Blood Studies: A Contribution to the Study of the Blood and Circulation in Horse-sickness.

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INTRODUCTION.

In a previous paper (1) some data in respect of the blood of healthy horses were presented. The main points emphasized were the extraordinary effect of prolonged vigorous exercise upon the erythrocyte content of jugular blood and the unequal distribution of blood-cells through the body during rest.

Examples:—

(1) Stabled horse not used to work: jugular blood contained 18 per cent. by volume of red cells.

Racehorse, fully trained: jugular blood contained 52 per cent. by volume of red cells.

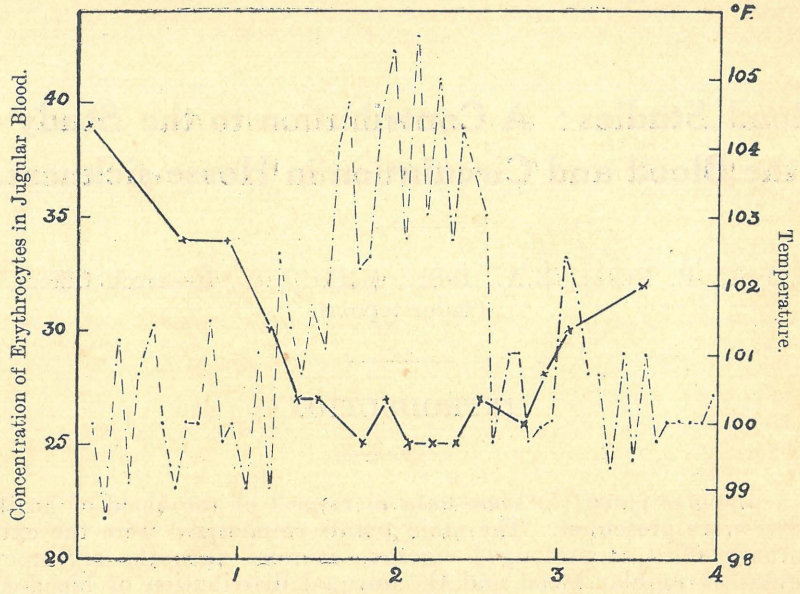
(2) Horse feeding quietly: ear red count, $9 \cdot 52 \times 10^6$; jugular red count, $6 \cdot 63 \times 10^6$.

Same horse threatened for 20 minutes: ear red count, $7 \cdot 89 \times 10^6$; jugular red count, $7 \cdot 54 \times 10^6$.

For these reasons it was pointed out that "average" figures obtained from a number of miscellaneous horses did not form a suitable basis for comparison with data obtained in diseases, and that the only sound way of obtaining reliable comparative data was to study the blood of one and the same animal first in health and then in disease. It was further pointed out that, owing to variations in the data recorded for the same animal from time to time in health, it became imperative to make observations as often as possible during disease.

A technique for taking blood-samples and making determinations on them was described. By the use of this technique blood-samples can be taken anywhere, relative errors are reduced to negligible proportions, and determinations can be made at leisure in the laboratory; even repeated, if necessary, without resampling. For differential counts, smears, specially made for the purpose, gave better results than smears made in any other way. For details of the technique the reader is referred to the original paper (*l.c.* 1).

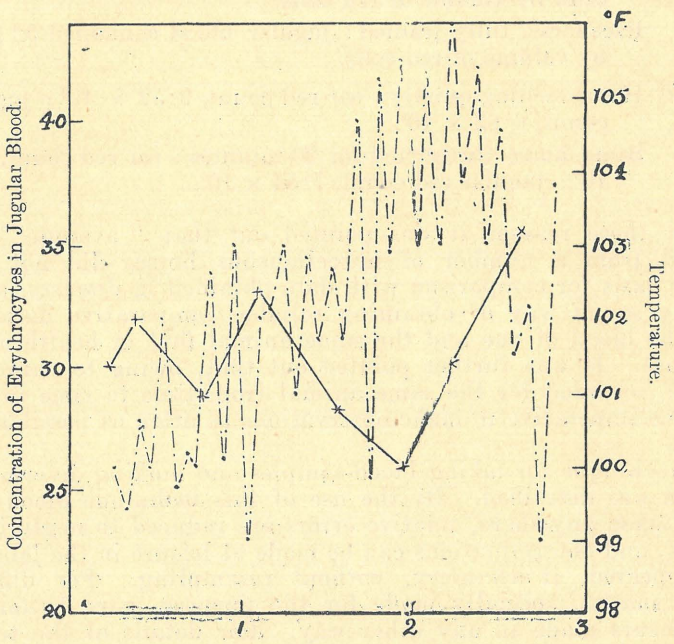
Horse No. 12214. August, 1920.



Weeks under Observation.

Graph I.

Horse No. 13854. October, 1920.



Weeks under Observation.

Graph II.

De Kock (2), working on infectious anaemia in horses, has already presented data in which the principles referred to were considered, and the technique used. In the present paper certain data obtained under the individual conditions emphasized above are presented for "horse-sickness." It is hoped subsequently to present similar and other data for various diseases of domestic animals.

Erythrocytes.

The morphology and staining reaction of the erythrocytes remain unchanged during the entire course of horse-sickness. In a few instances slight crenation was noted in smears made at the time when the jugular blood was very hydraemic, and this was probably caused by slow drying of such smears. In moist preparations no structural changes whatsoever could be seen in individual cells, but the tendency towards rouleaux formation and clumping is much more pronounced than in health.

The diameters of the red cells do not change to any appreciable extent during the course of the disease, and even at the fever acme the average approximates to 5.5μ, the figure obtained for healthy horses.

The volume of the erythrocyte is not changed in horse-sickness, as the ratio between percentage volume and count of erythrocytes remained approximately constant at the figure established for healthy horses, viz., $4.35 \times k$.

The haemoglobin content of the red cells showed minor adventitious fluctuations as in health, these being undoubtedly due to unavoidable errors with the use of the Sahli haemometer. The data certainly do not suggest that the haemoglobin content of the erythrocytes becomes changed in horse-sickness.

In respect of the above points, therefore, the blood in horse-sickness remains normal.

The concentration of erythrocytes in jugular blood, however, is in most cases subject to marked variations during the course of horse-sickness. These variations may be discussed by reference to graphs I and II, in which the data in respect of temperature and erythrocyte concentration are plotted for horses 12214 and 13854.

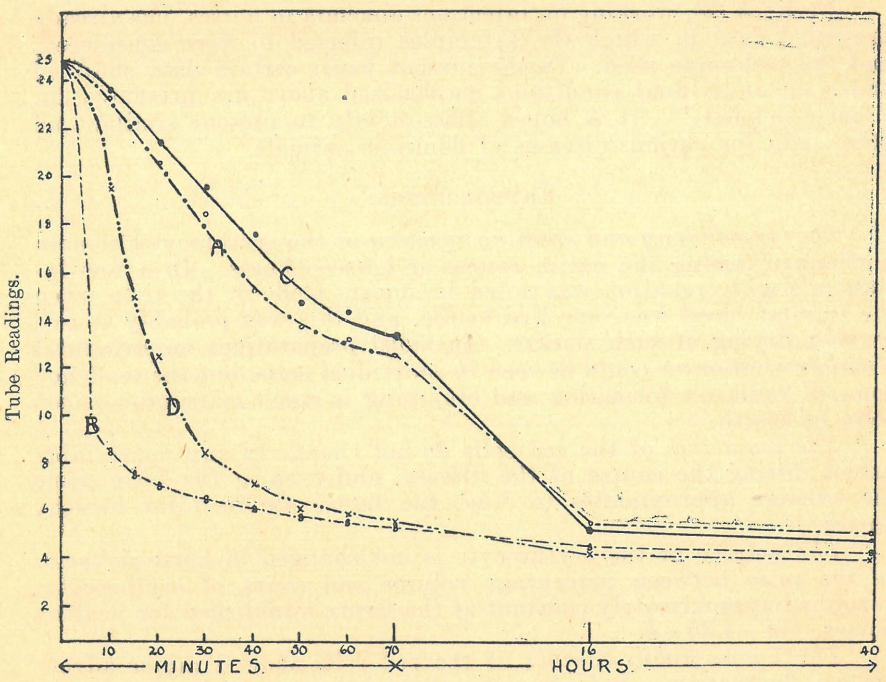
From the data given in graph I it is seen that in this particular animal (No. 12214) the concentration of erythrocytes in jugular blood progressively decreased as the temperature rose, and then again rose as the temperature fell.

In this animal the disease ran a fairly severe course, but was not attended by any complications, nor was oedema formation rapid or severe. The same phenomenon was noted in all cases in which the disease ran a similar course, e.g. in horses 13857, 13859, 13886.

From graph II, representing a clinically different case, it is seen that during the first two weeks there was a tendency for the data to show a relationship as in graph I, but that later on the concentration of erythrocytes in jugular blood increased even to above the figure for health.

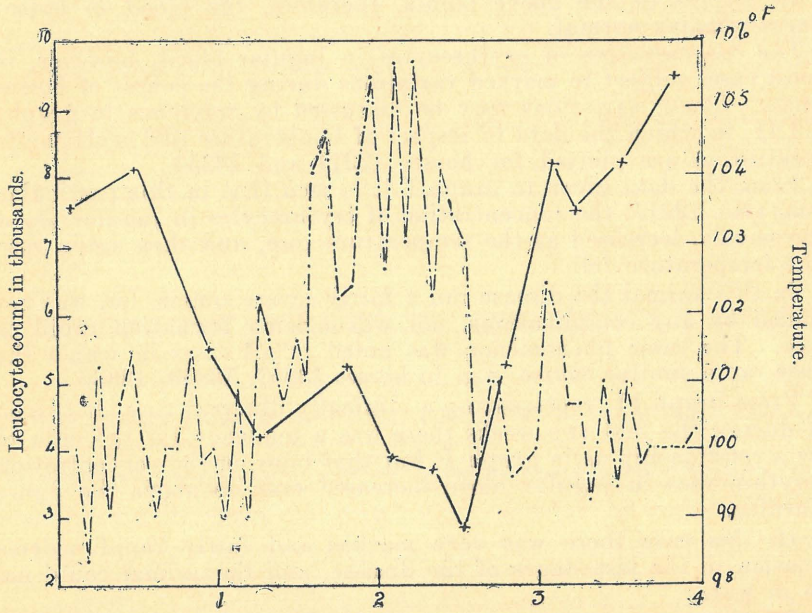
In this case there was very marked and fairly rapid oedema formation in the last stages of the disease, and the animal could not take in water.

In other cases, especially where the disease runs a peracute course (e.g. horse 13913), or a very mild course (e.g. horse 13850), the



Graph III.

Horse No. 12214. August, 1920.



Graph IV.

data do not show the interesting relationship noted above. Indeed, the concentration of erythrocytes in jugular blood may remain more or less constant, or may show irregular fluctuations.

Sedimentation.—The sedimentation of red corpuscles in horse-blood is normally fairly rapid. In certain diseases, as shown by Noltze (3), for infectious anaemia, and subsequently by other workers for other diseases of the horse, the rate of sedimentation is increased. In horse-sickness this is also true; in fact, at the fever acme of the dikkop form, and for some time thereafter, the rate of sedimentation is very rapid indeed. This is well shown in graph III. The data were obtained by collecting blood from a sick and a healthy animal in citrate (oxalate and citrate give approximately the same results). The blood in each case was centrifuged, and plasma and corpuscles separated. The corpuscles were then submitted to three washings in saline, and thereafter mixed with the citrated plasma, as under:—

Plasma from—	*Corpuscles from—*
A. Healthy horse.	Sick horse.
B. Sick horse.	Sick horse.
C. Healthy horse.	Healthy horse.
D. Sick horse.	Healthy horse.

Sedimentation was carried out as described by Noltze (3).

For practical purposes B and C give the rates of sedimentation of the red corpuscles in the sick and healthy animal respectively. In B the sedimentation was approximately 80 per cent., completed in ten minutes, whereas in the same time the sedimentation in C was less than 10 per cent. completed. This rapid rate of sedimentation was due mainly, if not exclusively, to a property acquired by the plasma during the disease; large clumps of corpuscles, plainly visible to the naked eye, are formed, and these fall rapidly.

The Leucocytes.

No abnormal white cells appear in the circulation during the course of horse-sickness. Now and again cells which could not be classified with certainty were seen, but such cells were also seen in smears of healthy horses, and quite as frequently.

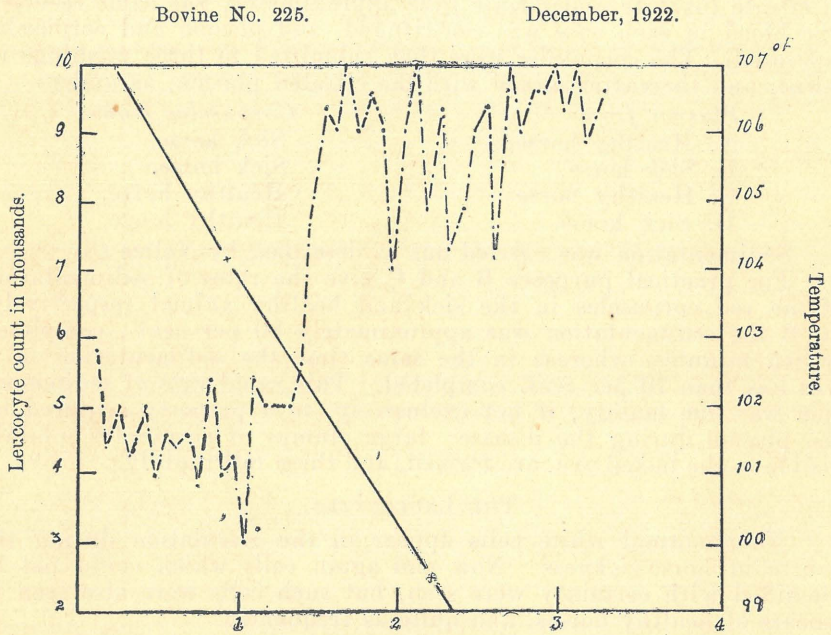
The number of leucocytes in jugular blood varies in an interesting and characteristic manner during the course of the disease. These variations may be discussed by reference to graph IV, in which the data in respect of the temperature and the leucocyte count are given for horse 12214.

The sequence of events may be briefly summarized thus. Following the injection of the virus, the leucocyte count of jugular blood was slightly increased. Soon, however, there was a decrease in the number of leucocytes, and this became progressively more marked as the temperature rose, until the low figure of 3,200 cells per c.mm. of blood was reached. The height of the disease had now been passed, and during the decremental stage the number of leucocytes in the jugular blood rapidly increased, until finally there was again a slight excess over the normal.

These striking changes were noted without exception in every case of horse-sickness. Even in cases where the temperature chart left one very doubtful whether the animal was undergoing a reaction, these changes were observed, e.g. horse 13886. But more remarkable still, the phenomenon was still in evidence, even in cases complicated by wounds, e.g. horse 13963.

To some extent the degree of jugular hypoleucocytosis is proportionate to the severity of the disease. But the data also suggest that all other factors being the same, the jugular leucopenia is more marked if the disease runs a slow course.

It is interesting that in East Coast fever, a tick-borne disease of cattle caused by *Theileria parva*, the leucocytes disappear from the circulation as in horse-sickness; indeed, in some cases to such an extent that it may be very difficult to find any white cells in blood smears. (The data in respect of temperature and jugular leucocyte count in East Coast fever are given for two animals in graphs V and VI.)



Graph V.

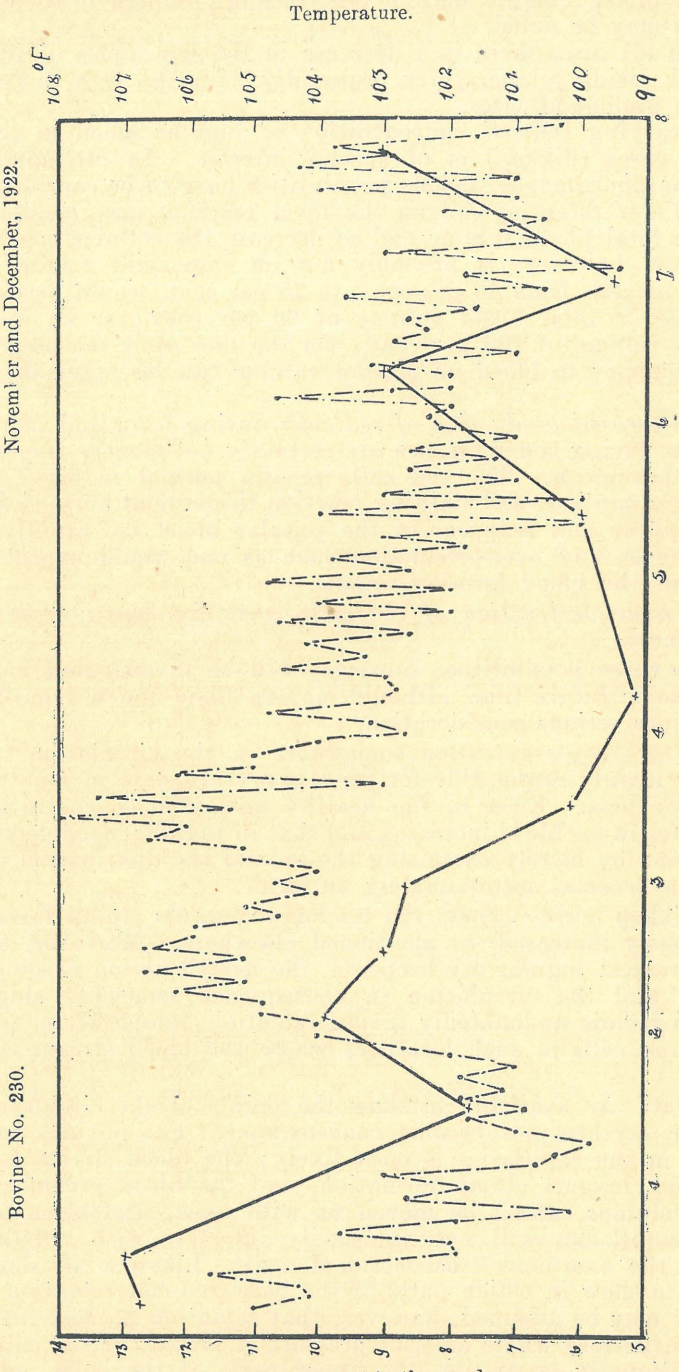
The differential counts recorded show that in the cases which came under observation there is a tendency towards a relative neutrophilia. In view of the fact that most horses get slightly hurt during the course of the disease it is probable that this relative neutrophilia is not due to the horse-sickness virus.

Discussion.

The data here presented are of interest not only in connexion with horse-sickness itself, but also as illustrative of a mechanism which is probably common to all those equine diseases in which haemagglutination becomes marked.

The data may be briefly summarized thus:—

(*a*) In the typical dikkop form of horse-sickness, and in all cases where the disease runs a prolonged course, we note decreased erythrocyte content of jugular blood and increased rate of sedimentation about and after the fever acme; sometimes the erythrocyte content of jugular blood may suddenly increase for reasons indicated below.


Temperature.
°F.
108
107
106
105
104
103
102
101
100
99
Bovine No. 230.
November and December, 1922.
14
13
12
11
10
9
8
7
6
5
Leucocyte count in thousands.
1
2
3
4
5
6
7
8
Weeks under Observation.

Graph VI.

(*b*) In peracute and very mild cases, the red-cell concentration of jugular blood remains more or less constant, though in some cases a decrease may be noted.

(*c*) In all cases there is a decrease in the leucocytes of jugular blood with rising temperatures, especially with the very acute and the severe prolonged cases.

The varying red-cell concentration of jugular blood in the (*a*) group of cases (dikkop) is of special interest. In attempting to explain the phenomena, various possibilities have to be considered.

(1) *Water retention* during the fever reaction may possibly increase the total blood volume and so decrease the cellular concentration. This, however, is probably not an important factor, as a reduction of cells from 40 per cent. to 25 per cent. would necessitate an increase in total blood volume of 60 per cent., or an increase in plasma volume of 100 per cent. On the face of it the possibility of such changes in blood or plasma volume can be reasonably discarded.

(2) *Decreased production of red cells* during fever and increased production during convalescence are certainly not largely responsible for the phenomena. The red cells remain normal in size, shape, haemoglobin content, and staining reaction throughout horse-sickness; they disappear and reappear in the jugular blood too rapidly, and there is never any over-production such as one would expect with stimulus of the blood forming organs.

(3) *Undue destruction of red cells* certainly does not occur in horse-sickness.

These three possibilities, however, will be investigated in more detail at some future time, although no one alone is now regarded as entering into serious consideration.

(4) *Erythrocyte retention* somewhere in the circulation is undoubtedly mainly responsible for jugular erythropenia in some forms of horse-sickness. Even in the healthy horse the concentration of erythrocytes in ear blood increases, and that in jugular blood decreases, during rest; by merely exercising the animal the distribution of red cells again becomes approximately uniform.

In dikkop horse-sickness the tendency towards clump formation is enormously increased, as mentioned elsewhere. Moreover, at the time of greatest jugular erythropenia, the heart's action is somewhat impaired, and the circulation in consequence somewhat sluggish. These two factors undoubtedly favour retention, temporary or permanent, of red cells in such localities where the blood stream is very slow.

No data are available to show the points of the circulation at which the erythrocytes become concentrated. For obvious reasons retention in the capillaries is most likely; the blood stream is very slow, which favours clump formation, and the blood pressure is so low that clumps cannot be moved on with ease. Retention of red cells in capillaries will not necessarily interfere with circulation, owing to the enormous cross-sectional area. Likewise no data are available to show in which parts of the body red-cell retention takes place. It may be assumed, however, that retention is most favoured in those situations where arterial pressure is lowest, or the capillary bed exceptionally large, i.e. the upper parts of the body, and the splanchnic area in particular.

(5) *Oedema formation*, especially if it is extensive and rapid and the horse cannot drink water, results in withdrawal of fluid from the circulation, and thus brings about concentration of the cellular elements. This is undoubtedly the explanation for those cases in which jugular blood suddenly becomes very rich in red cells. Moreover, oedema formation is probably responsible also for the very marked fluctuations in the red-cell content of jugular blood noted in many cases of horse-sickness, even though the animal can still drink water normally.

It is unlikely that the interesting phenomena in respect of erythrocyte content are confined to jugular blood. Owing to mixture of blood from all sources in the heart, arterial and venous blood generally must be influenced in the same way, even if retention of red cells is confined to one part of the body. Expressed differently, in dikkop horse-sickness *the circulating blood* becomes less concentrated in red cells, excepting if oedema formation is rapid and extensive and accompanied by inability to drink water, when it may become more concentrated.

The decreased number of red cells in the circulating blood is not necessarily adverse. In the healthy horse, unused to any work, the erythrocyte content of jugular blood may fall to below 20 per cent. by volume. It thus seems feasible to assume that while the circulating blood contains more than 20 per cent. by volume of red cells, its oxygen carrying capacity for ordinary needs is sufficient.

From the point of view of bulk circulation a decreased erythrocyte content is a positive advantage. The work of the heart is expended mostly in overcoming peripheral resistance, which, to a very large extent, is due to the viscosity of circulating blood (Bayliss). It was therefore considered of interest to determine experimentally the relationship between viscosity and cell volume for horse-blood. The viscosity of citrated blood of an arbitrarily selected horse was found to vary at 23° C. with different concentrations of erythrocytes, according to the accompanying graph VII.

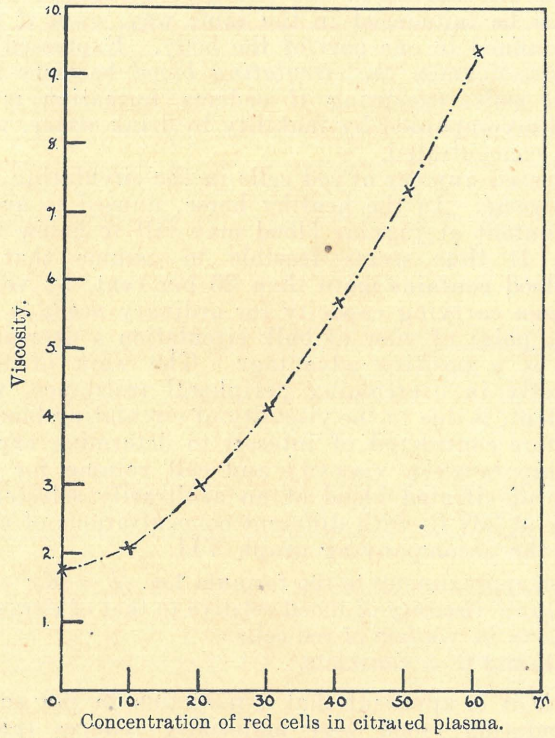
This graph approximates to the formula log. $\eta b = Kx + C$, where
ηb = viscosity of blood relative to that of citrated plasma.
x = % volume of red cells.
K and C = constants.

From this it is apparent that a reduction in the number of red cells in circulating blood very materially reduces the peripheral resistance. This enables the heart to maintain a large bulk circulation with a small expenditure of energy.

Sir Arnold Theiler (4), in recording clinical observations upon horse-sickness, says: —

"The pulse during the onset of the fever reaction is perhaps slightly stronger and fuller than normally, but its rate is not yet increased. Approaching the acme it increases very slightly, it may rise from 40 to 44 or 48, and may remain at this figure during the descending portion of the fever curve. Usually when the acme is passed and the symptoms of dikkop begin to appear the quality of the pulse alters, it becomes softer and weaker and thready, almost imperceptible or wholly so. Then the rate increases to 50 and 60, rarely more. Sometimes the rhythm is also altered; dicrotic or deficient pulses can be registered. The increase in rate and the weakening of the beats stand in some direct relation to the severity

of the swellings present. The heart action also undergoes alterations when the symptoms of dikkop are developing. Generally speaking the area of impulse increases, although the impulse is rarely so pronounced as to shake the wall; in some horses it is distinctly noticeable by hand, in others only faintly or not at all, viz., it may disappear during the course of the disease to appear again when recovery has taken place. The sounds, at the beginning strong and distinct, become during the course frequently weaker and diffused. The respiration during the ascent of the fever shows no changes in frequency or quality and in a great number of cases, that show the dikkop symptoms only mildly, never any interference is noted.



Graph VII.

Frequently, however, the respirations increase in number and become distinctly abdominal, particularly in cases that end fatally. Generally the frequency remains below thirty per minute; the groove along the costal arch becomes well pronounced and the double movement of the abdominal wall is distinct; the costal movement is, however, less prominent; these symptoms pass with the approaching recovery, viz., when the oedematous swellings are disappearing or have disappeared, but they increase with the increase of the swelling, and stand in a certain relation to the impaired heart action."

From this quotation it is seen that the marked decrease in the red-cell content of the circulating blood is noted precisely during that time when cardiac disturbance is in evidence. Expressed differently, the weakened heart can maintain a circulation of sorts

because the peripheral resistance has been decreased through a reduction of the number of erythrocytes in the circulating blood. In many cases, in spite of weakened heart's action, the circulation remains so good that dyspnœa is very slight or absent. Marked dyspœna is only noted in those cases in which the heart's action is much impaired, and it is then due to improper circulation rather than insufficient oxygen carrying capacity of the blood, i.e. a true cardiac dyspnœa.

If during the time that the heart's action is impaired the circulating blood becomes concentrated in red cells, peripheral resistance is increased and cardiac failure is threatened. A sudden increase in the number of erythrocytes in jugular blood must thus be regarded as of adverse prognostic significance.

In consideration of the points mentioned, the decrease in the red-cell content of circulating blood is a very fortunate occurrence in dikkop horse-sickness. While not seriously threatening the oxygen supply to the tissues, the lessened number of red cells in the circulating blood brings about a marked decrease in peripheral resistance and so enables the heart, even though its action be weakened, to maintain a fair circulation. From this point of view the decrease in erythrocyte-content of the circulating blood must be considered in the light of a compensatory mechanism, which enables the weakened heart to carry on a circulation where otherwise it would not be able to do so.

Several workers have recently shown that haemagglutination is marked in many diseases of horses. It is thus probable that the phenomena in connexion with the red cells noted in horse-sickness may also be noted in other equine diseases.

The progressive jugular leucopenia is very characteristic of horse-sickness and may actually be used for diagnostic purposes. What happens to the leucocytes is not known. The fact that they rapidly reappear, sometimes in abnormal numbers, with defervescence, speaks against specific destruction by the virus. It is probable that the leucocytes merely find their way into the tissues during the fever reaction, and that they re-enter the blood stream as soon as the temperature falls. There is certainly no great stimulus to the organs forming the leucocytes, for the hyperleucocytosis, when noted, is never comparable with that seen in bacterial diseases such as broncho-pneumonia, strangles, etc.

The data indicate that a rapid fall in jugular leucocyte count is of adverse prognostic significance, even in cases where the temperature is falling (H. 13836), whereas an increased leucocyte count is always a favourable sign.

Summary.

1. In the dikkop form of horse-sickness—

(*a*) sedimentation of red corpuscles is much accelerated in the later stages of the disease, owing to clumping of the cells. This clumping is due to a property acquired by the plasma;

(*b*) at the time of the fever acme and for some time thereafter, the jugular blood becomes poor in red cells, probably mainly on account of the tendency towards clumping

and the weak action of the heart. These favour erythrocyte retention in the capillary system. It is possible that water retention and decreased formation of red cells also play some subsidiary part in the production of this phenomenon;

(*c*) the decreased erythrocyte content of venous blood decreases peripheral resistance considerably, and allows the heart to maintain a fairly bulky circulation in spite of mechanical interference with its action (hydropericardium) and weakening of the myocardium;

(*d*) the dyspnœa which is noted in some cases is due not so much to the decreased oxygen carrying capacity of the blood, as to the inability of the heart to maintain a sufficiently bulky circulation and so ensure the efficient removal of wastes;

(*e*) if oedema formation is rapid and extensive and if complications such as paralysis of the oesophagus or pharynx persist for a long time, jugular blood may again become rich in red cells. This increases peripheral resistance and favours sudden cardiac failure.

2. In peracute and very mild cases of horse-sickness no marked or constant changes in erythrocyte content of jugular blood are noted—probably because there is no time for the formation of haemagglutinins, or because these are formed only to a very slight extent.

3. In all cases of horse-sickness, leucocytes disappear progressively from the jugular blood during the incremental stages of the disease; the rate at which leucocytes disappear is an index of the severity of the disease, but the extent of disappearance is to some extent also dependent upon the duration of the disease.

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(1) 1923, Neser, "The Blood of Equines," Department of Agriculture, Union of South Africa, April, 1923, Ninth and Tenth Reports of the Director of Veterinary Education and Research.

(2) 1923, De Kock, "A Contribution to the Study of the Virus, Haematology, and Pathology of Infectious Anaemia of Equines under South African Conditions," Department of Agriculture, Union of South Africa, April, 1923, Ninth and Tenth Reports of the Director of Veterinary Education and Research.

(3) 1921, Noltze, "Die Sedimentirungs geschwindigkeit der roten Blutkörperchen bei der infektiösen Anämie der Pferde als Diagnostikum," Monatshefte für praktische Tierheilkunde, Bd. 32, S. 481.

(4) 1921, Theiler, Arnold, "African Horse-sickness," Department of Agriculture, Union of South Africa, Science Bulletin No. 19.

(5) 1920, Bayliss, W. M., "Principles of General Physiology," Longmans & Co., London and New York.

Horse.	Date.	R.P.	R.C.	R.P./R.C.	Hb.	Hb./R.P.	Av.	Temp.	W.C.	L.	M.	N.	E.	B.	?	Remarks.
12214	11. 8.20	39	—	—	61	1·6	—	99·3 100·0	10·2	32	3	59	4	2	—	Injected 20 c.c. Tz. virus into the jugular vein.
	14. 8.20	34	7·6	4·5	60	1·8	5·6	101·4 100·0	11·0	31	3	64	1	1	—	
	15. 8.20	—	—	—	—	—	—	99·8 100·0	—	—	—	—	—	—	—	Injected 5 c.c. O. virus and 400 c.c. H.S. serum into the jugular vein.
	17. 8.20	34	7·6	4·5	57	1·7	5·4	100·0 100·2	7·2	—	—	—	—	—	—	
	19. 8.20	30	7·0	4·3	55	1·8	5·4	99·0 102·4	5·2	38	4	54	2	2	—	
	20. 8.20	27	—	—	50	1·9	—	101·4 100·6	—	—	—	—	—	—	—	
	21. 8.20	27	—	—	—	—	—	101·5 100·0	—	—	—	—	—	—	—	
	23. 8.20	25	—	—	—	—	5·2	104·0 102·6	6·7	28	5	65	1	1	—	Slight hollow.
	24. 8.20	27	6·5	4·2	55	2·0	5·1	104·0 105·5	—	—	—	—	—	—	—	
	25. 8.20	25	—	—	—	—	—	102·8 105·6	4·7	27	4	67	0	2	—	Flat.
	26. 8.20	25	—	—	50	2·0	—	103·2 105·6	—	—	—	—	—	—	—	
	27. 8.20	25	5·8	4·3	50	2·0	—	102·2 104·2	4·4	25	2	71	1	1	—	Slight D.K.
	28. 8.20	27	—	—	52	1·9	—	103·4 103·2	3·2	23	1	76	0	0	—	Slight D.K.
	30. 8.20	26	5·8	4·5	50	2·0	—	101·0 99·8	6·5	22	1	75	1	1	—	
	31. 8.20	27	—	—	52	2·0	—	99·8 100·2	8·0	23	1	75	1	0	—	
	1. 9.20	30	7·0	4·3	—	—	—	102·4 100·6	11·0	26	1	72	1	0	—	
	2. 9.20	—	—	—	—	—	—	100·6 100·6	11·0	25	2	71	0	2	—	
	4. 9.20	32	—	—	—	—	—	—	11·0	27	2	68	2	1	—	
	6. 9.20	—	—	—	—	—	—	—	13·0	25	4	65	3	2	1	Discharged: recovered.
13854	30. 9.20	—	—	—	—	—	—	—	—	—	—	—	—	—	—	Injected 25 c.c. Tz. virus into the jugular vein.
	1.10.20	30	7·0	4·3	59	2·0	5·4	100·0 100·6	13·4	34	5	54	6	1	—	
	2.10.20	32	7·1	4·5	59	1·8	5·4	100·0 100·0	10·2	38	7	52	3	—	—	

Horse.	Date.	R.P.	R.C.	R.P./R.C.	Hb.	Hb./R.P.	Av.	Temp.	W.C.	L.	M.	N.	E.	B.	?	Remarks.
13854—*contd.*	5.10.20	29	6·9	4·2	55	1·9	5·4	102·6 102·0	10·0	32	6	57	5	—	—	Injected 5 c.c. O. virus and 360 c.c. H.S. serum into the jugular vein.
	7.10.20	33	7·2	4·6	55	1·7	—	102·2 101·6	9·0	30	5	60	4	1	—	Injected 270 c.c. serum and 5 c.c. Relapse virus into the jugular.
	11.10.20	28	6·4	4·4	48	1·7	—	104·6 102·8	7·0	25	6	62	5	2	—	Hollow.
	13.10.20	26	6·0	4·3	—	—	—	105·6 103·0	6·5	29	4	64	2	1	—	
	15.10.20	30	—	—	—	—	—	106·0 101·5	6·0	30	3	66	1	—	—	Slight hollow.
	18.10.20	36	8·0	4·5	—	—	—	102·2	5·0	12	3	85	—	—	—	D.K. Throat and neck swollen.
	20.10.20	—	—	—	—	—	—	—	—	—	—	—	—	—	—	Animal died.
13857																Gelding, 6 years.
	7.10.20	—	—	—	—	—	—	99·0 101·6	—	—	—	—	—	—	—	50 c.c. Tz. virus injected into the jugular vein.
	11.10.20	30	7·1	4·2	55	1·8	—	100·0 100·2	—	—	—	—	—	—	—	
	12.10.20	—	—	—	—	—	—	99·2 101·0	—	—	—	—	—	—	—	5 c.c. O. virus and 360 H.S. serum injected into the jugular vein.
	13.10.20	30	7·2	4·2	54	1·8	—	100·0 102·0	12·7	26	4	64	5	1	—	
	14.10.20	30	7·0	4·3	—	—	—	100·4 102·2	15·0	18	4	73	5	—	—	270 c.c. H.S. serum injected into the jugular vein.
	15.10.20	27	6·0	4·5	43	1·6	—	101·0 105·4	14·6	—	—	—	—	—	—	
	16.10.20	—	—	—	—	—	—	100·2 104·6	—	—	—	—	—	—	—	Slight D.K. swelling head, neck, and sternum.
	18.10.20	20	4·6	4·3	33	1·6	—	101·6 104·0	—	—	—	—	—	—	—	Slight hollow and swelling head, neck, and sternum.
	20.10.20	21	5·1	4·1	35	1·7	—	103·6 105·0	11·5	16	3	74	5	1	1	Slight hollow and swelling head, neck, and sternum.
	22.10.20	22	5·4	4·1	37	1·7	—	103·4 104·0	7·5	19	2	69	8	1	1	
	25.10.20	24	5·5	4·4	40	1·7	—	102·0 104·4	8·3	—	—	—	—	—	—	Slight hollow and swelling head, neck, and sternum.
	27.10.20	25	—	—	—	—	—	100·0 102·4	8·6	15	3	71	11	—	—	Hollow swellings more or less disappeared.
	29.10.20	—	—	—	—	—	—	Normal	13·4	23	3	66	4	2	2	Small abscess on neck.
	24.11.20	30	7·0	4·3	46	1·5	—	Normal	13·2	41	3	43	11	2	—	Discharged: recovered.

Horse.	Date.	R.P.	R.C.	R.P./R.C.	Hb.	Hb./R.P.	Av.	Temp.	W.C.	L.	M.	N.	E.	B.	?	Remarks.
13859																Gelding, 8 years.
	7.10.20	—	—	—	—	—	—	99·0	—	—	—	—	—	—	—	50 c.c. Tz. virus injected into the jugular vein.
								100·4								
	8.10.20	30	6·9	4·3	55	1·8	5·6	100·0	—	—	—	—	—	—	—	
								101·0								
	11.10.20	30	7·0	4·3	54	1·8	—	98·4	—	—	—	—	—	—	—	
								101·6								
	12.10.20	—	—	—	—	—	—	100·6	—	—	—	—	—	—	—	5 c.c. O. virus and 360 c.c. H.S. serum injected into the jugular vein.
								101·8								
	13.10.20	28	6·4	4·4	44	1·6	—	99·6	8·9	39	4	52	4	1	—	
								101·6								
	14.10.20	28	6·4	4·4	45	1·6	—	100·0	7·9	—	—	—	—	—	—	270 c.c. H.S. serum injected into the jugular vein
								102·8								
	15.10.20	28	6·4	4·4	—	—	—	100·0	—	—	—	—	—	—	—	
								103·2								
	18.10.20	28	—	—	—	—	—	101·0	—	—	—	—	—	—	—	
								104·0								
	20.10.20	24	5·7	4·2	38	1·6	—	100·6	5·9	35	4	56	3	2	—	
								104·0								
	22.10.20	27	6·7	4·0	44	1·6	—	100·0	6·9	39	4	41	5	1	—	
								101·6								
	25.10.20	24	5·7	4·4	39	1·6	—	98·6	7·3	—	—	—	—	—	—	Slight swellings.
								100·0								
	27.10.20	26	—	—	44	1·7	—	98·6	6·7	30	6	54	6	3	1	
								100·6								
	29.10.20	29	—	—	—	—	—	99·0	9·5	29	7	61	2	1	—	Swelling disappeared.
								100·0								
	1.11.20	28	—	—	—	—	—	98·6	7·6	—	—	—	—	—	—	
								101·6								
	3.11.20	25	—	—	—	—	—	98·0	6·9	34	4	60	2	1	—	
								101·0								
	8.11.20	29	6·5	4·5	48	1·7	—	Normal	9·5	41	4	50	4	1	—	
	19.11.20	—	—	—	—	—	—	—	10·5	—	—	—	—	—	—	Discharged : recovered.
13836																Mare, 11 years, heavy in foal.
	14.10.20	34	7·6	4·5	53	1·6	—	99·8	17·5	19	4	70	5	1	1	Injected 50 c.c. Tz. virus into the jugular vein.
								101·4								
	15.10.20	35	7·8	4·5	57	1·6	—	101·0	21·0	21	3	73	2	1	—	
								102·0								
	18.10.20	30	7·0	4·3	55	1·8	—	101·6	13·6	24	4	68	3	1	—	
								104·4								
	19.10.20	—	—	—	—	—	—	—	—	—	—	—	—	—	—	5 c.c. O. virus and 360 c.c. H.S. serum injected into the jugular vein.

Horse.	Date.	R.P.	R.C.	R.P./R.C.	Hb.	Hb./R.P.	Av.	Temp.	W.C.	L.	M.	N.	E.	B.	?	Remarks.
13836 *Contd.*	20.10.20	29	6·7	4·3	53	1·8	—	101·0 104·6	11·2	26	3	69	1	1	—	
	22.10.20	26	6·1	4·3	44	1·7	—	101·4 105·4	9·4	29	3	64	4	—	—	
	25.10.20	25	5·5	4·5	46	1·8	—	106·0 104·0	9·2	30	6	59	4	1	—	
	27.10.20	29	7·0	4·2	—	—	—	101·2 105·6	8·5	29	4	63	2	1	1	Mare foaled during day.
	29.10.20	23	5·0	4·6	40	1·7	—	102·0 104·0	6·3	19	2	76	1	1	1	
	1.11.20	37	8·4	4·4	64	1·7	—	102·2 103·4	4·9	17	1	82	—	—	—	Animal died during night of 1st.
13886	29.10.20	47	10·8	4·4	66	1·4	—	100·2	10·5	34	3	60	2	1	—	Gelding, 6 years.
	30.10.20	35*	8·0	4·4	60	1·7	—	99·0 100·2	10·1	31	4	60	4	1	—	25 c.c. Tz. virus injected into the jugular vein.
	1.11.20	36	8·1	4·4	60	1·7	—	99·6 100·4	10·0	30	3	61	5	1	—	
	3.11.20	32	—	—	—	—	—	100·0 101·0	9·1	33	5	58	3	1	—	
	4.11.20	—	—	—	—	—	—	—	—	—	—	—	—	—	—	5 c.c. O. virus and 360 c.c. H.S. serum injected intravenously.
	5.11.20	32	7·2	4·4	50	1·6	—	103·0 101·4	8·2	25	5	69	1	—	—	
	6.11.20	27	—	—	—	—	—	100·0 102·0	7·5	29	4	67	—	—	—	270 c.c. H.S. serum injected intravenously.
	8.11.20	31	—	—	55	1·8	—	101·8 101·0	7·6	26	5	62	6	1	—	
	10.11.20	35	8·1	4·3	59	1·7	—	99·2 100·2	8·5	38	3	51	6	2	—	
	12.11.20	36	—	—	—	—	—	99·2 100·0	11·5	28	3	62	5	2	—	
	17.11.20	34	7·9	4·3	57	1·7	—	99·0 100·0	12·2	46	2	46	5	1	—	
	24.11.20	41	9·3	4·4	62	1·5	—	99·6 100·4	11·2	38	4	51	3	3	1	
	26.11.20	43	9·6	4·5	66	1·5	—	100·0 100·6	8·8	37	2	57	2	2	—	
	30.11.20	38	—	—	—	—	—	100·0 102·4	15·9	28	3	68	1	—	—	
	2.12.20	43	—	—	—	—	—	98·4 100·4	9·0	46	2	46	4	2	—	Discharged.

* Animal bled shortly after a heavy drink.

Horse.	Date.	R.P.	R.C.	R.P./R.C.	Hb.	Hb./R.P.	Av.	Temp.	W.C.	L.	M.	N.	E.	B.	?	Remarks.
13912	28.12.20	42	9·9	4·2	74	1·8	—	98·0 100·6	7·5	—	—	—	—	—	—	25 c.c. Tz. virus injected into the jugular vein.
	4. 1.21	34	7·7	4·4	64	1·9	—	99·6 101·2	7·2	42	2	51	3	2	—	
	6. 1.21	32	7·2	4·4	62	1·9	—	100·4 103·2	7·1	37	3	56	3	1	—	
	7. 1.21	32	7·2	4·4	60	1·9	—	101·4 104·4	5·7	44	1	50	5	—	—	
	9. 1.21	34	7·7	4·4	68	2·0	—	99·0 102·6	5·3	33	2	62	2	1	—	Marked D.K. After the blood was taken the animal was bled 10 litres.
	10. 1.21	—	—	—	—	—	—	—	—	—	—	—	—	—	—	Animal was bled 6 litres.
	12. 1.21	25	6·0	4·2	38	1·5	—	101·8 103·6	9·4	31	3	63	3	—	—	
	14. 1.21	21	—	—	—	—	—	100·0 101·6	11·9	30	8	57	4	—	1	
	17. 1.21	23	—	—	36	1·6	—	98·4 100·8	9·8	22	3	71	3	1	—	
	24. 1.21	37	8·4	4·4	60	1·6	—	99·0 100·2	13·0	20	5	75	—	—	—	
	26. 1.21	—	—	—	—	—	—	—	10·6	26	2	69	2	1	—	
	7. 2.21	29	—	—	—	—	—	—	7·7	33	2	57	7	1	—	Discharged: recovered.
13913																Gelding, 8 years.
	3. 1.21	30	7·6	4·0	55	1·8	—	98·8 101·2	6·6	42	2	50	5	1	—	5 c.c. O. virus injected into the jugular vein.
	5. 1.21	30	7·5	4·0	55	1·8	—	99·0 99·8	9·1	37	4	54	4	—	1	
	6. 1.21	29	—	—	—	—	—	102·0 104·0	6·9	21	2	74	3	—	—	
	7. 1.21	29	7·0	4·2	55	1·9	—	103·0 105·0	5·7	25	5	66	3	1	—	
	8. 1.21	30	7·0	4·3	55	1·8	—	103·6 103·6	4·1	23	—	76	—	1	—	
	9. 1.21	31	—	—	—	—	—	103·0 104·2	3·0	18	—	82	—	—	—	Died.
13914	3. 1.21	37	8·8	4·2	70	1·9	—	99·6 101·6	10·7	36	2	53	7	2	—	5 c.c. O. virus injected into the jugular vein.
	5. 1.21	35	8·0	4·4	68	1·9	—	98·6 99·2	9·7	42	4	45	8	—	1	

Horse.	Date.	R.P.	R.C.	R.P./R.C.	Hb.	Hb./R.P.	Av.	Temp.	W.C.	L.	M.	N.	E.	B.	?	Remarks.
13914 *contd.*	6. 1.21	32	7·3	4·4	60	1·9	—	100·8 105·0	9·1	36	1	55	8	—	—	
	7. 1.21	30	7·0	4·3	56	1·9	—	104·6 105·8	7·5	28	1	69	1	—	1	
	8. 1.21	31	7·1	4·4	55	1·8	—	104·6 105·6	5·5	16	—	83	—	—	1	After blood was taken the animal was bled 10 litres.
	9. 1.21	—	—	—	—	—	—	105·0	—	—	—	—	—	—	—	Died.
13963	13. 1.21	—	—	—	—	—	—	—	—	—	—	—	—	—	—	15 c.c. Tz. virus injected into the jugular vein.
	14. 1.21	31	7·6	4·1	55	1·8	—	—	12·8	36	2	58	3	1	—	
	17. 1.21	31	—	—	48	1·6	—	—	11·9	30	3	56	10	1	—	
	19. 1.21	34	—	—	55	1·6	—	—	11·2	26	2	59	12	1	—	5 c.c. O. virus and 270 c.c. H.S. serum injected into the jugular vein.
	21. 1.21	35	—	—	59	1·7	—	—	9·1	29	3	58	—	1	—	Animal badly kicked. 360 c.c. H.S. serum into the jugular vein.
	24. 1.21	32	—	—	56	1·7	—	—	11·4	40	2	49	8	1	—	
	26. 1.21	35	—	—	—	—	—	—	8·7	40	4	46	9	1	—	Badly swollen where kicked.
	28. 1.21	31	—	—	52	1·7	—	—	9·5	23	3	71	1	2	—	
	31. 1.21	33	—	—	—	—	—	—	7·0	28	3	66	1	2	—	
	2. 2.21	36	—	—	—	—	—	—	7·4	21	2	76	1	—	—	Swelling burst.
	4. 2.21	34	—	—	—	—	—	—	8·0	31	5	60	3	1	—	
	7. 2.21	31	—	—	55	1·8	—	—	20·0	31	3	64	2	—	—	
	9. 2.21	29	—	—	—	—	—	—	23·0	24	4	67	1	1	3	
	11. 2.21	31	7·4	4·2	—	—	—	—	16·0	25	3	64	4	1	3	
	14. 2.21	31	—	—	—	—	—	—	16·0	30	5	58	6	1	—	Discharged.
13850	30. 9.20	—	—	—	—	—	—	—	—	—	—	—	—	—	—	Injected 25 c.c. Tz. virus subcutaneously.
	1.10.20	27	6·4	4·2	55	2·0	5·5	100·2	12·5	35	5	56	3	1	—	
	2.10.20	30	6·6	4·5	58	1·9	—	100·0 100·4	—	—	—	—	—	—	—	
	5.10.20	30	6·6	4·5	57	1·9	—	100·0 102·4	9·0	35	7	55	3	—	—	360 c.c. H.S. serum and 5 c.c. O. virus injected into the jugular vein.
	7.10.20	26	6·1	4·3	50	2·0	—	99·6 102·2	8·6	—	—	—	—	—	—	270 c.c. H.S. serum injected into the jugular vein.
	11.10.20	25	6·0	4·2	45	1·8	—	99·0 100·6	8·0	—	—	—	—	—	—	
	13.10.20	27	—	—	—	—	—	99·6 102·6	7·8	—	—	—	—	—	—	
	15.10.20	29	—	—	—	—	—	100·0 101·0	7·0	—	—	—	—	—	—	
	18.10.20	26	—	—	—	—	—	100·0 104·4	6·8	—	—	—	—	—	—	

Horse.	Date.	R.P.	R.C.	R.P./R.C.	Hb.	Hb./R.P.	Av.	Temp.	W.C.	L.	M.	N.	E.	B.	?	Remarks.
13850—*contd.*	20.10.20	25	—	—	—	—	—	101·4 104·2	5·8	40	4	53	2	1	—	
	22.10.20	26	6·2	4·2	—	—	—	100·4 101·2	6·3	—	—	—	—	—	—	
	25.10.20	26	—	—	—	—	—	99·8 100·6	8·9	—	—	—	—	—	—	
	27.10.20	28	—	—	—	—	—	99·6 100·2	9·8	27	3	65	4	1	—	
	1.11.20	27	—	—	—	—	—	—	13·0	31	7	60	2	—	—	Discharged.
15226	9. 6.22	37	8·6	4·3	—	—	—	—	9·7	—	—	—	—	—	—	**Injected 20 c.c. Tz. virus.**
	12. 6.22	38	8·6	4·4	—	—	—	99·8 103·8	15·2	—	—	—	—	—	—	
	16. 6.22	30	7·0	4·3	—	—	—	101·4 101·6	14·6	—	—	—	—	—	—	Injected 5 c.c. O. virus and 200 c.c. serum.
	17. 6.22	30	7·0	4·3	—	—	—	101·4 103·0	15·7	—	—	—	—	—	—	
	19. 6.22	32	7·0	4·4	—	—	—	101·2 104·0	17·0	—	—	—	—	—	—	Injected 300 c.c. serum.
	20. 6.22	31	7·2	4·3	—	—	—	102·0 104·0	15·8	—	—	—	—	—	—	
	21. 6.22	28	6·5	4·3	—	—	—	102·0 104·0	19·7	—	—	—	—	—	—	
	26. 6.22	28	6·5	4·3	—	—	—	104·0 106·0	12·2	—	—	—	—	—	—	
	28. 6.22	24	5·0	4·8	—	—	—	103·0 104·0	9·8	—	—	—	—	—	—	
	30. 6.22	31	7·2	4·3	—	—	—	101·0 102·0	9·6	—	—	—	—	—	—	
	7. 7.22	33	7·6	4·3	—	—	—	100·0	16·1	—	—	—	—	—	—	Discharged: recovered. **Very mild reactor.**
15232	9. 6.22	30	7·0	4·3	—	—	—	—	10·4	—	—	—	—	—	—	Injected 20 c.c. Tz. virus.
	12. 6.22	30	7·0	4·3	—	—	—	98·8 99·8	10·6	—	—	—	—	—	—	
	16. 6.22	29	6·7	4·3	—	—	—	101·0 101·8	8·5	—	—	—	—	—	—	Injected 5 c.c. O. virus and 200 c.c. serum.
	17. 6.22	27	6·3	4·3	—	—	—	99·6 102·8	9·6	—	—	—	—	—	—	
	19. 6.22	27	6·3	4·3	—	—	—	100·6 103·0	7·9	—	—	—	—	—	—	Injected 300 c.c. serum.
	20. 6.22	25	—	—	—	—	—	101·0 104·6	6·5	—	—	—	—	—	—	

Horse.	Date.	R.P.	R.C.	R.P./R.C.	Hb.	Hb./R.P.	Av.	Temp.	W.C.	L.	M.	N.	E.	B.	?	Remarks.
15232—*contd.*	21. 6.22	23	5·3	4·3	—	—	—	102·4 104·0	7·4	—	—	—	—	—	—	
	25. 6.22	28	6·5	4·3	—	—	—	100·0 102·0	9·1	—	—	—	—	—	—	Dikkop.
	28. 6.22	24	5·6	4·3	—	—	—	103·6 103·8	10·5	—	—	—	—	—	—	Dikkop disappeared; both fore legs swollen and animal very lame.
	30. 6.22	26	6·0	4·3	—	—	—	101·0 101·2	16·9	—	—	—	—	—	—	Discharged. Mild reactor.
15234	9. 6.22	30	7·0	4·3	—	—	—	—	11·2	—	—	—	—	—	—	Injected 20 c.c. Tz. virus.
	12. 6.22	27	6·3	4·3	—	—	—	98·4 100·0	13·0	—	—	—	—	—	—	
	16. 6.22	25	6·0	4·3	—	—	—	102·0 102·0	9·8	—	—	—	—	—	—	Injected 5 c.c. O. virus and 200 c.c. H.S. serum.
	17. 6.22	26	6·0	4·3	—	—	—	101·2 104·0	8·5	—	—	—	—	—	—	
	19. 6.22	26	6·0	4·3	—	—	—	101·0 103·0	9·3	—	—	—	—	—	—	Injected 300 c.c. H.S. serum.
	20. 6.22	26	6·0	4·3	—	—	—	101·2 101·0	10·7	—	—	—	—	—	—	
	21. 6.22	24	5·6	4·3	—	—	—	99·4 101·0	10·5	—	—	—	—	—	—	
	27. 6.22	29	6·7	4·3	—	—	—	99·4 100·8	9·1	—	—	—	—	—	—	
	30. 6.22	30	7·0	4·3	—	—	—	101·0 103·0	8·5	—	—	—	—	—	—	
	7. 7.22	25	6·0	4·3	—	—	—	99·4	12·3	—	—	—	—	—	—	Discharged. Very mild. Double reaction at ± 17·6 and 30·6.
15271	9. 6.22	27	6·3	4·3	—	—	—	—	12·5	—	—	—	—	—	—	Injected 20 c.c. Tz. virus.
	12. 6.22	30	7·0	4·3	—	—	—	—	13·0	—	—	—	—	—	—	
	16. 6.22	31	7·2	4·3	—	—	—	—	9·0	—	—	—	—	—	—	Injected 5 c.c. O. virus and 270 c.c. serum.
	17. 6.22	32	7·4	4·3	—	—	—	—	8·5	—	—	—	—	—	—	
	19. 6.22	28	6·5	4·3	—	—	—	—	10·2	—	—	—	—	—	—	Injected 360 c.c. serum.
	20. 6.22	29	6·7	4·3	—	—	—	—	10·7	—	—	—	—	—	—	
	21. 6.22	29	6·7	4·3	—	—	—	—	11·7	—	—	—	—	—	—	
	27. 6.22	27	6·3	4·3	—	—	—	—	9·0	—	—	—	—	—	—	
	30. 6.22	27	6·3	4·3	—	—	—	—	8·9	—	—	—	—	—	—	
	7. 7.22	32	7·4	4·3	—	—	—	—	11·9	—	—	—	—	—	—	Discharged. Mild. Double reaction at ± 17·6 and 29·6.

Horse.	Date.	R.P.	R.C.	R.P./R.C.	Hb.	Hb./R.P.	Av.	Temp.	W.C.	L.	M.	N.	E.	B.	?	Remarks.
15273	9. 6.22	42	9·7	4·3	—	—	—	—	11·6	—	—	—	—	—	—	Injected 20 c.c. Tz. virus.
	12. 6.22	41	9·5	4·3	—	—	—	99·6 100·2	11·1	—	—	—	—	—	—	
	16. 6.22	38	9·0	4·2	—	—	—	100·0 101·4	10·8	—	—	—	—	—	—	Injected 5 c.c. O. virus and 200 c.c. serum.
	17. 6.22	38	9·0	4·2	—	—	—	100·2 103·4	8·5	—	—	—	—	—	—	
	19. 6.22	32	7·4	4·3	—	—	—	100·0 103·6	9·7	—	—	—	—	—	—	Injected 360 c.c. serum.
	20. 6.22	25	6·0	4·2	—	—	—	101·0 102·0	8·5	—	—	—	—	—	—	
	21. 6.22	33	7·6	4·4	—	—	—	100·0 102·0	10·8	—	—	—	—	—	—	
	27. 6.22	37	8·6	4·3	—	—	—	100·0 100·4	13·7	—	—	—	—	—	—	
	30. 6.22	43	10·0	4·3	—	—	—	100·0 99·4	12·9	—	—	—	—	—	—	
	7. 7.22	42	9·7	4·3	—	—	—	99·0	14·1	—	—	—	—	—	—	Discharged. Very mild. Single reaction ± 15·6 and 20·6.