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Osteofibrosis in Equines.

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Cases of osteofibrosis are not infrequently reported from race-horse stables where the animals are fed on cereal concentrates, and where the hay ration is kept relatively low. Unless legume hay is fed, or a correction is made in the diet by the supplementation of calcium, rations consisting for the most part of cereals may prove to have a high phosphorus and a low calcium content.

The occurrence of osteofibrosis has been reported from various countries, but has assumed a magnitude of considerable economic importance in India and the Philippine Islands, where the native feeds supplied to animals are relatively low in calcium. Although the disease is well known, the nomenclature used by different writers in describing it, differs considerably. The result is that a certain degree of confusion exists in regard to the various terms applied to this and related osteodystrophic disorders.

An attempt was, therefore, made to produce cases of osteofibrosis in horses. The material collected would then form a useful basis of bone pathological comparisons in osteodystrophic diseases.

LITERATURE.

Theiler et al. (1937) describe rickets or osteomalacia as a phosphorus deficiency disease, and suggest that calcium deficiency produces osteofibrosis. Their observations were carried out mainly on cattle.

Niimi (1927) considered that a deficiency of calcium salts in the diet was the cause of osteomalacia, and actually produced what he believed to be osteomalacia, but which in reality was osteofibrosis, in two old horses. The ration given to these horses consisted of barley and contained 2.09 gms. CaO and 27.57 gms. P₂O₅.

In a subsequent trial Niimi and Aoki (1927) produced the disease in horses receiving 3.6 gms. CaO and 47.4 gms. P_2O_5 daily. The control group remained normal when receiving a ration that contained 28.0 gms. CaO and 41.6 gms. P_2O_5 .

Marek (1922) points out that osteodystrophic diseases develop in pigs receiving a ration which does not show an Erdalkali-Alkalizität (CaO+MgO- P_2O_5 in mgm. equivalents) of 20 to 25 mgm. equivalents. This author apparently does not distinguish aetiologically between osteomalacia and osteofibrosis.

Kintner and Holt (1932) describe what they considered to be osteomalacia in horses and conclude that, "osteomalacia will develop in horses when the ratio of calcium oxide to phosphorus pentoxide is 1:2.9". According to the pictures of the bone pathology, these authors were dealing with osteofibrosis. It was further shown that, "the condition did not develop during the nine-month period when this ratio was 1:1.9. Evidence is presented showing that the calcium content of the drinking water was of particular importance".

It is apparent from the work of these authors that the indigenous feeds generally given to horses in the Philippines are so low in calcium, that unless limestone is added to the ration, equines are liable to contract osteofibrosis.

The work of Sturgess (1927) definitely showed that the possible lack of the vitamins contained in greenfeed could not be considered as contributory factors in the occurrence of osteofibrosis. This worker produced the disease on a ration consisting of bran and 10 pounds of green grass, and came to the conclusion that osteoitis fibrosa was a possible deficiency of calcium, the condition being intensified when there was a co-existing excess of phosphorus.

Crawford (1927) while working in Ceylon, came to the conclusion that the determining factor in the production of osteoporosis was the proportion of calcium to phosphorus present in the diet.

Olver (1933) in describing a case of osteoporosis in an aged gelding in India, gives the predisposing cause of the disease as diet composed mainly of cereals, such as bran and oats, with inadequate fodder of leguminous nature.

It is evident from the work of Theiler (1934) that the disease which was produced by Crawford and also by Olver, was not osteoporosis but osteofibrosis.

It is quite possible, according to Mitchell (1935), that a large number of common horse ailments such as arthritis of the knees and fetlocks, may be attributed to varying degrees of osteodystrophic diseases.

It is evident from the literature on osteofibrosis, that uncertainty exists in regard to the actual causation of the disease. Certain authors hold that osteofibrosis in horses is associated with abnormal calcium metabolism. The dietary calcium may be insufficient for the requirements of the animal, or the deficiency may be accentuated by a relatively large excess of phosphorus, or both factors may operate. The result is the same, viz., that the animal is starved for calcium.

EXPERIMENTAL.

Five yearling fillies, numbers 20722, 20723, 20721, 20739 and 20745, were procured for the purpose of producing clinical symptoms of osteofibrosis.

A common basal ration consisting of 1,000 gms. crushed oats, 1,000 gms. yellow maize, 1,000 gms. wheaten bran, 400 gms. greenteed, 500 gms. teff hay and 2 ounces of CaCO_a, was fed to the animals for a pre-experimental period of 8 months in order to ensure uniformity of pre-experimental treatment. The horses were then grouped and received mineral supplements as shown in Table 1:—

Table 1.

Experimental Plan.

No.	Group.	Supplement.	CaO : P ₂ O ₅ .	Remarks.
20723 20739	1	$64~\mathrm{gms.~Na_2HPO_4}$	1:12.06	The basal ration remained unchanged, with the exception
$20721 \\ 20722$	2	75 gms. CaCO ₃	1.8:1	that 2 ounces of CaCO ₃ was omitted.
20745	3	0	1:6.3	

The quantity of hay had necessarily to be limited to a daily intake of 500 gms. per horse in order to keep the CaO content of the ration low. The basal ration contained 370 gms. protein, 4.5 gms. CaO, 28.4 gms. P_2O_5 , and a CaO: P_2O_5 ratio of 1:6.

The concentrate mixture, greenfeed and mineral supplement were fed to the horses at 2 p.m. daily. The hay was supplied early in the morning, and when it had been consumed the animals were muzzled and allowed to run in an open air, cement-floored paddock until 2 p.m.

In order to avoid possible complications due to internal parasitic infection, all the animals were treated for worms periodically. Daily inspection of the health and development of clinical symptoms of the horses were made. The animals were weighed monthly, and blood was drawn for the chemical determinations of calcium, phosphorus and phosphatase. Metabolism trials were carried out during the latter four months of the experiment. In order to ensure the daily consumption of all the minerals supplied, a system of feeding was adopted whereby each horse received only as much feed as it would eat. It was necessary, however, occasionally to weigh feed back.

Rubber matting was used in the stable in order to obviate the consumption of bedding by the animals.

RESULTS.

The horses, without exception, showed osteophagia or "pica" to a marked degree, and unless muzzled, would greedily consume each other's faeces. Lack of total bulk in the ration may have contributed towards the deprayed appetites shown by the animals.

The clinical progress of the disease may be given as follows:-

Horse 20723, Group 1. Intake CaO = 4.5 gms. $P_2O_5 = 54.29$ gms.

Date. Remarks.

- 11.9.34. Commenced supplementation of 64 gms. Na₂HPO₄ daily.
- 14.5.35. A shortened stride was noticeable.
- 16.5.35. Facial swellings just visible.
- 4.6.35. Animal reluctant to rise in the morning.
- 6.6.35. Rear feet rested alternately when standing.
- 11.6.35. Facial enlargements marked, involving submaxillary bones. Horse would frequently lie down with legs outstretched. Abscess on point of left shoulder.
- 13.6.35. Chewing became painful and quidding of the food was observed.
- 3.7.35. Stride stiff and short, face greatly enlarged.
- 17.7.35. Unable to rise, animal put into a sling.
- 18.7.35. Supplement changed to 75 gms. CaCO3 daily.
- 21.7.35. Feed consumption improved, animal made attempts to stand.
- 22.7.35. Able to walk with difficulty, thrust in off fore.
- 2.8.35. Greatly recovered, kept on control ration for a year in order to observe facial swelling.

Horse 20739, Group 1. Intake CaO = 4.5 gms. $P_2O_5 = 54.29$ gms.

Date Remarks.

- 11.9.34. Commenced supplementation of 64 gms. Na₂HPO₄ daily.
- 14.5.35. When trotted a slightly shortened stride noticeable.
- 21.5.35. Ringbone appeared on near rear pastern.
- 28.6.35. Stiff gait and rested rear feet alternately.
- 25.7.35. Stiff, shortened stride, pasterns painful, off feed.
- 9.8.35. Facial enlargement very sudden and marked.
- 23.8.35. Abscess developed on near knee.
- 11.9.35. Unable to rise unassisted.
- 13.9.35. Fell and broke back behind sacral vertebra. Microscopical examination of bones showed osteofibrosis.

Horse 20722, Group 2. Intake $CaO = 52 \cdot 29$ gms. $P_2O_5 = 28 \cdot 42$ gms.

Date. Remarks.

11.9.34. Commenced supplementation of 75 gms. CaCO₃ daily.

11.9.35. Animal remained in good health and thrifty throughout the duration of the experiment. No facial enlargements developed, and she was retained on the same ration for an additional period of a year in order to act as further control to 20723.

Horse 20721, Group 2. Intake $CaO = 52 \cdot 29$ gms. $P_2O_5 = 28 \cdot 42$ gms.

Date.

Remarks.

11.9.34. Commenced supplementation of 75 gms. CaCO3 daily.

11.9.35. Discharged from experiment in excellent health and condition. No facial enlargements were shown.

Horse 20745, Group 3. Intake CaO = 4.5 gms. $P_2O_5 = 28.42$ gms.

11.9.34. Basal ration only, i.e. no mineral supplement.

16.5.35. Slight swelling of left sub-maxillary bone.

 12.7.35. Enlargement of bone on left side of face pronounced, poor, coat stary and gait stiff.

12.11.35. Discharged from experiment in poor condition.

The general condition and appearance of the fillies when at first admitted to the experiment, as well as at the conclusion of the experimental period, may be seen from the photographs of these animals given in Appendix I. As further illustrated in these photographs the facial enlargements involve mainly the submaxillary bones. The enlargements being particularly clear in the case of 20723, and remained unchanged after this animal received a calcium carbonate supplement for a year. The poor condition of 20723 and 20739 is apparent at the conclusion of the experiment. The characteristic way in which 20739 draws up her hind leg, and the slightly protruding tongue, is worthy of note.

The curves indicating the weights of the fillies are given in Appendix 2, and indicate clearly that only in the case of the control group, 20721 and 20722, was there any gain in weight during the experimental period. The consistent loss in weight of 20723 was checked when calcium carbonate was supplied in the ration.

Feed consumption may be regarded as satisfactory. Animals only went off feed when exhibiting severe clinical symptoms of osteofibrosis.

The figures indicating chemical blood determinations that were made are given in Appendix 3, and show no marked deviation in any particular group or constituent. It may, therefore, be concluded that blood analyses will give no assistance in the diagnoses of the

disease. No indication of anaemia could be registered from the haemoglobin determinations made when the animals in group 1 were in the concluding stages of the disease.

The temperatures were recorded in every case throughout the experimental period and were not in any way affected by the progress of the disease.

The actual mineral intake of each horse is given in Table 2.

Table 2.

The Ca and P supplied to the horses.

Number.	P intake.	Supp.	Total P.	Ca intake.	Supp.	Total Ca
20723	12-41	11.3	23.71	3.41	0	3.41
20739	12.41	11.3	23.71	3 - 29	0	3 - 29
20721	12.41	0	12.41	3.35	34.0	37.35
20722	12-41	0	12.41	3.17	34.0	37 - 17
20745	12.41	0	12.41	3 · 23	0	3.23

Metabolism trials were carried out at monthly intervals during the last four months of the experiment. The results of these trials are given in Tables 3 and 4.

The quantity of calcium supplemented to horses 20721 and 20722 had necessarily to be sufficient to enable a $CaO:P_2O_5$ of 1.8:1 to be obtained. It does not, therefore, have any bearing upon the calcium requirement of horses.

Table 3.

Calcium Metabolism.

				Ca ou				
Date.	Number.	Group.	Ca intake.	Faeces.	Urine.	Balance.		
6/35	20723 20739 20721 20722 20745	$\left.\begin{array}{cccccccccccccccccccccccccccccccccccc$	$ \begin{array}{r} 3 \cdot 41 \\ 3 \cdot 29 \\ 37 \cdot 35 \\ 37 \cdot 17 \\ 3 \cdot 23 \end{array} $	$7 \cdot 49$ $5 \cdot 51$ $23 \cdot 32$ $20 \cdot 60$ $5 \cdot 44$	0.88 0.72 2.93 4.48 0.38	$\begin{array}{ c c c c c c }\hline - & 4 \cdot 19 \\ - & 2 \cdot 94 \\ + & 11 \cdot 10 \\ + & 12 \cdot 09 \\ - & 2 \cdot 59 \\ \hline\end{array}$		
7/35	20723 20739 20721 20722 20745	$ \begin{cases} 1 & {1 \atop 2} \\ 3 \end{cases} $	2·70 2·68 37·35 30·24 2·67	$7 \cdot 12$ $5 \cdot 88$ $30 \cdot 40$ $24 \cdot 00$ $5 \cdot 43$	0.44 0.53 2.17 4.30 0.64	$\begin{array}{r} - & 4.86 \\ - & 4.31 \\ + & 4.72 \\ + & 1.94 \\ - & 3.40 \end{array}$		
8/35	20723 20739 20721 20722 20745	$ \begin{cases} 1 & \begin{cases} \\ 2 & \end{cases} \\ 3 & \end{cases} $	37 · 94 2 · 66 37 · 42 31 · 28 2 · 65	$14 \cdot 20$ $3 \cdot 18$ $30 \cdot 40$ $24 \cdot 20$ $5 \cdot 66$	0·48 0·51 3·36 4·60 0·87	$\begin{array}{r} + 22 \cdot 26 \\ - 1 \cdot 03 \\ + 3 \cdot 66 \\ + 2 \cdot 48 \\ - 3 \cdot 88 \end{array}$		
9/35	20723 20739 20721 20722 20745	$ \begin{cases} 1 & \begin{cases} 2 & \begin{cases} 3 \end{cases} \end{cases} $	37·96 2·60 37·41 33·36 2·64	$27 \cdot 70$ $3 \cdot 82$ $31 \cdot 40$ $27 \cdot 40$ $3 \cdot 80$	2·95 0·62 2·73 5·04 0·75	$\begin{array}{ c c c c }\hline + & 7 \cdot 29 \\ - & 1 \cdot 80 \\ + & 3 \cdot 28 \\ + & 5 \cdot 52 \\ - & 1 \cdot 91 \\\hline \end{array}$		

Table 4.

Phosphorus Metabolism.

				P ou		
Date.	Number.	Group.	P intake.	Faeces.	Urîne.	Balance.
6/35,	20723 20739 20721 20722 20745	} 1 { } 2 { 3	23.71 23.71 12.41 12.41 12.41	7·72 6·60 10·15 9·02 5·06	4.53 4.34 0.11 0.22 0.80	+ 11·46 + 12·77 + 2·16 + 3·17 + 6·55
7/35	20723 20739 20721 20722 20745	$\left.\begin{array}{ccc} 1 & \left\{ & 2 & \left\{ & 2 & \left\{ & 2 & \left[& &$	19.60 16.47 12.41 10.68 6.31	$11 \cdot 20$ $7 \cdot 56$ $12 \cdot 10$ $7 \cdot 62$ $4 \cdot 47$	2·42 2·70 0·06 0·06 1·54	+ 5.98 + 6.21 + 0.21 + 3.00 + 0.30
8/35	20723 20739 20721 20722 20745	$\left.\begin{array}{ccc} & 1 & \left\{\\ & 2 & \left\{\\ & 3 & \end{array}\right.\right.\right.$	$\begin{array}{c} 16 \cdot 46 \\ 10 \cdot 40 \\ 12 \cdot 41 \\ 12 \cdot 41 \\ 8 \cdot 32 \end{array}$	$7 \cdot 30$ $5 \cdot 39$ $9 \cdot 70$ $6 \cdot 95$ $5 \cdot 23$	1·05 1·51 0·03 0·06 0·91	$\begin{array}{c ccccc} + & 8.11 \\ + & 3.50 \\ + & 2.68 \\ + & 5.40 \\ + & 2.18 \end{array}$
9/35	20723 20739 20721 20722 20745	$\left.\begin{array}{ccc} & 1 & \left\{\\ & 2 & \left\{\\ & 3 & \end{array}\right.\right.\right.$	16·53 16·62 12·41 12·41 6·82	10·48 8·41 9·60 8·51 5·00	0·17 1·78 0·08 0·10 1·68	+ 5.88 + 6.33 + 2.74 + 3.80 + 0.14

The slight variations in Ca and P intakes for different metabolism periods may be attributed to the lowered food consumption of the animal concerned. A total chemical analysis of the ration prior to each metabolism trial similarly discloses slight variations in the Ca and P contents.

It may be seen from the metabolism figures that a positive phosphorus balance was retained in all cases. The retention of phosphorus was greater in 20723 and 20739, where P intake was highest. Where the calcium was deficient in the ration, groups 1 and 3, a negative calcium balance was recorded. The retention of Ca was greatly increased immediately after Ca had been supplied to 20723, as is evident from a comparison of period 8/35 in Table 3, with the Ca retentions of 20723 for the previous periods.

A record of the X-ray photographs taken of the metacarpal bones two months prior to the conclusion of the experiment, are given in Appendix 4. The lesser defined lines are clearly shown in the metacarpal bone pictures of 20723, 20739 and 20745, as compared to the distinctly denser bones of 20721 and 20722.

DISCUSSION.

Clinical symptoms of osteofibrosis were produced in both horses, 20723 and 20739 fed a ration containing 4.5 grams CaO and 54.29 grams P_2O_5 , i.e. a ratio of 1:12.06.

The disease was produced to a lesser degree in horse 20745 receiving in its ration 4.5 grams CaO and 28.4 grams P_2O_5 , i.e. a ratio of 1:6.3.

In these horses, kept under laboratory conditions where no work was performed, the first clinical symptoms of osteofibrosis were seen eight months after the commencement of the experiment. When once clinical symptoms became clearly established, the disease took its course in an alarmingly rapid fashion. At first facial swellings became noticeable, these involved mainly the submaxillary bones, the enlargement of which became most marked during the last month of the experiment. There was also a thickening of the jaw hones, which became especially marked in the case of 20723.

Both horses 20723 and 20739 displayed characteristically shortened strides, the rear pasterns became painful and the animals would draw up the rear feet alternately as shown by 20739 in Appendix 1. Apparently 20723 had difficulty, or pain, in properly masticating its feed, as quidding of partly masticated hay was frequently seen in the manger.

Bone sections were taken for histopathological examination according to the technique described by Thomas and van der Wath (1937). Although this method of bone sampling was devised after the conclusion of the experiment, sections were nevertheless taken from 20723 and 20745 on the 3.11.36 and 5.11.36 respectively. The rib sections in both cases showed that osteofibrosis had been present. When horse 20739 had to be destroyed on the 13.9.35, microscopical examination of the bones left no doubt as to the presence of osteofibrosis.

The weight curves shown in Appendix 2 indicate clearly that only for the animals in group 2 (20721 and 20722), was there a steady gain in weight recorded throughout the experimental period. In group 1, horse 20739 showed a satisfactory gain in weight for seven months on the experimental ration. During the last three months, however, a rapid loss in weight occurred. A steady loss in weight was recorded in the case of 20723 and 20745.

From the figures available for blood analyses which are given in Appendix 3, it would appear that a calcium deficiency, as in 20723, 20739 and 20745, was not reflected by a lowered blood calcium during 1935. The inorganic phosphorus, phosphatase, and haemoglobin content of the blood and serum remained normal in all cases. Blood analysis is not, therefore, considered a reliable guide for diagnostic purposes in the case of osteofibrosis suspects.

An analysis of the results of the balance trials given in Table 3 shows that horses 20723, 20739 and 20745, remained on a negative calcium balance while on a daily average Ca intake of about 2.85 grams. It is evident, therefore, that the skeletons of these animals must have suffered a continual depletion of calcium salts. Such a drain upon the system must eventually lead to serious consequences.

It is significant that immediately after horse 20723 had its Ca intake raised from 2.7 gms. to 37.94 gms., the balance was 22.26 gms. as compared to -4.86 for the previous month as shown in

periods 8/35 and 7/35 respectively. The demand for calcium, which a period of starvation had created, was rapidly satisfied by the sudden abundant supply, as may be seen in period 9/35 when only 7.29 gms. Ca was retained.

From a study of the results of the phosphorus (P) balance trials given in Table 4, it may be seen that all the animals showed a positive phosphorus balance. The control animals 20721 and 20722 showed an average retention of phosphorus of 23 per cent. When the phosphorus intake was 23.71 gms. as is shown in trial 6/35 in the case of 20723 and 20739, the balance was 11.46 and 12.77 respectively, or about 50 per cent. The retention of phosphorus was, therefore, considerably increased when the intake was more. Subsequent periods show a lower phosphorus balance for 20723 and 20739 because food consumption became irregular during the latter three months with the result that the intake had to be reduced during the metabolism periods.

The presence of large amounts of phosphorus and its increased utilization, may be the reason for the aggravation of the disease when there is a wide CaO: P₂O₅ ratio, as well as a deficiency of calcium.

When horse 20723 was given a supplement of 37.94 grams Ca at the conclusion of the experimental period, its recovery was phenomenal. As a result, it was decided to continue giving the control diet to this horse in order to note whether there would be any lessening of the facial swelling. Although a remarkable gain in condition was noted, the facial enlargement did not subside, as may be seen from the photographs in Appendix 1. These pictures were taken a year later, together with those of the control horse 20722. The latter received its original control ration until discharged on the 17.2.37.

The X-ray photographs of the metacarpal bones shown in Appendix 4 illustrates clearly that the bone in the case of horses 20721 and 20722 is more compact and thicker than that of horses 20723, 20739 and 20745. The various lines of demarcation are far more clearly defined in the case of the former animals. The medullary cavity is smaller and consequently the outer shell thicker. In the latter cases there is a wider, less dense cortex which is poorly defined.

SUMMARY.

- (1) Clinical symptoms of osteofibrosis were brought about, and later definitely shown to be the disease by histo-pathological examination, in three 2-year-old fillies receiving a ration which contained 4.5 grams CaO and 54.29 grams P_2O_5 in the case of two horses, and 28.42 grams P_2O_5 in the case of the other horse.
- (2) The two control fillies received in their ration 52.3 grams CaO and 28.42 grams P_2O_3 . The CaO intake had necessarily to be high in order to rectify the otherwise abnormal CaO: P_2O_3 ratio.
- (3) The control animals gained in weight, whereas the horses receiving a deficiency of CaO in their diet lost weight, became emaciated and poor in condition.

- (4) Balance trials showed that the skeletons in the case of those horses receiving 4.5 gms. CaO, were continually being depleted of calcium.
- (5) The phosphorus retention was greatest for the horses receiving most phosphorus in their ration.
- (6) From the data available it would appear that blood Ca determinations are of little value for diagnostic purposes in cases of suspected osteofibrosis.
- (7) Facial enlargements, when once established by the disease, were not reduced in size by feeding a ration which was supplemented with CaCO₃.

ACKNOWLEDGMENT.

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APPENDIX 1.

PHOTOGRAPHS OF HORSES.

Control horses 20721 and 20722: Intake: $-CaO = 52 \cdot 29$ gm., $P_{s}O_{s} = 28 \cdot 42$ gm.

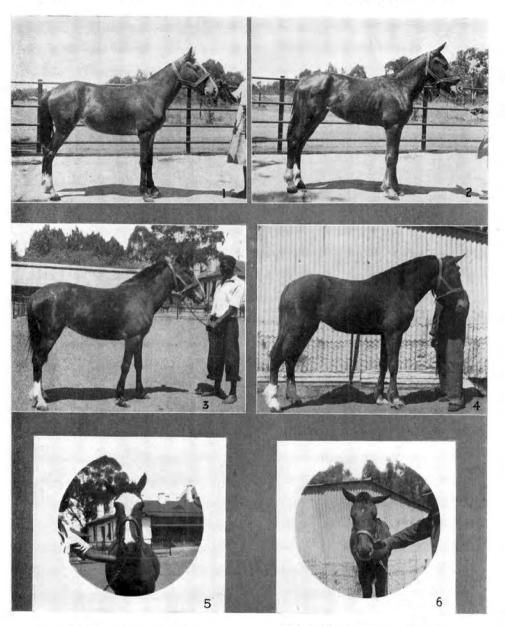


Fig. 1. Horse 20721 (29.10.34). Fig. 3. , (9. 9.35). Fig. 5. , (9. 9.35) Face.

Horses 20723 and 20739: Intake:— $CaO = 4 \cdot 5$ gm., $P_2O_5 = 54 \cdot 29$.

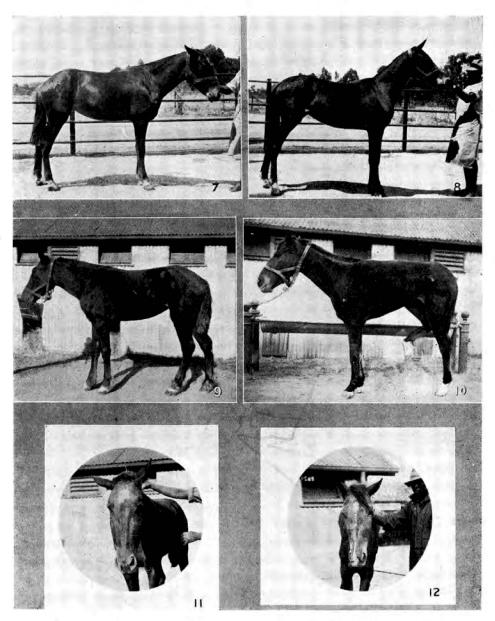


Fig. 7. Horse 20723 (29.10.34). Fig. 9. " (9. 9.35). Fig. 11. ", (9. 9.35)

9.35) Face.

Fig. 8. Horse 20739 (29.10.34). Fig. 10. , (9. 9.35). Fig. 12. , (9. 9.35) Face.

Horse 20745: Intake:— $CaO = 4.5 \ gm., \ P_2O_5 = 28.42 \ gm.$



Fig. 13. Horse 20745 (29.10.34).

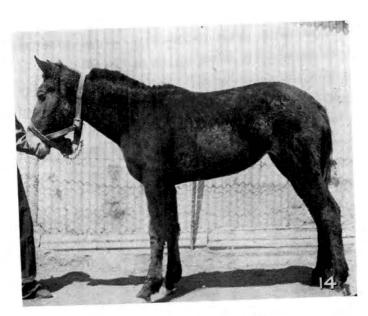


Fig. 14. Horse 20745 (9. 9.35).

 $Horse \ 20745: \ Intake:-CaO\!=\!4\cdot 5 \ gm., \ P_{2}O_{5}\!=\!28\cdot 42 \ gm.$



Fig. 15. Horse 20745 (9. 9.35)

Horses 20722 and 20723 photographed on 17.2.37 after receiving an intake of 52·29 gm. CaO and 28·42 gm. P_2O_3 since the conclusion of the experiment on 12.11.35.



Fig. 16. Horse 20722.

Horses 20722 and 20723 photographed on 17.2.37 after receiving an intake of $52\cdot29$ gm. CaO and $28\cdot42$ gm. P_2O_3 since the conclusion of the experiment on 12.11.37.

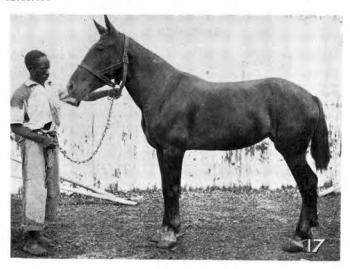


Fig. 17. Horse 20723.



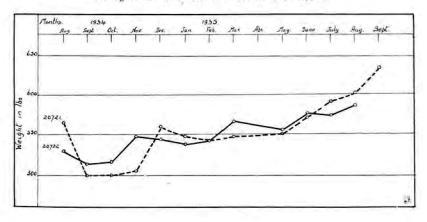
Fig. 18. Face of Horse 20722.



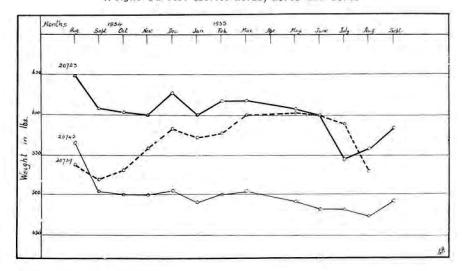
Fig. 19. Face of Horse 20723.

APPENDIX 2.

Weight Curves: Horses 20721 and 20722.



Weight Curves: Horses 20723, 20739 and 20745



APPENDIX 3.

Inorganic Phosphorus in mgm./100 c.c. blood.

		Mont	hs:	1935.				Months: 1936.						
Number.		1	1	1	-	-	î.	T.	1	1	1		1	
	5.	6.	9.	10.	11.	1.	3.	4.	5.	6.	7.	8.	9.	10.
20723*,	2.9	4.9	3.6	3-9	4.0	3-4	3.5	3.7	3.6	3.7	3.3	4.0	4.0	3.9
20739	3.3	4.4	3.4	die			0.0	1	0.0	100.0	0.0	1		
20721	1.8	2.9	3.3	die						1 .			N 1	
20722	1.9	4.0	3.6	3.6	2.8	5.1	2.9	3.7	3.8	3.4	3.7	3.5	3.9	3.5
20745	3.1	3.4	3.9	4.0	5.1	4.6	8.5	2.6	5.6	5.0		5.8	5.5	5.9
	* 2	0723	recei	ved a	CaC		4,400	nent	in 19	936.				
	7		1	1.	I , k	seru;	no.	Ŷ	1		-	-	ř	1
20723	5.2	5.2	5.2	4.7	5.0	4.8	4.9	5.2	5.0	4.0	5.1	5.3	5:0	1
20739	6.1	5:1	4.9	die		3.0	10	0.2	0.0	- M	2.1	10.00	0.0	
20721	4.8	4.1	4.0	die			1				1			
20722	4.1	3.6	5.1	4.5	4.2	-5.	3.9	5.0	5.1	4:5	4.6	4.5	5.2	100
20745	5.3	4.7	4.7	4.4	6.8		10.5	104 100	6.4	6.3	5.8	6.9	7.2	Ξ
20740	3.3	4.4	4.4	474	0.8		10:5	0.7	0.4	0.9	3.9	0.9	1.2	
2				P	hosp	hata	se.							
20723	10.9	12.4	8.1	9-1	12.2	_	13-2	10-1	13-0	11.5	11.0	9:0	13.3	
20739	9.5	11.0	11.6	die	100		1000				1000	1		
20721	7.1	8.4	7.3	die							1	H 1	100	
20722	11.8	10.1	10:0		12.8	-	11.5	10.1	15.6	11.8	13.0	11.1	13.4	1
20745	6.5	6.5	6-7	7.7	9.4	=	8.5	8.1	10.8	8.0	10.8	11.4	9.8	-
		1		1	Calc	ium.							1	
201-200	1				10.0	1	l	[.,			100		1.,	
20723	_	-	10.9	2.7	100	-	11-1	8.3	7.9	10.1	9.6	9.4	9.1	-
20739	-	-	11-I	die	1000		100	1	15.3					
$20721\ldots\ldots$	-	-	9.6	die		1	1	150	100	100.0	W	74.4	lac at	
20722	-	-	9.4		10.8	-	10.1			10.1	9.7	10·I	9.5	-
20745		-	11.2	10.7	8.8	-	9-0	8.4	8.4	8.3	7.2	9.4	6.9	-
				Н	aeme	oglob	rin.							
20723			12.1	19.4	11.3		-							
20739			11.7	die			1	1	1	1000				
20721		Œ	11-9				1							1
	1		20,000	21.00	200							1		
			11.5	19.1	11.9.01									
20722 20745	1-	-	11.5	200	12·0 12·0		1	-	-	-				

Appendix 4.

Radiographs of Metacarpal Bones of Horses 20722, 20723, 20721, 20739 and 20745.

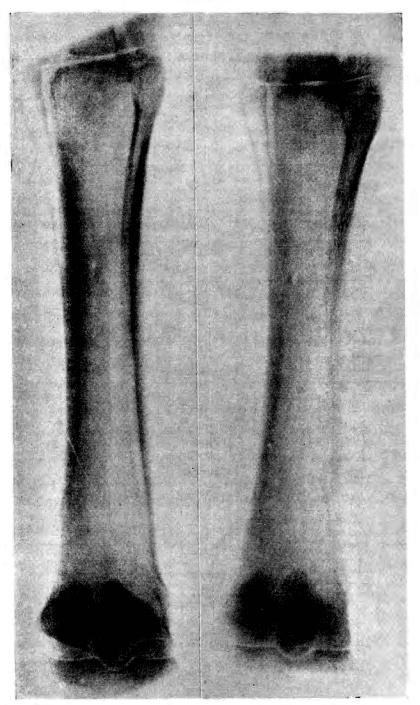


Fig. 20. Horse 20722.

Fig. 21. Horse 20723.

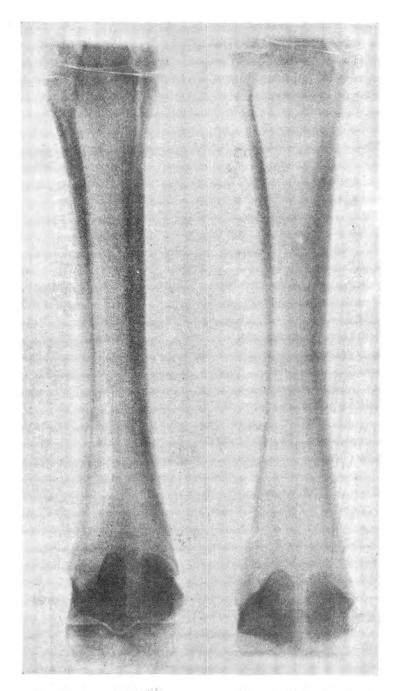


Fig. 22. Horse 20721.

Fig. 23. Horse 20739.

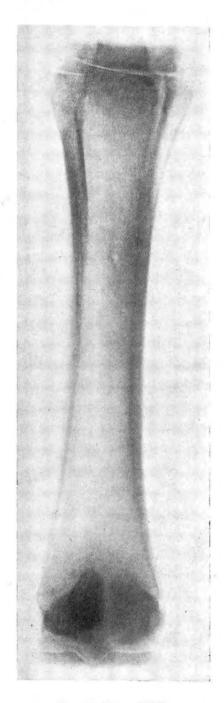


Fig. 24. Horse 20745.