
18/7/77	Dusky	Mebendazole
	Moruisq	"
2/8/77	Moruisq	Fenbendazole

TABLE 18

TABLE SHOWING SPECIES DISTRIBUTION FROM LARVAL CULTURE RESULTS

STRONGYLID SPP.	OVER ALL PREVALENCE	MEAN LEVEL OF INFECTION WHEN SPECIES PRESENT
Trichonema spp.	100%	99.6%
Strongylus vulgaris	13%	2.5%
S. edentatus	5%	2%
Triodontophorus spp.	18.3%	14%

TABLE 36

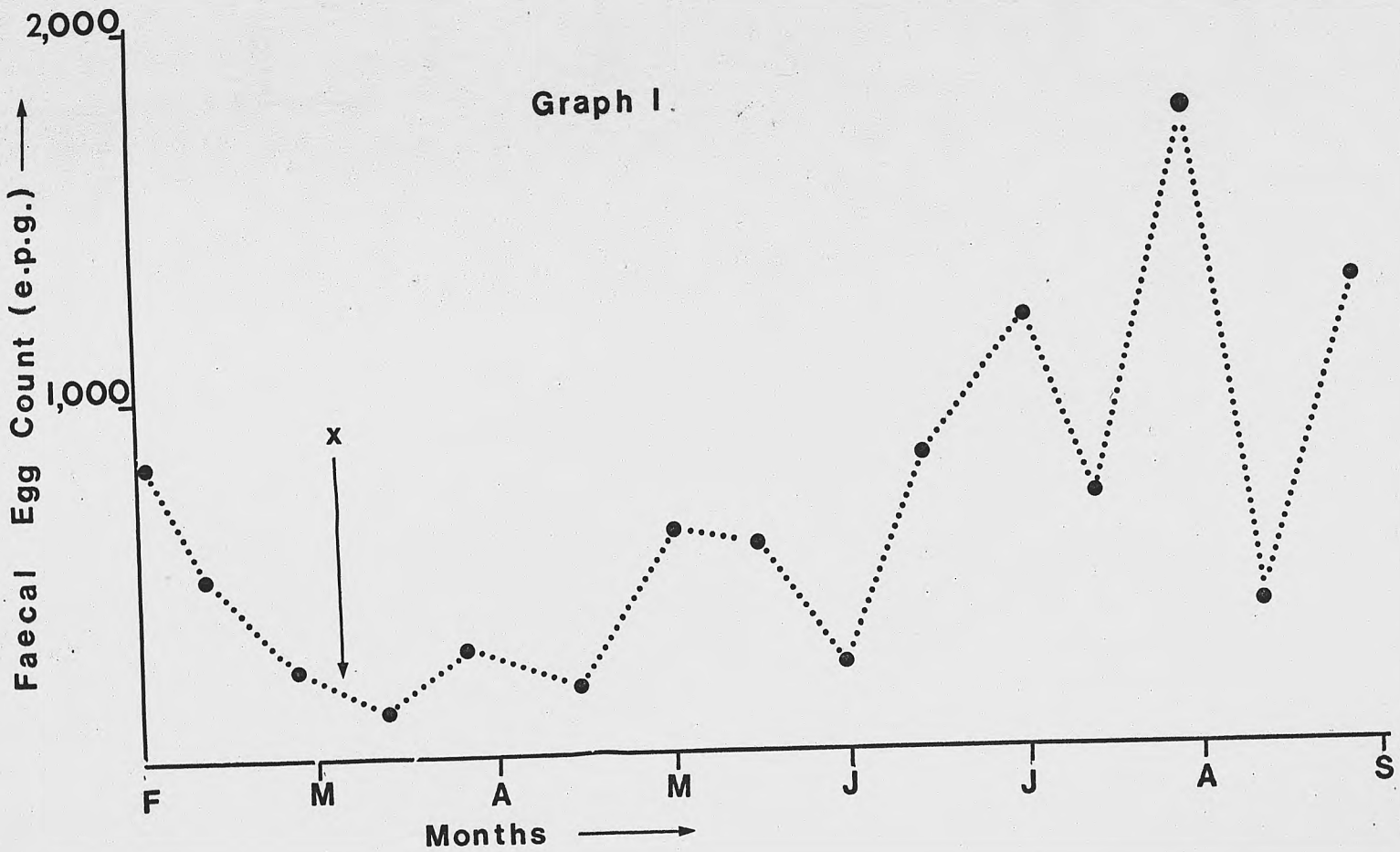
POST MORTEM RESULTS

DATE	ANIMAL	NO. OF ADULT STRONGYLES AND SPECIES		NO. OF INHIBITED LARVAE	FAECAL EGG COUNT RESULTS	
		Caecum	Large Intestines		Mc	Salt
30/11/76	7 yr. female pony	none	none	200-400 Trichonema species	0	0
12/1/77	8 yr. male pony	400 Trichonema spp.	256 Trichonema spp.	v. large number	0	10
2/3/77	2½ yr. female pony	none	20 Trichonema spp.	none	50	6

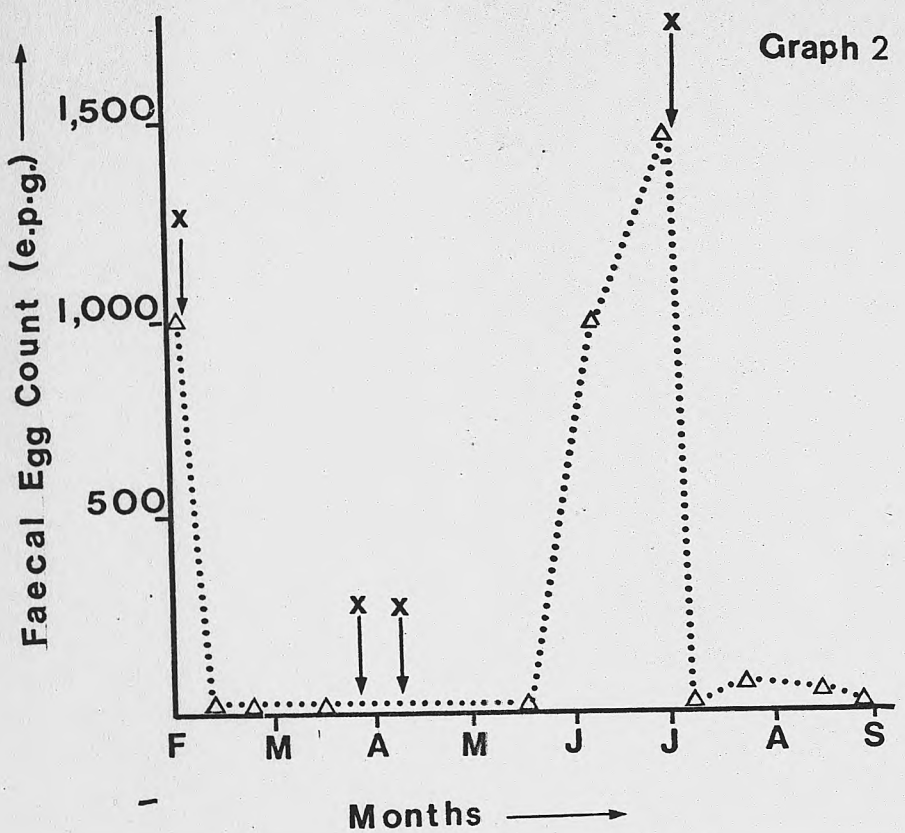
Graph 1

Average faecal egg counts of horses at Unit B, during the study period.

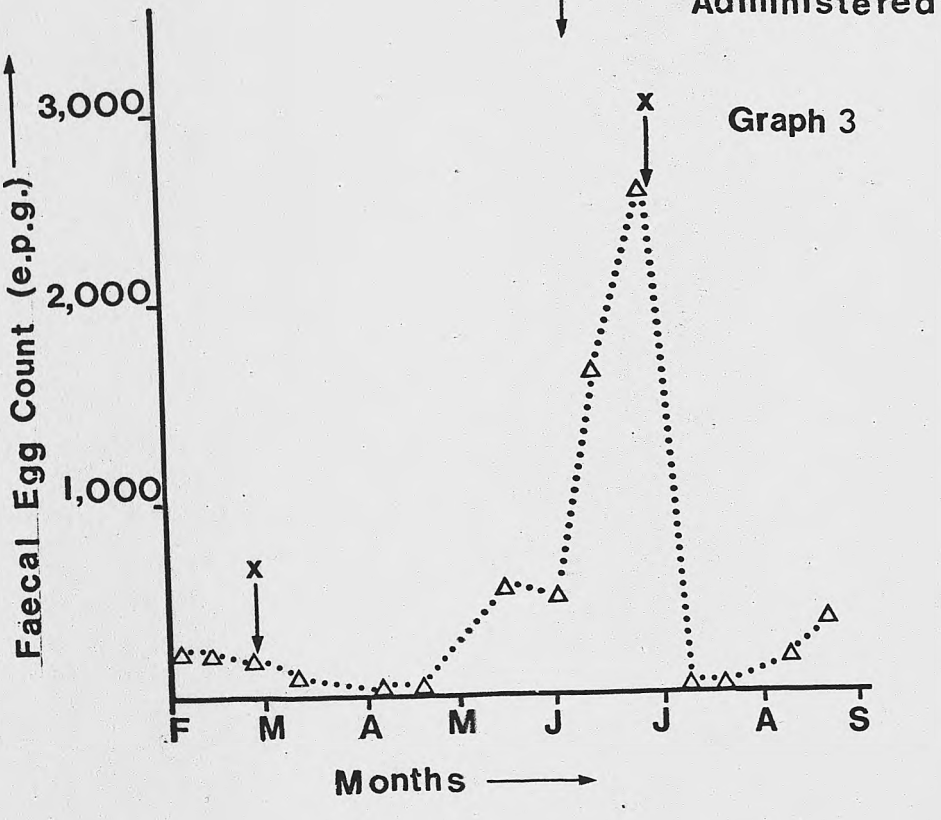
Animals treated with Dichlorvos


Graph I.
2,000
1,000
Faecal Egg Count (e.p.g.)
X
F
M
A
M
J
J
A
S
Months

Graph Showing Faecal Egg Count Monthly Of Cloudie And Greta, Unit B, During The Study Period.



x Dichlorvos Administered



Graphs 4, 5 and 6

Faecal egg counts (e.p.g.) of mares at Unit B during study period.

Graph 4 - Story

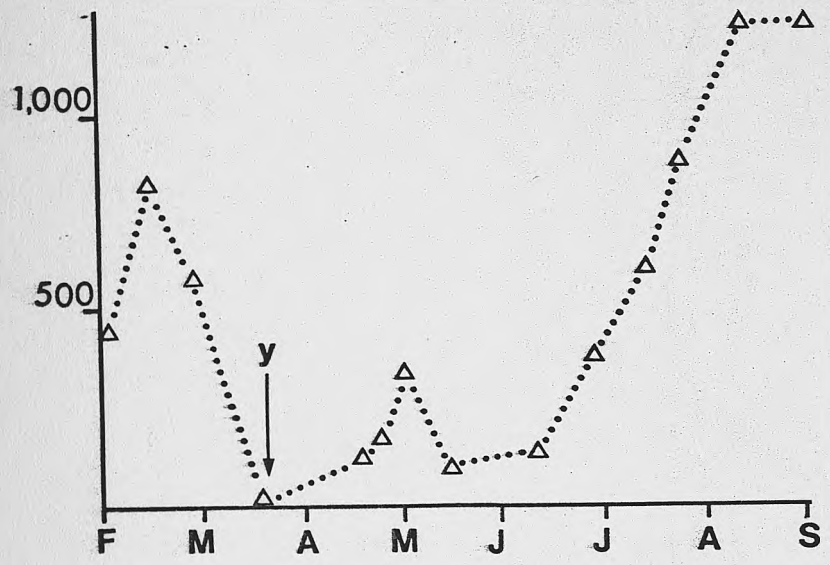
Graph 5 - Nimbus

Graph 6 - Velvet

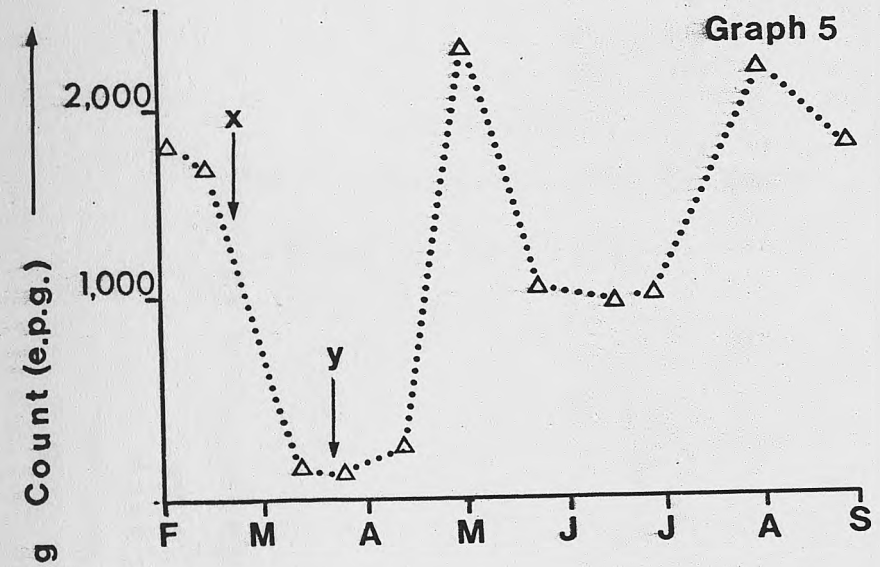
x ⟶ Thiabendazole administered

y ⟶ Dichlorvos administered

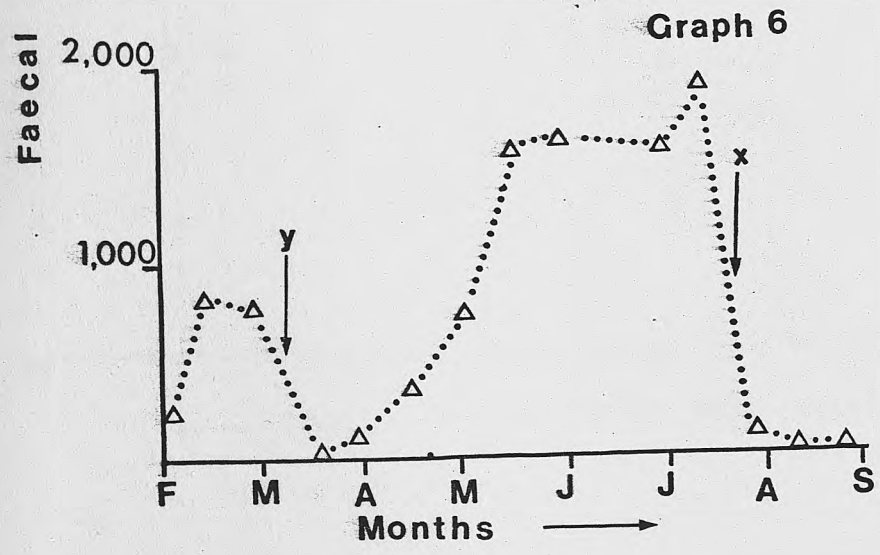
Graph 4



Graph 5

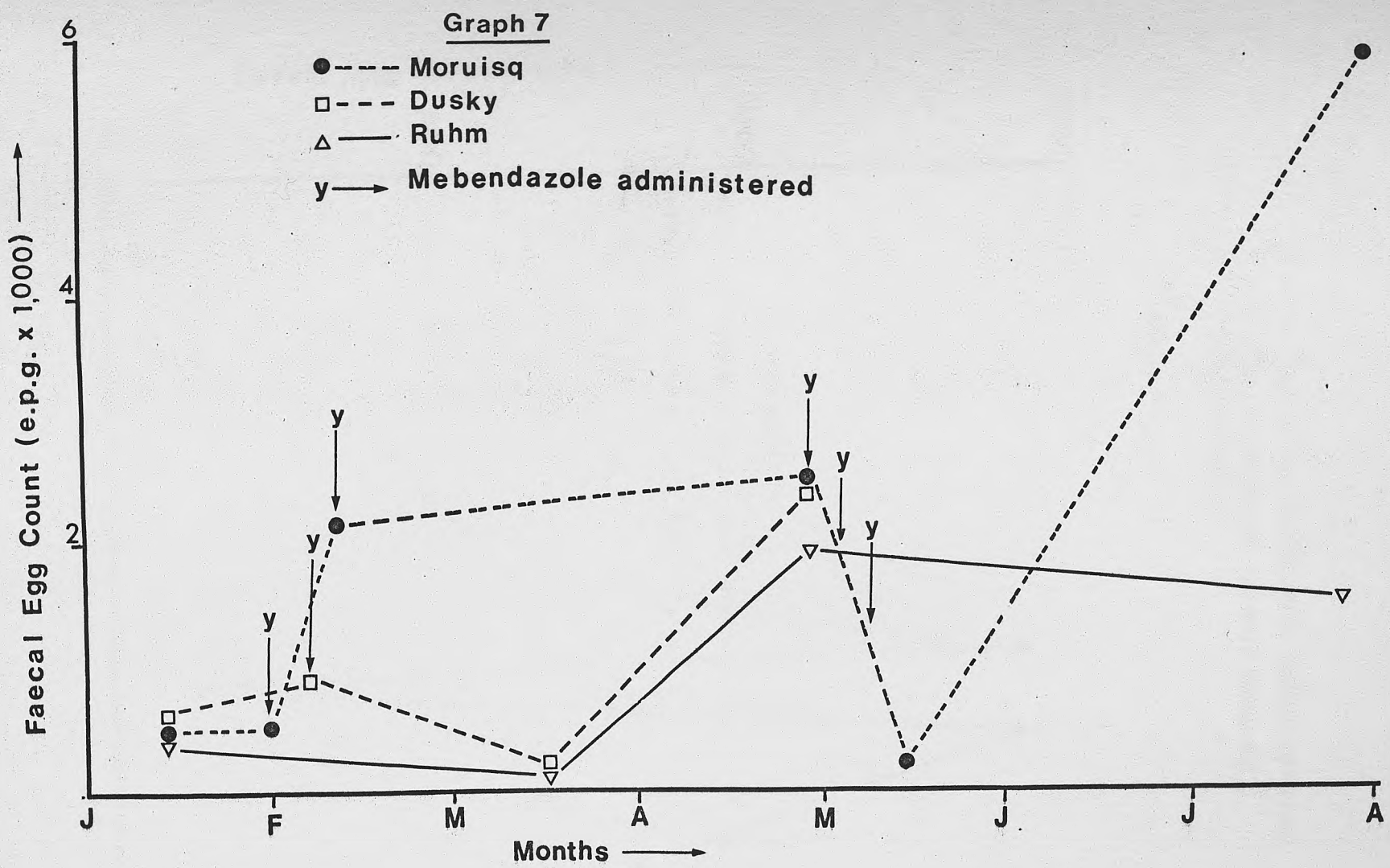


Graph 6



Graph 7

Faecal egg counts (e.p.g.) of horses at Unit C during the study period.

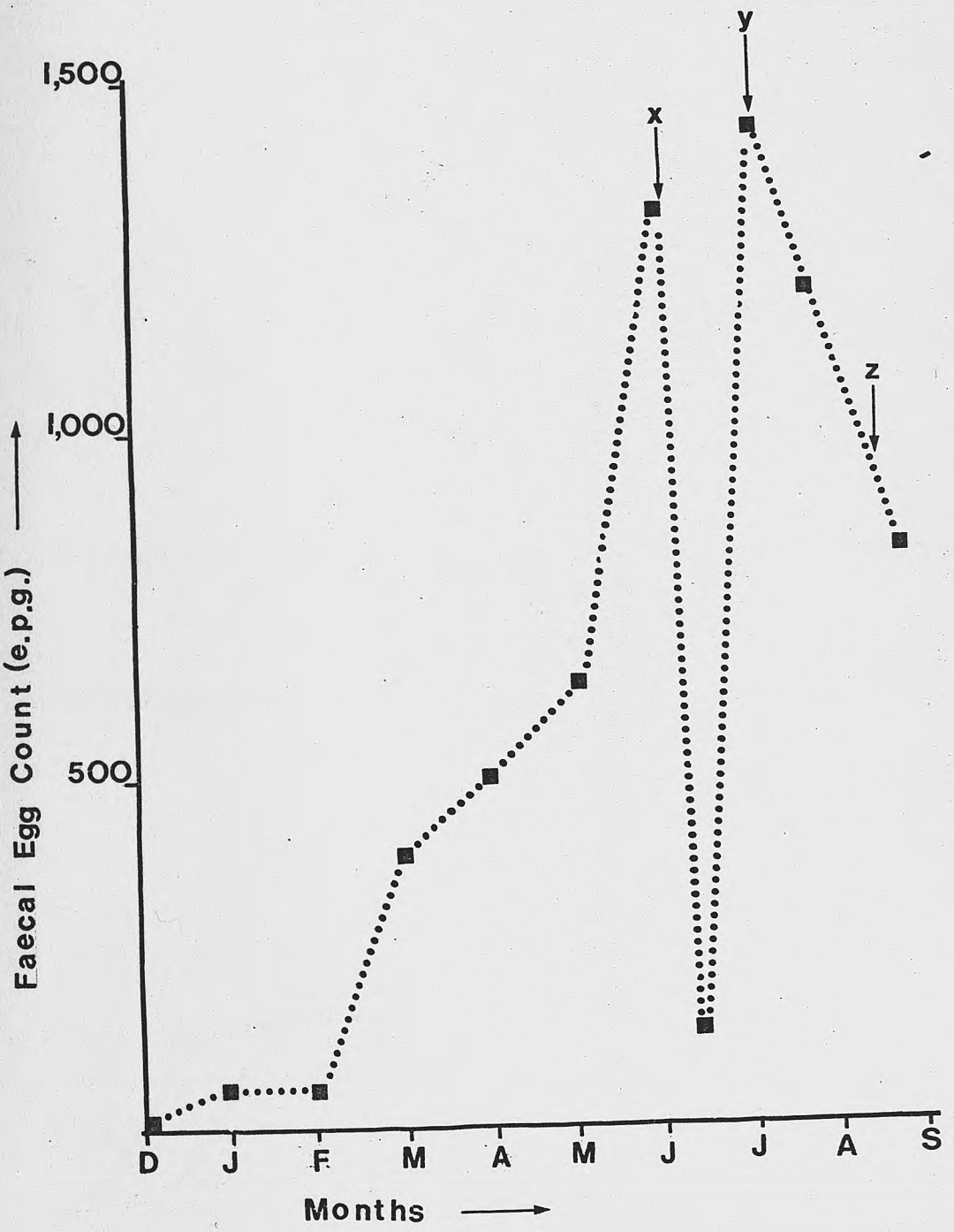

Graph 7
Moruisq
Dusky
Ruhm
y Mebendazole administered
Faecal Egg Count (e.p.g. x 1,000)
6
4
2
J
F
M
A
M
J
J
A
Months
y

Graph 8

Faecal egg count (e.p.g.) of horse no. 10, Unit A, during study period.

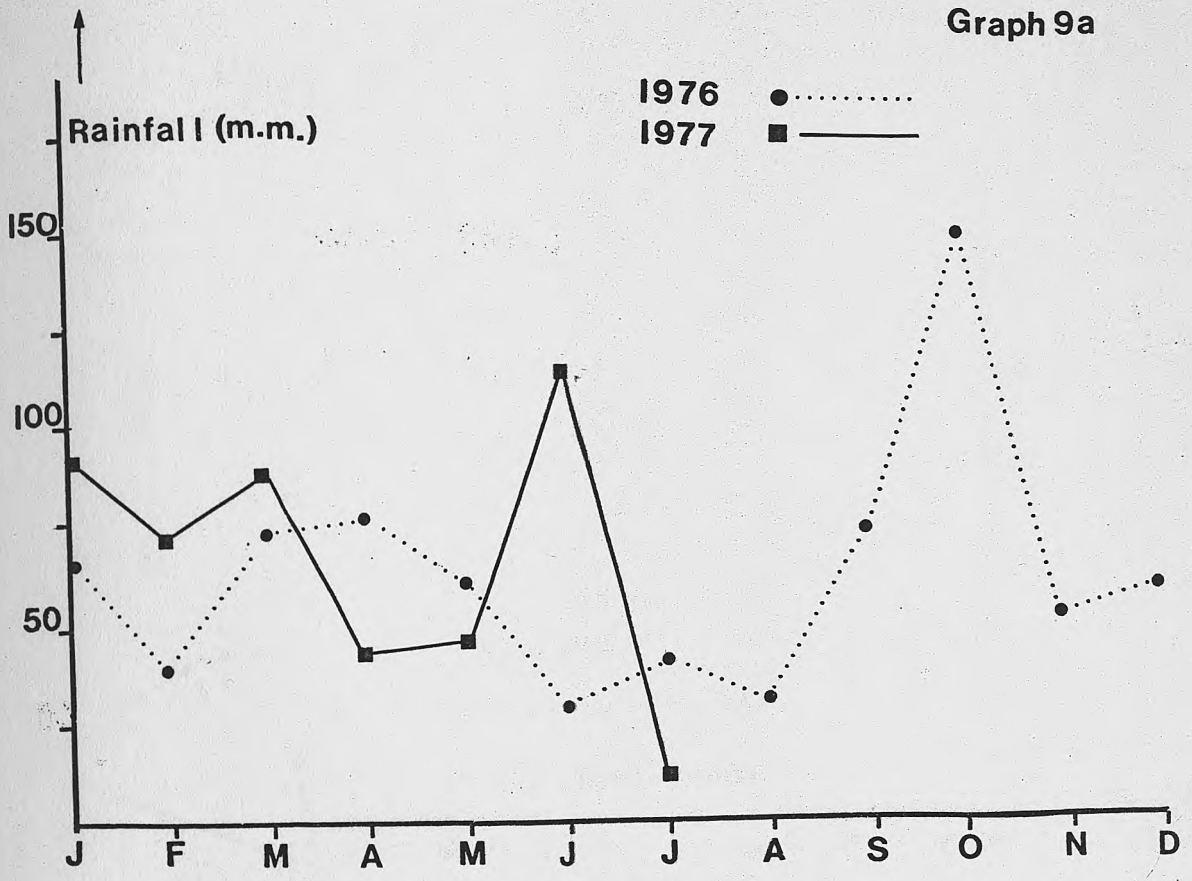
x ⟶ Pyrantel Embonate Administered
y ⟶ Fenbendazole Administered
z ⟶ Turned Out

Graph 8



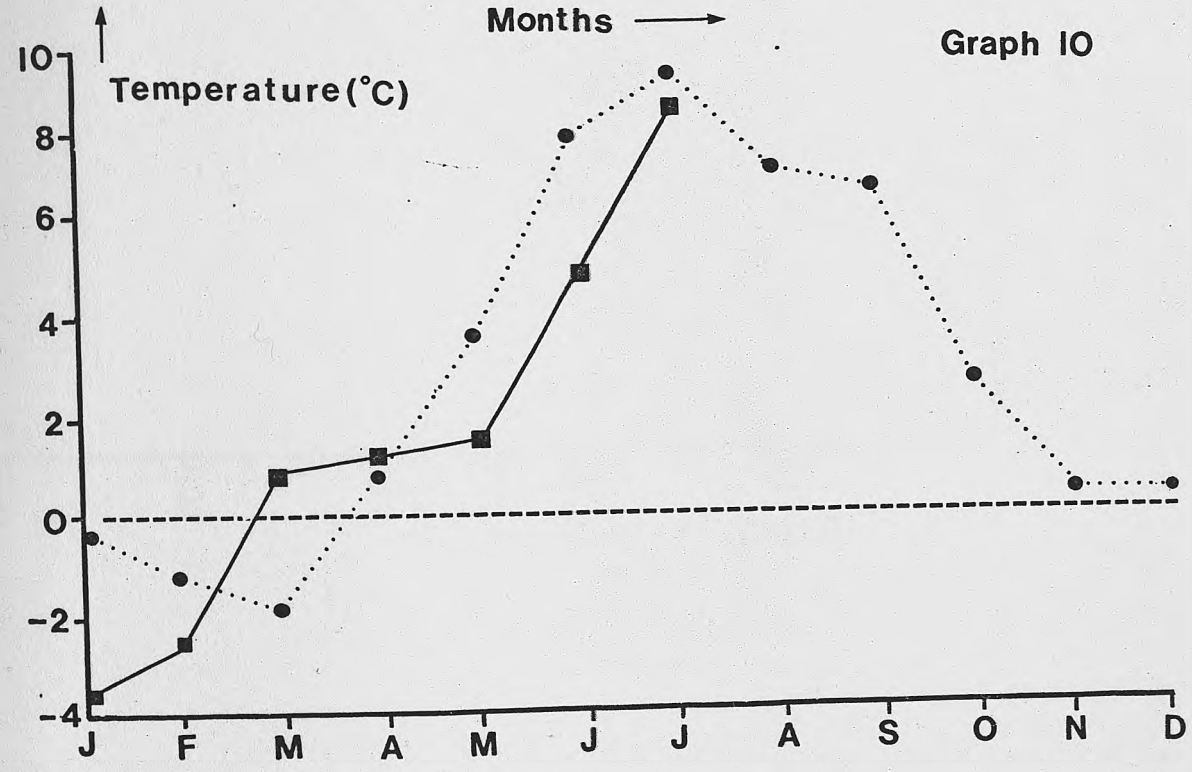
Graph Showing Mean Monthly Rainfall (m.m.) And Mean Monthly Minimum Grass Base Temperature (°C) 1976/77

Graph 9a



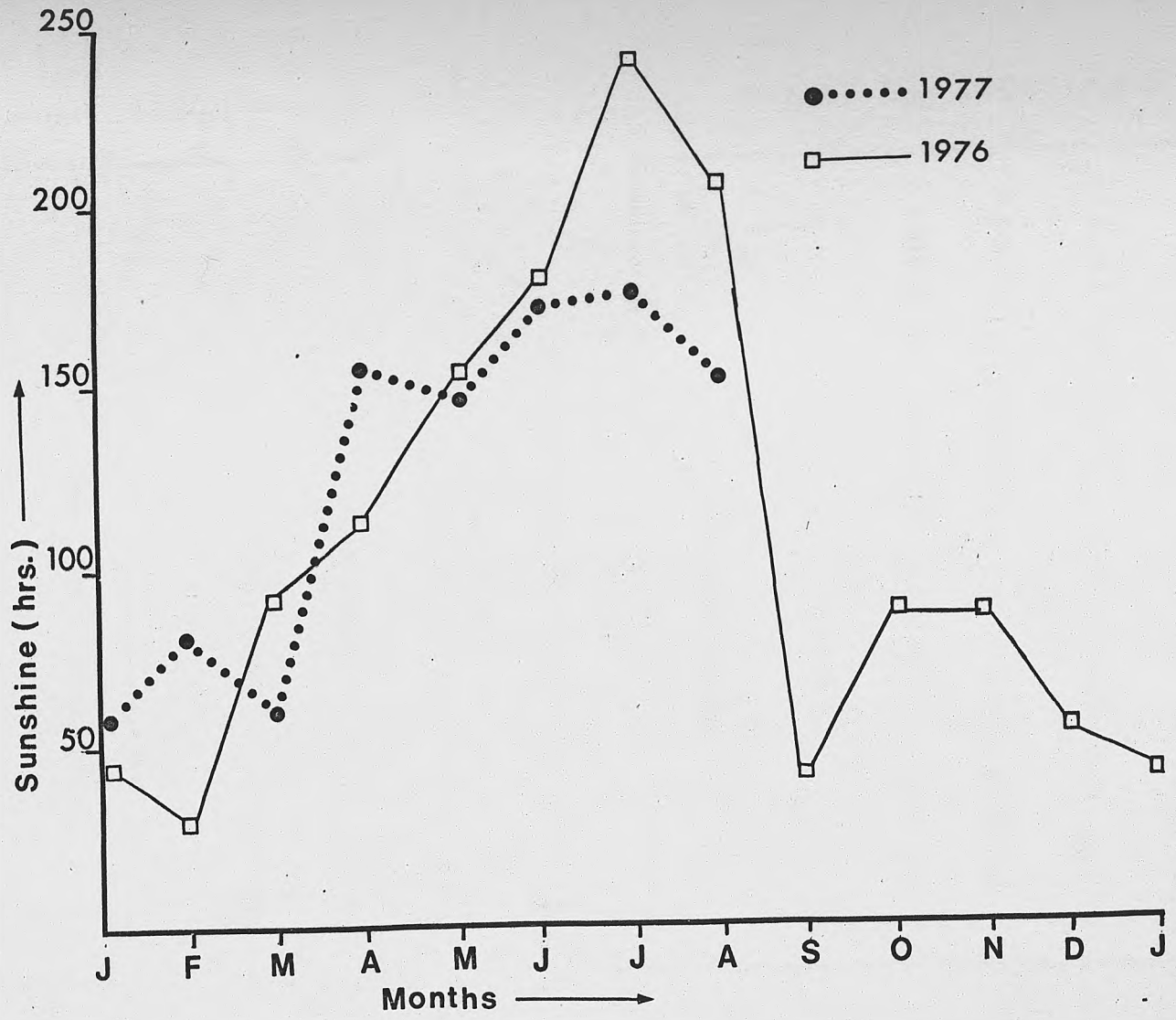
Months →

Graph 10



Graph 9b

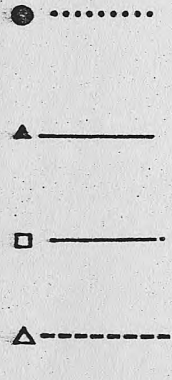
Number of hours sunshine per month during 1976 and 1977.


250
200
150
100
50
Sunshine (hrs.)
1977
1976
J
F
M
A
M
J
J
A
S
O
N
D
J
Months

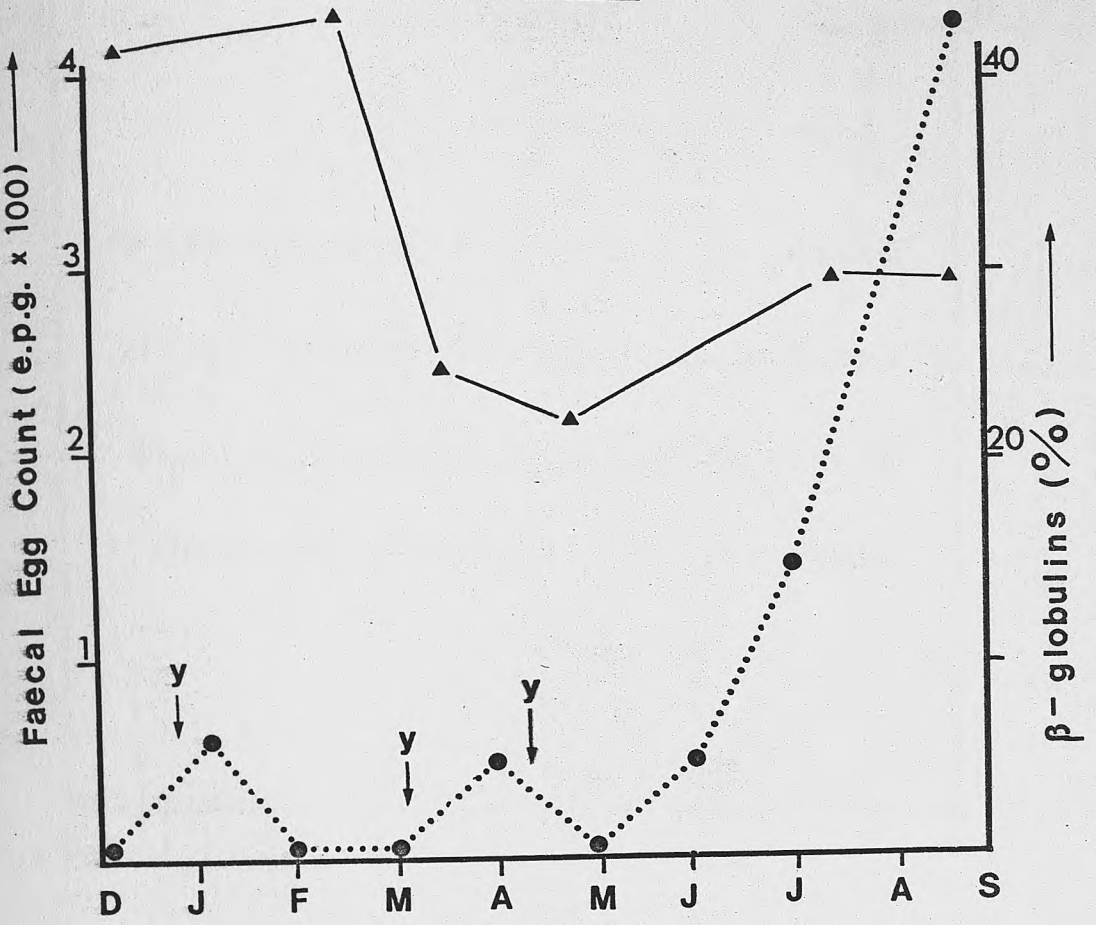
Graph 9b

Graphs of Results of Monthly Faecal Egg Counts (e.p.g.) and Monthly Levels of ß-globulins (%), Eosinophils (%) and Neutrophils (%), Throughout the Study Period.

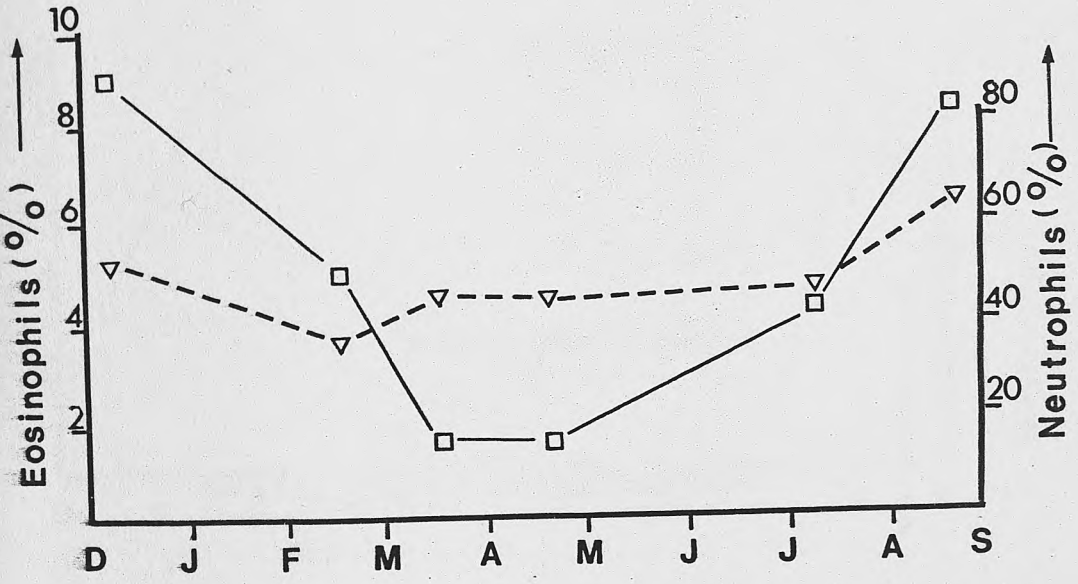
Horse No. YOI



Graph 11



Graph 12



Graphs of Results of Monthly Faecal Egg Counts(e.p.g.) and Monthly Levels of ß-globulins (%), Eosinophils (%) and Neutrophils (%), Throughout the Study Period.

Horse No. YII

● ····· Faecal Egg Counts.

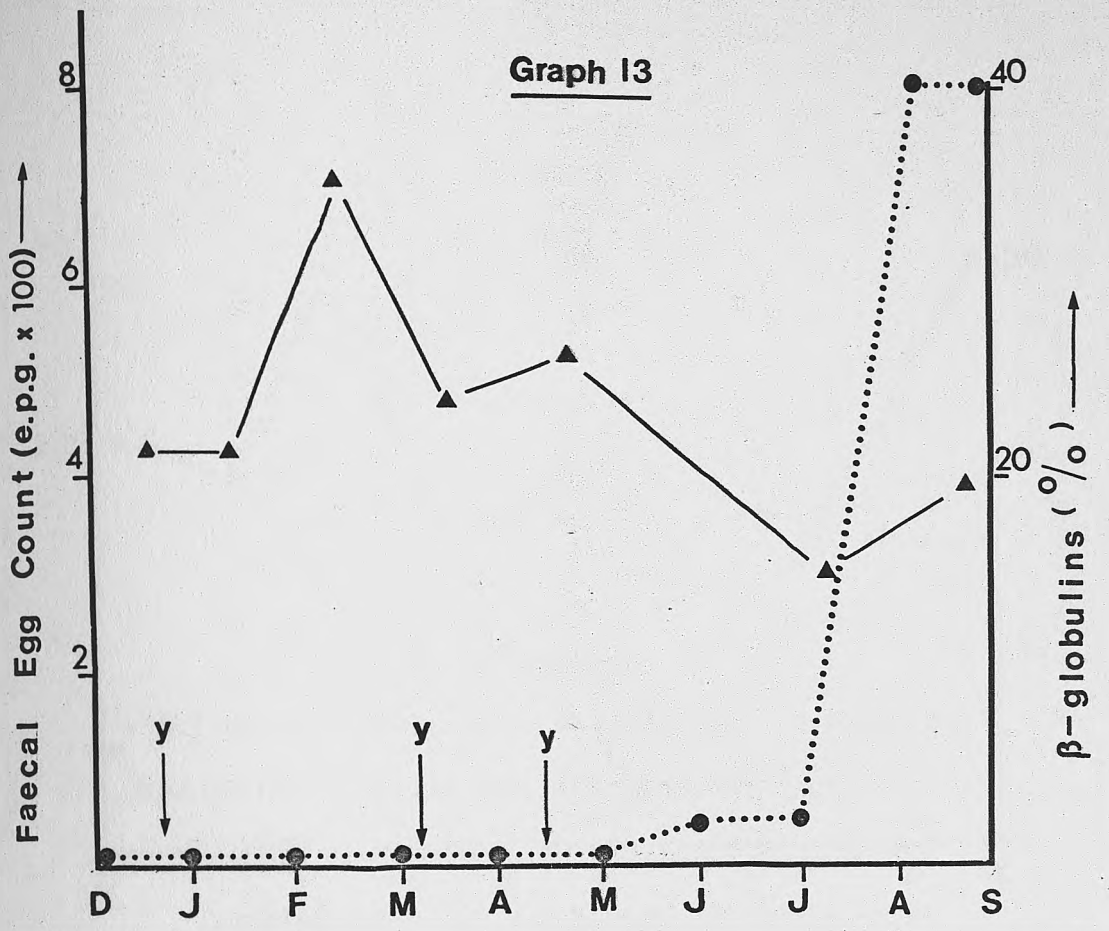
▲——— ß-globulins.

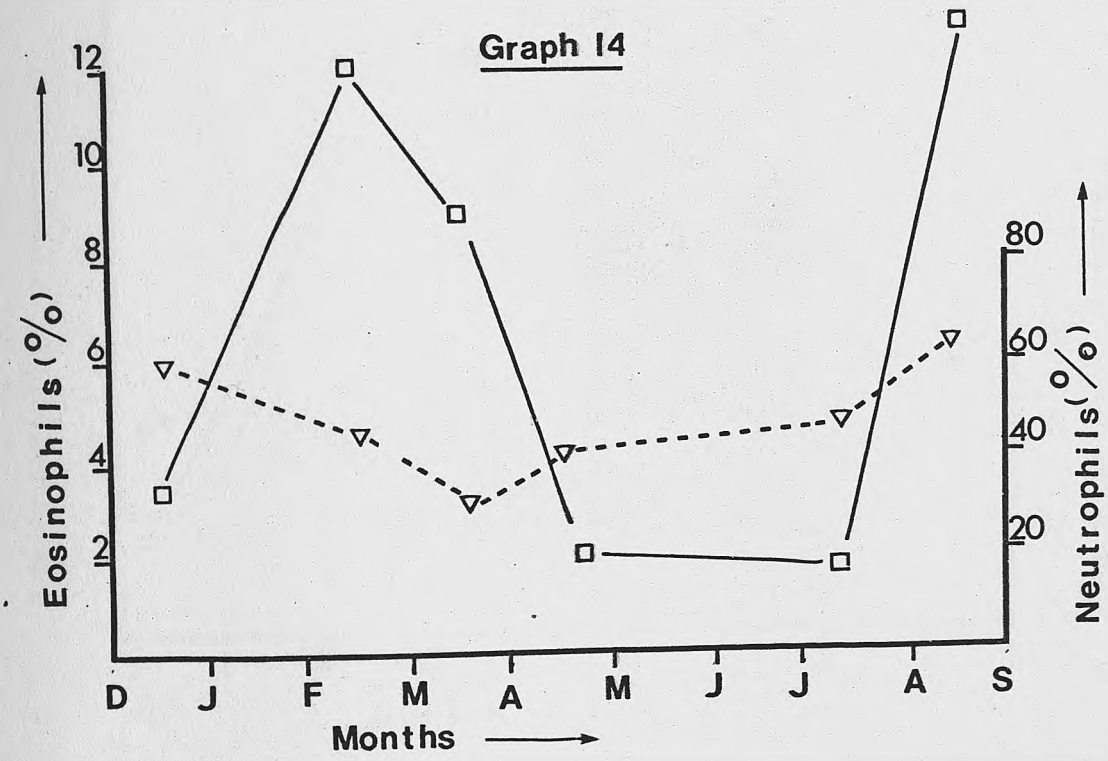
□ ———· Eosinophils.

△-------- Neutrophils.

x
|
▽ Pyrantel Embonate

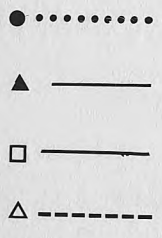
y
|
▽ Fenbendazole Administered


Graph 13
Faecal Egg Count (e.p.g. x 100)
β-globulins (%)
8
6
4
2
40
20
y
y
y
D
J
F
M
A
M
J
J
A
S


Graph 14
Eosinophils (%)
Neutrophils (%)
12
10
8
6
4
2
80
60
40
20
D
J
F
M
A
M
J
J
A
S
Months

Graphs 15 & 16

Average of results from three foals born in 1976, of monthly faecal egg counts (e.p.g.) and monthly levels of β globulins (%) eosinophils (%) and neutrophils (%) throughout the study period.

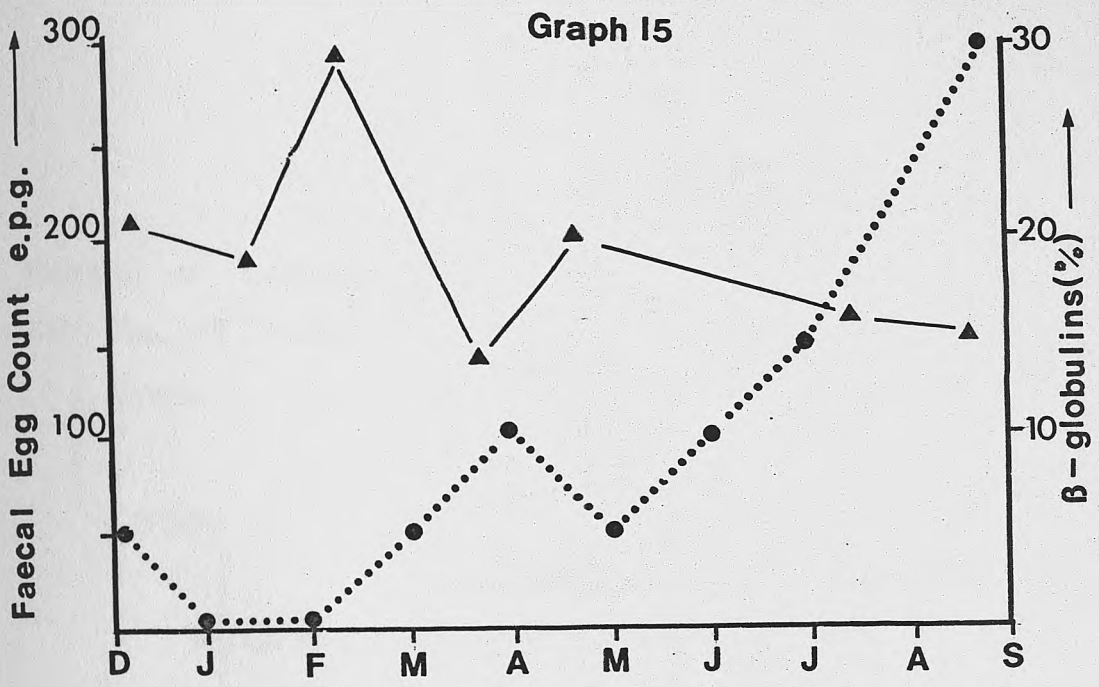


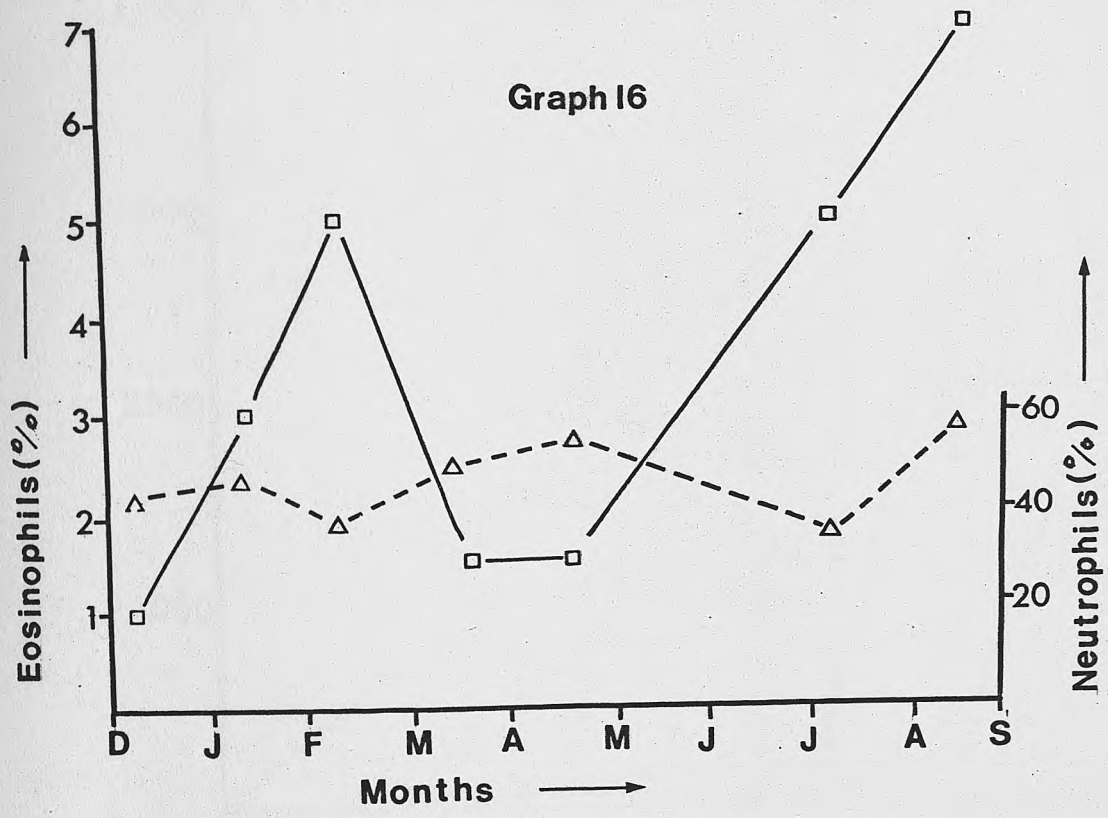
Faecal Egg Counts

β globulins

Eosinophils

Neutrophils


Graph 15
Faecal Egg Count e.p.g.
300
200
100
30
20
10
β-globulins(%)
D
J
F
M
A
M
J
J
A
S


Graph 16
Eosinophils(%)
7
6
5
4
3
2
1
60
40
20
Neutrophils(%)
D
J
F
M
A
M
J
J
A
S
Months

Graph 17

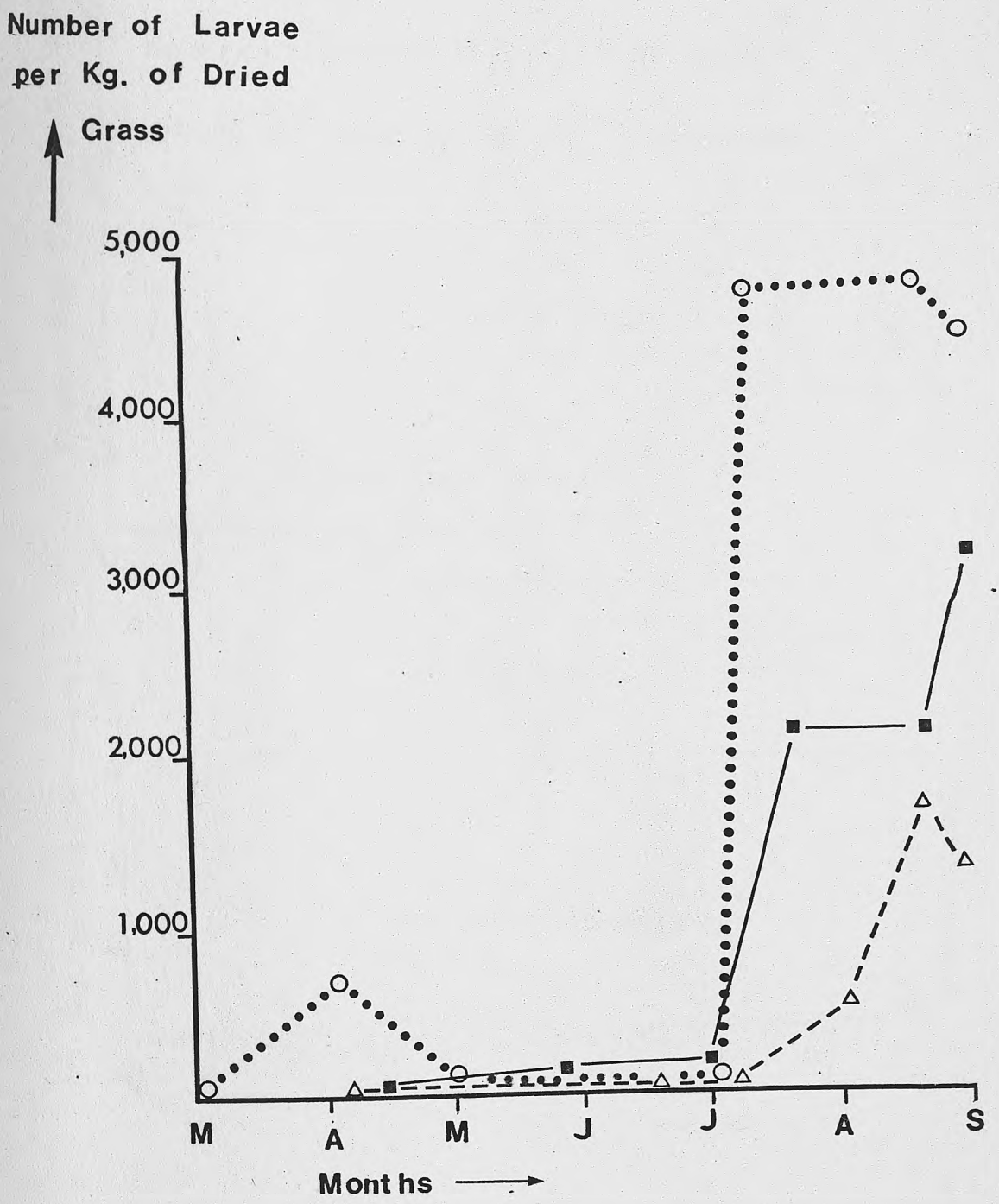
Number of infective larvae on the selected pastures during the study period.

○ ••••• Unit B pasture of mares and foals

■ —— Unit A pasture of mares and foals

△ – – – Unit A pasture of 1yr. & 2yr. olds

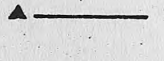
Graph 17



Graphs of Results of Monthly Faecal Egg Counts (e.p.g.) and Monthly Levels of ß-globulins (%), Eosinophils (%) and Neutrophils (%), Throughout the Study Period.

Horse No. 00

Faecal Egg Counts.



ß-globulins

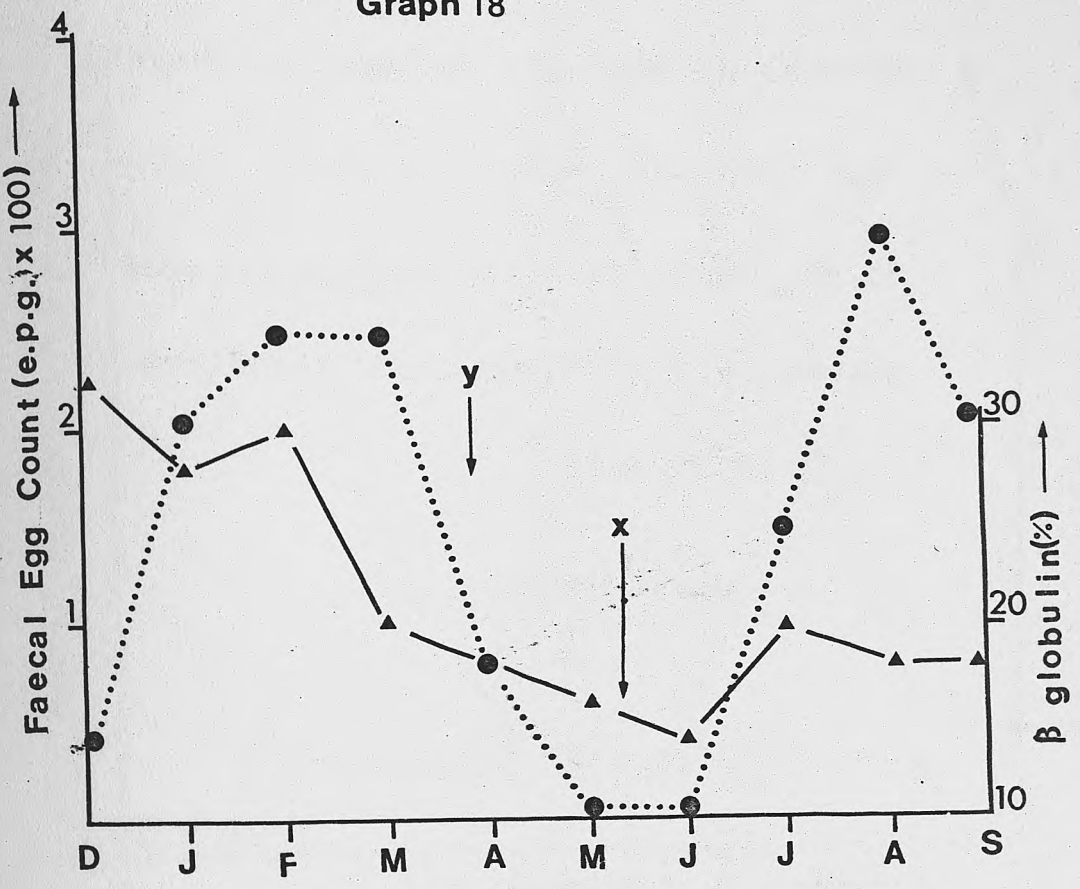
Eosinophils.

Neutrophils.

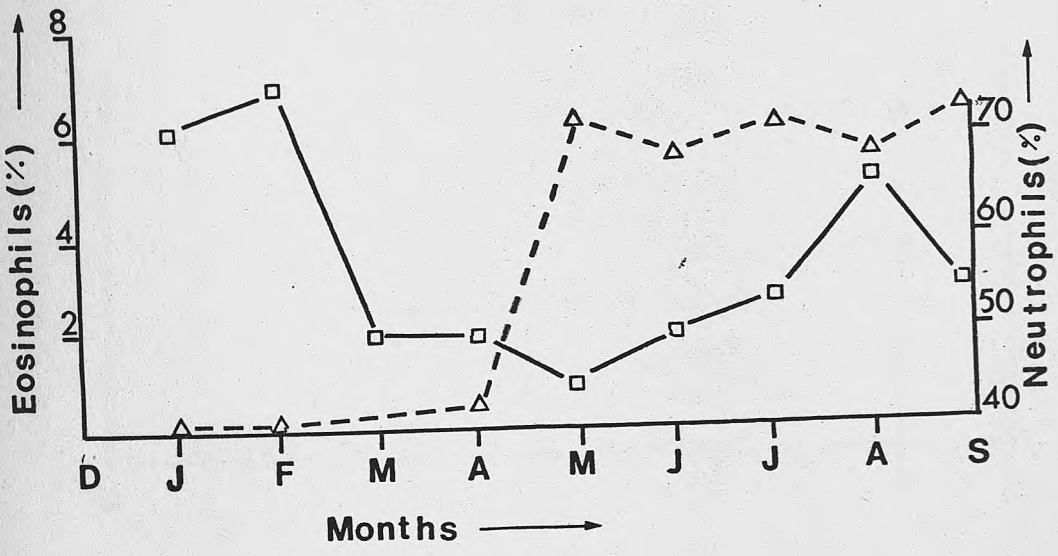
Pyrantel Embonate

Fenbendazole Administered

Graph 18



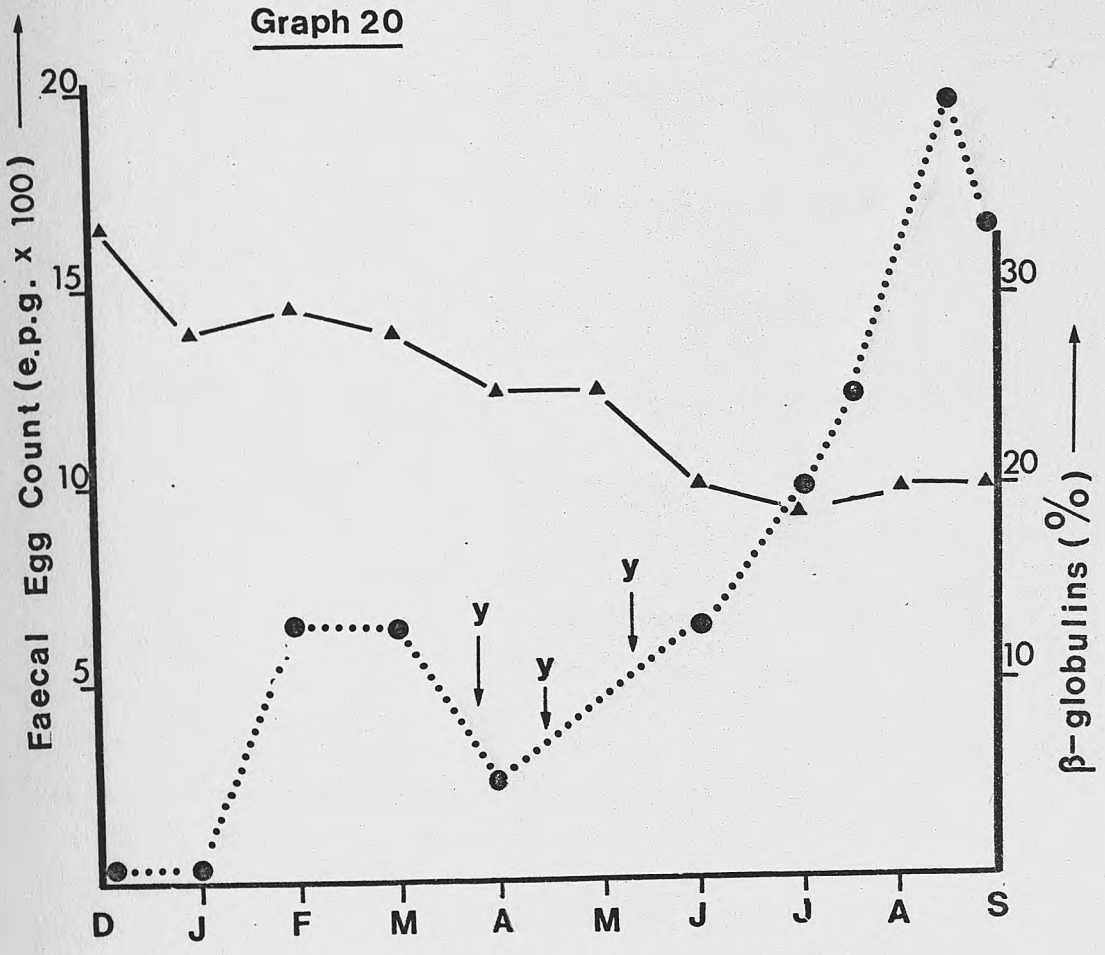
Graph 19



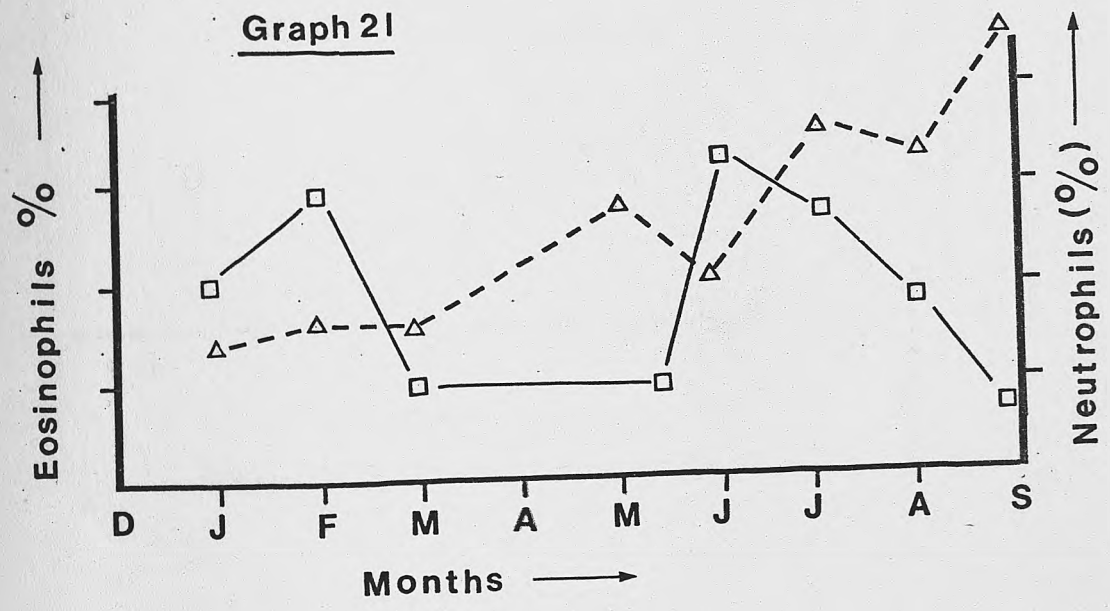
Graphs of Results of Monthly Faecal Egg Counts (e.p.g.) and Monthly Levels of ß-globulins (%), Eosinophils (%) and Neutrophils (%), Throughout the Study Period.

Horse No. 08

Graph 20



Graph 21



Graphs of Results of Monthly Faecal Egg Counts (e.p.g.) and Monthly Levels of β-globulins (%), Eosinophils (%) and Neutrophils (%), Throughout the Study Period.

Horse No. 18


Faecal Egg Counts.


β-globulins

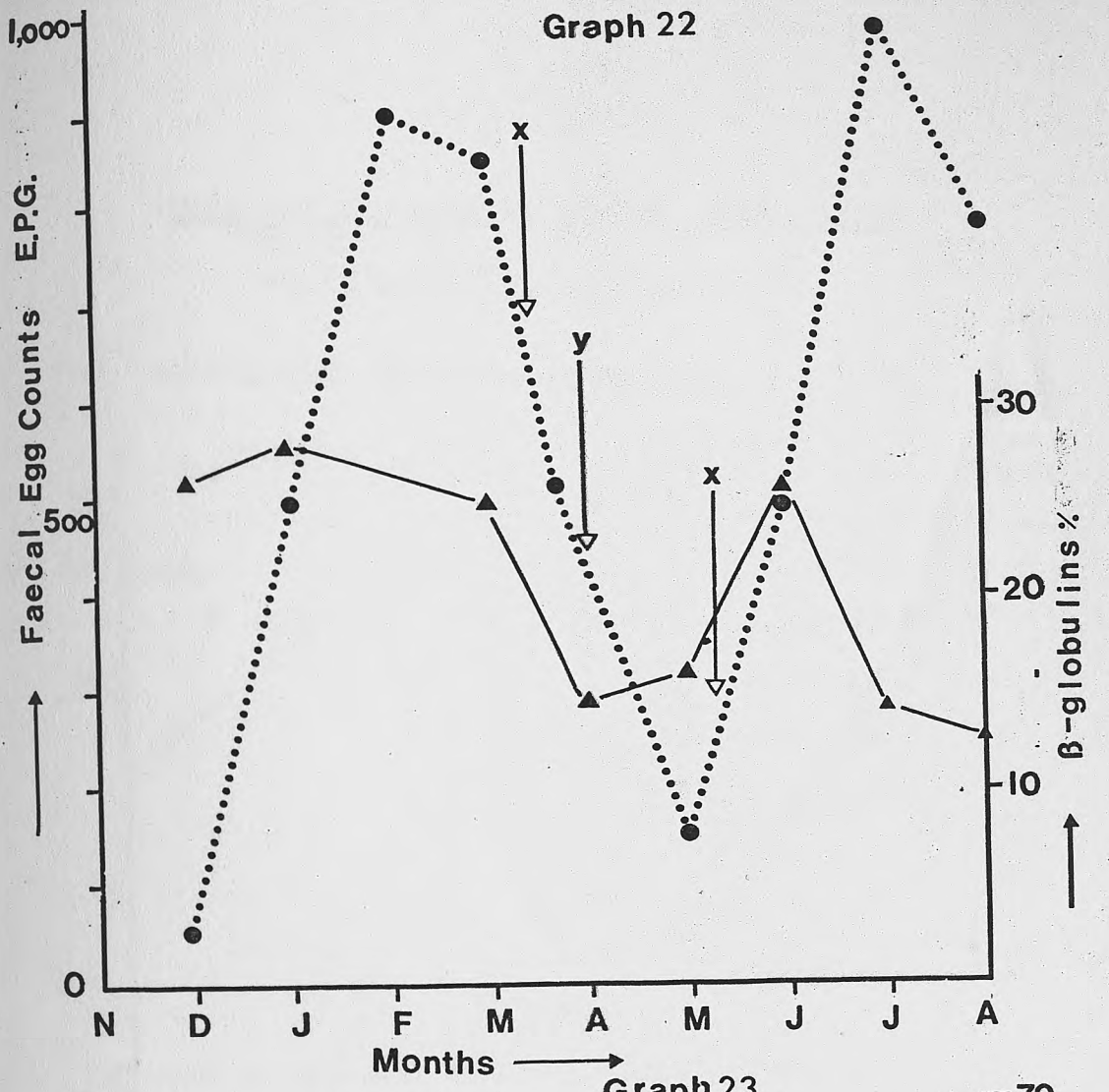

Eosinophils.


Neutrophils.

Pyrantel Embonate

Fenbendazole Administered

Graph 22



Graph 23

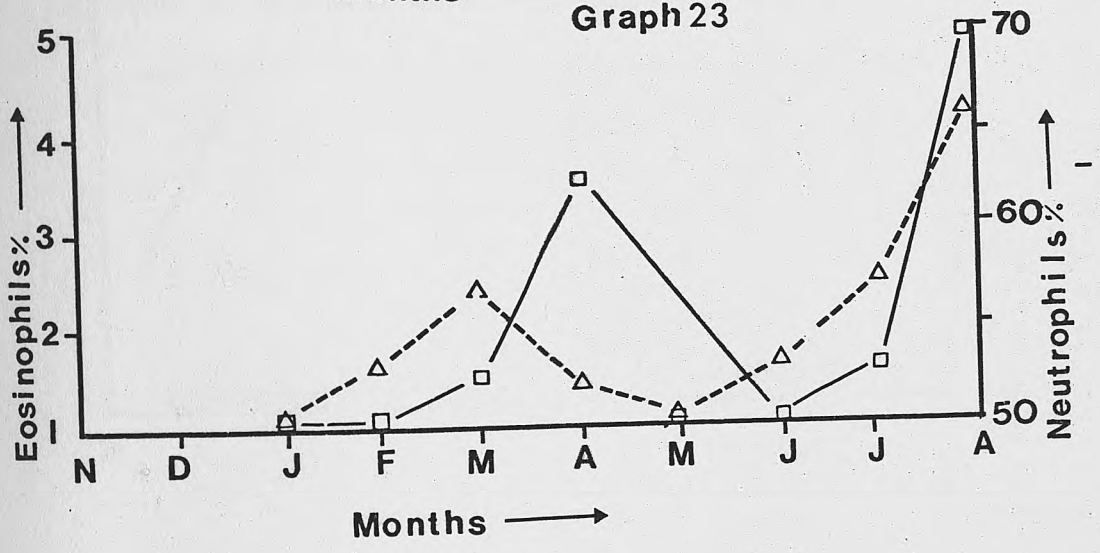


TABLE SHOWING STRONGYLOIDES FAECAL EGG COUNT

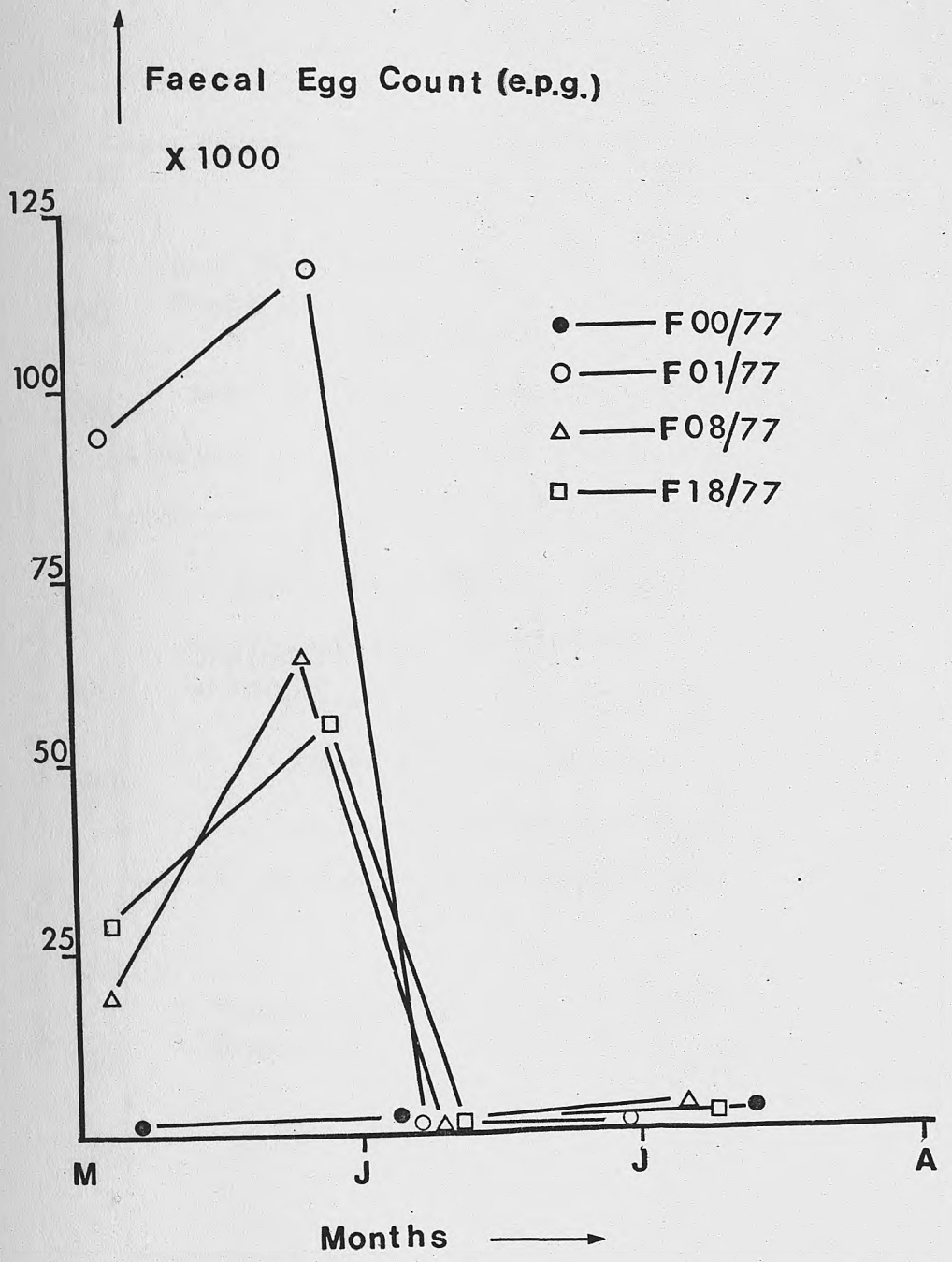
OF FOALS BORN IN 1977 AT UNIT A.

Date / Horse no.	7/5	20/5	26/5	2/6	8/6	7/7	14/7	30/7	7/8
F00/77	-ve				200		200		
F01/77	94,000	113,500		0		0	0	0	0
F08/77	16,800	60,500			0		1,250	150	150
F18/77	26,600		54,500	0		1,500	0	0	0

Graph 24

Strongyloides faecal egg counts (e.p.g.) of foals born in 1977 at Unit A, during the study period.

Graph 24

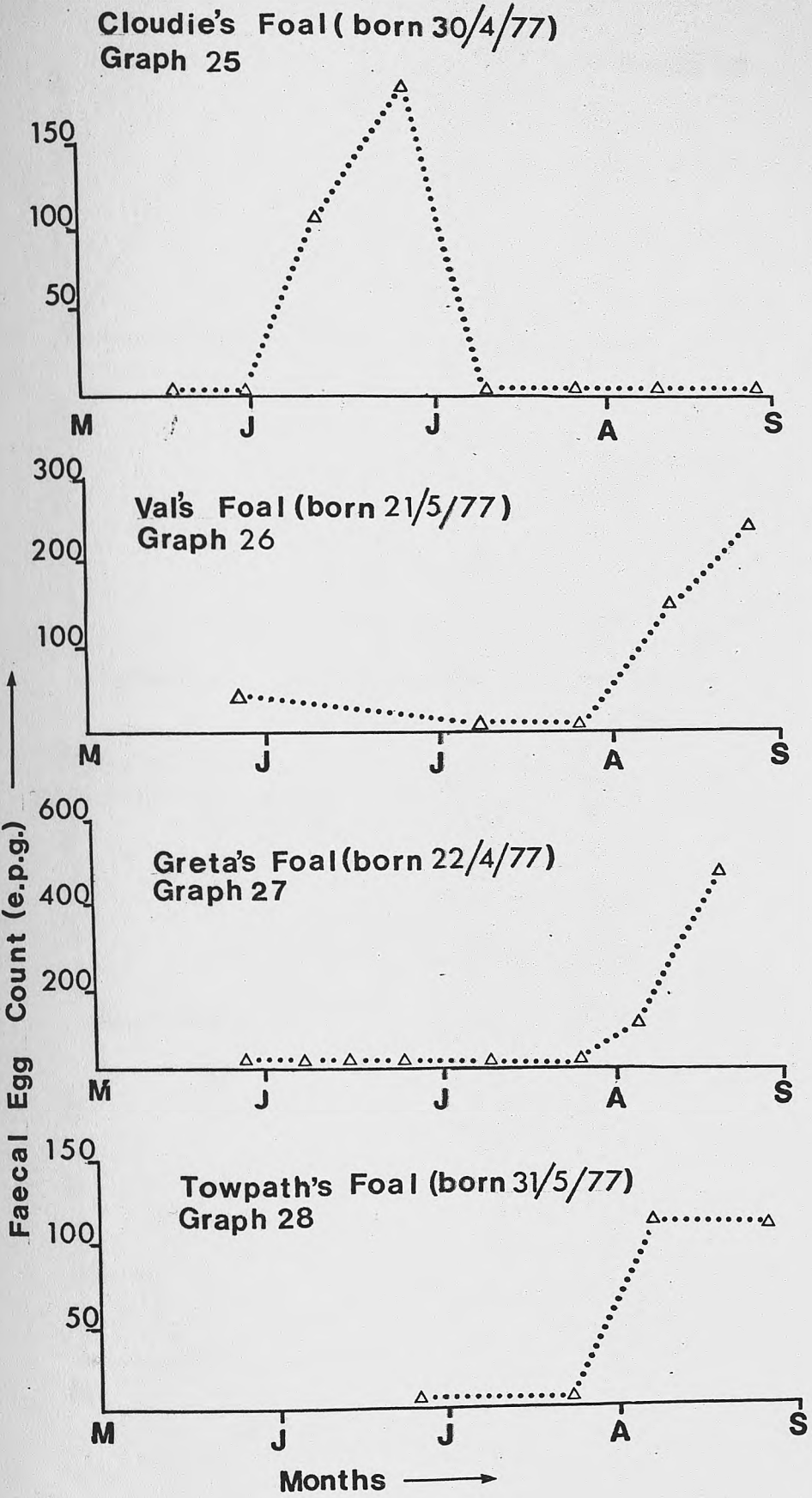

Faecal Egg Count (e.p.g.)
X 1000
125
100
75
50
25
F 00/77
F 01/77
F 08/77
F 18/77
M
J
J
A
Months

Graphs 25-28

Faecal egg counts (e.p.g.) of foals born at Unit B during the study period.

Prepatent Periods Established

Cloudie's Foal	6 weeks
Val's Foal	9 weeks
Greta's Foal	13 weeks
Towpath's Foal	9 weeks
Average	9-2 weeks

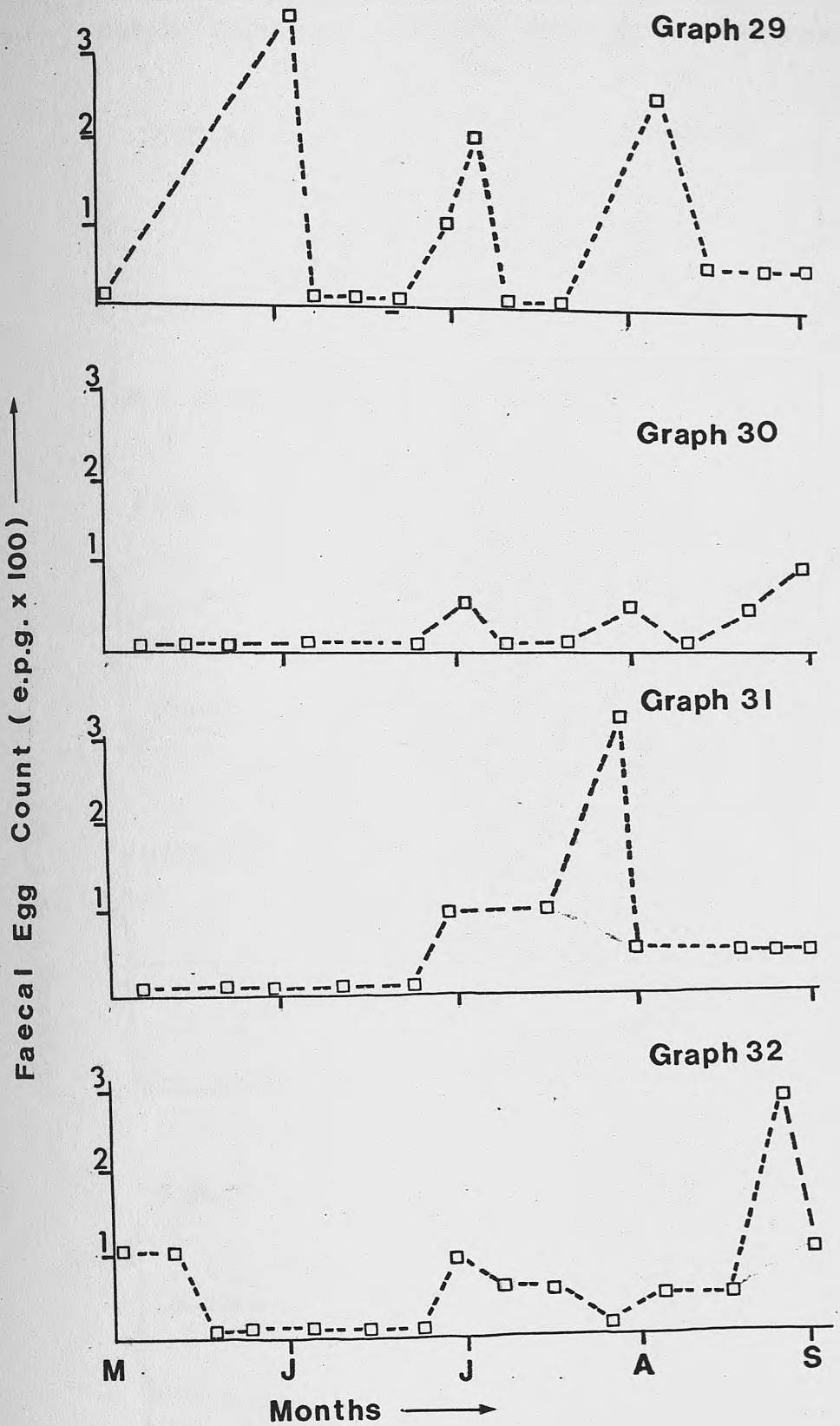

Cloudie's Foal (born 30/4/77)
Graph 25
150
100
50
M
J
J
A
S
300
200
100
Val's Foal (born 21/5/77)
Graph 26
600
400
200
Greta's Foal (born 22/4/77)
Graph 27
150
100
50
Towpath's Foal (born 31/5/77)
Graph 28
Faecal Egg Count (e.p.g.)
Months

Graphs 29-32

Faecal egg counts (e.p.g.) of

foals born in 1977 at Unit A.

GRAPH NO.	HORSE NO.	DATE BORN	ESTABLISHMENT OF PATENT STRONGYLE INFECTION FROM BIRTH (WKS.)
29	F00/77	29/4/77	9
30	F01/77	27/4/77	9
31	F08/77	29/4/77	9
32	F18/77	15/4/77	10


Graph 29
Graph 30
Graph 31
Graph 32
Faecal Egg Count (e.p.g. x 100)
3
2
1
M
J
J
A
S
Months

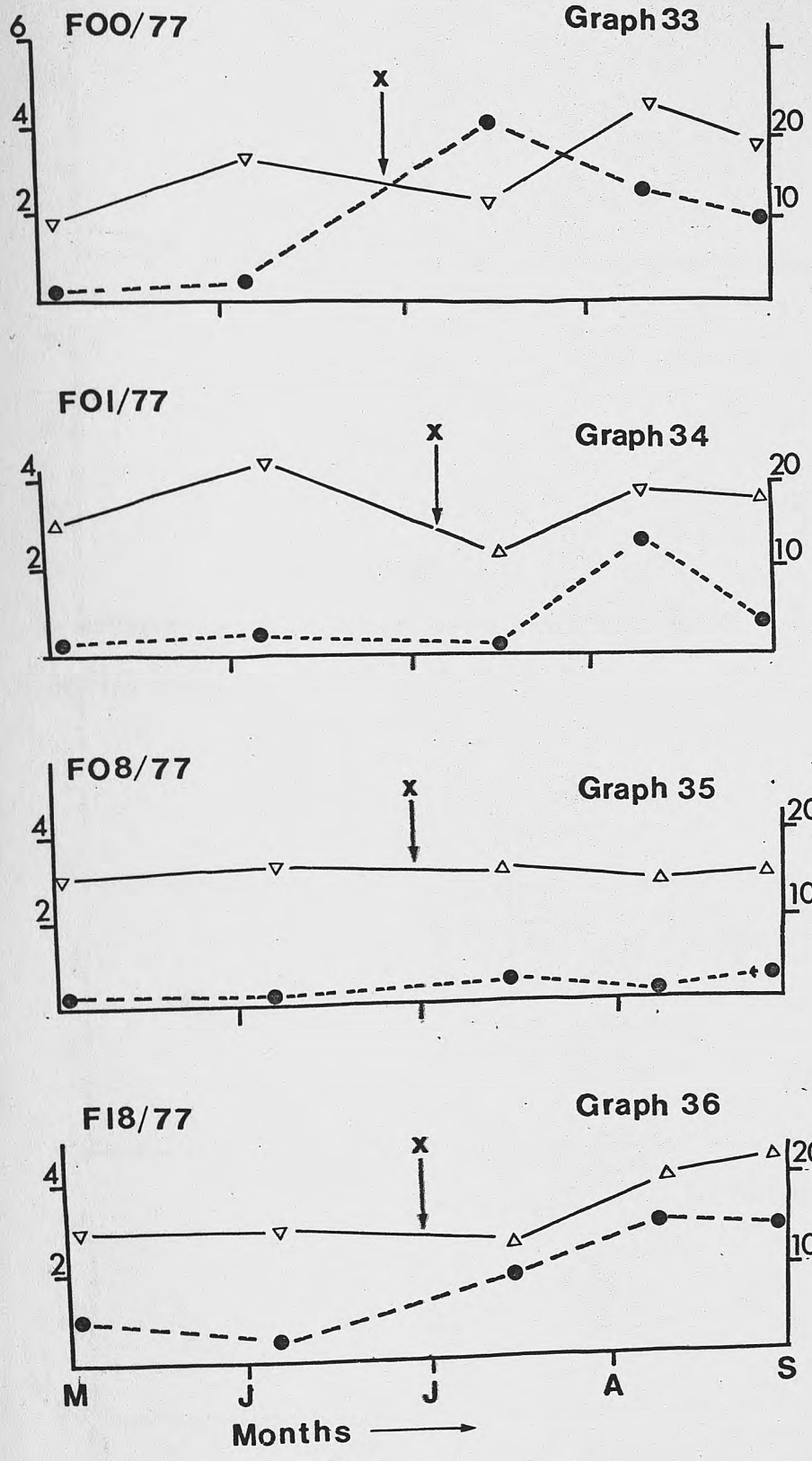
Graphs 33-36

β globulin (%) and eosinophil (%) levels of foals born in 1977 at Unit A.

▽— β-globulins

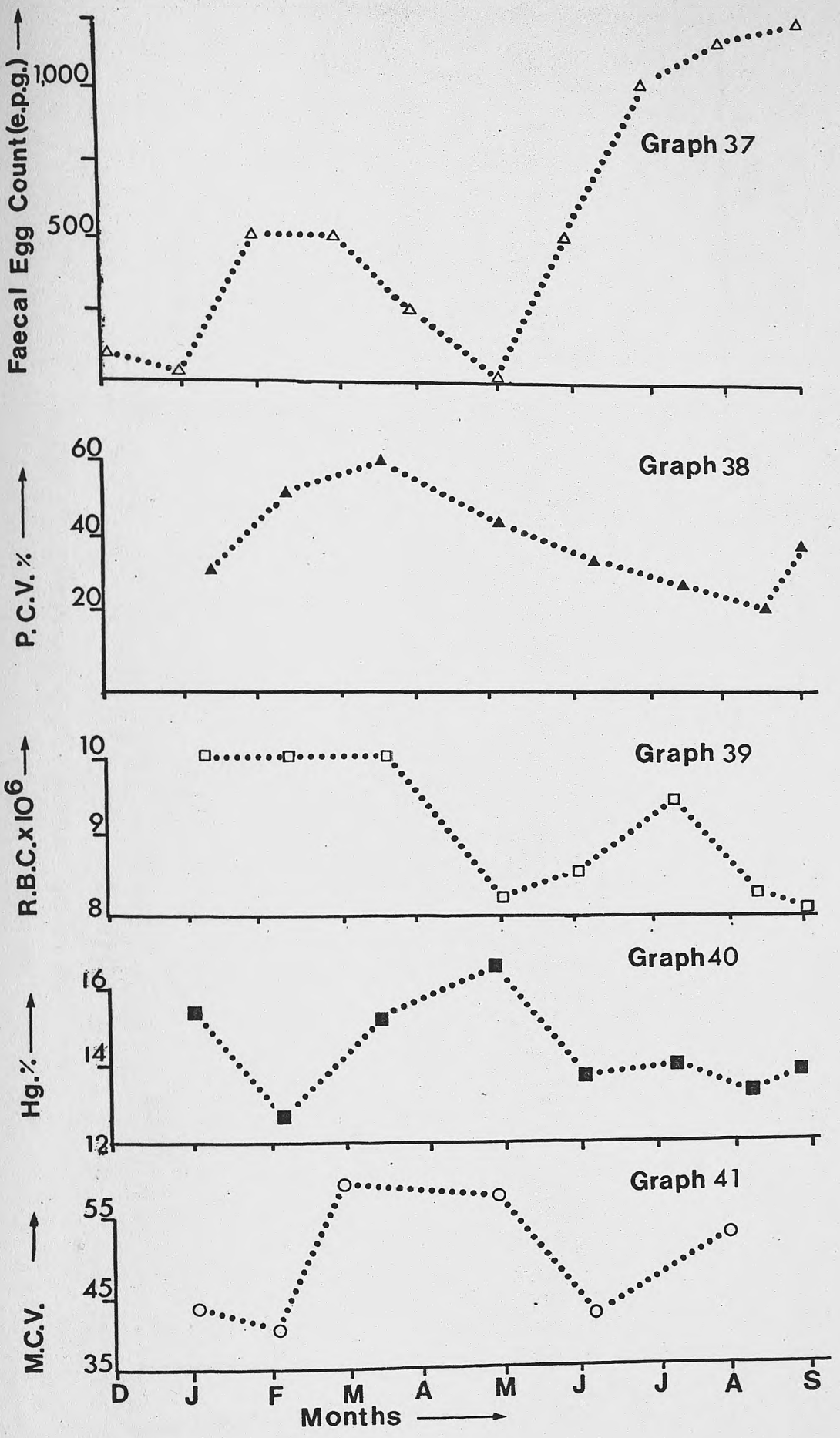
●---- eosinophils

x ——► establishment of patent strongyle infection



Graphs 37-41

Faecal egg counts and haematological results of horse no. 08 at Unit A during the study period.

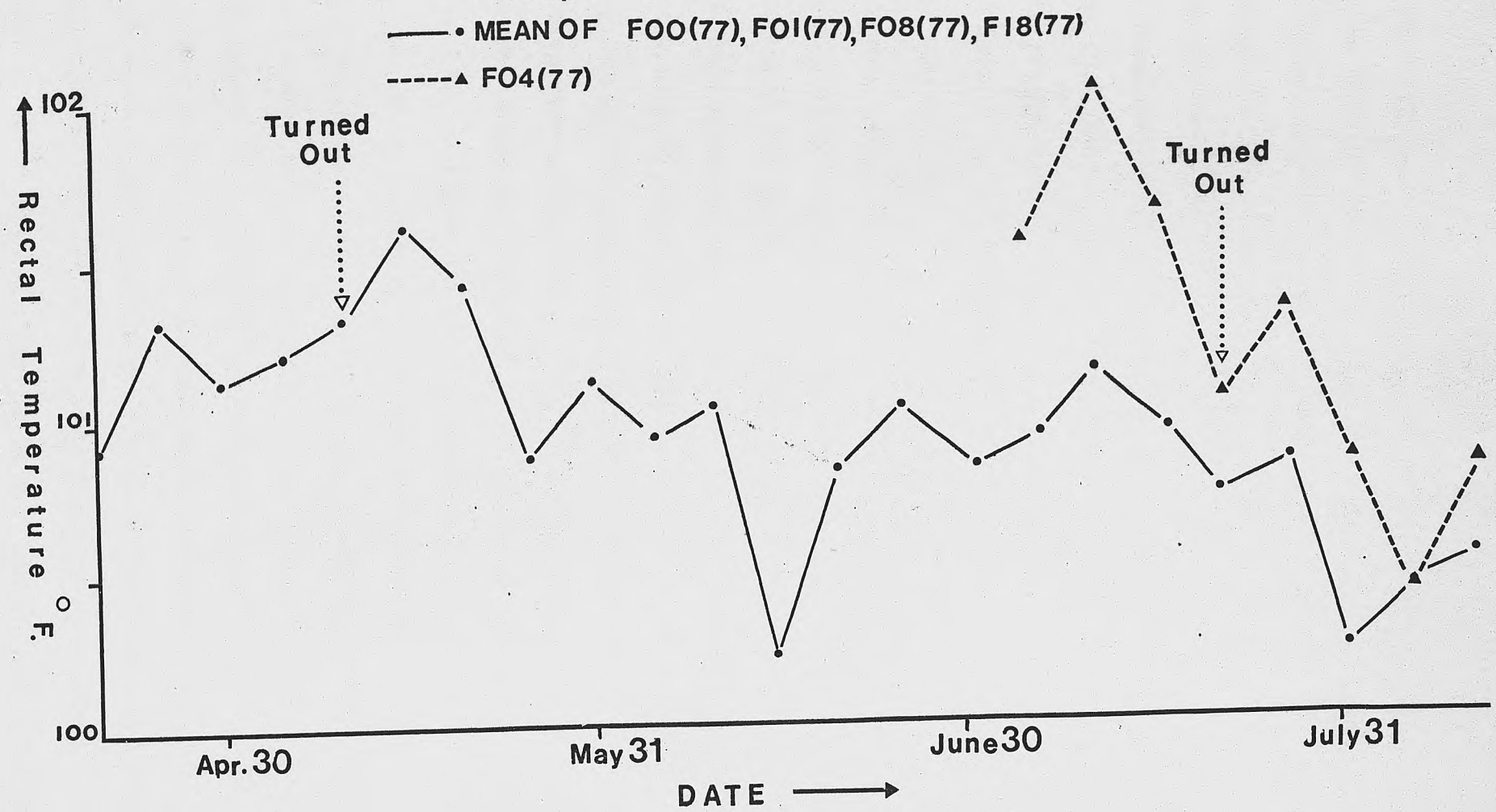

Faecal Egg Count(e.p.g.)
1,000
500
Graph 37
P.C.V.%
60
40
20
Graph 38
R.B.C.x10⁶
10
9
8
Graph 39
Hg.%
16
14
12
Graph 40
M.C.V.
55
45
35
Graph 41
D
J
F
M
A
M
J
J
A
S
Months

Graph Showing Rectal Temperatures

Of Foals Since Birth Against

Time

Graph 42


MEAN OF FOO(77), FOI(77), FO8(77), F18(77)
FO4(77)
Turned Out
Turned Out
102
101
100
Rectal Temperature °F.
Apr. 30
May 31
June 30
July 31
DATE

DISCUSSION

In assessing an epidemiological survey of strongyliasis in the horse one must ask if the helminths present in the studied animals present a potential hazard to these or other animals. This necessitates a diagnosis of subclinical infection, a knowledge of the bionomics of the free-living and parasitic stages of the helminths and a consideration of the host environment, past, present and future. This data must then be correlated to derive an epidemiological prognosis.

There is so little knowledge of the epidemiology of strongyliasis, that work badly needs to be conducted using animals free of strongyles, infecting them with single species of the strongylids, and then holding them on previously ungrazed pastures. Careful monitoring of the disease as it then develops should produce much needed data on these clinical entities. However the high cost of the horse as an experimental animal and the long prepatency of the strongylids has so far limited work of this nature.

For these reasons, throughout this study it was only possible to monitor naturally acquired mixed infections using locally owned horses. Two main problems beset this approach.

1. The difficulty of obtaining routine samples. The collection of faecal samples from horses at pasture requires either the individual stabling of animals for a period long enough for a faecal sample to be obtained, or routine collection of rectal samples. The former is time consuming, requires adequate nearby buildings, approachable animals and to some extent interferes with the natural grazing habits. The latter soon renders most animals unmanageable. Similarly routine blood collection requires both good lay assistants

and co-operative owners.

2. A lack of control over important epidemiological factors.

To obtain the co-operation needed in collecting the samples, information of levels of infection must be routinely fed back to the owner, and this produces a tendency to indulge in haphazard treatment with a variety of drugs, over which little control can be exercised. Animals also were placed on certain pastures for reasons other than control of strongyle infection so that the animals studied were meeting varying levels of pasture infection throughout the study period.

To reduce the severity of these problems two main lines of approach were taken,

(a) As many samples, taken as regularly as possible, were collected and although there are gaps in the series of results, which has largely precluded the use of statistical analysis, enough results were obtained to enable some conclusions to be drawn.

(b) Three equine units representing different management regimes were chosen thus giving a more balanced epidemiological picture.

Faecal Egg Counts

A great number of faecal egg counts were carried out and although the natural course of the infection in many animals was interrupted by anthelmintic treatment (Tables 4, 6 and 8), an overall trend can be seen. Graph 1 shows the average faecal egg output of horses at Unit B, which increased steadily from May onwards. Graphs 2 and 3 show individual levels of Cloudie and Greta respectively, at Unit B. Again faecal egg output increases from mid May onwards and then climbs rapidly to a peak in late July. Levels only fell after treatment with dichlorvos(1). Graphs 4, 5 and 6 show the faecal egg

output of three 2 year old horses at Unit B and again a distinct seasonal rise from May onwards can be seen. In the case of Story (Graph 4), treatment with dichlorvos in mid March (Table 6) will have removed any adult strongyles (Drudge and Lyons, 1969), but these will have rapidly been replaced from the inhibited trichoneme larvae in the mucosal wall (Ogbourne, 1975), whose presence was verified by subsequent larval culture (Table 14). It can be seen from graph 4 that faecal egg output then steadily increased to reach levels of over 1,000 (e.p.g.) by August. Pasture larval levels at this farm (graph 17) were sufficient in April to have infected animals and further increased the egg output by mid July, assuming previously established prepatent periods of the Trichonematidae (Russell, 1948; Poynter, 1958; Round, 1969b). This summer rise in faecal egg output is also shown at Unit C, (graph 7), where despite treatment with mebendazole(2) (Table 8), high levels of faecal egg output were reached in July and August by one horse, Moruisq.

Horse No. 10 at Unit A was retained inside until late August, yet faecal egg output increases steadily from March onwards (graph 8). Larval culture showed these eggs to be of Trichonema spp. (Table 10). Hence there was either an emergence of developing overwintered larvae from the mucosal wall (Ogbourne, 1975), or an increase in egg output of existing adults in the intestinal lumen, (Poynter, 1954), during this period.

In mid May, treatment with mebendazole will have removed existing adults (Drudge & Lyons, 1969). Within five weeks the faecal

(1) Frisk (Thomas Pettifer and Co. Ltd., Guildford, Surrey)

(2) Telmin (Crown Chemicals Ltd., Guildford, Surrey)

egg output was above that of pretreatment levels and, as this animal had no access to infective larvae and the shortest prepatency of Trichonema spp. is six weeks, (Round, 1969b), egg laying adults must have been derived from inhibited larvae in the mucosal wall, thus verifying the work of Ogbourne (1975). It would seem that large numbers of inhibited larvae of Trichonema spp. move into the lumen, develop to adults and quickly produce a faecal egg output in the spring to infect pastures for animals turned out at this time. Thus treatment with drugs effective only against adult small strongyles, prior to turning the animals out, will do little to reduce pasture contamination.

At Unit A, the one and two year old horses were turned out onto a pasture previously ungrazed by horses for four years, immediately after having been treated with fenbendazole(3). Subsequently no treatment was administered. Fenbendazole is effective against adult Trichonematidae (100%) and also against developing and inhibited larvae in the mucosal wall (93%), (Duncan, McBeath, Best & Preston, 1977). However by mid August all the animals were showing above 300 e.p.g. and larval culture showed these to be derived from trichonemes (graphs 11, 13 and 15, table 10). Since the prepatent periods of these species are from 9-14 weeks (Round, 1969b), and few or no infective larvae were present on the pasture at turning out in May (graph 17), the few remaining larvae in the mucosa must have been sufficient to cause infection with worms and a heavy pasture contamination by mid July (graph 17). Thus it can be seen that treatment with a single dose of fenbendazole is not sufficient to

(3)Panacur (Hoechst Chemicals, Hounslow, Middlesex)

prevent significant infections by mid August. This picture is also seen in the cases of the mares at Unit A (graphs 18, 20 and 22).

Thus it was found that despite a wide range of treatment regimes and placing horses on pastures of varying levels of contamination, an increase in faecal egg output, and the resultant pasture contamination invariably enabled the foals grazing these to become infected.

Faecal examination for the presence of strongyle eggs in the case of the foals at Unit A and B, produced results in agreement with previous work. Strongyle eggs were found in the faeces of the newly born foals F00/77 and F18/77 (graphs 29 and 32) but disappeared for a period, before recurring and then steadily increasing. This agrees with previous workers (Russell, 1948 and Poynter, 1958) who found that coprophagia occurs in the very young animal. The first appearance of eggs due to a patent infection occured at an average of nine weeks at both Units A and B (graphs 25-32). Since larval cultures of eggs passed by the mares yielded almost 100% Trichonema spp. (Tables 10 and 15), it is concluded that the prepatent period of the species of Trichonemes present on these farms was of the order of 9 weeks. This assumes infection was acquired shortly after birth. Previous work (Russell, 1948; Poynter, 1958, Table 2, and Round 1969b) gives various prepatent periods for Trichonema spp. However there are many species of this genus (Table 1) and this may well account for the variety of results, the actual length depending on the predominant species in an area. Round (1969b) gives periods in the order of 8-10 weeks which agrees well with that seen in this study. It is instructive to note that whereas at Unit A the pastures on which the newborn foals were placed had not been previously grazed

and the mares of these animals were all treated with fenbendazole prior to foaling, the foals at Unit B were placed on a contaminated pasture with untreated animals present in the field. Despite these profoundly different infective situations both groups showed patent infections at about the same time, thus showing that even low levels of pasture contamination with trichoneme larvae in May and June (graph 17) are sufficient to infect fully susceptible animals.

During the study period it was also possible to monitor the output of Strongyloides eggs during the early period of the foals life. These are clearly different from strongyle eggs, as they are larvated in fresh faeces and somewhat smaller. Numbers of eggs in a faecal sample can be very large. From the results (graph 24) it can be seen that egg output starts within 2 weeks of birth and is at its maximum at about 3 and 4 weeks after birth. Thereafter it rapidly declines to be negligible by 6 weeks of birth. This is presumably the result of the loss of adults of this species present in the intestinal lumen at this time.

Although this nematode is not a member of the family Strongylidea and thus not within the confines of the title of this dissertation, little work has been done on the epidemiology of Strongyloides infection in the horse and these results merit more detailed study.

The foals were housed during the early part of their lives and these boxes were thoroughly cleaned prior to occupation by the mare and foal and daily thereafter. Furthermore no Strongyloides eggs were found in the mares' faeces. Thus skin penetration was an unlikely source of infection. Hence infection was either trans-placental or transcolostral. Recent work by Drudge, Lyons &

Tolliver (1969) has suggested that the latter is the more likely route, but further work needs to be done on this aspect. Previous workers (Russell, 1948; Poynter, 1958) have suggested that the pathogenesis of this nematode is extremely low but in the cases of F01/77, F08/77 and F18/77, a severe diarrhoea occured at about 3 weeks of age, corresponding with peak Strongyloides egg output. Diarrhoea at this time has often been attributed to the presence of the foaling heat occuring in the mares at the same time, but to further elucidate this point F04/77 was treated with fenbendazole 10 days after birth. No diarrhoea occured in this animal during the following four weeks. This work is by no means conclusive but does emphasise the need for a more careful study of this type of infection.

In approaching this study it was decided to use a centrifugal flotation technique on faecal samples showing less than 200 eggs per gram by the modified McMaster egg counting technique. It was hoped that low numbers of strongyle eggs passed in the faeces would thus be detected, signifying the presence of a small but patent strongyle infection. However the results of the centrifugal flotation technique (Table 9) were not satisfactory and individual results differed widely from those obtained by the modified McMaster technique, and were often considerably less (Tables 9, 13 and 16). Although the technique did at times show up light infections, little extra information was obtained and the technique was time consuming. The accuracy of the technique may be improved by the use of Zinc Sulphate solution in place of a saturated salt solution, but as at present, in a study on naturally acquired infections this technique has little to offer.

Larval Culture

From the results of the larval cultures (Table 18), the overall prevalence of the different species and the numbers of the individual species in any one horse differ markedly from those described by previous workers (Russell, 1948; Poynter, 1954, 1958; Mathieson, 1969). These workers all showed a much higher prevalence of species other than Trichonema and lower proportions of Trichonema species in individual horses. Although they all agree that all horses carried Trichonema species the proportion of Trichonema eggs was only about 70%, much less than the 99.6% found in this survey. Indeed, in the previous work, S. vulgaris, S. edentatus and Triodontophorus spp. reached levels over 50% of total egg output in many animals (Russell, 1948 and Poynter, 1959). Two reasons could account for this: (a) the techniques employed in this study may have favoured the survival in culture and the identification of Trichonema spp. (b) recent environmental conditions may have favoured the survival of trichonemes as against other strongylids. To test the former hypothesis the culture recovery time was varied from 5 to 10 days, with no noticeable difference in results. The problem was also discussed with another worker in the field, whose recent observations suggested that such a differential favouring of the trichonemes was unlikely (Duncan, 1977, Personal Communication).

Thus it was thought that the environmental conditions on the pastures, under which the development and survival from the egg to the infective larval stage of these strongylids had occured, enabled Trichonema spp. to predominate. Since at the beginning of the study period, trichonemes were already present at very high levels (Table 18) the weather conditions prevailing in 1976 were examined

(graph 9a, 9b, 10). Temperatures were suitable for larval development of all species from March onwards (Ogbourne, 1972). Rainfall however, although adequate up to May, was at a very low level during June, July and August, while the numbers of hours of sunshine were very high. Although egg development and larval survival will occur in a faecal pat even in dry conditions, in exceptionally hot, dry conditions both egg and larval mortality can be very high (Ogbourne, 1972). It can be seen that just such conditions prevailed in the summer of 1976. Rainfall adequate for egg and larval development occured from September 1976 onwards, but by October the temperatures became too low for the free-living stages to develop (Ogbourne, 1973).

In the spring and early summer the larval stages of the strongyles invade the lumen of the large intestines to become adults and commence egg laying (Poynter, 1954; Ogbourne, 1972) and these adults are highly susceptible to anthelmintics (Drudge & Lyons, 1969). In the case of the large strongyles, this movement is well regulated and entirely seasonal (Duncan, 1973; Ogbourne, 1975a). Thus the susceptible egg laying females of the large strongyles would almost all have been removed by anthelmintic treatment in the spring and summer of 1976 and the eggs they had passed prior to such treatment would have failed to survive due to the unusual environmental conditions during that spring/summer period. Since the migration of developing larvae into the lumen to form adults would have ceased after the spring/summer period, no more adults would develop and hence no more eggs, would have been available to contaminate the pastures when conditions improved in September.

However in the case of the small strongyles, larvae remain inhibited in the mucosal wall and after anthelmintic treatment some

of these will mature and invade the lumen to become egg laying adults (Ogbourne, 1976) and this cycle will have been repeated throughout the spring and summer of 1976. Thus although their eggs deposited in the spring and summer will also have failed to survive, inhibited trichoneme larvae, maturing in September, would have become egg laying adults and provided pasture egg contamination at this time. Since most strongylid eggs and infective larvae are resistant to low temperatures (Ogbourne, 1973) this reinfection of the pastures during the autumn will have enabled the Trichonema spp. to have reinfected horses in the spring of 1977. Furthermore some of the Trichonema larvae will have survived through the winter as inhibited forms in the mucosal wall and then matured in the spring of 1977 to provide additional reinfection of the pastures at this time.

In this way it is possible to account for both the exceptionally high incidence of trichonematodiasis in 1977 and the increase, albeit in still small numbers, of other species in later larval cultures during 1977 (Tables 10, 14 and 17).

It is felt that the overall incidence of species other than trichonemes was found to be so low that their effect on the parameters measured in this study was negligible and the rest of this discussion should thus be limited to a consideration of the effects of an infection with these small strongyles.

Pasture Larval Levels

The number of infective larvae on a pasture obtained at any one time will depend on

(a) The numbers of eggs of that species contaminating the pasture

(b) The environmental conditions, primarily those of

temperature and rainfall, occuring at that time

(c) The acuracy of the technique employed for recovery of these larvae.

The results of pasture larval surveys at Units A and B are shown in graph 17. Since both pastures at Unit A were initially clean the results were negative in May, but a sudden rise in the numbers of infective larvae on the pasture occured in July and was sustained throughout August. At Unit B a small rise in pasture larval numbers occurred in April. This was probably due to the presence of infected horses contaminating the pasture in the previous autumn, when the larvae could develop to the infective stage, in which they remained in the pasture matt throughout the winter. The increase in temperature in spring (graph 10) then led to these larvae becoming more active and thus to their recovery in grass samples. A similar sharp rise in July and sustained throughout August, was seen at Unit B.

Providing adequate moisture is available development from the egg to the infective larva is temperature dependant and as the temperature rises so this development period shortens (Ogbourne, 1973). Thus eggs deposited in the spring will take much longer to reach the infective larval stage than those in early and mid summer. This will result in a large number of larvae reaching the infective stage at about the same time in summer. Furthermore the persistence thereafter of high temperatures resulting in minimum development times will produce a levelling out in numbers of infective larvae. Thus from graph 17, it can be seen that levels of infective larvae climb rapidly in July and then maintain this level throughout August. The sudden increase in rainfall in July (graph 9a) was probably also

significant as it will have improved conditions for larval development at this time. Although egg output was increasing at all three units in mid June (graphs 1, 2, 18 and 20) the rainfall was still low (graph 9) and this would tend to reduce the rate at which the larvae leave the faeces and pass on to the pasture. On the other hand the increased rainfall and higher temperatures in August will then have encouraged rapid development to infective larval stages.

Although studies on development temperatures and larval survival have been carried out on S. vulgaris and on mixed strongyle egg batches (Ogbourne, 1973), little has been done on the Trichonema spp. It is not therefore possible to give more details on pasture larval survival and egg development. However the faecal egg levels in May and early June, although still quite low, were adequate to infect the young susceptible horses going onto the pastures at this time. The resultant effect on these animals of high burdens in early July and August can be primarily seen on the haematological indices.

Haematological and Plasma Protein Studies

Clinical disease caused by strongyle infections can be due to the presence of adults in the lumen of the large intestines, to the effects of migrating larvae of the large strongyles (Duncan, 1973) or to those of developing and inhibited larvae of the small strongyles in the mucosal wall (Round, 1969a). The presence of adult strongyles can most easily be detected by faecal egg counts, supported by haematological examinations, but the presence of migrating or inhibited larvae can only be satisfactorily diagnosed in the living animal by haematological and biochemical examinations (Round, 1968a). Thus the blood from the horses at Unit A was routinely examined.

Unfortunately, the low number of animals studied, the lack of

data on United Kingdom normal variations, and the absence of a control group did not allow a statistical analysis of these results and therefore only major changes from normal values (Table 35) could be taken as significant.

In mare no. 18 (graph 22) the faecal egg count varied co-incidentaly with the levels of β globulin, eosinophils and neutrophils. The β globulin increase is related to the end of the prepatent period in strongyle infections (Round, 1969) and in the case of a small strongyle infection the β globulin reactions to the initial infection is varied in young animals, but occurs more commonly in subsequent infections (Round, 1969a). It can be seen that the peak levels of β globulins are reached before the peak levels of faecal egg counts, thus supporting the work of Round (1969a) and Duncan (1973). This effect is also seen in the cases of horses nos. 00 (graph 18), 08 (graph 20) and the three foals born in 1976, F10/76, F11/76 and F18/76, (graph 15). In the case of the two yearlings Y11 and Y01 a somewhat different result was obtained, there being no rise in May or June, corresponding to the increase in faecal egg output in July and August (graphs 11 and 13). However high β globulin levels occurred in December and January in both animals, probably due to large numbers of inhibited Trichonema in the mucosal wall of the large intestines at this time.

Graphs 33 to 36 show the effects of a first infection with a small strongyle species on the foals born in the study year. A more varied result is seen with an increase in β globulins occurring in F00/77 (graph 33) and F01/77 (graph 34) but not in F08/77 (graph 35) and F18/77 (graph 36), prior to the establishment of a patent strongyle infection. This agrees with the findings in initial

infections made by Round (1969a).

Eosinophilia has been recorded, caused by the tissue stages of strongyle infection in the horse, (Round, 1968a; Duncan, 1973) and the results obtained in this study closely agree with these results. Horse nos. 18 (graph 23) 00 (graph 19) and 08 (graph 21) showed double peaks of eosinophil numbers apparently associated with an increase in faecal egg numbers, caused by the movement of inhibited larvae into the lumen and becoming egg laying adults (Ogbourne, 1975). Both Y01 and Y11 had high eosinophil levels in December and January (graphs 12 and 14 respectively) and into February in the case of Y11, corresponding to the β globulin increases at this time and again probably due to large numbers of inhibited trichoneme larvae in the mucosal wall. Rises in eosinophil numbers in these two animals are also seen in July and August with the establishment of a patent infection at this time.

Similar results were obtained with the three foals born in 1976 (graph 16), their mean eosinophil count showing this double peak. In the cases of foals born in 1977 (graphs 33 to 36), none of the animals showed an eosinophilia prior to the establishment of a patent infection, but an increase in eosinophil numbers occurred in all four during August, probably related to the increased numbers of infective larvae on the pastures at this time (graph 17) and hence an increased intake and penetration of the mucosal wall by these infective larvae, rather than the establishment of a patent infection. The phenomenon may be in part also responsible for the eosinophilia seen in the other animals.

Duncan (1973), Round (1968a) and Archer & Poynter (1957) described an increase in white blood cell numbers and a neutrophilia

associated with the tissue stages of the horse strongyles. They also described a decrease in red blood cell numbers and packed cell volume whilst haemoglobin values and mean cell corpuscular volume remains normal in patent strongyle infections in the horse. Although these parameters were measured throughout the study period the results were inconclusive (graphs 37 to 41). Because of the small numbers involved, the lack of control groups and large individual variations no real trends could be observed. Further on two occasions the Coulter Counter was not available (14th July and 11th August 1977) and manual techniques were employed, producing yet more inconsistency. In a naturally acquired infection the level of intake of infective larvae fluctuates in contrast to an experimentally induced infection, where large numbers of infective larvae may be given at one time (Duncan, 1973). This will produce observable changes in these parameters, whereas in naturally acquired infections other factors operate to obscure these changes. Thus it would seem that changes in eosinophil numbers and β globulin levels are the most useful haematological indicators of a small strongyle infection in the horse.

Rectal Temperature Changes

Pyrexia has been observed in young horses as infective larvae of S. vulgaris penetrate the intestinal wall (Ooms, Oyart, Mylle, Vanden, Hende & Decraemere, 1976; Duncan, 1973) and also on re-entry prior to the establishment of a patent infection (Ooms et al., 1976). In an attempt to show a similar effect with a small strongyle infection, the rectal temperatures of the foals born at Unit A in 1977 were periodically recorded (Table 12). The result of these recordings (graph 42) do not indicate any significant increase in rectal temperature at any time. This may be due to either no such

change occurring as a result of the presence of infective or prepatent trichineme larvae or the numbers of such larvae present at any one time not being sufficient to elicit the response.

It is an important clinical factor to determine if a pyrexia does occur at these times as a temperature increase is not normally associated with a helminth infection and pyrexia may serve to confuse the differential diagnosis. It would appear from this short study that this problem does not occur in trichonemiasis per se.

Pathogenesis

When assessing the total pathogenic effect of a strongyle infection, one must take into account the effect caused by migrating and developing larvae and that caused by an adult infection in the lumen of the caecum and colon.

The resultant effect of a patent strongyle infection may primarily be observed as a loss of condition with no other clinical manifestations. Throughout the study period the horses at Unit B were consistently reported by their owners to have improved in condition after treatment with an anthelmintic, but no other effect of an adult infection was observed.

However in the case of an infection with trichonemes it has recently been observed that large numbers of inhibited and developing larvae in the mucosal wall may give rise to intermittent diarrhoea, (Chiejina & Mason, 1977). During this study Y01 and Y11 both manifested this condition during the winter of 1976/77. A progressive loss of condition, with intermittent diarrhoea and a degree of anorexia occurred in the case of Y01 during November 1976. The faecal egg count in these yearlings was very low at this time (graph 11). Larval culture from the faeces of the older horses grazing on

the same pastures showed only the presence of eggs of Trichonema spp. Haematological and plasma protein analysis on December 9th (Table 25) showed elevated eosinophil, basophil, neutrophil and β globulin levels and low lymphocyte and albumin levels. It was considered that this was suggestive of severe prepatent strongyliasis (Round, 1969; Duncan, 1973). Y01 was treated with 50 mg./kg. fenbendazole on the 20th December and the response to treatment was dramatic. Within a week the diarrhoea ceased, the appetite improved and the animal quickly gained weight. The results of successive leucocyte and plasma protein analysis showed a progressive return to normality (Table 25), although this was slower than the clinical recovery. These results have previously been reported and discussed by Jeggo & Sewell (1977).

Similar results were obtained with Y11, although the clinical manifestations of diarrhoea, loss of condition and anorexia were less noticeable (Table 26, graphs 13 and 14).

It was hoped that other pathogenic effects would be observed from the post mortems carried out during the study period. Unfortunately suitable post mortem material was rarely available and all that could be obtained from the post mortem results (Table 36) was that numbers of inhibited trichoneme larva were observed in the mucosa of the caecum and colon and that there was little evidence of either adults or migrating larvae of the large strongyle spp. during the winter months, supporting therefore the conclusions of the larval culture studies.

CONCLUSIONS

1. Conditions prevailing in the study area allow the development of strongyliasis in the local horses.

2. Infections were primarily of Trichonema spp. during the study period due to environmental conditions prevailing in the previous year.

3. Measurement of subclinical and clinical infection of Trichonema spp. is best carried out by faecal egg counts, haematological and biochemical analysis, primarily of peripheral eosinophils and β globulin levels.

4. There was a clear cut seasonal variation in numbers of adults and inhibited larvae of Trichonema spp. with large numbers of inhibited larvae occurring in the winter and movement of these into the lumen of the caecum and colon to produce a patent infection in the spring.

5. A consequent seasonal variation in faecal egg output and pasture contamination with infective trichoneme larvae occurred, with high levels of both being reached by July and August.

6. This phenomenon allows for rapid infection of susceptible animals on the same pasture at the same time.

7. Even low levels of pasture contamination are sufficient to establish an infection by Trichonema spp.

8. Turning out animals onto clean pastures does little to reduce the establishment of a patent infection by Trichonema spp. because of their pre-existing burden of inhibited larvae.

9. It is unlikely that pyrexia occurs as a result of infection by Trichonema spp.

10. Control of infection by Trichonema spp. should be by

(a) Monitoring both faecal egg output and blood values

(b) Treatment of inhibited larvae in winter with drugs effective against these

(c) Strategic treatment in the summer based on the routine laboratory findings.

11. Strongyloides westeri occurs in very young animals and may be responsible for the diarrhoea seen at this time.

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Easter Bush, Scotland

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APPENDIX

Tables of All Individual Results

From Routinely Collected Samples

TABLE 9

Faecal Egg Counts of Unit A

DATE	25/11/76		3/12/76		10/12/76		17/12/76	
HORSE NO.	Mc	Salt	Mc	Salt	Mc	Salt	Mc	Salt
F10	0	0	0	0	0	0	100	0
F11	0	0	0	0	100	1	0	1
F18	0	0	0	0	100	0	100	2
Y01	100	0	0	0	0	0	0	0
Y11	0	0	0	0	0	0	0	0
00	0	2	0	0	0	0	150	0
01	200	6	0	0	0	0	200	2
04	100	0	100	10	250	23	0	0
08	0	0	0	1	0	0	100	1
10	0	0	0	0	0	0	0	0
18	0	0	0	0	0	0	100	0

DATE	24/12/76		31/12/76		7/1/77		14/1/77	
F10	0	0	0	8	0	1	0	1
F11	0	2	0	0	50	2	0	2
F18	100	1	200	0	0	0	0	0
Y01	0	0	0	0	100	0	50	1
Y11	0	0	100	22	100	0	0	1
00	0	0	100	1	50	10	0	0
01	100	7	200	28	0	2	0	0
04	0	2	0	0	0	6	0	0
08	200	5	250	13	0	2	0	0
10	0	0	0	0	0	0	0	0
18	100	15	0	4	0	9	0	0

DATE	21/1/77		28/1/77		4/2/77		11/2/77	
F10	0	0	0	0	0	0	0	1
F11	0	0	0	0	0	0	0	0
F18	0	9	50	21	0	0	50	12
Y01	0	0	0	0	0	0	0	1
Y11	0	0	0	0	0	0	0	0
00	350	22	350	120	250	0	150	40
01	50	7	250	135	350	176	350	218
04	0	0	150	6	150	13	50	13
08	150	92	100	50	650	-	400	-
10	300	3	0	12	0	4	50	24
18	1,100	217	900	275	700	-	500	-
	Mc	Salt	Mc	Salt	Mc	Salt	Mc	Salt

Mc - McMaster Technique (eggs per gram)
Salt - Salt Flotation Technique (eggs per gram)

Faecal Egg Counts of Unit A (contd.)

DATE	18/2/77		25/2/77		1/3/77		11/3/77	
HORSE NO.	Mc	Salt	Mc	Salt	Mc	Salt	Mc	Salt
F10	0	5	50	0	50	0	0	0
F11	50	4	0	0	0	1	0	0
F18	0	9	0	1	50	7	100	23
Y01	0	0	0	2	0	0	0	0
Y11	0	0	0	0	0	3	0	0
00	0	14	500	-	350	-	50	18
01	1,000	-	1,400	-	1,800	-	1,500	-
04	300	-	250	15	350	-	350	-
08	500	-	500	-	100	10	350	-
10	0	0	200	28	50	45	200	-
18	900	-	1,500	-	1,000	-	1,300	-

DATE	18/3/77		24/3/77		25/3/77		1/4/77	
F10	0	13			50	8	0	2
F11	50	0			0	5	0	5
F18	50	10			0	5	0	4
Y01	0	0			0	0	0	0
Y11	0	0			0	0	0	0
00	250	19	150	45	150	48	100	66
01	3,300	-	1,100	-	1,050	-	850	-
04	100	59			350	-	0	88
08	400	48	1,100	-	400	-	600	-
10	250	53			650	-	250	-
18	250	8			900	-	0	12

DATE	8/4/77		14/4/77		22/4/77		29/4/77	
F10	100	6	0	6	150	0	50	2
F11	0	1	0	2	0	0	100	6
F18	0	1	50	0	0	9	50	7
Y01	0	0	0	0	200	0	0	0
Y11	0	0	0	9	0	0	0	0
00	300	-	0	2	0	0	0	0
01	1,400	-	0	9	0	8	50	48
04	200	63	0	0	250	-	200	4
08	0	9	0	0	50	6	100	14
10	450	-	800	-	750	-	800	-
18	450	-	450	-	400	-	250	-
	Mc	Salt	Mc	Salt	Mc	Salt	Mc	Salt

Faecal Egg Counts of Unit A (contd.)

DATE	2/5/77		11/5/77		17/5/77		20/5/77	
HORSE NO.	Mc	Salt	Mc	Salt	Mc	Salt	Mc	Salt
F10	50	5			50	5		
F11	0	3			50	7		
F18	50	7			0	7		
Y01					50	0		
Y11					0	0		
00	0	0	50	1	0	0		
01	200	24	200	10	150	10		
04	50	22	150	11			50	8
08	0	26	100	16	150	0		
10	700	-	700	-			500	-
18	150	85	300	-	250	0		
F18/77	100	5	100	6			0	0
F01/77			0	0				
F00/77	0	0						
F08/77	0	0					0	0

DATE	26/5/77		2/6/77		8/6/77		14/6/77	
F10			100	5				
F11			150	12				
F18			0	1				
Y01			0	6	50	31		
Y11			50	5				
00	0	0	0	0	0	2	0	0
01	100	38			0	60	300	-
04	150	23	0	0	150	20	150	-
08	50	9	750	-	300	-	250	-
10	700	-	1,500	-	1,100	-	100	23
18	860	-	250	-	500	-		
F18/77	0	0			0	0	0	0
F01/77	0	0	0	3	0	0	0	0
F00/77			350	0	0	0	0	0
F08/77	0	0			0	0	0	0
ALBERT	2,500	-						
	Mc	Salt	Mc	Salt	Mc	Salt	Mc	Salt

Faecal Egg Counts of Unit A (contd.)

DATE	27/6/77		7/7/77		14/7/77	
HORSE NO.	Mc	Salt	Mc	Salt	Mc	Salt
F10	50	17				
F11	0	1				
F18	450	-				
Y01	150	51				
Y11	50	1				
00	50	9	50	9	300	-
01	300	-			100	94
04	100	56			200	42
08	800	-			1,150	-
10	1,450	-			1,700	-
18	350	-	-	-	300	-
F18/77	100	1	50	0	0	5
F01/77	0	2	50	4	0	0
F00/77	100	10	200	4	0	6
F08/77	100	2	-	-	100	2
ALBERT	1,250	-	5,600			

DATE	21/7/77		31/7/77		7/8/77	
F10						
F11					200	15
F18						
Y01						
Y11						
00	200	18	300	-	150	16
01	1,000	-	250	-		
04	50	11	-	32	-	17
08	1,000	-	1,200	-	1,250	-
10	1,000		450	-	400	-
18	700	-	1,800	-	750	-
F18/77	50	3			0	0
F01/77	0	0	50	3		
F00/77	0	6	0	0		
F08/77	350	-	50	5	0	0
F04/77	0	0	0	1		
F10/77	0	0			0	0
	Mc	Salt	Mc	Salt	Mc	Salt

Faecal Egg Counts of Unit A (contd.)

DATE	12/8/77		18/8/77		23/8/77	
HORSE NO.	Mc	Salt	Mc	Salt	Mc	Salt
F10					700	-
F11					500	-
F18					250	-
Y01					500	-
Y11					800	-
00	860	-	50	13		
01			600	-		
04	250	-	100	15		
08	1,100	-	1,350	-		
10	700	-	950			
18	860	-	600	-		
F18/77	50	2	50	1		
F01/77	0	1	50	0		
F00/77	250	-	50	3		
F08/77			50	4		
F04/77	0	0				
F10/77	0	0				
ALBERT			2,550	-		

DATE	26/8/77		31/8/77	
00	200	-	200	
01	450	-	700	
04	0		200	
08	1,900	-	1,650	
10	300		700	-
18			400	
F18/77	800		100	
F01/77	50		100	
F00/77	50	10	50	
F08/77	0	10	50	
F04/77			0	0
F10/77			0	0
	Mc	Salt	Mc	Salt

TABLE 10 (contd.)

Date	Horse No.	Infective Larvae spp. Distribution (%)
4/7/77	08	97% Trichonema spp. 3% Triodontophorus spp.
	ALBERT	75% Trichonema spp. 25% Triodontophorus spp.
19/7/77	ALBERT	95% Trichonema spp. 4% Triodontophorus spp. 1% S. vulgaris
21/7/77	01	100% Trichonema spp.
	08	99% Trichonema spp. 1% Triodontophorus spp.
24/7/77	10	100% Trichonema spp.
	04	"
31/7/77	10	98% Trichonema spp. 2% S. vulgaris
	08	100% Trichonema spp.
	18	"
8/7/77	10	"
	08	"
	18	"
18/8/77	10	"
	18	"
	ALBERT	97% Trichonema spp. 3% S. vulgaris

TABLE 11

LARVAL PASTURE COUNTS OF UNIT A

DATE 1977	FIELD OCCUPANTS	NO. OF INFECTIVE LARVAE COUNTED	WT. OF GRASS DRIED	NO. OF LARVAE PER Kg. DRIED GRASS
23/4	Vacant field to be occupied by mares and foals	none	80g.	none
16/5	Vacant. To be yearlings' field.	none	375g.	none
24/5	Mares and foals	2	50g.	40
19/6	Yearlings and 2 year olds	none	240g.	none
6/7	Mares and foals	1	525g.	2
7/7	Yearlings and 2 year olds	4	800g.	5
2/8	Yearlings and 2 year olds	143	275g.	519
5/8	Mares and foals	491	245g.	2004
17/8	Yearlings and 2 year olds	656	387g.	1695
18/8	Mares and foals	190	95g.	2000
31/8	Mares and foals	378	118g.	3200
31/8	Yearlings and 2 year olds	314	230g.	1365

TABLE 12

RECTAL TEMPERATURES OF FOALS BORN IN 1977 AT UNIT A

(TEMPERATURES RECORDED IN °F)

Horse Identification	F00(77)	F01(77)	F04(77)	F08(77)	F18(77)
Date					
16/4/77					101.6
18/4/77					100.3
21/4/77					101.2
23/4/77					101.2
24/4/77					101.6
27/4/77		101.6			101.0
29/4/77		101.4			101.2
30/4/77	100.2	101.3		101.4	101.4
2/5/77	101.8	101.6		100.8	100.6
4/5/77	100.8	101.4		101.8	101.0
6/5/77	101.6	101.4		101.7	101.0
8/5/77	101.0	100.8		101.0	101.4
10/5/77					101.2
11/5/77					101.4
12/5/77	101.4				102.0
13/5/77	102.0	101.4		101.6	101.2
15/5/77	102.5				101.2
17/5/77	101.0	101.2		101.6	102.1
20/5/77	100.6	101.6		101.0	101.0
23/5/77	100.0	102.0		101.0	100.0
24/5/77	101.0	101.0		101.2	100.8
27/5/77				101.3	100.8
31/5/77	100.2	100.1		101.1	100.1
1/6/77	100.8	102.0		101.5	101.0
2/6/77	100.6	100.6		100.4	101.2
3/6/77	100.6	101.8		101.0	101.0
6/6/77		101.0			99.2
7/6/77	100.9	101.2		102.0	101.6
8/6/77	101.5	101.6		101.6	101.0

TABLE 12 (contd.)

Horse Identification	F00(77)	F01(77)	F04(77)	F08(77)	F18(77)
Date					
9/6/77	101.6	101.9		101.0	101.0
10/6/77	100.8	101.0		100.2	99.8
12/6/77	100.8	100.2		98.0	100.6
14/6/77	100.0	100.4		100.8	100.0
22/6/77	101.4	100.8		100.8	100.8
29/6/77	100.8	101.0		100.8	100.6
5/7/77	101.3	100.6	101.5	101.2	100.3
6/7/77	101.0	101.0	101.8	100.6	100.4
8/7/77	100.1	100.8	102.1	100.8	100.2
11/7/77	101.0	101.0	101.8	101.0	101.0
12/7/77	100.2	100.6	100.4	101.4	100.0
14/7/77	101.5	100.6	101.8	101.4	100.6
20/7/77	100.6	100.2	101.0	100.0	100.8
21/7/77	101.0	101.0	102.0	100.8	100.2
22/7/77	100.8	101.0	101.2	100.8	100.8
25/7/77	100.6	100.4	100.8	101.0	100.0
26/7/77	101.2	98.6	101.2	100.0	100.0
27/7/77	100.0	98.0	100.2	100.4	98.0
28/7/77	100.0	100.4	100.2	100.2	100.0
29/7/77	101.0	101.4	101.4	101.0	100.6
2/8/77	100.0	100.4	100.4	100.2	100.0
3/8/77	100.2	100.4	100.8	100.0	100.4
5/8/77	100.8	100.6		100.4	100.2
9/8/77	100.4	100.6	100.8	100.6	100.2

TABLE 13

FAECAL EGG COUNT RESULTS OF UNIT B

DATE	1/2/77		11/2/77		25/2/77		13/3/77	
Horse Name	Mc	Salt	Mc	Salt	Mc	Salt	Mc	Salt
Lollipop			100	3	50	16	0	2
Blue	0	3	50	4	100	4	50	1
Cressie	700	-	400	-	50	39	50	7
Festival	100	2	150	6	50	5	550	-
Cadger	250	-	50	21	50	15	50	28
Mais Belle	1,900		250	-	700	-	0	0
Greta	150	29	150	66	150	38	100	4
Velvet	150	80	700	-	650		0	1
Prince	1,750	-	1,650	-	50	6	50	9
Nimbus	1,600	-	1,500	-			150	2
Val	850	-	400	-	300	-	150	2
Towpath	150	30	500	-	300	-	0	1
Story	400	-	850	-	600	-	0	0
Cloudie	1,000	-	50	6	50	7	0	0

DATE	27/3/77		14/4/77		16/4/77		1/5/77	
Lollipop	0	12			100	1		
Blue	1,350	-			50	1		
Cressie	200	16			150	6		
Festival	850	-			0	1		
Cadger								
Mais Belle								
Greta	0	14	50	2	50	4		
Velvet	100	1	300	-	300	-	700	-
Prince	0	10			950	-		
Nimbus	100	2	200	58			2,300	
Val	100	2					200	35
Towpath	0	0	0	3	200	4	150	4
Story			100	6	200	18	350	-
Cloudie							0	1
Cloudie's Foal								

FAECAL EGG COUNT RESULTS OF UNIT B (contd.)

DATE	15/5/77		29/5/77		12/6/77		27/6/77	
Horse Name	Mc	Salt	Mc	Salt	Mc	Salt	Mc	Salt
Cadger	[illegible]				200	-	400	
Mais Belle							1,500	-
Greta	650	-	600	-	1,700	-	2,600	
Velvet	1,600	-	1,700	-			1,550	
Prince	750	-			0	0		
Nimbus	1,050	-			1,100	-	900	-
Val			500	-			1,500	
Towpath	150	2	50	6	100	6	50	
Story	150	32			200	-	400	-
Cloudie	0	0			1,000		1,450	-
Cloudie's Foal	0	0	0	6	100	10	300	-
Val's Foal			50	10				
Greta's Foal			0	0	0	0	0	0
Towpath's Foal							0	0

DATE	8/7/77		24/7/77		7/8/77		22/8/77	
Blue	450	-						
Cressie	850	-						
Festival			3,550	-				
Cadger	0	2						
Mais Belle	2,600	-	10,000		0	0	0	
Greta	0	0	0	9	150	3	350	
Velvet	1,900	-	50	1	0	0	0	
Prince			550	-			10,700	
Nimbus			2,400	-			1,750	
Val	50		0	2	0	25	550	
Towpath	0	2	50	5	100	1	0	
Story	650	-	900	-	1,350	-	1,350	
Cloudie	0	4	50	-	50	6	0	
Cloudie's Foal	0	0	0	0	0	0	0	
Val's Foal	0	2	-	3	150	-	250	
Greta's Foal	0	0	0	0	100	4	400	
Towpath's Foal			0	0	100	6	100	-

TABLE 14

LARVAL CULTURE FROM FAECAL SAMPLES SHOWING OVER 400 e.p.g. BY McMASTER TECHNIQUE FROM UNIT B

Date	Horse Name	Infective Larvae spp. Distribution (%)
1/2/77	Prince	88% Trichonema spp. 5.3% S. vulgaris 11.4% Trichostrongylus axei
4/3/77	Mais Belle	100% Trichonema spp.
	Velvet	"
	Cloudie	"
11/3/77	Festival	"
27/3/77	Festival	"
	Blue	"
15/4/77	Velvet	"
20/4/77	Prince	87% Trichonema spp. 12% Triodontophorus spp. 1% S. edentatus
	Velvet	100% Trichonema spp.
	Strike	70% Trichonema spp. 20% Triodontophorus spp. 10% S. vulgaris
1/5/77	Strike	100% Trichonema spp.
	Nimbus	"
	Festival	"
22/6/77	Cloudie	"
	Greta	"
	Nimbus	"
4/7/77	Greta	"
	Mais Belle	"
	Val	"
	Cloudie	70% Trichonema spp. 30% Triodontophorus spp.
	Velvet	98% Trichonema spp. 1% S. vulgaris 1% Triodontophorus spp.
24/7/77	Mais Belle	100% Trichonema spp.

TABLE 14 (contd.)

Date	Horse Name	Infective Larvae spp. Distribution (%)
24/7/77	Festival	99% Trichonema spp. 1% S. edentatus
	Prince	68% Trichonema spp. 32% Triodontophorus spp.
25/7/77	Nimbus	100% Trichonema spp.
8/8/77	Story	80% Trichonema spp. 19% Triodontophorus spp. 1% S. vulgaris
17/7/77	Strike	100% Trichonema spp.
	Velvet	"
	Greta	98% Trichonema spp. 2% T. axei
	Mais Belle	99% Trichonema spp. 1% T. axei
	Prince	87% Trichonema spp. 6% Triodontophorus spp. 4% S. vulgaris 3% T. axei
23/8/77	Nimbus	100% Trichonema spp.
	Prince	"
	Val	"
	Greta's Foal	40% Strongyloides westeri 60% Trichonema spp.
	Story	98% Trichonema spp. 2% S. vulgaris

TABLE 15

LARVAL PASTURE COUNTS OF UNIT B

DATE 1977	FIELD OCCUPANTS	NO. OF INFECTIVE LARVAE COUNTED	WT. OF GRASS DRIED	NO. OF LARVAE PER Kg. DRIED GRASS
28/2	1-4 yr. olds	3	267g.	11
28/2	Mares	1	118.5g.	9
28/2	Yearlings	66	77g.	857
4/4	Mares	14	25g.	560
29/4	Mares	6	137g.	44
9/7	Mares and foals	4	800g.	5
7/8	Mares and foals	1,262	265g.	4,760
16/8	Mares and foals	2,900	596g.	4,860
1/9	Mare and foals	468	105g.	4,450

TABLE 16

FAECAL EGG COUNT RESULTS OF UNIT C

DATE	13/1/77		26/1/77		9/2/77		11/2/77	
HORSE NAME	Mc	Salt	Mc	Salt	Mc	Salt	Mc	Salt
Dusky	500	-						
Moruisq	500	-	600	-			2,100	-
Rhum	450	-			900	-		

DATE	4/3/77		14/3/77		26/4/77		16/5/77	
HORSE NAME	Mc	Salt	Mc	Salt	Mc	Salt	Mc	Salt
Dusky	0	4	100	2	1,900	-		
Moruisq	0	0	50	14	2,650	-	200	13
Rhum			0	6	2,500	-		

DATE	26/7/77		30/8/77	
HORSE NAME	Mc	Salt	Mc	Salt
Dusky	1,450	-		
Moruisq	5,800	-	250	-
Rhum				

TABLE 17

LARVAL CULTURE OF FAECAL SAMPLES SHOWING OVER

400 e.p.g. BY McMASTER TECHNIQUE from UNIT C.

Date	Horse Name	Infective Larvae Species Distribution (%)
30/4/77	Dusky	100% Trichonema spp.
	Ruhm	"
	Moruisq	"
25/7/77	Moruisq	95% Trichonema
	"	5% S. edentatus
	Dusky	100% Trichonema
13/1/77	Moruisq	"
	Dusky	"
	Ruhm	"

TABLE 18(b)

LARVAL PASTURE COUNT RESULTS from UNIT C

DATE	FIELD OCCUPANTS	NO. OF INFECTIVE LARVA COUNTED	WT. of GRASS DRIED (g.)	NO. OF LARVA PER kg. DRIED GRASS
10/4	Moruisq	17	540	32
10/4	Dusky Ruhm	36	460	78

TABLE 19

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. 00

	1976	1977			
Date	24/12	14/1	11/2	18/3	22/4
Parameter					
P.C.V. (%)		39.2	47	41.1	42.4
R.B.C. ($x10^6$/cmm.)		9.36	8.69	7.96	8.7
M.C.V. (fl.)		43	40.5	52	45.2
Hb (g./dl.)		14.5	14.3	17.4	15.4
W.B.C. ($x10^3$/cmm.)					
Total		7.6	8.1	9.7	8.5
Lymphocytes (%)		3.9 (51)	4.5 (55.5)	4.5 (46)	4.3 (50)
Neutrophils (%)		3.0 (40)	3.1 (38½)	4.9 (50)	3.6 (42)
Eosinophils (%)		0.46 (6)	0.57 (7)	0.19 (2)	0.17 (2)
Basophils (%)		0.76 (1)	- (-)	0.97 (1)	0.85(1)
Monocytes (%)		1.5 (2)	- (-)	0.97 (1)	0.43 (5)
Serum Protein (g./l.)					
Total		5.7	5.7	5.7	x
Albumin (%)	38	2.5 (43)	1.9 (34)	2.3 (41)	x
α globulin (%)	15	0.8 (14)	1.2 (21)	1.0 (18)	x
β globulin (%)	32	1.6 (28)	1.7 (30)	1.1 (20)	x
γ globulin (%)	15	0.9 (15)	0.9 (15)	1.2 (21)	x

x Blood sample not available

TABLE 19 (contd.)

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. 00

	1977				
Date	2/5	8/6	14/7	11/8	30/8
Parameter					
P.C.V. (%)	51.8	36.5	35	27	40
R.B.C. ($x10^6$/cmm.)	0.36	9.18	10.45	7.73	7.81
M.C.V. (fl.)	50	40	33	35	51
Hb (g./dl.)	18.7	18.2	14.5	18.6	14.9
W.B.C. ($x10^3$/cmm.)					
Total	13.0	11.0	8.0	8.1	10.7
Lymphocytes (%)	3.3(25)	3.0(27)	2.0(24½)	2(26)	2.6(24½)
Neutrophils (%)	9.2(71)	7.5(68)	5.7(71)	5.6(68)	7.7(72)
Eosinophils (%)	0.13(1)	0.22(2)	0.2(2½)	0.4(5)	0.32(3)
Basophils (%)	- (-)	(-)	- (-)	- (-)	0.16(1½)
Monocytes (%)	0.39(3)	0.33(3)	0.18(2)	0.04(½)	0.59(5½)
Serum Protein (g./l.)					
Total	5.7	5.7	5.7	5.7	5.6
Albumin (%)	3.4(59)	3.0(53)	(46)	3.2(56)	2.8(50)
α globulin (%)	0.8(14)	0.97(17)	1.0(18)	0.9(15)	0.95(17)
β globulin (%)	0.91(16)	0.8(14)	1.1(20)	1.0(18)	1.03(18)
γ globulin (%)	0.63(11)	0.91(16)	0.91(16)	0.63(11)	0.86(15)

TABLE 20

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. 01

	1977				
Date	14/1	11/2	18/3	22/4	2/5
Parameter					
P.C.V. (%)	36.4	48	54	57	44
R.B.C. ($x10^6$/cmm.)	8.3	8.6	9.14	9.23	9.0
M.C.V. (fl.)	44	41	50	70	49
Hb (g./dl.)	13.3	13.7	17.0	13.7	15.2
W.B.C. ($x10^3$/cmm.)					
Total	9.74	8.3	8.5	11.9	10.44
Lymphocytes (%)	x	x	4.3(50)	4.6(39)	3.3(32)
Neutrophils (%)	x	x	4.0(47)	6.6(55)	6.1(58½)
Eosinophils (%)	x	x	0.04(½)	0.36(3)	0.52(5)
Basophils (%)	x	x	- (-)	0.2(1)	0.1(1)
Monocytes (%)	x	x	0.17(2)	0.16(2)	0.42(4)
Serum Protein (g./l.)					
Total	5.89	4.9	5.4	5.7	4.42
Albumin (%)	2.5(42)	2.3(46)	2.7(50)	2.6(45)	2.3(53)
α globulin (%)	0.9(16)	0.93(19)	0.86(16)	1.1(20)	0.75(17)
β globulin (%)	1.2(20)	0.93(19)	1.0(19)	1.1(20)	0.88(20)
γ globulin (%)	1.3(22)	0.78(16)	0.81(15)	0.86(15)	0.66(10)

TABLE 20 (contd.)

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. 01

	1977			
Date	8/6	14/7	11/8	30/8
Parameter				
P.C.V. (%)	41	33	27	39
R.B.C. ($x10^6$/cmm.)	10.5	11.1	8.8	6.8
M.C.V. (fl.)	39			5.6
Hb (g./dl.)	16.6	14.7	15.9	14.3
W.B.C. ($x10^3$/cmm.)				
Total	9.5	6.75	8.53	10.0
Lymphocytes (%)	3.9(41)	2.3(34½)	2.1(25)	1.9(19)
Neutrophils (%)	5.2(55)	4.1(60)	5.8(68)	7.5(75)
Eosinophils (%)	0.2(2)	0.3(4½)	0.3(3½)	4.5(4½)
Basophils (%)	0.1(1)	- (-)	0.09(1)	- (-)
Monocytes (%)	0.24(2½)	0.14(2)	0.21(2½)	0.15(1½)
Serum Protein (g./l.)				
Total	5.84	5.42	5.84	5.84
Albumin (%)	3.0(51)	2.8(52)	2.8(48)	2.8(48)
α globulin (%)	0.82(14)	0.81(15)	0.99(17)	0.88(15)
β globulin (%)	1.2(20)	1.1(20)	1.2(20)	0.19(17)
γ globulin (%)	0.88(15)	0.7(13)	0.88(15)	1.2(20)

TABLE 21

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. 04

	1977		
Date	14/7	11/8	30/8
Parameter			
P.C.V. (%)	28	26	32
R.B.C. ($x10^6$/cmm.)	9.5	8.12	8.3
M.C.V. (fl.)	29	32	40
Hb (g./dl.)	12.4	12.6	15.4
W.B.C. ($x10^3$/cmm.)			
Total	10.5	11.4	11.1
Lymphocytes (%)	2.3(22)	1.6(14)	1.5(13½)
Neutrophils (%)	7.6(72)	8.6(75)	8.3(75)
Eosinophils (%)	0.42(4)	1.0(9)	1.2(11)
Basophils (%)	0.11(1)	- (-)	- (-)
Monocytes (%)	0.11(1)	0.23(2)	0.05(½)
Serum Protein (g./l.)			
Total	5.42	6.42	6.4
Albumin (%)	3.0(56)	3.7(58)	2.4(38)
α globulin (%)	0.43(8)	1.1(17)	0.96(15)
β globulin (%)	0.65(12)	1.3(20)	1.09(17)
γ globulin (%)	1.3(24)	0.37(5)	1.92(30)

TABLE 22

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. 08

	1976	1977			
Date	24/12	14/1	11/2	18/3	22/4
Parameter					
P.C.V. (%)	x	32	52	60	x
R.B.C. ($x10^6$/cmm.)	x	5.2	10.1	10.1	x
M.C.V. (fl.)	x	44	36.3	59	x
Hb (g./dl.)	x	15.5	12.3	15.1	x
W.B.C. ($x10^3$/cmm.)					
Total	x	7.34	9.76	7.0	x
Lymphocytes (%)	x	2.8(38)	3.7(38)	2.73(39)	x
Neutrophils (%)	x	3.2(44)	4.5(46)	3.2(46)	x
Eosinophils (%)	x	0.44(6)	0.78(8)	0.28(4)	x
Basophils (%)	x	0.59(8)	0.4(4)	0.21(3)	x
Monocytes (%)	x	0.3(4)	0.4(4)	0.28(4)	x
Serum Protein (g./l.)					
Total	x	5.7	5.4	5.5	5.42
Albumin (%)	35	2.17(38)	1.9(36)	2.0(36)	2.2(40)
α globulin (%)	12	0.97(17)	0.92(17)	0.83(15)	0.81(15)
β globulin (%)	33	1.6(28)	1.6(29)	1.5(28)	1.4(25)
γ globulin (%)	15	0.97(17)	0.97(18)	1.2(21)	1.1(20)

TABLE 22 (contd.)

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. 08

	1977				
Date	2/5	8/6	14/7	11/8	30/8
Parameter					
P.C.V. (%)	44.1	34.6	30	23	35
R.B.C. ($x10^6$/cmm.)	8.14	8.6	9.42	8.29	6.5
M.C.V. (fl.)	54	4.0	32	27	53
Hb (g./dl.)	16.5	13.8	13.9	13.4	13.6
W.B.C. ($x10^3$/cmm.)					
Total	14.0	9.5	5.2	9.58	9.3
Lymphocytes (%)	4.2(30)	3.61(38½)	1.2(24)	2.6(27)	1.95(21)
Neutrophils (%)	8.2(58½)	4.9(51½)	3.4(65)	6.0(63)	6.9(74½)
Eosinophils (%)	0.56(4)	0.8(8½)	0.39(7½)	0.53(5½)	0.3(3)
Basophils (%)	0.28(2)	0.05(½)	0.05(1)	0.19(2)	- (-)
Monocytes (%)	0.77(5½)	0.1(1)	0.13(2½)	0.24(2½)	0.15(1½)
Serum Protein (g./l.)					
Total	6.98	5.7	6.7	5.56	6.4
Albumin (%)	2.5(36)	2.7(48)	3.6(53)	3.0(53)	3.07(48)
α globulin (%)	1.0(15)	0.91(16)	1.1(16)	1.0(18)	1.28(20)
β globulin (%)	1.8(25)	1.1(20)	1.3(19)	1.1(20)	0.89(14)
γ globulin (%)	1.7(24)	0.91(16)	0.8(12)	0.46(9)	0.15(18)

TABLE 23

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. 10

	1976	1977		
Date	21/12	14/7	11/8	30/8
Parameter				
P.C.V. (%)	x	30	30	38
R.B.C. ($x10^6$/cmm.)	x	11.12	8.7	7.14
M.C.V. (fl.)	x	15.7	16.1	14.3
Hb (g./dl.)	x	27	34	52
W.B.C. ($x10^3$/cmm.)				
Total	x	6.3	5.25	7.8
Lymphocytes (%)	x	2.6(41)	1.8(33½)	1.13(14½)
Neutrophils (%)	x	3.6(56½)	2.8(53½)	6.0(77)
Eosinophils (%)	x	0.09(1½)	0.47(9)	0.55(7)
Basophils (%)	x	- (-)	0.05(1)	- (-)
Monocytes (%)	x	0.03(½)	0.15(3)	0.12(1½)
Serum Protein (g./l.)				
Total	x	5.7	4.99	6
Albumin (%)	46	3.3(58)	2.8(57)	3(50)
α globulin (%)	14	0.57(10)	0.6(12)	0.84(14)
β globulin (%)	26	0.86(15)	0.75(15)	0.72(12)
γ globulin (%)	14	0.97(17)	0.8(16)	1.44(24)

TABLE 24

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. 18

	1976	1977			
Date	24/12	14/1	11/2	18/3	22/4
Parameter					
P.C.V. (%)		43	44	45.9	52
R.B.C. ($x10^6$/cmm.)		10.9	10.11	9.64	9.79
M.C.V. (fl.)		50.2	49.7	48	51
Hb (g./dl.)		17.1	18.7	19.9	19.2
W.B.C. ($x10^3$/cmm.)					
Total		11.2	10.7	8.86	12.0
Lymphocytes (%)		4.6(41)	4.1(38)	3.1(35½)	4.7(39)
Neutrophils (%)		5.6(50)	5.7(53)	5.1(57)	6.24(52)
Eosinophils (%)		0.112(1)	0.112(1)	0.13(1½)	0.42(3½)
Basophils (%)		- (-)	0.3(3)	0.18(2)	0.12(1)
Monocytes (%)		0.9(8)	0.5(5)	0.27(3)	0.54(4½)
Serum Protein (g./l.)					
Total	6	6.01	5.7	5.4	5.84
Albumin (%)	2.8(40)	2.6(43)	x	2.8(52)	3.0(51)
α globulin (%)	0.84(14)	1.0(17)	x	0.76(14)	0.93(16)
β globulin (%)	1.6(26)	1.7(28)	x	1.4(25)	0.92(15)
γ globulin (%)	0.84(14)	0.8(14)	x	0.49(9)	1.1(18)

TABLE 24 (contd.)

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. 18

	1977				
Date	2/5	8/6	14/7	11/8	30/8
Parameter					
P.C.V. (%)	60.4	53.1	26	26	44.5
R.B.C. ($x10^6$/cmm.)	2.11	10.4	8.4	10.67	8.24
M.C.V. (fl.)	48	42	31	24	53
Hb (g./dl.)	16.0	16.2	13.1	15.9	13.2
W.B.C. ($x10^3$/cmm.)					
Total	12.75	9.7	8.4	9.96	9.05
Lymphocytes (%)	5.5(43½)	3.9(40)	3.2(38)	2.24(22½)	2.26(25)
Neutrophils (%)	6.5(51)	5.1(53)	4.8(57)	6.6(66)	6.15(68)
Eosinophils (%)	0.13(1)	0.1(1)	0.13(1½)	0.5(5)	0.63(7)
Basophils (%)	0.13(1)	0.3(3)	0.13(1½)	0.05(½)	- (-)
Monocytes (%)	0.38(3½)	0.3(3)	0.25(3)	0.6(6)	- (-)
Serum Protein (g./l.)					
Total	5.99	6.27	5.7	5.7	6.3
Albumin (%)	3.1(51)	2.8(45)	3.0(53)	3.42(60)	2.7(43)
α globulin (%)	0.83(14)	0.94(15)	0.97(17)	0.8(14)	1.1(17)
β globulin (%)	0.95(16)	1.6(26)	0.8(14)	0.74(13)	1.5(24)
γ globulin (%)	1.0(17)	0.91(14)	0.91(16)	0.74(13)	1.0(16)

TABLE 25

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. Y01

	1976	1977		
Date	9/12	11/2	18/3	22/4
Parameter				
P.C.V. (%)	35.5	35.8	28.7	43
R.B.C. ($x10^6$/cmm.)	9.96	8.48	6.54	10.09
M.C.V. (fl.)	40	43	44	43
Hb (g./dl.)	13.4	14.3	12.2	11.6
W.B.C. ($x10^3$/cmm.)				
Total	13.6	11.9	11.1	11.3
Lymphocytes (%)	3.4(25)	6.6(55)	5.1(46)	5.3(47)
Neutrophils (%)	6.8(52)	4.3(36)	5.4(49)	5.3(47)
Eosinophils (%)	1.2(9)	0.6(5)	0.2(1½)	0.2(1½)
Basophils (%)	1.1(8)	0.1(1)	0.2(1½)	0.2(1½)
Monocytes (%)	0.5(4)	0.3(3)	0.2(2)	0.3(3)
Serum Protein (g./l.)				
Total	6.5	6.0	5.9	5.6
Albumin (%)	1.9(30)	1.8(30)	3.0(50)	3.1(55)
α globulin (%)	1.3(20)	1.0(17)	0.8(14)	0.9(17)
β globulin (%)	2.7(41)	2.6(43)	1.5(25)	1.2(22)
γ globulin (%)	0.7(10)	0.6(10)	0.6(11)	0.3(6)

TABLE 25 (contd.)

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. Y01

	1977	
Date	12/7	28/8
Parameter		
P.C.V. (%)	40	38
R.B.C. ($x10^6$/cmm.)	8.0	9.84
M.C.V. (fl.)	50	40
Hb (g./dl.)	13.5	18.8
W.B.C. ($x10^3$/cmm.)		
Total	7.45	12.8
Lymphocytes (%)	3.2(43)	3.2(25)
Neutrophils (%)	3.6(48)	8.2(64)
Eosinophils (%)	0.34(4½)	1.1(8½)
Basophils (%)	0.3(4)	- (-)
Monocytes (%)	0.03(½)	0.32(2½)
Serum Protein (g./l.)		
Total	4.56	6.7
Albumin (%)	2.1(45)	3.0(44)
α globulin (%)	0.87(19)	1.14(17)
β globulin (%)	1.2(26)	1.7(26)
γ globulin (%)	0.46(10)	0.87(13)

TABLE 26

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. Y11

	1976	1977		
Date	17/12	14/1	11/2	18/3
Parameter				
P.C.V. (%)	36.8	37.6	30.6	38.8
R.B.C. ($x10^6$/cmm.)	9.99	10.02	8.31	8.96
M.C.V. (fl.)	38	38	38	43
Hb (g./dl.)	13.5	12.3	11.9	10.06
W.B.C. ($x10^3$/cmm.)				
Total	11.3	13.1	12.4	9.1
Lymphocytes (%)	3.3(29)	x	5.4(43½)	4.5(49)
Neutrophils (%)	6.8(60)	x	5.8(46½)	2.9(32)
Eosinophils (%)	0.4(3½)	x	1.5(12)	0.82(9)
Basophils (%)	0.62(5½)	x	0.62(5)	0.18(2)
Monocytes (%)	0.11(1)	x	0.5(4)	0.64(7)
Serum Protein (g./l.)				
Total	5.7	5.7	5.8	5.8
Albumin (%)	2.3(40)	2.1(37)	2.1(36)	2.2(38)
α globulin (%)	1.1(20)	1.2(21)	0.52(9)	1.28(22)
β globulin (%)	1.3(22)	1.3(22)	2.0(35)	1.4(24)
γ globulin (%)	1.0(18)	1.0(20)	0.58(10)	0.93(16)

TABLE 26 (contd.)

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. Y11

	1977		
Date	22/4	12/7	24/8
Parameter			
P.C.V. (%)	37.7	26	38
R.B.C. ($x10^6$/cmm.)	9.18	10.13	9.24
M.C.V. (fl.)	41	26	44
Hb (g./dl.)	12.1	14.3	17.5
W.B.C. ($x10^3$/cmm.)			
Total	8.95	9.55	14.6
Lymphocytes (%)	3.9(43½)	4.2(44)	3.3(22½)
Neutrophils (%)	4.1(46)	4.7(49)	9.1(62)
Eosinophils (%)	0.18(2)	0.14(1½)	1.9(13)
Basophils (%)	0.13(1½)	0.24(2½)	- (-)
Monocytes (%)	0.18(2)	0.29(3)	0.03(2½)
Serum Protein (g./l.)			
Total	6.27	5.26	6.4
Albumin (%)	2.6(42)	3.4(64)	3.1(48)
α globulin (%)	1.1(18)	0.89(16)	1.3(20)
β globulin (%)	1.6(26)	0.79(15)	1.3(20)
γ globulin (%)	0.89(14)	0.26(5)	0.7(12)

TABLE 27

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. F10/76

	1976	1977		
Date	17/12	14/1	11/2	18/3
Parameter				
P.C.V. (%)	40	35	73	36.3
R.B.C. ($x10^6$/cmm.)	8.44	10.17	10.22	8.63
M.C.V. (fl.)	34	42.5	73	42
Hb (g./dl.)	14.1	16.4	11.0	13.5
W.B.C. ($x10^3$/cmm.)				
Total	16.2	14.8	12.1	12.1
Lymphocytes (%)	6.6(41)	x	7.0(57½)	5.6(46½)
Neutrophils (%)	9.1(56)	x	4.1(34)	5.9(48½)
Eosinophils (%)	- (-)	x	- (-)	0.3(2½)
Basophils (%)	- (-)	x	- (-)	- (-)
Monocytes (%)	0.49(3)	x	1.0(8½)	0.3(2½)
Serum Protein (g./l.)				
Total	5.56	5.3	5.3	5.5
Albumin (%)	3.3(60)	2.5(48)	2.1(40)	x
α globulin (%)	1.1(20)	1.2(22)	1.3(24)	x
β globulin (%)	1.1(20)	1.0(19)	1.3(25)	x
γ globulin (%)	- (-)	0.58(11)	0.5(10)	x

TABLE 27 (contd.)

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. F10/76

	1977	
Date	22/4	28/8
Parameter		
P.C.V. (%)	40.5	36.5
R.B.C. ($x10^6$/cmm.)	9.57	9.36
M.C.V. (fl.)	4.2	53
Hb (g./dl.)	12.1	17.5
W.B.C. ($x10^3$/cmm.)		
Total	11.5	16.5
Lymphocytes (%)	5.4(47)	5.3(32)
Neutrophils (%)	5.7(50)	9.6(58)
Eosinophils (%)	0.23(2)	0.52(8)
Basophils (%)	- (-)	- (-)
Monocytes (%)	0.11(1)	0.5(3)
Serum Protein (g./l.)		
Total	5.13	6.3
Albumin (%)	2.7(52)	3.02(48)
α globulin (%)	1.1(21)	1.25(20)
β globulin (%)	0.97(19)	1.13(18)
γ globulin (%)	0.41(8)	0.88(14)

TABLE 28

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. F11/76

	1976	1977		
Date	17/12	14/1	11/2	18/3
Parameter				
P.C.V. (%)	42	33.6	30.8	31
R.B.C. ($x10^6$/cmm.)	6.23	10.59	8.69	8.49
M.C.V. (fl.)	34	32	36	36
Hb (g./dl.)	13.4	11.1	12.9	11.5
W.B.C. ($x10^3$/cmm.)				
Total	12.7	6.87	9.81	10.1
Lymphocytes (%)	6.6(52)	x	5.8(59½)	5.6(55½)
Neutrophils (%)	5.6(44)	x	3.8(39)	4.1(40½)
Eosinophils (%)	0.19(1½)	x	0.29(3)	0.1(1)
Basophils (%)	- (-)	x	- (-)	- (-)
Monocytes (%)	0.32(2½)	x	0.39(4)	0.3(3)
Serum Protein (g./l.)				
Total	6.042	5.7	5.7	5.56
Albumin (%)	3.1(52)	3.1(54)	2.2(38)	3.2(58)
α globulin (%)	1.6(26)	1.4(25)	1.3(23)	0.89(16)
β globulin (%)	1.1(18)	1.0(18)	1.5(26)	0.78(14)
γ globulin (%)	0.24(4)	0.29(5)	0.74(13)	0.67(12)

TABLE 28 (contd.)

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. F11/76

	1977		
Date	22/4	12/7	28/8
Parameter			
P.C.V. (%)	35.3	30.0	35
R.B.C. ($x10^6$/cmm.)	9.07	11.2	10.6
M.C.V. (fl.)	39	-	41
Hb (g./dl.)	12.2	12.7	18.6
W.B.C. ($x10^3$/cmm.)			
Total	10.4	15.9	16.6
Lymphocytes (%)	5.3(51)	8.1(51)	4.2(25½)
Neutrophils (%)	4.9(47)	6.4(40½)	9.4(56½)
Eosinophils (%)	0.1(1)	0.95(6)	2.6(15½)
Basophils (%)	- (-)	- (-)	- (-)
Monocytes (%)	0.1(1)	0.4(2½)	0.42(2½)
Serum Protein (g./l.)			
Total	5.13	5.7	6.4
Albumin (%)	2.3(44)	2.8(49)	3.4(53)
α globulin (%)	1.3(25)	1.1(21)	1.22(19)
β globulin (%)	1.1(22)	1.1(20)	0.77(12)
γ globulin (%)	0.46(9)	0.57(10)	1.02(16)

TABLE 29

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. F18/76

	1976	1977		
Date	17/12	14/1	11/2	18/3
Parameter				
P.C.V. (%)	35	34	34	43.9
R.B.C. ($x10^6$/cmm.)	9.89	10.96	9.34	10.8
M.C.V. (fl.)	33	40.2	35	41
Hb (g./dl.)	13.0	14.2	15.6	11.7
W.B.C. ($x10^3$/cmm.)				
Total	10.4	11.3	12.0	10.04
Lymphocytes (%)	7.8(75)	6.1(54)	6.5(54)	5.6(55½)
Neutrophils (%)	2.4(23)	5.2(46)	5.5(46)	4.0(40)
Eosinophils (%)	0.1(1)	1.1(10)	1.2(10)	0.15(1½)
Basophils (%)	0.1(1)	0.79(7)	0.84(7)	0.05(½)
Monocytes (%)	- (-)	- (-)	- (-)	0.2(2)
Serum Protein (g./l.)				
Total	5.13	6.01	6.4	5.26
Albumin (%)	2.6(50)	2.8(46)	2.1(32)	2.7(52)
α globulin (%)	0.97(19)	1.7(28)	1.2(18)	1.1(20)
β globulin (%)	1.3(25)	0.9(15)	2.2(35)	0.74(14)
γ globulin (%)	0.3(6)	0.66(11)	0.96(15)	0.74(14)

TABLE 30

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. F00/77

	1977				
Date	2/5	8/6	14/7	11/8	30/8
Parameter					
P.C.V. (%)	43	44.7	36	36	40
R.B.C. ($x10^6$/cmm.)	13.0	10.4	7.08	12.2	10.89
M.C.V. (fl.)	53	36	51	30	37
Hb (g./dl.)	19.3	17.2	15.8	18.1	16.1
W.B.C. ($x10^3$/cmm.)					
Total	7.78	10.33	10.5	10.2	10.3
Lymphocytes (%)	1.8(22½)	3.6(34½)	3.15(30)	4.5(44)	3.9(38)
Neutrophils (%)	5.8(74)	6.5(62½)	6.5(62)	5.0(49½)	5.6(54)
Eosinophils (%)	- (-)	0.05(½)	0.42(4)	0.26(2½)	0.2(2)
Basophils (%)	0.39(5)	0.26(2½)		0.05(½)	0.3(3)
Monocytes (%)	0.23(3)	- (-)	0.42(4)	0.36(3½)	0.3(3)
Serum Protein (g./l.)					
Total	5.7	5.27	5.42	4.85	5.7
Albumin (%)	3.7(65)	2.7(52)	3.3(60)	2.7(53)	2.7(48)
α globulin (%)	1.0(18)	1.2(22)	0.98(18)	0.87(18)	1.4(25)
β globulin (%)	0.51(9)	0.84(16)	0.65(12)	1.1(22)	1.1(19)
γ globulin (%)	0.46(8)	0.53(10)	0.54(10)	0.34(7)	0.8(14)

TABLE 31

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. F01/77

	1977				
Date	2/5	8/6	14/7	11/8	30/8
Parameter					
P.C.V. (%)	55.8	44.7	31	33	23.6
R.B.C. ($x10^6$/cmm.)	8.82	10.4	13.07	10.7	5.78
M.C.V. (fl.)	44	36	24	30.8	40
Hb (g./dl.)	17.7	17.2	14.7	14.0	11.7
W.B.C. ($x10^3$/cmm.)					
Total	11.2	10.33	11.6	15.8	6.96
Lymphocytes (%)	2.8(25)	3.7(35½)	5.3(46)	4.8(30½)	1.6(23)
Neutrophils (%)	8.0(71½)	7.0(68)	6.03(52)	10.1(64)	5.1(72½)
Eosinophils (%)	0.006(½)	0.05(½)	- (-)	0.4(2½)	0.03(½)
Basophils (%)	0.11(1)	- (-)	0.17(1½)	0.16(1)	- (-)
Monocytes (%)	0.22(2)	0.15(1½)	0.11(1)	0.32(2)	0.38(5½)
Serum Protein (g./l.)					
Total	5.84	5.13	5.13	5.84	5.9
Albumin (%)	3.2(55)	2.5(49)	4.1(80)	3.1(53)	2.8(47)
α globulin (%)	0.93(16)	0.92(18)	0.46(9)	1.1(19)	1.4(24)
β globulin (%)	0.99(17)	1.1(21)	0.56(11)	1.1(19)	1.1(19)
γ globulin (%)	0.7(12)	0.62(12)	- (-)	0.52(9)	0.59(10)

TABLE 32

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. F04/77

	1977			
Date	20/6	14/7	11/8	30/8
Parameter				
P.C.V. (%)	x	27	27	41
R.B.C. ($x10^6$/cmm.)	x	9.39	9.45	10.2
M.C.V. (fl.)	x	28	29	40
Hb (g./dl.)	x	12.9	17.3	15.2
W.B.C. ($x10^3$/cmm.)				
Total	x	17.5	13.45	13.47
Lymphocytes (%)	x	3.9	4.3(32)	0.67(25½)
Neutrophils (%)	x	13.1	8.7(64½)	4.7(72)
Eosinophils (%)	x	-	- (-)	0.67(½)
Basophils (%)	x	0.35	- (-)	- (-)
Monocytes (%)	x	0.088	0.47(3½)	0.27(2)
Serum Protein (g./l.)				
Total	4.70	4.99	5.42	5.3
Albumin (%)	2.6(55)	2.74	2.4(45)	3.02(57)
α globulin (%)	0.94(20)	0.9	1.5(27)	0.9(17)
β globulin (%)	0.89(19)	1.0	1.08(20)	0.95(18)
γ globulin (%)	0.28(6)	0.25	0.43(8)	0.42(8)

TABLE 33

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. F08/77

	1977				
Date	2/5	8/6	14/7	11/8	30/8
Parameter					
P.C.V. (%)	41.8	35.6	28	30	47.2
R.B.C. ($x10^6$/cmm.)	9.97	10.9	11.96	10.8	10.96
M.C.V. (fl.)	42	29	23.4	28	43
Hb (g./dl.)	15	17.8	14.7	14.4	16.0
W.B.C. ($x10^3$/cmm.)					
Total	10.4	10.65	10.5	17.4	13.9
Lymphocytes (%)	2.7(26)	3.7(34½)	4.1(39)	2.1(12)	4.5(32½)
Neutrophils (%)	7.54(72½)	6.6(62½)	6.0(57)	14.8(85)	9.1(65½)
Eosinophils (%)	- (-)	0.54(½)	- (-)	- (-)	0.07(½)
Basophils (%)	0.052(½)	0.27(2½)	0.37(3½)	- (-)	0.07(½)
Monocytes (%)	0.104(1)	- (-)	0.53(½)	0.52(3)	0.14(1)
Serum Protein (g./l.)					
Total	5.56	4.85	5.26	5.7	5.84
Albumin (%)	2.9(52)	2.4(50)	2.7(52)	3.0(52)	2.8(48)
α globulin (%)	0.89(16)	0.87(18)	0.94(18)	1.1(20)	1.17(20)
β globulin (%)	0.83(15)	0.82(17)	0.89(17)	0.97(15)	0.82(14)
γ globulin (%)	0.94(17)	0.73(15)	0.68(13)	0.74(13)	1.05(18)

TABLE 34

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. F10/77

	1977	
Date	11/8	30/8
Parameter		
P.C.V. (%)	29	42
R.B.C. ($x10^6$/cmm.)	9.0	10.54
M.C.V. (fl.)	32	41
Hb (g./dl.)	17	17.6
W.B.C. ($x10^3$/cmm.)		
Total	11.3	11.2
Lymphocytes (%)	1.8(16)	2.0
Neutrophils (%)	9.3 (82)	8.7
Eosinophils (%)	- (-)	0.11
Basophils (%)	- (-)	-
Monocytes (%)	0.226(2)	0.33
Serum Protein (g./l.)		
Total	4.85	5.84
Albumin (%)	2.6	3.2
α globulin (%)	0.73	1.17
β globulin (%)	0.49	1.17
γ globulin (%)	1.07	0.35

TABLE 35

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. F18/77

	1977			
Date	22/4	2/5	8/6	14/7
Parameter				
P.C.V. (%)	45.4	41.1	33.9	40
R.B.C. ($x10^6$/cmm.)	9.37	10.9	10.94	12.73
M.C.V. (fl.)	48	38	28	
Hb (g./dl.)	15.4	13.7	17.6	16.1
W.B.C. ($x10^3$/cmm.)				
Total	11.12	11.04	9.07	10.7
Lymphocytes (%)	4(36)	4.6(42)	3.9(43½)	2.3(21)
Neutrophils (%)	6.7(60)	6.6(60)	4.8(53)	7.6(71)
Eosinophils (%)	0.5(4½)	0.11(1)	0.41(½)	0.21(2)
Basophils (%)	0.33(3)	- (-)	- (-)	0.42(4)
Monocytes (%)	0.27(2½)	0.11(1)	0.32(3½)	0.42(2)
Serum Protein (g./l.)				
Total	5.13	5.84	4.85	5.27
Albumin (%)	3.5(69)	3.4(59)	2.5(52)	2.8(53)
α globulin (%)	0.76(14)	0.64(11)	1.1(22)	1.1(20)
β globulin (%)	0.56(11)	0.82(14)	0.68(14)	0.63(12)
γ globulin (%)	0.31(6)	0.93(16)	0.58(12)	0.79(15)

TABLE 35 (contd.)

HAEMATOLOGICAL RESULTS UNIT A

HORSE NO. F18/77

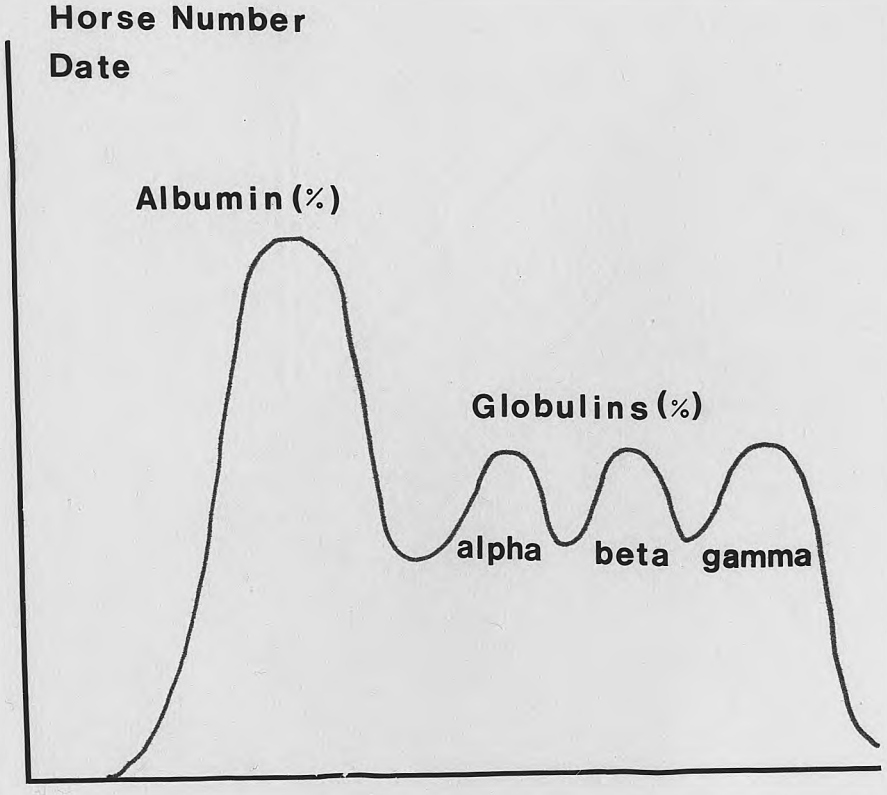
	1977	
Date	11/8	30/8
Parameter		
P.C.V. (%)	33	45
R.B.C. ($x10^6$/cmm.)	15.3	9.84
M.C.V. (fl.)		37
Hb (g./dl.)	17.8	15.6
W.B.C. ($x10^3$/cmm.)		
Total	11.4	17.29
Lymphocytes (%)	5.3(46½)	7.3(42½)
Neutrophils (%)	5.13(45)	9.2(53½)
Eosinophils (%)	0.34(3)	0.52(3)
Basophils (%)	0.052(½)	- (-)
Monocytes (%)	0.51(4½)	0.26(1½)
Serum Protein (g./l.)		
Total	4.85	5.6
Albumin (%)	2.7(55)	2.7(49)
α globulin (%)	0.87(18)	1.12(20)
β globulin (%)	0.97(20)	1.29(23)
γ globulin (%)	0.34(7)	0.45(8)

TABLE 35

TABLE SHOWING NORMAL HAEMATOLOGICAL VALUES

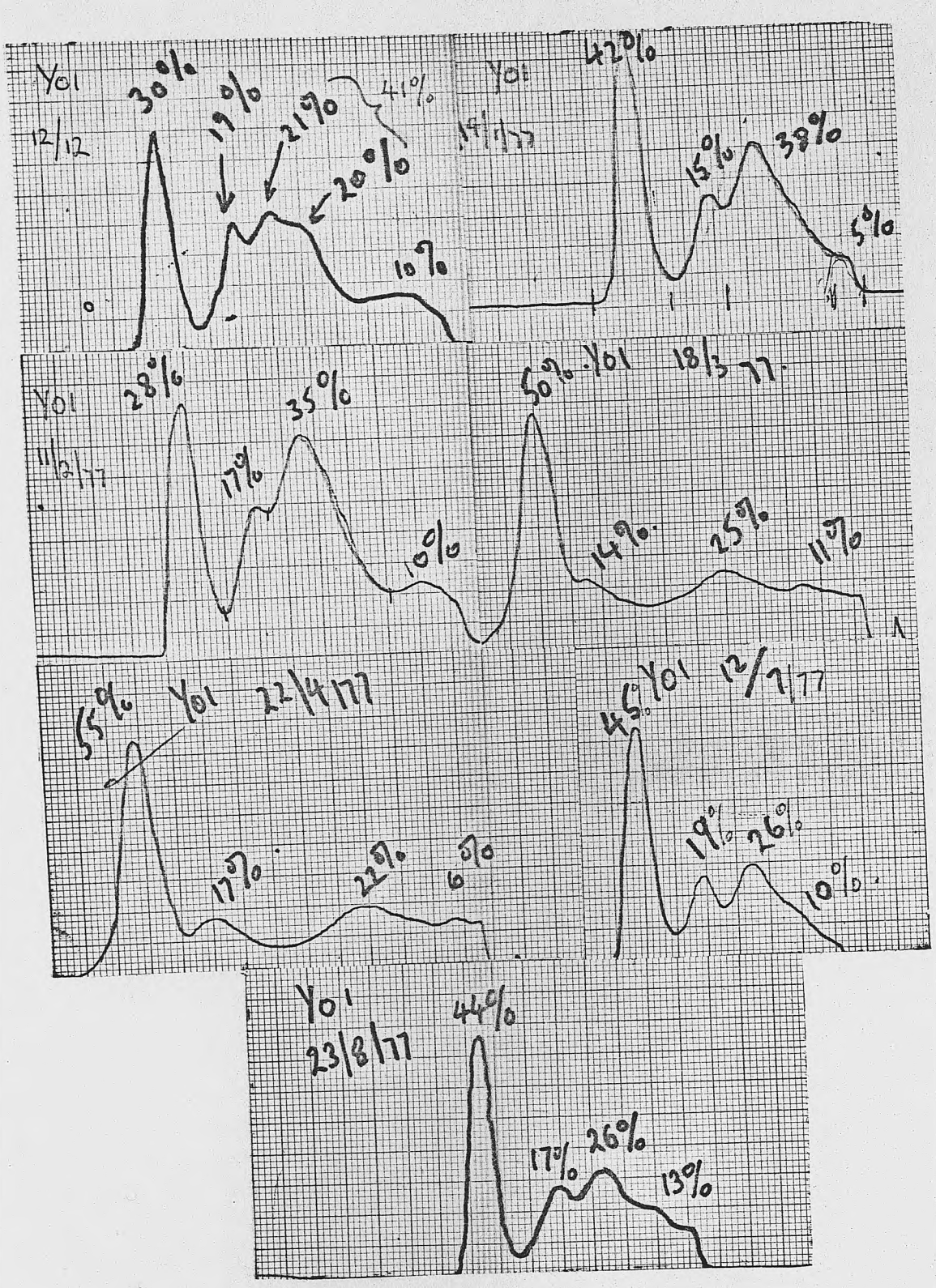
(Schalm, Jain & Carroll, 1975)

BREED / PARAMETER	THOROUGHBRED (From 26 animals)	STANDARD BREED (From 15 animals)
Red Blood Cells (RBC) ($x10^6/\mu l.$)	9.64 ± 1.06	8.32 ± 0.72
Haemoglobin (Hb.) (g./dl.)	15.2 ± 1.4	13.7 ± 0.9
Packed Cell Volume (PCV) (%)	43.6 ± 3.9	39.3 ± 2.5
Mean Cell Volume (MCV) (fl.)	45.6 ± 5.2	47.3 ± 3.4
Serum Protein (g./dl.)	7.0 ± 0.7	6.8 ± 0.2
White Blood Cells (WBC) ($x10^3/\mu l.$)	9.815 ± 1,449	7.913 ± 0.988
Neutrophils ($x10^3/\mu l.$) (%)	5.365 ± 1.056 (54.6)	4.456 ± 0.789 (56.7)
Lymphocytes ($x10^3/\mu l.$) (%)	3.684 ± .975 (37.6)	2.933 ± 0.818 (36.7)
Monocytes ($x10^3/\mu l.$) (%)	0.371 ± .215 (3.8)	0.397 ± 0.24 (4.9)
Eosinophils ($x10^3/\mu l.$) (%)	0.310 ± .208 (3.1)	0.084 ± 0.063 (1.1)
Basophils ($x10^3/\mu l.$) (%)	0.031 ± 0.056 (0.3)	0.042 ± 0.046 (0.5)



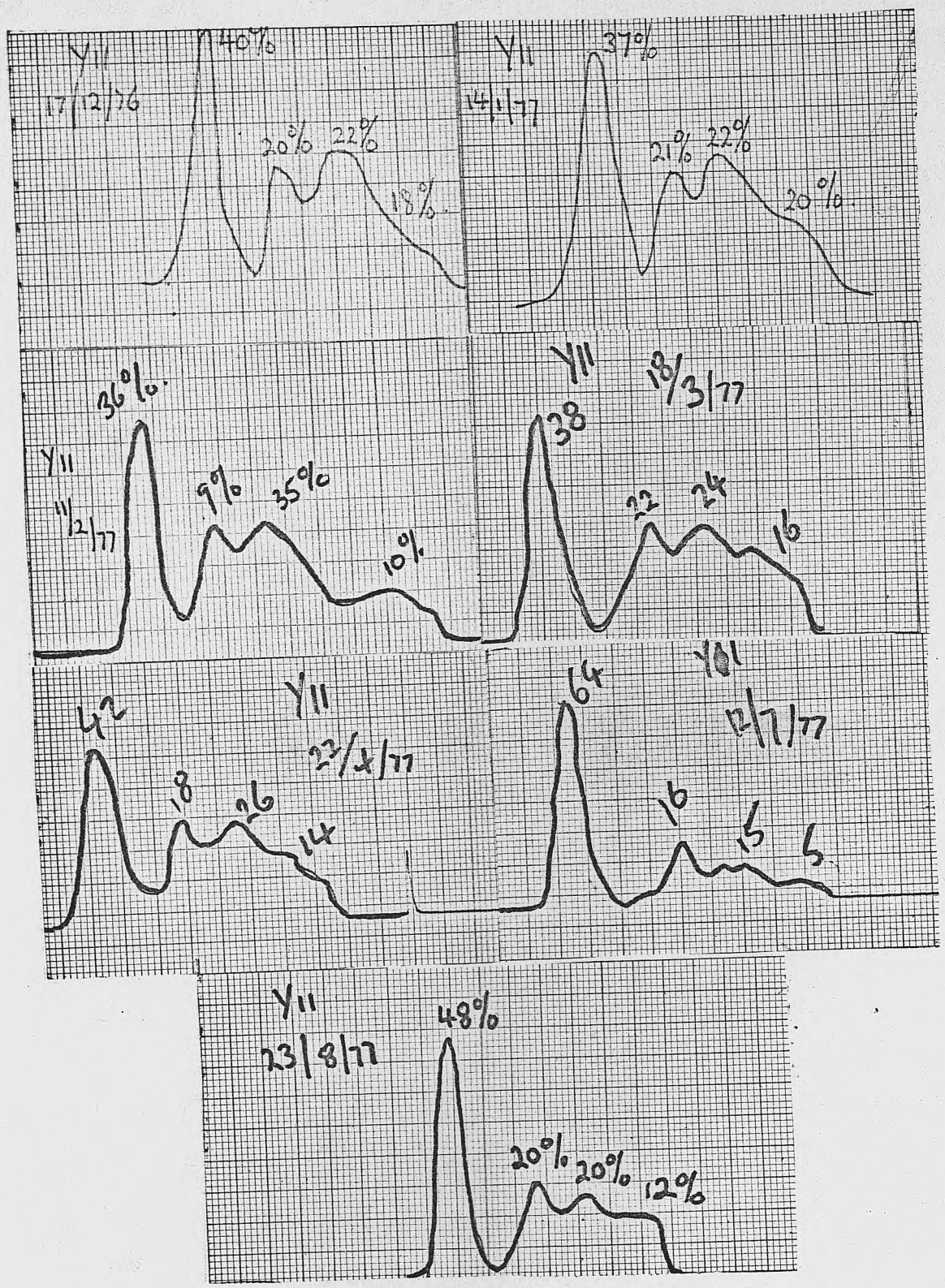
KEY 1

ELECTROPHORETIC GRAPH



ELECTROPHORETIC RESULTS OF

HORSE NO YOI



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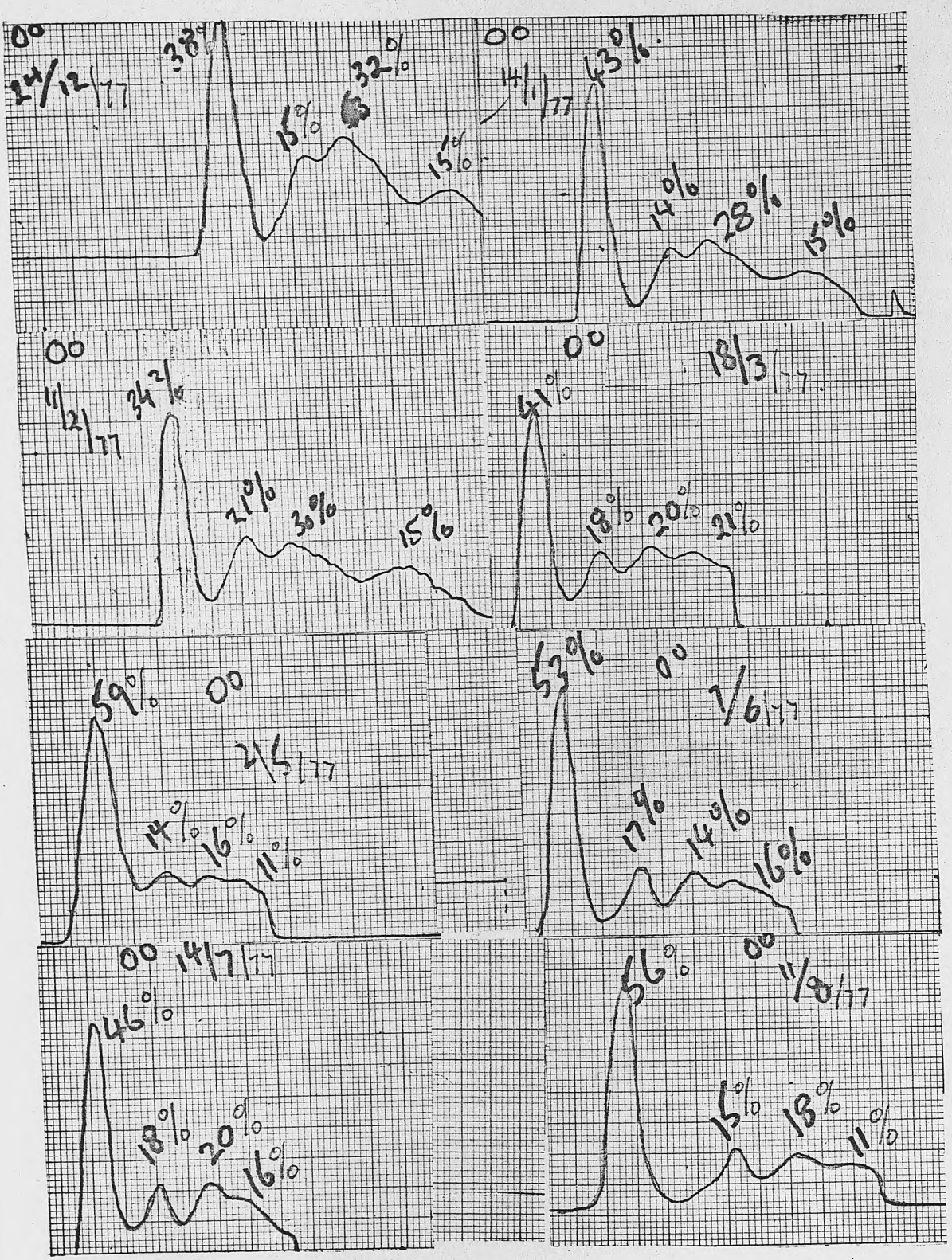

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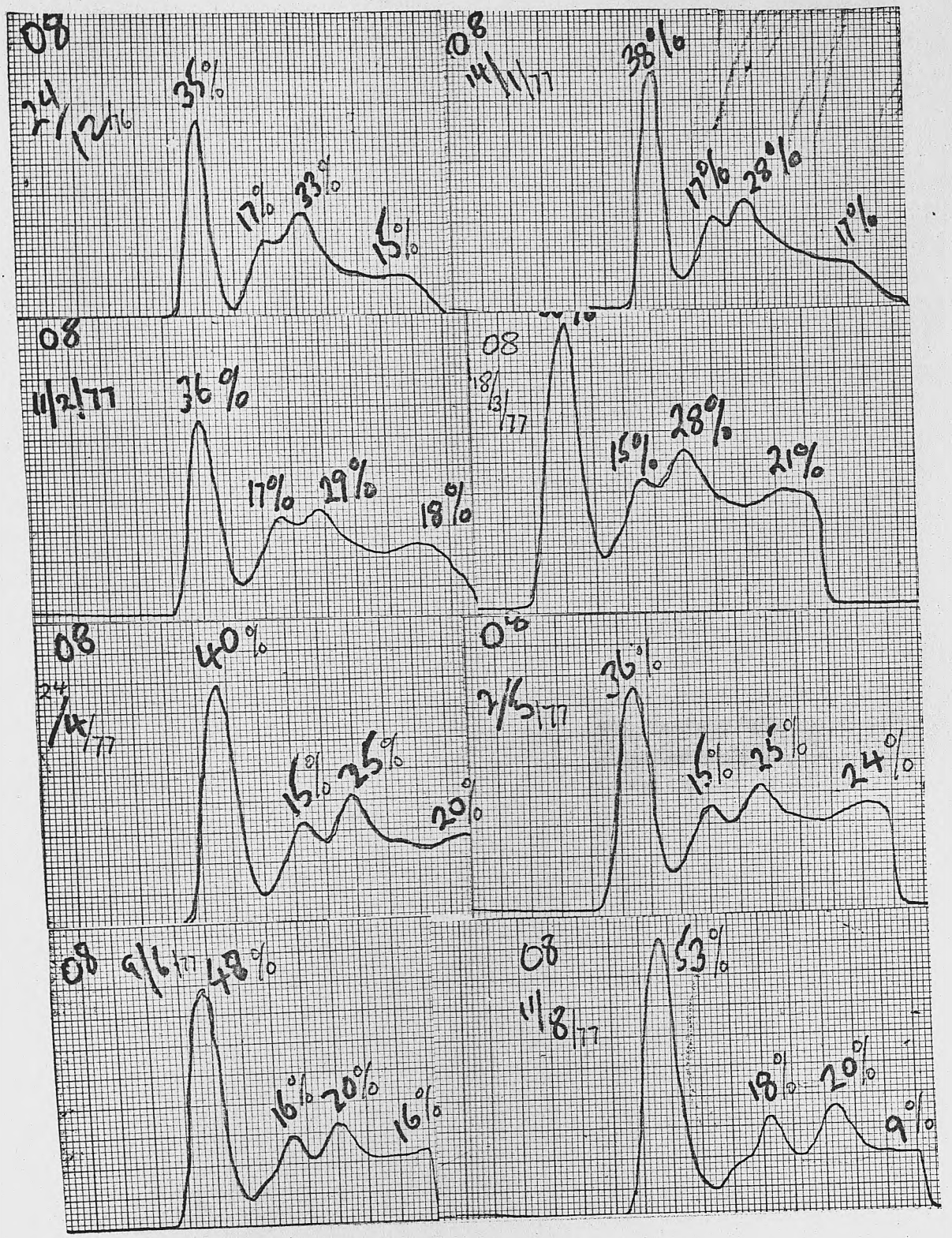

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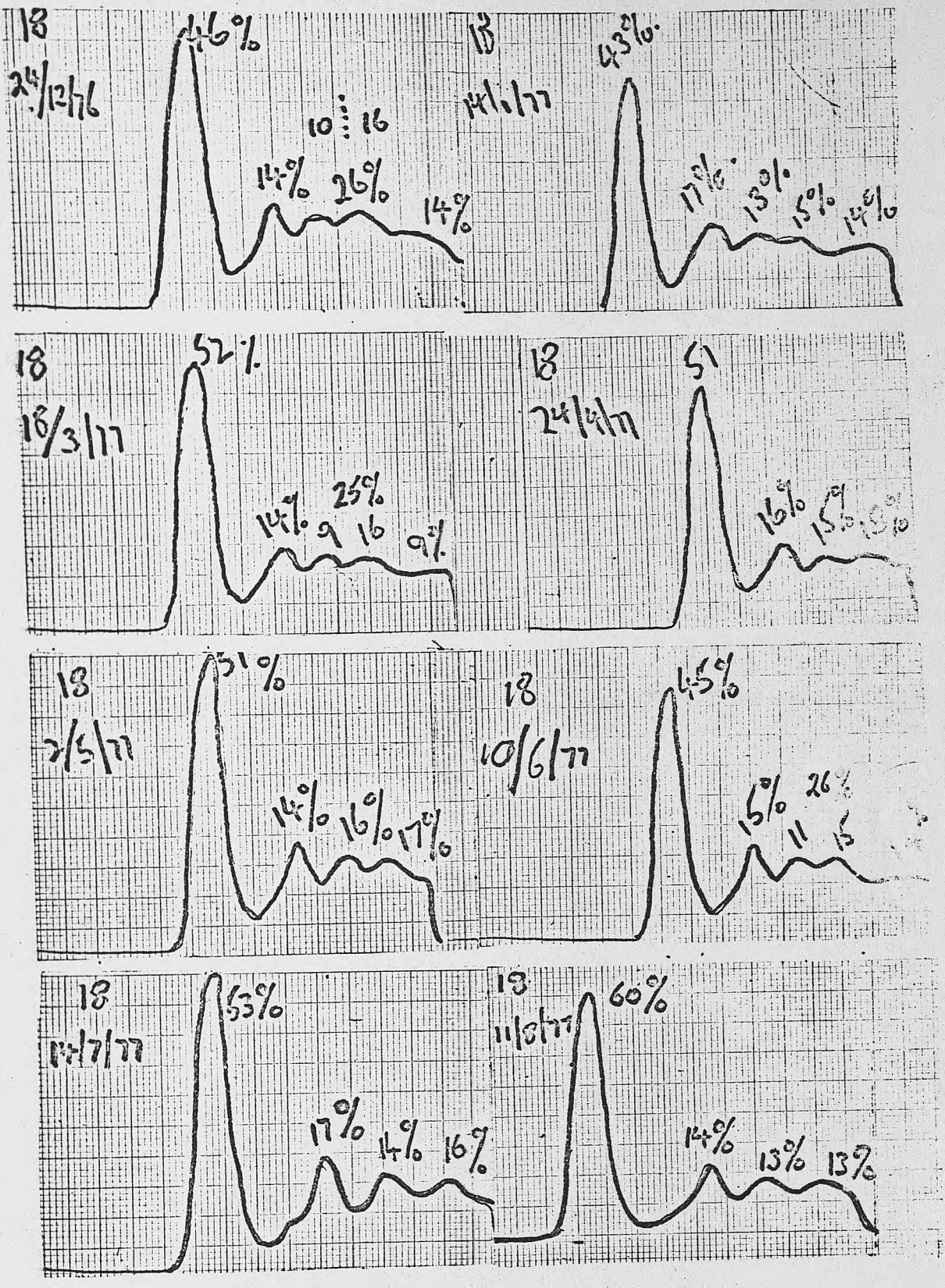
HORSE NO. 08



ELECTROPHORESIS RESULTS OF

HORSE NO. 18