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SWEATING SICKNESS: THE PRESENT STATE OF OUR KNOWLEDGE

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DEFINITION

Sweating sickness is a peracute, acute, subacute or mild tick-borne toxicosis of cattle, particularly young calves, transmitted by some strains of *Hyalomma transiens* Schulze (Neitz, 1953, 1954, 1955, 1956). It is characterized by pyrexia, anorexia, hyperaemia and hyperaesthesia of the skin and visible mucous membranes, salivation, lachrymation, serous or crupous rhinitis, epistaxis, localized or generalized profuse moist eczema, diarrhoea, and diphtheroid stomatitis, pharyngitis, laryngitis, oesophagitis, vaginitis or posthitis. Recovered animals develop a durable immunity.

SYNONYMS

The popular term " sweating sickness " owes its origin to the most noticeable symptom of the disease incorrectly interpreted as a " profuse perspiration " over parts of the skin. This symptom is in reality a " profuse moist eczema ".

Sweetsiekte, Nat kalwersiekte (Wet calf disease), Vuursiekte (Fire disease) (Afrikaans); Schwitzkrankheit (German); La dyhydrose tropicale (French); Foma (Swazi); Ol masheri (Masai of Kenya).

HISTORY

The history of sweating sickness is a fascinating one. This disease was known to European and African stockowners long before it was described by Bevan (1920) and Sinclair (1920) from Southern Rhodesia, and by Du Toit (1923) from Swaziland and the Union of South Africa.

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Little is known about its origin but Du Toit (*loc. cit.*) and Lawrence (1946) suggested that it might have been introduced into Southern Africa from the north. The present knowledge on the mode of transmission, and the feeding habits of the vector, the stripe-legged tick *Hyalomma transiens*, make this suggestion plausible. Infective ticks could have been disseminated over vast areas by mammals and birds. This becomes clear if one considers the fact that the immature stages of the two host *Hyalomma* spp. have often been encountered on migratory birds. Schulze (1930) recorded the introduction of *Hyalomma* nymphae into Scandinavia from Egypt, while Enigk (1944) observed annual introductions of such ticks into Germany from subtropical regions. It is thus conceivable that migratory birds could have brought the immature stages of infective *H. transiens* into Southern Africa.

There has been a great deal of speculation in the past about the mode of transmission of sweating sickness. The fact that this disease cannot be communicated to susceptible animals by means of blood inoculations (Bevan, 1940; Du Toit, 1923) caused the latter investigator to suggest that transmission is probably effected by a blood-sucker.

Many European and African stockowners incriminated the bontpoot or stripe-legged ticks (*Hyalomma rufipes rufipes* Koch, *H. rufipes glabrum* Delpy and *H. transiens* Schulze) as likely vectors. Bevan (1920) recorded the observations of a farmer which suggested that ticks could have played a rôle in the transmission of sweating sickness. This farmer stated that "it is significant that one large mob (of cattle) running on burned veld, though in the midst of the area furnishing the bulk of the cases, had not one case." Lewis (1934) gave an interesting account submitted to him by the Masai of Kenya. He recorded that "ol masheri is a native name, not only of a tick but also for a disease which has been known to the Masai for many years. The natives state that the disease is tick-borne, hence its native name. It is said to occur in calves ranging from three to six months, and is more common during the rainy season. Some cases of this disease were seen in October, 1931, on a European farm bordering the Masai Reserve. An affected calf showed drooping of the ears. Embedded in a mass of purulent matter in the ear, and attached to the ears, were ticks of the species *Hyalomma aegyptium impressum*. The skin around the ear and on the neck was inflamed, and the inflammation showed a tendency to spread. The skin, in parts, was thickened and hardened, which condition was preceded by the appearance of vesicles containing a clear fluid. Later 'scabbiness' appeared and the hair fell off in tufts. The farmer stated that the temperature reached 108° F, and a sweating appeared in the mornings. Whether this disease bears any relation to sweating sickness of calves in Rhodesia and South Africa has not been determined." Later in his report he stated that "this disease has now been diagnosed by the Chief Veterinary Research Officer as sweating sickness."

Further observations in support of sweating sickness being a tick-borne disease have been recorded from Southern and Northern Rhodesia. Hooper Sharpe (1935, 1936) mentioned that "short interval dipping appears to be the best preventive for this disease in calves." Le Roux (1943) noticed *Hyalomma aegyptium* to be very prevalent in areas of infection during the rainy season.

The hypothesis that sweating sickness is a tickborne disease was not generally accepted. Clark (1933) failed to find any evidence in support of this theory. He mentioned that "introduction of a five day dipping and hand-dressing in the Hluhluwe Settlement (Zululand) has tended to discredit this idea as no falling off in the number of cases has been noted since its inception." Lawrence (1946) gave an account of this disease in two animals kept in a stable which he believed to be free from blood-sucking arthropods.

The position remained obscure until Neitz (1953, 1954, 1955*a*) found that the aetiological agent could be transmitted to susceptible calves by adult *H. transiens* ticks, the progeny of a single female obtained from a sick calf in the Mhlosinga area in Zululand.

The cause of sweating sickness also led to a great deal of controversy. Bevan (1920) deduced from the lesions at autopsy that it might be a toxaemia. Although Du Toit (1923) failed to transmit the disease artificially he nevertheless believed that an infectious agent may be the cause. This view was shared by Neitz (1954) and R. du Toit (1955). Alexander (1955) expressed a more guarded opinion in that he thought that "in the case of lumpy skin disease, sweating sickness and possibly tick paralysis, there has been unearthed a group of disease-producing entities which lie between the toxins and viruses, just as the rickettsias lie between the viruses and the bacteria." Daubney (1936) on the other hand advanced another theory, and suggested that sweating sickness is a deficiency disease such as pellagra. Lawrence (1946) stated that "there are certain facts that point to its being an error of metabolism (which may be loosely put as a dietetic disturbance or digestive derangement) or possibly a vitamin deficiency."

To this review on the various theories advanced on the aetiology of sweating sickness, it is of interest to quote a remark made by Du Toit (1923). He stated that "it would be futile at this stage of the investigations to speculate about the possible mode of transmission of sweating sickness."

As long as the aetiology remains obscure there is little hope of solving the transmission problem. History, however, relates that the reverse took place. The mode of transmission was established first, and this led to the determination that the causal agent is a toxin (Neitz, 1956). This observation also gives an explanation why the artificial transmission from affected to healthy calves by means of blood and organ emulsions failed.

Distribution

Sweating sickness is widely distributed in Central, Eastern and Southern Africa. Records on its distribution will be found in the annual reports of the veterinary departments of the various African territories mentioned in the bibliography (*vide infra*). The occurrence of this disease in Southern India and Ceylon is based on the clinical observations made 25 years ago by Crawford (1956).

Countries from which the occurrence has been recorded are listed in the appended Table 1.

TABLE 1

Country	References
AFRICA—	
Angola	Gerlach, 1956.
Bechuanaland Protectorate	Bechuanaland Annual Reports, 1934, 1935.
Belgian Congo	Leplae and Tobback, 1933.
Kenya	Walker, 1932; Daubney, 1931, 1936, 1943. Lewis, 1934.
Moçambique	de Abreu, 1954.
Northern Rhodesia	Turnbull, 1929; Smith, 1931; Purchase, 1936; le Roux, 1936; 1943; Morris, 1937–1941; Fern, 1943.
Nyasaland	Matson, 1958.
Southern Rhodesia	Bevan, 1920, 1931; Sinclair, 1921–1933; Hooper Sharpe, 1930–1936; Lawrence, 1939–1946.
South West Africa	Report of the Union of South Africa to League of Nations, 1935, 1936, 1937; Von Ostertag and Kulenkampff, 1941.
Swaziland	Du Toit, 1923; Annual Reports, 1931–1936; Elder, 1939; Nilsen, 1940–1941; Faulkner, 1942–1943; Barnard, 1942–1952; van Heerden, 1953.
Tanganyika	*
Union of South Africa—	
Transvaal	Du Toit, 1923.
Natal	Du Toit, 1923; Clark, 1933.
Western Cape Province	Bisschop, 1940.
Eastern Cape Province	Diesel, 1949.
Orange Free State	Diesel, 1949.
Uganda	*
ASIA—	
Ceylon	Crawford, 1956.
Southern India	Crawford, 1956.

* Present but apparently no references in literature.

AETIOLOGY

It has been established recently that the causal agent of sweating sickness is a toxin and that it is transmitted transovarially for at least seven generations by the vector (Neitz, 1955, 1956). The conception that the agent is a toxin became evident during the course of a series of tick transmission experiments conducted on calves (Table 2) and piglets (Table 3). The results showed that the causal agent can be transmitted by the adult stage of the vector (*H. transiens*) as early as 72 hours but usually between 72 and 96 hours after attachment. Tick-feeding periods of 72, 96 or 120 hours are followed by either an inapparent form of the disease, relatively mild or severe reactions and recovery. Periods of 144 hours and longer, on the other hand, are followed by severe or very severe reactions terminating fatally in 75 per cent of cases. This available evidence points to the fact that the causal agent of sweating sickness develops or multiplies in the invertebrate host, and the timely removal of attached "infective" ticks results in the subsidence of the symptoms with recovery and development of immunity. A similar phenomenon is known to occur in the case of tick paralysis, and in this disease it is generally accepted that the cause is a toxin. Consideration of the results following the interrupted feeding of "infective" ticks leaves no doubt that the behaviour of the causal agent of sweating sickness differs materially from that observed with true viruses, and that sufficient evidence is available that it is a toxin.

TABLE 2
Observations on Calves

No. of Calf	Ticks fed for	No. of Ticks Attached	Nature of Reaction	Result	Immunity Test				
					Interval	Ticks fed for	No. of Ticks Attached	Nature of Reaction	Result
	Days				Days	Days			
7653.....	1	16	NR	—	29	11	28	+++++	Died
7702.....	2	12	NR	—	29	8	8	+++++	Died
7810.....	3	7	NR	—	23	9	8	+++++	Died
7643.....	3	12	NR	—	29	11	6	NR	Immune
					77	15	5	NR	Immune
7805.....	4	15	NR	—	23	8	8	NR	Immune
7642.....	4	17	NR	—	29	11	7	NR	Immune
					77	15	6	NR	Immune
7807.....	4	24	+	Recovered.	112	10	15	NR	Immune
7969.....	5	13	+++	Recovered.	112	10	8	NR	Immune
7939.....	5	19	+++	Recovered.	112	10	10	NR	Immune
7971.....	6	14	+++++	Died.....	—	—	—	—	—
7972.....	8	8	+++++	Died.....	—	—	—	—	—
7637.....	11	12	+++++	Died.....	—	—	—	—	—
8065.....	12	6	++++	Recovered.	—	—	—	—	—
7633.....	15	6	++++	Recovered.	—	—	—	—	—

NR signifies no reaction.
+++++ degree of severity of reaction from definite but very mild to very severe.

TABLE 3
Observations on Pigs

No. of Pig	Ticks fed for	No. of Ticks Attached	Nature of Reaction	Result	Immunity Test				
					Interval	Ticks fed for	No. of Ticks Attached	Nature of Reaction	Result
	Days				Days	Days			
1429	3	12	NR	—	19	20	14	++	Recovered
1431	3	28	NR	—	19	20	4	++++	Recovered
1432	3	20	NR	—	19	20	7	++++	Recovered
1430	3	28	NR	—	19	12	10	+++++	Died
1396	4	10	+	Recovered.	19	20	8	+++	Recovered
1408	4	20	+++	Recovered.	19	20	3	NR	Immune
1409	4	21	++++	Recovered.	19	20	5	NR	Immune
1410	4	27	++++	Recovered	19	20	9	NR	Immune
1411	5	17	+	Recovered.	63	12	8	++	Recovered
1412	5	12	++++	Recovered.	63	12	2	NR	Immune
1419	5	13	++++	Recovered.	63	12	3	NR	Immune
1413	5	19	+++++	Recovered.	63	12	4	NR	Immune
1433	6	13	++++	Recovered.	96	13	8	NR	Immune
1425	6	27	+++++	Died.....	—	—	—	—	—
1426	6	17	+++++	Died.....	—	—	—	—	—
1427	6	22	+++++	Died.....	—	—	—	—	—
1428	10	10	+++++	Died.....	—	—	—	—	—
1434	10	15	+++++	Died.....	—	—	—	—	—
1372	10	8	+++++	Died.....	—	—	—	—	—
1373	10	40	+++++	Died.....	—	—	—	—	—
1441	15	9	+++++	Died.....	—	—	—	—	—
1438	20	6	+++++	Died.....	—	—	—	—	—
1442	20	6	++++	Recovered.	77	9	6	NR	Immune
1445	20	4	++++	Recovered.	—	—	—	—	—

TABLE 4

Failure to Transmit Sweating Sickness with the Progeny of Hyalomma transiens *Adult Females which Fed on Reacting Calves*

Animal No.	Species	Age	Infested with Adult Ticks		History of Ticks	Result	Immunity Test							
			Gen.	Batch No.			Interval	Infested with Adult Ticks: Gen.	Infested with Adult Ticks: Batch No.	Incubation Period	Duration of Disease	Total Period	Nature of Reaction	Result
										Days	Days	Days		
6647	Calf...	2 mths...	1	2094 Ca 1	Progeny of a female from Mhlosinga, Zululand	NR	—	—	—	—	—	—	—	—
6680	Calf...	1 mth...	1	2094 Ca 2	Progeny of a female from Mhlosinga, Zululand	NR	6 mths...	2	2118 Ca 1	5	5	10	+++++	Died
8545	Calf...	2 mths...	1	2236 Ga 4	Progeny of a female from Candover, Zululand	NR	2½ mths..	7	2246 Ga 1	6	7	13	+++++	Died
2486	Sheep.	12 mths..	1	2236 Ca 2	Progeny of a female from Candover, Zululand	NR	4½ mths..	6	2243 Da 1	7	10	17	+++++	Died
1513	Pig....	5 mths...	1	2236 Ga 1	Progeny of a female from Candover, Zululand	NR	3 mths...	7	2244 Aa 1	6	8	14	+++	Recovered
1525	Pig....	4 mths...	1	2236 Ga 2	Progeny of a female from Candover, Zululand	NR	3 mths..	7	2244 Ba 1	8	3	11	++	Recovered

Further evidence in support of the causal agent of sweating sickness being a toxin is based on the following observations: Tick-feeding experiments conducted at Onderstepoort have shown that not all strains of *H. transiens* harbour the aetiological agent. Experiments have also shown that the progeny of two adult females of this species which had fed to repletion on reacting calves failed to transmit this disease. One of these females (Batch No. 2094) was received from the Mhlosinga area, and the other (Batch No. 2236) from the Candover area in Zululand. The larvae and nymphae were reared on rabbits while the ensuing adults were allowed to feed on either calves, sheep or pigs listed in Table 4. None of these animals reacted and on challenging their immunity they were found to be fully susceptible to sweating sickness.

Consideration of these results shows that even though the two adult females had every opportunity of infecting themselves, the causal agent failed to establish itself in the potential host. This again points to the fact that the behaviour of the agent differs materially from that observed with true viruses, and that further evidence is available that it is a toxin harboured by some strains of *H. transiens*.

TRANSMISSION

A. Biological transmission

Although the three species of the two host bontpoot or stripe-legged ticks (*Hyalomma rufipes rufipes* Koch, H. *rufipes glabrum* Delpy = *H. turanicum* Pomerantzev, and *H. transiens* Schulze = *H. truncatum* Koch) have been incriminated as possible vectors, it was only recently that successful transmission was achieved by means of *H. transiens* (Neitz, 1953, 1954).

A fully engorged *H. transiens* female adult tick was obtained in April, 1953, from a calf reacting to sweating sickness in the Mhlosinga area in Zululand. Since the larval and nymphal stages do not feed on cattle they were reared on a rabbit maintained in a warm room during winter, July, 1953. Of the ensuing adults twenty were placed on the ear of a three-month-old calf in October, 1953. The ticks attached readily, and after nine days the calf developed typical symptoms of sweating sickness. It died three days later. Three fully engorged females were collected between the ninth and eleventh day after tick infestation. These ticks served as the basic stock from which approximately 15,000 adult ticks were reared.

During the investigations, it was established that ticks reared on rabbits and on either cattle, sheep or pigs can transmit the aetiological agent hereditarily for seven generations. Furthermore, it was found that this agent is retained by the vector irrespective of whether the adult stage feeds on a susceptible calf, immune calf, or on an insusceptible animal, the horse.

Tick-feeding experiments have shown that not all strains of *H. transiens* harbour the causal agent. Furthermore it has been established that the progeny of such ticks are incapable of transmitting the disease even though the adult females had fed to repletion on affected calves. Similarly it has been found, in a single attempt, that the progeny of adult *H. rufipes rufipes* ticks which had engorged on a reacting calf failed to transmit sweating sickness.

Tests have shown that four to six adult *H. transiens* ticks, consisting of males and females, will transmit the disease regularly. In one instance the disease was transmitted by a male and a female. Although the impression has been gained that the severity of the reaction is not dependent upon the number of ticks that have attached, it is nevertheless suggested that more work be conducted before final conclusions can be drawn. Mild, severe or very severe reactions followed when the number of ticks varied from four to forty. Sweating sickness has also been transmitted by adult males.

B. Tick-breeding

For the sake of completeness a short description of the methods of rearing of ticks will be given. Field observations recorded by Bedford (1932, 1936) have shown that the immature stages of *Hyalomma* spp. occurring in South Africa feed on the hedgehog, rat, various species of hares and birds (Table 5). Under laboratory conditions the immature stages can be fed on guinea-pigs but these animals are not suited for raising large numbers. Rabbits are far better hosts. For this purpose approximately 200 larvae are transferred into a small tube which is then closed with a cotton wool plug. This tube is placed into a tick-bag which is drawn over the ears of the rabbit. The open end of the bag is fixed around the base of the ears with adhesive plaster. By withdrawing the cotton wool plug the larvae are released into the confined space. Two days later the tick-bag is slit open and the glass tube, cotton wool plug and other debris removed. The opening in the bag is stitched and the ticks are allowed to continue feeding. Experience has shown that three to six week-old larvae attach readily while younger or older ones are less inclined to do so. They usually die after a few days. The reason for the mortality needs to be determined. Larvae commence to moult six to eight days later. They often detach to reattach at new sites. It has frequently been observed that small abscesses develop at the sites of attachment. This greatly interferes with the engorgement of the nymphae and may even lead to the death of the rabbit. Engorged nymphae drop 14 to 35 days after the infestation while the majority detach between the fifteenth and twentieth day. Nymphae moult after a period varying from three to six weeks, and the adults are ready for feeding when they are six to eight weeks old. The engorgement period of adult females on domestic animals varies from five to fifteen days, and the majority detach between the seventh and tenth day. Adult males remain attached for periods of up to eight weeks and longer. (The end point has not been determined.)

It is self-evident that the maintenance of ticks harbouring the causal agent of sweating sickness is of great importance. Experience has shown that if adequate numbers of engorged females are required for perpetuating the strain, it is essential to infest the ears of domestic animals with a relatively large number (20—30) of ticks. By following this procedure the chances of overcoming several hazards of tick-feeding become greater. Some of the adult ticks are sometimes disinclined to attach. Furthermore, the period of engorgement (5 to 15 days) almost coincides with the period of incubation and the duration of sweating sickness (7 to 15 days) in cases where the disease has a fatal termination. When a large number of ticks feed one can expect that at least a few females will have engorged before either the profuse moist eczema, the development of abscesses at the site of tick attachment or the death of the host animal interrupts the feeding. It is of course far simpler to feed adult ticks on either immune or insusceptible animals. However, at the time when the vector of sweating sickness was identified, nothing was known about the behaviour and the nature of the causal agent.

TABLE 5

The Natural Hosts of the South African Hyalomma *spp.*

Class and Order	Family	Zoological Names	Vernacular Names
MAMMALIA			
Lipotyphla	Erinaceidae	*Aethechinus frontalis frontalis* (A. Smith) *Atelerix frontalis* (A. Smith)	South African Hedgehog, Krimpvarkie
Rodentia	Muridae	*Rattus rattus* (Linnaeus)	Black rat, Swartrot
Lagomorpha	Leporidae	*Lepus capensis capensis* (Linnaeus) *Lepus capensis ochropus* (Wagner) *Lepus saxatilis zuluensis* (Thos. and Schw.) = *Lepus zuluensis* (Thos. and Schw.)	Cape hare, Vlakhaas Cape hare, Vlakhaas Zululand hare, Zoeloelandhaas
AVES			
Struthioniformes	Struthionidae	*Struthio camelus australis* (Gurney) = *Struthio camelus* (Gurney)	Ostrich, Volstruis
Galliformes	Phasianidae Meleagrididae	*Gallus domesticus* (Linnaeus) *Meleagris* sp.	Fowl, Hoender Turkey, Kalkoen
Passeriformes	Alaudidae	*Calendula magnirostris magnirostris* (Steph.)	Thick billed lark, Dikbek lewerkie
	Dicruridae	*Dicrurus adsimilis adsimilis* (Bechstein) = *Buchanga adsimilis*	Fork tailed drongo, Mikstert byvanger
	Turdidae	*Cossypha caffra caffra* (Linnaeus) = *Caffrornis caffra caffra* (Linnaeus)	Cape robin-chat, Janfrederik
Coraciiformes	Strigidae	*Bubo africanus* (Temm)	Cape spotted eagle owl, Gevlekte ooruil

It was, therefore, of great importance to gain reliable evidence whether or not the strain of ticks would retain the causal agent of sweating sickness from one generation to another. Tests have shown that the causal agent, a toxin, is an inherent property of the vector, and that it is transmitted transovarially.

Adult females, reared at Onderstepoort, lay approximately 3,000 to 5,000 eggs. Observations made on the duration of the various developmental stages in ticks maintained in an acaridarium in which the temperature was 26° C and the relative humidity 80 per cent are as follows:—

Pre-oviposition period ... ... 6 to 30 days.

Oviposition lasts ... ... 10 to 40 days.

Larvae hatch ... ... 28 to 50 days.

Larvae and nymphae on rabbits ... ... 14 to 35 days (Average 15—20 days).

Nymphae moult ... ... 21 to 42 days.

Females engorge ... ... 5 to 15 days (Average 7–10 days).

Unfed larvae survive ... ... 3 to 6 months.

Unfed adults survive ... ... 9 to 12 months.

C. Artificial transmission

All attempts at transmitting sweating sickness to susceptible calves by either the intravenous or subcutaneous injection of blood or organ emulsions of diseased animals have failed (Bevan, 1920; Sinclair, 1920; Du Toit, 1923; Le Roux, 1943; Quin, 1943; Lawrence, 1946). Du Toit was unable to communicate the disease by either rubbing the diseased skin on the scarified skin of healthy calves, or by rubbing the diphtheritic membranes and saliva of affected calves on the gums of healthy animals. Contact between sick and healthy calves also failed to bring about infection (Du Toit, *loc. cit.*; Lawrence, 1946).

Although the abovementioned negative results appeared to be convincing that sweating sickness cannot be transmitted artificially they did, however, not indicate whether an inapparent form of the disease had taken place. This doubt suggested that the work be repeated and that the immunity of the experimental animals be challenged before drawing final conclusions. This investigation was undertaken by the writer in collaboration with Dr. D. Haig at the Onderstepoort Veterinary Laboratory. For this purpose blood, and various organs were collected from two calves (No. 7483 and 7513) and sheep (No. 86978) on either the third or fourth day of the disease. The blood and emulsions prepared from the organs were injected by either the intravenous, subcutaneous, intradermal or intracerebral route into 18 fully susceptible Merino sheep reared at Onderstepoort. The experimental observations are mentioned in the appended Table 6. No reactions were observed in any of the recipients except in the case of sheep No. 88596 which developed a fever (104–107° F) for three days on the eleventh day after the injection. Blood of this animal, injected into sheep No. 89156, failed to produce any visible symptoms. On challenging the immunity of all the recipients including sheep No. 89156 after intervals varying from 21 days to four months after the artificial infection, all died except sheep No. 86977 which contracted a relatively mild form of sweating sickness and recovered. The five controls all died. These experiments confirmed the observations of previous investigators that sweating cannot be transmitted artificially, and eliminated the doubt that an inapparent form of the disease could have taken place.

TABLE 6

Attempts to Transmit Sweating Sickness Artificially to Sheep

Animal	Breed	Age of Animal	Injected from	Inoculum	Result	Immunity Test							
						Interval	Infested with Adult Ticks		Incubation Period	Duration of Disease	Total Period	Nature of Reaction	Result
							Gen.	Batch No.					
									Days	Days	Days		
88444	Merino..	20 days..	Calf 7483....	Kidney, liver, spleen 10 c.c. i.v.	NR	21 days..	4	2148 Ja 5..	6	5	11	+++++	Died
88523	Merino..	3 mths..	Calf 7483....	Liver, 10 c.c. i.v.....	NR	21 days..	4	2148 Ja 1..	5	3	8	+++++	Died
86977	Merino..	3 mths..	Calf 7483....	Spleen, 10 c.c. i.v.....	NR	21 days..	4	2148 Ja 2..	5	9	14	+++	Recovered
88117	Merino..	5 mths..	Calf 7483....	Kidney, 10 c.c. i.v.....	NR	21 days..	4	2148 Ja 3..	4	6	10	+++++	Died
86696	Merino..	5 mths..	Calf 7483....	Kidney, liver, spleen 10 c.c. i.v.	NR	21 days..	4	2148 Ja 4..	5	4	9	+++++	Died
88596	Merino..	5 mths..	Calf 7513....	Kidney, liver, spleen 10 c.c. i.v.	Fever..	21 days..	4	2148 Ha 2.	5	6	11	+++++	Died
88597	Merino cross	5 mths..	Calf 7513....	Brain, 0·25 c.c. i.c.....	NR	21 days..	4	2148 Ia 1..	4	5	9	+++++	Died
88581	Merino cross	6 mths..	Calf 7513....	Adrenal, 10 c.c. i.v.....	NR	21 days..	4	2148 Ga 2.	5	5	10	+++++	Died
88589	Merino..	6 mths..	Calf 7513....	Skin, 5 c.c. i.v. 0·25 c.c. i.d...	NR	21 days..	4	2148 Ga 3.	6	6	12	+++++	Died
88590	Merino..	6 mths..	Calf 7513....	Lymph gland, 10 c.c. i.v.....	NR	21 days..	4	2148 Ha 1.	5	5	10	+++++	Died
89156	Merino..	3 mths..	Sheep 88596..	Blood, 10 c.c. i.v.....	NR	28 days..	4	2148 Ia 2..	7	4	11	+++++	Died
88021	Merino..	15 mths..	Sheep 86978..	Adrenal, 5 c.c. i.v. 2 c.c. s.c...	NR	4 mths..	3	2142 Aa 3..	8	6	14	+++++	Died
88030	Merino..	15 mths..	Sheep 86978..	Blood, 10 c.c. i.v.....	NR	4 mths..	3	2142 Aa 8..	6	2	8	+++++	Died
87497	Merino..	16 mths..	Sheep 86978..	Salivary gland, 2 c.c. i.v. 2 c.c. s.c.	NR	4 mths..	3	2142 Aa 4..	6	3	9	+++++	Died
87494	Merino..	16 mths..	Sheep 86978..	Brain, 2 c.c. i.v., 2 c.c. s.c....	NR	4 mths..	3	2142 Aa 5..	7	5	12	+++++	Died
87495	Merino..	16 mths..	Sheep 86978..	Blood, 2 c.c. i.v., 2 c.c. s.c....	NR	4 mths..	3	2142 Aa 6..	7	2	9	+++++	Died
86698	Merino..	17 mths..	Sheep 86978..	Spleen, 2 c.c. i.v., 2 c.c. s.c...	NR	4 mths..	3	2142 Aa 7..	7	7	14	+++++	Died
86979	Merino..	16 mths..	Sheep 86978..	Tick emulsion.....	NR	4 mths..	3	2142 Aa 9..	6	3	9	+++++	Died
875	Merino..	1 mth..	Control.....	—	—	—	2	2195 Ja 1..	8	3	11	+++++	Died
88116	Merino..	9 mths..	Control.....	—	—	—	2	2118 Ba 7..	8	4	12	+++++	Died
86975	Merino..	11 mths..	Control.....	—	—	—	2	2118 Ca 5..	7	4	11	+++++	Died
86721	Merino..	11 mths.	Control.....	—	—	—	2	2118 Ca 6..	8	4	12	+++++	Died
87715	Merino..	16 mths..	Control.....	—	—	—	2	2118 Ga 3.	4	5	9	+++++	Died
86978	Merino..	11 mths..	Donor.....	—	—	—	2	2118 Ca 8..	7	3	10	++++	Killed
7483	Calf.....	7 mths..	Donor.....	—	—	—	4	2148 Ga 1.	4	3	7	++++	Killed
7513	Calf.....	4 mths..	Donor.....	—	—	—	4	2148 Aa 1..	5	4	9	++++	Killed

EPIZOOTOLOGY

In common with other diseases which require an arthropod vector, sweating sickness is characteristically a disease of place. The density of vectors and the number of susceptible cattle determine the incidence of the disease. Observations recorded from Southern Rhodesia and Swaziland (Table 7) show that there is a marked variation in the cyclical incidence of this disease. This may be attributable to either unfavourable climatic conditions interfering with the normal development of the vector, intentional or accidental veld-burning causing the destruction of large numbers of ticks, or systematic dipping as recorded by Hooper Sharpe (1935, 1936). *H. transiens* ticks harbouring the toxin are capable of transmitting this agent transovarially for seven generations irrespective of whether they feed on a susceptible calf, immune calf or an insusceptible animal, the horse. It thus becomes evident that vectors can retain the toxin in the complete absence of susceptible mammals. The sudden appearance of the disease in calves in apparently clean areas is, therefore, not necessarily due to a fresh introduction of toxin-harbouring ticks. On the other hand, it should also be remembered that migratory birds may introduce the vector into regions free of the disease.

In tropical and subtropical regions sweating sickness can be expected at any time of the year (Clark, 1933; Morris, 1938). The highest incidence is during the summer months, January, February and March. It appears that the disease is more prevalent in regions with a high rainfall, but its occurrence in semi-arid areas is by no means uncommon. The occurrence of sweating sickness during the summer months is dependent upon the life-cycle of the vector.

Adult *H. transiens* ticks are encountered chiefly during the summer months. If one considers the duration of the different developmental stages (*vide supra*-Transmission) in conjunction with the climatic conditions prevailing during autumn, winter and spring it becomes apparent that it takes nine to twelve months for the vector to complete its life-cycle in nature.

Altitude does not seem to be an important factor in the incidence of sweating sickness. It has been encountered in areas varying from 200 to 4,000 feet above sea-level (Du Toit, 1923).

Sweating sickness may occur in association with either anaplasmosis, babesiosis, East Coast fever, heartwater, nagana, paratyphoid, white scours, pneumonia and myiasis (Thomas, 1933). In these circumstances it is difficult to estimate the direct losses due to sweating sickness unless systematic blood-smear examination and careful clinical observations are made on all animals, particularly young calves, that die within an area.

TABLE 7

Variation in the Cyclical Incidence of Sweating Sickness

Country	Period	Incidence	Reference
Swaziland	1932–1936	++	Annual Veterinary Reports
	1937–1938	?	Annual Veterinary Reports
	1939–1941	+	Annual Veterinary Reports
	1942–1943	++	Annual Veterinary Reports
	1944–1947	+	Annual Veterinary Reports
	1948	++	Annual Veterinary Reports
	1949	+	Annual Veterinary Reports
	1950–1951	++	Annual Veterinary Reports
	1952	+	Annual Veterinary Reports
	1953	++	Annual Veterinary Reports
Southern Rhodesia	1919–1920	++	Annual Veterinary Reports
	1921	?	Annual Veterinary Reports
	1922–1932	+	Annual Veterinary Reports
	1933–1935	++	Annual Veterinary Reports

\+ signifies low incidence
++ signifies high incidence
? signifies no information available

PATHOGENICITY

Sweating sickness is a tick-borne disease transmissible to several members of the order Artiodactyla (Neitz, 1955). In nature it has so far only been encountered in cattle. It is essentially a disease of calves, usually affecting those between the ages of a week and nine months (Bevan, 1920; Sinclair, 1920; Du Toit, 1923; Hooper Sharpe, 1930; Thomas, 1932; Walker, 1932; Clark, 1933; Lewis, 1934; Daubney, 1936; Morris, 1938; Bisschop, 1942; Lawrence, 1946; Henning, 1932, 1949, 1956; Neitz, 1955). The disease has been encountered in young stock (age not mentioned) by Bevan (1920), in cattle up to two years old (Du Toit, 1923; Clark, 1933), in three-year-old cattle (Clark, 1933; Lawrence, 1947), and in animals (Table 8) varying from three-and-a-half to thirteen years of age (Neitz, 1955).

TABLE 8

Observations on the Susceptibility of Cattle to Sweating Sickness

Animal No. (Cattle)	Infested with Adult Ticks		Age of Animal	Incubation Period	Duration of Disease	Total Period	Nature of Reaction	Result
	Gen.	Batch No.						
				Days	Days	Days		
7445	2	2118 Ca 4..	2 days....	5	5	10	+++++	Died
7793	4	2171 Aa 1..	7 days....	5	5	10	+++++	Died
7503	3	2141 Ca 1..	10 days....	5	6	11	+++++	Died
6842	2	2118 Ra 1..	21 days....	8	7	15	+++	Recovered
6808	2	2118 Ea 1..	1 month..	4	8	12	+++++	Died
7521	4	2160 Aa 1..	1 month..	5	7	12	+++++	Died
8423	6	2224 Ca 2..	1½ months.	4	8	12	++++	Recovered
7668	4	2171 AAa 1	2 months.	4	2	6	+++++	Died
7486	3	2142 Aa 1..	2 months.	5	7	12	+++++	Died
8408	6	2224 Da 1.	2 months.	6	6	12	+++	Recovered

TABLE 8 (*continued*)

Animal No. (Cattle)	Infested with Adult Ticks		Age of Animal	Incubation Period	Duration of Disease	Total Period	Nature of Reaction	Result
	Gen.	Batch No.		Days	Days	Days		
8154	4	2182 Ba 1..	2 months.	6	11	17	+++	Recovered
7491	3	2142 Aa 11	2 months.	7	9	16	++++	Recovered
8639	6	2242 Aa 2..	2 months.	9	6	15	+++++	Died
8349	6	2224 Ca 1..	3 months.	4	7	11	+++	Recovered
7449	3	2141 Ba 1..	3 months.	5	7	12	+++++	Died
7527	3	2142 Ea 2..	3 months.	6	9	15	+++++	Died
7529	3	2142 Ea 3..	3 months.	6	8	14	+++++	Died
6880	2	2118 Ca 9..	3 months.	7	7	14	+++++	Died
7523	3	2142 Ga 2.	4 months.	5	8	13	+++++	Died
7526	4	2148 Aa 2..	4 months.	5	7	12	+++++	Died
7530	3	2141 Da 2.	4 months.	5	6	11	+++++	Died
7512	4	2159 Ga 1.	4 months.	6	6	12	+++++	Died
6722	2	2118 Ba 2..	4 months.	6	7	13	+++++	Died
7511	4	2159 Aa 1..	4 months.	7	11	18	+++++	Died
6618	1	2093 Aa 1..	4 months.	8	4	12	+++++	Died
7518	3	2142 Ga 1.	4 months.	8	12	20	++++	Recovered
8555	6	2241 Aa 1..	4 months.	9	20	29	+++++	Died
7528	4	2148 Ia 5..	5 months.	5	5	10	+++++	Died
7971	5	2198 Ba 6..	5 months.	5	8	13	++++	Recovered
8545	7	2246 Ga 1.	5 months.	6	7	13	+++++	Died
7972	5	2198 Ba 7..	5 months.	6	9	15	+++++	Died
8065	6	2216 Ca 1..	5 months.	6	14	20	++++	Recovered
7707	5	2195 Fa 2..	6 months.	6	12	18	++++	Recovered
6690	2	2118 Ba 2..	6 months.	7	3	10	+++++	Died
7704	5	2195 Fa 1..	6 months.	7	11	18	+++	Recovered
6696	2	2118 Ca 2..	6 months.	8	4	12	+++++	Died
6680	2	2118 Ca 1..	7 months.	5	5	10	+++++	Died
7525	4	2160 Ba 1..	7 months.	5	5	10	+++++	Died
6688	2	2118 Ca 3..	7 months.	5	9	14	++++	Recovered
7702	5	2200 Ea 1..	7 months.	6	6	12	+++++	Died
7517	4	2148 Ia 4..	8 months.	5	7	12	+++++	Died
7637	5	2196 Fa 5..	8 months.	5	8	13	+++++	Died
7612	5	2195 Ba 1..	8 months.	5	12	17	+++++	Died
7653	5	2200 Da 3.	8 months.	6	6	12	+++++	Died
6843	3	2142 Ba 5..	8 months.	7	7	14	+++	Recovered
7656	4	2183 Da 5.	10 months.	6	4	10	+++++	Died
7633	4	2183 Ba 1..	11 months.	6	5	11	++++	Recovered
6697	3	2142 Ba 3..	11 months.	6	10	16	+++	Recovered
7421	3	2142 Ba 1..	14 months.	7	8	15	+++++	Died
7422	3	2142 Ca 4..	15 months.	6	9	15	++++	Recovered
6698	3	2142 Aa 7..	17 months.	7	7	14	+++++	Died
7810	5	2198 Oa 2..	19 months.	4	8	12	+++++	Died
7419	3	2142 Ba 2..	20 months.	6	16	22	++++	Recovered
7638	5	2198 Oa 3..	23 months.	5	14	19	++++	Recovered
5138	2	2118 Ea 2..	44 months.	4	4	8	+++++	Died
4254	2	2118 Qa 2..	61 months.	6	2	8	+	Recovered
3665	3	2142 Ba 1..	81 months.	7	5	12	+++	Recovered
3065	3	2142 Ca 2..	83 months.	7	4	11	+++	Recovered
5300	2	2118 Ba 10.	84 months.	6	16	22	++++	Recovered
6793	4	2148 Ia 6..	120 months.	5	9	14	++++	Recovered
45	3	2142 Ea 1..	162 months.	5	16	21	++++	Recovered

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Information quoted in the literature shows a marked variation in the mortality rate in calves raised under field conditions. Bevan (1920) and Sinclair (1920) assumed a mortality of 75 per cent in Southern Rhodesia. Du Toit (1923) recorded a morbidity rate of nine per cent and a mortality of 30 per cent in Swaziland. Clark (1933) reported that the morbidity in Zululand varied from 10 to 100 per cent, while the death-rate varied from 30 to 77 per cent. Careful observations made over a period of eleven years at Armoedsvlakte in the Vryburg District of the Western Cape Province by Bisschop (1942) revealed a morbidity rate of 2·2 per cent, and a mortality of 29 per cent in calves varying in age from 11 to 349 days.

The marked variation in the mortality rate recorded by various investigators made it necessary to study the degree of susceptibility of calves under more or less uniform laboratory conditions. The availability of a large number of *H. transiens* adult ticks derived from a single "infective" female, and a relatively large number of calves raised under conditions in which stripe-legged ticks could not maintain themselves, made this project possible. During the course of these investigations it was found that two calves (No. 7487 and 7700, Table 13) proved to be refractory. As an adequate number of ticks had engorged to repletion on both these animals no explanation can be given for this phenemenon. On testing their immunity subsequently (Tables 12 and 13) they were found to be solidly immune. Tick transmission experiments conducted on 48 calves (Table 8) varying in age from two days to eleven months were followed by a mortality of 69 per cent. Typical reactions were also observed in six head of cattle which were 14 to 23 months old. Of these three died at the ages of either 14, 17 or 19 months.

Consideration of these results shows that experimental evidence is now available, which supports the conclusion of previous investigators, that in uncomplicated cases of sweating sickness the mortality rate may be as high as 77 per cent. These results, however, give no indication why in some circumstances the mortality rate may be as low as 29 per cent. In order to elucidate this, it is suggested that tests be conducted to determine whether the fluctuation in the death-rates can be ascribed to either a variation in the degree of virulence of the toxins harboured by different strains of ticks, or to a variation in the amount of toxin liberated by the vectors during the process of feeding. The latter suggestion appears to be plausible if one considers the non-fatal reactions in calves and pigs which followed when the feeding periods of ticks were interrupted either at 96 or 120 hours after attachment.

During the course of these studies it was established that besides cattle, sheep, goats and pigs are also susceptible to sweating sickness. The observations made on the latter three species will be dealt with separately.

i. Susceptibility of sheep

Tick transmission experiments were conducted on 72 sheep reared in different areas of the Union of South Africa. Observations made on 21 Merino sheep and two Merino crosses, raised at Onderstepoort, are mentioned in Table 6. The ages of these animals varied from 20 days to 17 months. Tick infestation with adult *H. transiens* ticks was followed by a fatal reaction in all but one sheep, which recovered from a fairly severe reaction. Further observations, listed in Table 9,

were made on 34 Merino sheep raised in the Western Cape Province. With the exception of one five-year-old ram, all these sheep were approximatley two years old. Tick infestation showed that fourteen animals were refractory. Of the remaining 20 sheep five developed a very mild, one a mild, ten a fairly severe, and one a severe reaction, followed in all cases by recovery, while three contracted a fatal form of sweating sickness. Tests conducted on other breeds of sheep (Table 9), also raised in the Western Cape Province, revealed that they were highly susceptible. These animals were approximately 15 months old. Of the three Dorset Horn rams, one developed a fairly severe reaction and recovered, while two died from sweating sickness. The Dormer ram contracted a severe form of the disease and recovered, and the Karakul ewe died from this disease.

No satisfactory explanation can be given for the marked variation in the susceptibility in the Merino sheep raised in the Western Cape Province. Since there is no evidence that sweating sickness occurs naturally in sheep in this area, one cannot attribute this resistance to a naturally acquired immunity. Whether or not two-year-old Merino sheep possess a greater degree of resistance than 15-month-old animals needs to be determined. It is, however, significant that a five-year-old Merino ram was still fully susceptible.

ii. Susceptibility of goats

Observations have been made on a limited number of goats of unknown breeding. These animals were raised at Onderstepoort, and their ages varied from 12 to 29 months. The type of reactions following tick infestation are mentioned in Table 10. They were either very mild, mild or fairly severe but not fatal and give the impression that this species is very resistant.

iii. Susceptibility of pigs

A total of 21 Large White pigs was infested with "infective" ticks. The observations made on the animals are recorded in Table 11. The fourteen two-month-old pigs all reacted severely, and nine died from sweating sickness. Of the five six-month-old pigs one developed a mild, a second one a severe, a third one a fatal reaction, while two were found to be refractory. On challenging the immunity of the latter two pigs both were found to be immune. A seven-month-old pig contracted a mild, and an eight-month-old one a fairly severe reaction. Both animals recovered.

Consideration of the results of the transmission experiments of sweating sickness conducted on cattle, sheep, goats and pigs raised at Onderstepoort clearly indicates that there is a variation not only in the individual but also in the species susceptibility. In the individual animals the disease appears either in an inapparent, mild, severe or fatal form. Of the four species concerned, sheep are the most and goats the least susceptible, while cattle and pigs occupy an intermediate position. Although the number of animals representing the different age groups is limited, the impression is gained that in cattle, calves are more susceptible than adult stock.

TABLE 9

Observations on the Susceptibility of Sheep, raised in the Western Cape Province, to Sweating Sickness

Animal No. (Sheep)	Breed	Age of Animal	Origin	Infested with Adult Ticks		Incubation Period	Duration of Disease	Total Period	Nature of Reaction	Result
				Gen.	Batch No.	Days	Days	Days		
188	Merino wether..	24 mths..	Beaufort West Dist., W.C.P..	5	2196 Fa 3..	—	—	—	—	NR
510	Merino wether..	24 mths..	Beaufort West Dist., W.C.P..	5	2195 Ca 2..	—	—	—	—	NR
513	Merino wether..	24 mths..	Beaufort West Dist., W.C.P..	5	2195 Da 1.	—	—	—	—	NR
86757	Merino wether..	24 mths..	Beaufort West Dist., W.C.P..	2	2118 Ba 13.	—	—	—	—	NR
87721	Merino wether..	24 mths..	Beaufort West Dist., W.C.P..	2	2118 Qa 4..	—	—	—	—	NR
88428	Merino wether..	30 mths..	Beaufort West Dist., W.C.P..	2	2118 Ba 4...	—	—	—	—	NR
86748	Merino wether..	24 mths..	Beaufort West Dist., W.C.P..	2	2118 Ba 12.	7	2	9	+	Recovered
165	Merino wether..	24 mths..	Beaufort West Dist., W.C.P..	5	2195 HIJa 1	7	7	14	+++	Recovered
209	Merino wether..	24 mths..	Beaufort West Dist., W.C.P..	5	2196 Fa 4..	5	2	7	+++++	Died
87715	Merino wether..	24 mths..	Beaufort West Dist., W.C.P..	2	2118 Qa 3..	4	6	10	+++++	Died
89433	Merino wether..	24 mths..	Prieska District, W.C.P.....	4	2148 Da 1.	—	—	—	—	NR
89443	Merino wether..	24 mths..	Prieska District, W.C.P.....	4	2148 Da 2.	—	—	—	—	NR
89542	Merino wether..	24 mths..	Prieska District, W.C.P.....	4	2148 Da 3..	—	—	—	—	NR
89463	Merino wether..	24 mths..	Prieska District, W.C.P.....	4	2148 Ea 1..	—	—	—	—	NR
89349	Merino wether..	24 mths..	Prieska District, W.C.P.....	4	2148 Ba 1..	8	3	11	+	Recovered
89353	Merino wether..	24 mths..	Prieska District, W.C.P.....	4	2148 Ba 2..	7	4	11	+	Recovered
89413	Merino wether..	24 mths..	Prieska District, W.C.P.....	4	2148 Ca 2..	7	3	10	+	Recovered
89490	Merino wether..	24 mths..	Prieska District, W.C.P.....	4	2148 Ea 2..	5	3	8	+	Recovered

TABLE 9 (*continued*)

Animal No. (Sheep)	Breed	Age of Animal	Origin	Infested with Adult Ticks		Incubation Period	Duration of Disease	Total Period	Nature of Reaction	Result
				Gen.	Batch No.					
						Days.	Days.	Days.		
89378	Merino wether..	24 mths..	Prieska District, W.C.P.....	4	2148 Ba 3..	7	3	10	+++	Recovered
89381	Merino wether..	24 mths..	Prieska District, W.C.P.....	4	2148 Ca 1..	7	8	15	+++	Recovered
89494	Merino wether..	24 mths..	Prieska District, W.C.P.....	4	2148 Fa 1..	7	7	14	+++	Recovered
89506	Merino wether..	24 mths..	Prieska District, W.C.P.....	4	2148 Fa 2..	7	3	10	+++	Recovered
781	Merino wether..	25 mths..	Middelburg District, W.C.P..	5	2195 Da 2.	—	—	—	—	NR
784	Merino wether..	25 mths..	Middelburg District, W.C.P..	5	2195 Ea 1..	—	—	—	—	NR
785	Merino wether..	25 mths..	Middelburg District, W.C.P..	5	2195 Ea 2..	—	—	—	—	NR
783	Merino wether..	25 mths..	Middelburg District, W.C.P..	5	2195 Da 3.	11	6	17	+++	Recovered
787	Merino wether..	25 mths..	Middelburg District, W.C.P..	5	2195 Fa 3..	11	5	16	+++	Recovered
786	Merino wether..	25 mths..	Middelburg District, W.C.P..	5	2195 Ea 3..	7	5	12	+++++	Died
775	Merino ram....	60 mths..	Middelburg District, W.C.P..	5	2196 Ja 2..	7	10	17	+++	Recovered
777	Merino ram....	15 mths..	Middelburg District, W.C.P..	5	2196 Ca 1..	7	11	18	+++	Recovered
789	Merino ewe....	25 mths..	Colesberg District, W.C.P....	5	2195 Ea 4..	9	7	16	+++	Recovered
790	Merino ewe....	25 mths..	Colesberg District, W.C.P ...	5	2195 Ca 1..	—	—	—	—	NR
791	Merino ewe....	25 mths..	Colesberg District, W.C.P ...	5	2195 Ga 2.	10	5	15	+++	Recovered
792	Merino ewe....	25 mths..	Colesberg District, W,C.P ...	5	2195 Ga 3.	9	6	15	+++	Recovered
774	Dorset horn ram	15 mths..	Middelburg District, W.C.P..	5	2196 Ja 1..	6	4	10	+++++	Died
778	Dorset horn ram	15 mths..	Middelburg District, W.C.P..	5	2200 Fa 1..	7	14	21	+++++	Died
780	Dorset horn ram	15 mths..	Middelburg District, W.C.P..	5	2200 Da 1.	7	9	15	+++	Recovered
776	Dormer ram...	15 mths..	Middelburg District, W.C.P..	5	2196 Ea 1..	6	11	17	++++	Recovered
793	Karakul ewe...	15 mths..	Middelburg District, W.C.P..	5	2196 Ga 4.	6	3	9	+++++	Died

TABLE 10

Observations on the Susceptibility of Goats to Sweating Sickness

Animal No. (Goats)	Infested with Adult Ticks		Age of Animal	Incubation Period	Duration of Disease	Total Period	Nature of Reaction	Result
	Gen.	Batch No.						
				Days	Days	Days		
88593	3	2141 Ea 1..	12 months.	9	2	11	+	Recovered
88594	3	2141 Fa 1..	12 months.	6	5	11	+	Recovered
89844	3	2142 Ba 7..	12 months.	7	3	10	+	Recovered
89845	3	2142 Ba 8..	12 months.	6	5	11	++	Recovered
85412	3	2141 Aa 1..	29 months.	6	5	11	++	Recovered
85422	3	2141 Da 1.	29 months.	6	4	10	+++	Recovered
85400	3	2142 Ba 5..	29 months.	5	7	12	+++	Recovered
85403	3	2142 Ba 6..	29 months.	6	6	12	+++	Recovered

Attempts to transmit sweating sickness to horses have failed. No symptoms of sweating sickness have been observed in any of the 500 rabbits used for rearing the larvae and nymphae, the progeny of infective adults.

PATHOGENESIS

From the sites of attachment of the vectors, the toxin is conveyed by the blood-stream to various organs, and also to the mucous membranes and skin, where owing to the epitheliotropic properties, on sites of predilection, it causes hyperaemia and subsequently desquamation of the superficial layers of the epithelium. As sites of predilection the upper respiratory and alimentary tracts, and external genital tract must be taken into consideration. In affected animals one encounters a diphtheroid rhinitis, laryngitis, stomatitis, glossitis, pharyngitis, oesophagitis, vaginitis or posthitis, and a profuse moist eczema. The latter symptom is followed by desquamation of the superficial epithelial layers of the skin and alopecia.

SYMPTOMATOLOGY

Studies made on cattle, sheep, goats and pigs exposed to tick infestation established that the severity of sweating sickness depends upon either the resistance of animals or the duration of the feeding period of the vector (*vide supra*– Aetiology). The incubation period in the abovementioned animals varies from four to eleven days with an average of six days. Depending upon the nature of the reaction sweating sickness may be classified according to its symptoms into five types: (1) the peracute, (2) the acute, (3) the subacute, (4) the mild, and (5) the inapparent form.

(1) *The peracute form* has so far been observed in four per cent of calves (Table 8), and in 25 per cent of Merino sheep (Table 6) raised at Onderstepoort. It is difficult to distinguish it from the peracute forms of anthrax, anaplasmosis, babesiosis, heartwater, mineral and plant poisoning. The onset of the disease is sudden. The affected animal has a high temperature (106–108° F), shows hyperaemia and hyperaesthesia of the visible mucous membranes and skin, anorexia, thirst, lachrymation, serous rhinitis, salivation, muscular tremours, dysphagia, sluggish gait and dyspnoea. In sheep oedema of the lips is usually pronounced. A moist eczema is rarely seen in calves. Death occurs within 48 to 72 hours after the initial rise in temperature.

(2) *The acute form* is the usual type seen in calves of up to twelve months of age (Table 8), in sheep of up to fifteen months old (Table 6), and in two-month-old pigs (Table 11). Observations on older animals are limited. This form has been seen in 14, 17, 19 and 44 months-old cattle (Table 8), and in a six-months-old pig (Table 11). The symptoms exhibited by cattle, sheep and pigs are similar but not identical, and in order to avoid confusion they will be described separately.

TABLE 11

Observations on the Susceptibility of Pigs to Sweating Sickness

Animal No. (Pig)	Infested with Ticks		Age of Animal	Incubation Period	Duration of Disease	Total Period	Nature of Reaction	Result
	Gen.	Batch No.		Days	Days	Days		
1373	5	2198 Ba 1..	2 months.	5	7	12	+++++	Died
1426	5	2199 Ja 1..	2 months.	5	5	10	+++++	Died
1427	5	2199 Ia 1..	2 months.	6	4	10	+++++	Died
1430	4	2183 Ea 1..	2 months.	6	6	12	+++++	Died
1433	5	2198 Fa 1..	2 months.	6	10	16	++++	Recovered
1425	5	2199 Ka 1.	2 months.	7	8	15	+++++	Died
1434	5	2198 Fa 2..	2 months.	7	4	11	+++++	Died
1442	4	2183 Ga 3.	2 months.	7	24	31	++++	Recovered
1445	4	2183 Ca 4..	2 months.	7	15	22	++++	Recovered
1428	5	2200 Ba 1..	2 months.	8	6	14	+++++	Died
1431	4	2183 Ea 2..	2 months.	8	19	27	++++	Recovered
1432	4	2183 Ea 4..	2 months.	8	15	23	++++	Recovered
1441	4	2183 Ga 2.	2 months.	9	7	16	+++++	Died
1438	4	2183 Ga 1.	2 months.	11	14	25	+++++	Died
1282	4	2148 Fa 3..	6 months.	4	9	13	++++	Died
1283	4	2148 Fa 4..	6 months.	5	10	15	+++++	Recovered
1281	4	2148 Fa 2..	6 months.	—	—	—	—	NR
	4	2171 Wa 1.	9 months.	—	—	—	—	NR
1531	6	2243 Ea 1..	6 months.	—	—	—	—	NR
	7	2244 Ba 2..	9 months.	—	—	—	—	NR
1532	6	2240 Aa 4..	6 months.	6	5	11	++	Recovered
	7	2244 Ba 3..	9 months.	—	—	—	—	NR
1525	7	2244 Ba 1..	7 months.	8	3	11	++	Recovered
1513	7	2244 Aa 1..	8 months.	6	8	14	+++	Recovered

Cattle.—The onset of the disease is, as a rule, sudden. The course of the disease varies from four to twenty days with an average of six days. The affected animal usually has a high temperature (105–108° F). The fever is continuous for three to six days or irregularly intermittent for eight days. The animal appears dull, with drooping ears and staring coat. Hyperaemia of the visible mucous membranes and skin (if not pigmented), and salivation are characteristic primary symptoms. Hyperaesthesia soon follows as indicated by the disinclination of the calf to suck. In these circumstances the udder of the cow becomes distended with milk. The primary symptoms are soon followed by lachrymation, serous rhinitis, and sometimes by epistaxis. A few days later the serous discharge from the nose and eyes may become mucopurulent, and on drying crusts appear in and around the orifices. The margin of the eye-lids may be glued together, and respiration may be interfered with. In rare cases the cornea becomes affected, resulting in opacity. A moist eczema may appear as early as 48 hours but usually 72 hours after the onset of the symptoms. It may be confined to the ears, face, neck, axilla, flank or groin, but very often the whole skin becomes involved. An evil odour is emitted from the skin which attracts flies. Myiasis is a common complication. The skin is clammy with the hair matted, and later drops of exudate appear on the tips of the hair. Over the affected skin the hair can be pulled out very easily. It comes off in tufts with the epidermis attached, and leaves a red but not bleeding surface. The skin is extremely sensitive, and the animal instinctively seeks shade to protect itself against the rays of the sun. Handling will cause the animal to crouch. Cattle with unpigmented claws show distinct hyperaemia of the coronets. The sick calf stands with an arched back and shows a painful gait. Death may take place at this stage of the disease or alternatively the calf continues to live for a few days or longer. In the latter event the skin becomes dry and indurated. This is followed by desquamation of the superficial layers of the epidermis and a variable degree of alopecia.

Examination of the buccal cavity usually shows the presence of a diphtheroid stomatitis, glossitis, pharyngitis, and laryngitis. Severe erosions may be seen on the inside of the lips, on the hard palate, at the base and sides of the tongue, on the mucous membrane opposite the molar teeth, and on the pharynx and larynx. A continued or intermittent grinding of the teeth is a common symptom.

The intestinal tract may be severely affected, and animals show diarrhoea. A diphtheroid vaginitis or posthitis is not an uncommon symptom.

The central nervous system is not affected as a rule. Du Toit (1923) records a case in which a diseased calf became very aggressive. The writer observed a nervous case in a 44-months-old cow. This animal showed continuous nodding movements of the head for a period of 36 hours before death. Muscular tremours are often seen.

Sick animals often lie in a crouched position, and show dyspnoea, and abdominal pain. There is a rapid loss of condition accompanied by excessive dehydration. The temperature may be subnormal for several days before death supervenes. There may be a copious, frothy nasal discharge.

In the event of animals recovering, there is a rapid healing of the mucous membranes of the alimentary tract. Animals then commence to suck and to feed normally, and soon regain their former condition. The skin, however, is thickened and forms folds. Later it becomes covered with loose epidermis. The hair and epidermis then falls off leaving bare patches on the skin. Very often calves are almost devoid of hair. About three to four weeks later the hair starts to grow again. In animals with an unpigmented skin the hyperaemia is seen to persist for periods of up to eight weeks after recovery. During this period animals are photosensitive, and if exposed to the sun thin layers of epidermis are cast off.

Sheep.—The onset of the disease is sudden. The course varies from four to nine days with an average of six days. Although affected animals usually show a high temperature for three to five days, it should be remembered that the degree of hyperthermia cannot be correlated in any way with the severity of the clinical symptoms. A slight fever rising to 104·4° F may be accompanied by the most severe symptoms.

During the early stages of sweating sickness, the symptoms of oedema of the lips, hyperaemia of the visible mucous membranes, skin and coronets resemble those of blue-tongue very closely. A differential diagnosis becomes possible with progress of the disease. The hyperaemia of the visible mucous membranes and skin is far more pronounced than in blue-tongue. Another differentiating feature between these diseases is the nature of the hyperaemia of the coronets of the claws. In sweating sickness the hyperaemia is diffuse and uniform, while in blue-tongue it is accompanied by petechiae which coalesce to form vertical red streaks below the horny wall of the claws. This abnormality in blue-tongue remains visible for longer than ten weeks, and disappears with the growing horny layer. In sweating sickness the hyperaemia subsides when the animal recovers.

Besides the above-mentioned symptoms, affected sheep show salivation, anorexia, thirst, serous rhinitis, lachrymation, marked hyperaesthesia of the mucous membranes and skin, diphtheroid stomatitis, glossitis, laryngitis, pharyngitis, vaginitis or posthitis but no signs of a *moist* eczema. As in the case of calves, the superficial layers of the epidermis can also be detached mechanically.

As the disease progresses the oedema of the lips subsides, and the serous discharge from the nose and eyes becomes mucopurulent. On drying, crusts are formed in and around the orifices interfering with respiration or causing the margin of the eyelids to be gummed together.

Before death the temperature may become subnormal. Animals may lie for several days in a recumbent position before death supervenes. In the event of recovery a break in the wool is observed three to four weeks later. The whole fleece is shed, and hangs down in tatters.

Pigs.—Symptoms in this species resemble those of sheep. There is, however, no oedema of the lips. The course of the disease varies from four to fifteen days. Hyperthermia (105–106° F) for three to five days, and hyperaemia of the visible mucous membranes, skin and coronets of the claws are constant symptoms. In many instances flushing of the skin is preceded by the appearance of numerous circumscribed red patches varying from 0·5 to 1·5 cm. in diameter. A very moderate moist eczema may be seen in the axilla and groin. The epidermis, on the ventral surface of the chest and belly and on the snout, can be removed mechanically leaving a red but not bleeding surface.

The diphtheritic lesions in the buccal cavity are, as a rule, more pronounced than in sheep. The development of pharyngeal and laryngeal lesions can be judged by changes in the tone and pitch of the squeals on handling without resorting to the exceedingly painful manipulation of forcing the mouth open. Diarrhoea may be present.

Affected animals lose condition. They often lie in a recumbent position for several days and show signs of abdominal pain and dyspnoea. Before death the temperature becomes subnormal.

Recovery is followed by rapid healing of the buccal mucosa. The hyperaemia of the skin and visible mucous membranes subsides rapidly.

(3) *The subacute form.*—The symptoms of this form are similar to but not so pronounced as those of the acute form. It has been observed in cattle, sheep, goats and pigs. The thermal reaction shows no marked deviation from that occurring in the acute form. Anorexia persists for three to five days, and loss in condition is not so marked. The disease takes a non-fatal course. Symptoms abate after six to nine days and recovery is complete after 12 to 20 days.

(4) *The mild form.*—Affected ruminants and pigs show a mild fever (104–105° F) for two to three days. This is accompanied by slight inappetence, and a moderate hyperaemia of the skin and visible mucous membranes. The symptoms subside in four to five days.

(5) *The inapparent form.*—This form has been observed in two calves (Table 13), and two pigs (Table 11) on which ticks had fed to repletion. It has also been encountered in three calves (Table 2) where the feeding periods of ticks was interrupted after either 72 or 96 hours. Evidence that an inapparent form of sweating sickness had taken place was established by challenging the immunity with " infective " ticks. All these animals proved to be solidly immune.

PATHOLOGY

Pathological studies on sweating sickness have been recorded by Du Toit (1923). Observations made during the course of these studies on calves, sheep and swine have shown that a few hitherto undescribed lesions such as hyperaemia and oedema of the lungs, and atrophy of the spleen, also occur in affected animals. The nature of the skin lesions occurring in cattle differs from those observed in sheep and pigs; otherwise the lesions are fairly uniform, even though some local changes are more pronounced in one species than in another. The lesions also vary greatly according to the duration and the severity of the disease.

A. Macroscopical lesions

In the peracute form, observed in calves and sheep, the carcase does not show emaciation and dehydration. Subepicardial and subendocardial petechiae, hyperaemia and oedema of the lungs, hyperaemia of the conjunctiva and of the nasal, buccal, pharyngeal, laryngeal, vaginal or preputial, gastric and intestinal mucous membranes are fairly constant lesions. A variable degree of hyperaemia is visible in the unpigmented skin. Early stages of a moist eczema may be present in calves, and an oedema of the lips is often seen in sheep.

Typical lesions are observed in the acute form of the disease. Complications such as hypostatic pneumonia, myiasis, skin abrasions, traumatic injuries, decubitus lesions as result of prolonged prostration are not uncommon.

Depending upon the duration of the disease, and the presence of concurrent infectious diseases (anaplasmosis, babesiosis, nagana, heartwater and paratyphoid), the emaciation and dehydration may be pronounced. Frequently various species of ticks may be attached to the skin. The perineal region and tail may be soiled with faeces. The visible mucous membranes appear cyanotic, and the eyes are sunken. A bilateral muco-catarrhal discharge admixed with blood and forth may escape from the nostrils. The blood-vessels of the subcutaneous tissue may be injected. The skeletal muscles appear normal.

In calves the skin varies in appearance according to the duration of the disease. If the animal dies within a week after the onset of the disease, an acute dermatitis is present. If the course is prolonged the skin is thickened and indurated. Patches of skin are devoid of hair, and loose scales of epidermis cover these areas. Small vesicles may be present on the skin of some cases. In sheep and pigs the skin shows hyperaemia and a variable number of circumscribed red foci.

The visible mucous membranes show changes already described (*vide supra*-Symptomatology). The same diphtheroid membrane observed on external clinical examination, may also cover the mucous membranes of the nasal cavities, of the pharynx, larynx, oesophagus, oesophageal groove, and margins of the lamellae of the omasum. Sometimes a moderate oedema of the aryepiglottic folds is seen. The mucous membrane of the trachea may be hyperaemic. Oedema and hyperaemia of the lungs is a fairly common lesion; interstitial emphysema may be present; the apical, cardiac, and antero-ventral border of the diaphragmatic lobe may show bronchopneumonia.

The myocard is flabby and a variable number of petechiae may appear on the epicardium and endocardium. Hydrothorax and hydropericardium may be present; spontaneous coagulation of the fluid is frequently observed.

The liver is often congested and increased in size, soft, friable and at times greyish in colour; fatty degeneration may be evident. The spleen is usually atrophic and the pulpa firm; the Malpighian corpuscles are indistinct. The lymphatic glands, particularly the parotid, prescapular and mesenteric glands, may be enlarged and hyperaemic. The kidneys may be congested or show a variable number of petechiae in the cortex. Petechiae may also be visible in the cortex of the adrenals and in the mucosa of the urinary bladder.

Changes in the alimentary tract vary a great deal. The rumen and reticulum contain a relatively small amount of ingesta; the mucous membrane may be diffusely red in colour. The omasum contains partially dehydrated contents. The mucous membrane of the abomasum or stomach may be red in colour. In the small intestine hyperaemia is usually marked in the duodenum, patchy in the jejunum, and fairly pronounced in the ileum. The mucous membrane of the large intestine is often hyperaemic, particularly so in the case of the caecum.

Apart from hyperaemia of the meninges no characteristic changes are observed in the central nervous system.

B. Microscopical lesions

No detailed histopathological study is available. Du Toit (1923) gives the following brief account:—

> "The most interesting pathological changes are those of the kidneys. Signs of an acute inflammation are shown mainly in the form of a glomerulonephritis. Haemorrhages may be present in the cortex. Sometimes hyaline cylinders occlude the tubuli contorti, the epithelial lining of which shows cloudy swelling. The liver cells show fatty degeneration and cloudy swelling, and their arrangement is disturbed. The pancreas shows practically no change, although there may be a slight hyperaemia and slight hyperplasia of the Langerhans cells. The spleen may show haemosiderosis and hyperplasia. Hyperkeratosis and necrosis are observed in the deeper layers of the skin. The sweat glands are sometimes distended. The mucous membranes of the nares, buccal cavity, larynx, pharynx and oesophagus show signs of inflammation and necrosis."

Diagnosis

Epizootologically, sweating sickness is a disease of place. When investigations are being made it is essential to determine the presence of the vector, the local incidence of sweating sickness and other diseases likely to be confused therewith, the origin and age of the sick animals, and whether or not the usual routine immunization against various infectious diseases has been practised. These considerations should guide the investigational procedures.

In nature sweating sickness has so far only been observed in cattle. The establishment of the susceptibility of sheep, goats and pigs suggests that this disease may also occur in these species in the enzootic areas (*vide supra*-Distribution). Its possible natural occurrence in sheep is suggested by the relatively large number of solidly immune animals (Table 9) raised in the Western Cape Province.

A diagnosis of sweating sickness can be made by considering the epizootology, the clinical symptoms and lesions at autopsy. This is possible where the affected animals are suffering from either the acute or subacute form of the disease. In sheep, sweating sickness can be differentiated from bluetongue if the above described criteria, namely the degree of the hyperaemia of the visible mucous membranes and skin, and the nature of the hyperaemia of the coronets of the claws are taken into consideration (*vide supra*—Symptomatology).

Difficulty in making a diagnosis is experienced when affected animals are suffering from either the peracute or mild form of the disease. It should be borne in mind that sweating sickness cannot be transmitted artificially from affected to susceptible animals. The application of a biological test can thus only reveal the presence of diseases other than sweating sickness. The determination of concurrent infections is of great value in making a differential diagnosis.

The peracute form of sweating sickness in cattle may be difficult to distinguish from the peracute forms of anthrax, anaplasmosis, babesiosis and heartwater. Their presence can be determined microscopically, while their absence suggests that sweating sickness may be the cause of the disease. In sheep the symptoms and lesions of the peracute form of sweating sickness may be confused with those of bluetongue. By applying the biological test the former disease can be excluded if the experimental sheep develop symptoms of bluetongue.

In the case of the mild form of sweating sickness in cattle, sheep, goats and pigs a tentative diagnosis can be established or excluded by challenging the immunity of suspects with " infective " ticks. Presence of an immunity confirms the diagnosis of sweating sickness.

Hyperkeratosis or X disease of cattle due to pentachlorinated naphthalene compounds has not yet been encountered in South Africa. This disease bears some resemblance to sweating sickness. In making a differential diagnosis enquiries should be made whether or not these wood-preserving compounds have been made use of by the owner.

TREATMENT

A. Specific treatment

There is no known drug that has a specific action on the causal agent of sweating sickness. The report of cures should be considered with full knowledge that spontaneous recovery is not uncommon. The procedures of treatment instituted during the last four decades have been empirical. The various compounds that have been employed, and the results claimed are listed in the appended Table 12. Consideration of these results in conjunction with observations made in practice by veterinarians and stockowners, permits one to conclude that no really satisfactory treatment has yet been found, unless the curative effects of liver meal and sulphamezathine are fully confirmed. It is significant that treatment with liver is hardly ever employed at present. The beneficial influence of sulphamezathine on the course of the disease is in all probability attributable to its bactericidal property which prevents or alternatively cures complications due to secondary bacterial invasion of the respiratory and alimentary tracts.

TABLE 12

Drugs which have been tried in the Treatment of Sweating Sickness

Drug	Effect on the Course of the Disease	References
Aspirin	—	Morris, 1941
Aureomycin	—	Neitz, 1957
Ferrous sulphate	+	Daubney, 1943
Formalin	+	Purchase, 1936
	+	Lawrence, 1946
Liver meal	++	Hudson, 1938
	+-+	Quin, 1943
	++	Lawrence, 1946
Penicillin	—	Neitz, 1957
Phenanthridinium	—	Neitz, 1957
Phenargen	—	Neitz, 1957
Quinine	—	Morris, 1941
Sulphamezathine	+?	Barnard, 1950, 1951, 1952
Trypan blue	—	Morris, 1941

— = Of no apparent value
\+ = Beneficial influence
++ = Significant response
+? = Questionable value

Therapy of sweating sickness is still a major problem, particularly on farms were systematic tick control is not practised.

B. Symptomatic treatment

This form of treatment is of great value, and many calves affected may be saved (Thomas, 1932; Clark, 1933; Quin, 1943; Lawrence, 1946). It is, however, important that treatment of sweating sickness should begin as soon as possible after the onset of symptoms. In practice, careful examination of blood-smears should be conducted in order to exclude the possibility of intercurrent infections.

The sick animal should be protected against unfavourable weather conditions, and great care should be taken to keep it quiet and to prevent bodily exertion. Careful nursing throughout the course of the disease is essential. The animal must have free access to water. Although the sick calf cannot suck, it is nearly always able to drink. It should be permitted to drink milk out of a bucket, or alternatively be hand-fed.

Prevention or treatment of myiasis is essential. The skin may be washed with a very weak solution of a disinfectant, and then be rubbed gently with Carron oil. Treatment with either sulphonamides or antibiotics should be employed to counteract secondary bacterial invasion.

PROGNOSIS

Prognosis should always be guarded. The losses from sweating sickness due to mortality may be higher than 75 per cent. The financial losses as result of emaciation and unthriftiness are also important.

PROPHYLAXIS

Prophylaxis against sweating sickness is based upon the elimination of the arthropod vector. The only effective weapon for destruction of ticks is regular, systematic dipping or spraying combined with careful hand-dressing.

The dipping of cattle in an arsenical dip (0·16 per cent $As_2 O_3$) at five-day intervals has been successfully employed by Hooper Sharpe (1935, 1936) in Southern Rhodesia. Van Heerden (1953) states that toxaphene dips are more efficacious than arsenical dips in the destruction of bontpoot ticks (*Hyalomma* spp.), and that systematic dipping in toxaphene should reduce the incidence of sweating sickness.

IMMUNITY

Experimental evidence on the nature and duration of the immunity in sweating sickness is limited. Tests conducted on two calves (No. 7487, 7700; Tables 13 and 14) which had developed an inapparent reaction, and on ten calves (Table 13), which had recovered from a clinically detectable reaction, showed that the immunity persists for periods of up to 13 months. Observations on a single calf (No. 6842; Table 13) disclosed that it was fully susceptible when the immunity was challenged 21 months after recovery. Experiments on the duration of immunity in swine established that two pigs (No. 1281, 1531; Table 11) which contracted an inapparent reaction, and one pig (No. 1532; Table 11) which recovered from a mild form of the disease were solidly immune when the immunity was challenged three months later. In all these tests "infective" *H. transiens* adult ticks had been allowed to feed to repletion.

TABLE 13

Observations on the Duration of Immunity in Sweating Sickness

Animal No.	Infested with Adult Ticks		Age of Animal	Ticks fed	No. of Ticks Attached	Nature of Reaction	Result	Immunity Test								
								Interval	Infested with Adult Ticks		Ticks fed	No. of Ticks Attached	Incubation Period	Duration of Disease	Nature of Reaction	Result
	Gen.	Batch No.							Gen.	Batch No.						
				Days							Days		Days	Days		
7643	5	2196 Ba 1..	8 mths..	3	12	NR*	—	29 days..	5	2200 Da 2	11	6	—	—	NR	Immune
								77 days..	4	2183 Aa 2	15	5	—	—	NR	Immune
7642	5	2198 Aa 1..	8 mths..	4	17	NR*	—	29 days..	5	2196 Fa 6	11	7	—	—	NR	Immune
								77 days..	4	2183 Aa 1	15	16	—	—	NR	Immune
7805	5	2198 Ba 1..	15 days..	4	15	NR*	—	23 days..	5	2198 Oa 1	8	8	—	—	NR	Immune
7807	5	2198 Ba 2..	7 mths..	4	24	+	Recovered	112 days..	6	2216 Aa 1	10	15	—	—	NR	Immune
7969	5	2198 Ba 5..	5 mths..	5	13	+++	Recovered	112 days..	6	2216 Ba 1	10	8	—	—	NR	Immune
7939	5	2198 Ba 4..	6 mths..	5	19	+++	Recovered	112 days..	6	2216 Aa 2	10	10	—	—	NR	Immune
6688	2	2118 Ca 3..	4 mths..	13	9	++++	Recovered	6 mths..	3	2142 Ca 3	9	24	—	—	NR	Immune
7518	3	2142 Ga 1.	4 mths..	10	6	++++	Recovered	10 mths..	4	2183 Da 4	9	6	—	—	NR	Immune
7422	3	2142 Ca 4..	9 mths..	9	13	++++	Recovered	12 mths..	4	2183 Ba 2	16	6	—	—	NR	Immune
6697	3	2142 Ba 3..	11 mths..	12	13	+++	Recovered	13 mths..	4	2183 Ba 5	15	6	—	—	NR	Immune
6843	3	2142 Ba 5..	8 mths..	12	12	+++	Recovered	13 mths..	4	2183 Ba 4	15	6	—	—	NR	Immune
7419	3	2142 Ba 2..	20 mths..	12	16	++++	Recovered	13 mths..	4	2183 Ba 5	15	6	—	—	NR	Immune
7487	3	2142 Aa 10	2 mths..	13	12	NR	—	13 mths..	4	2183 Da 3	15	5	—	—	NR	Immune
7491	3	2142 Aa 11	2 mths..	11	10	++++	Recovered	13 mths..	4	2183 Da 2	15	20	—	—	NR	Immune
6842	3	2118 Ra 1..	24 mths..	10	18	+++	Recovered	21 mths..	4	2183 Da 1	15	6	8	10	+++	Susceptible
7702	—	Control....	7 mths..	—	—	—	—	—	5	2200 Ea 1	8	6	8	8	+++++	Died
7633	—	Control....	11 mths..	—	—	—	—	—	4	2183 Ba 1	15	5	6	5	+++	Recovered
8065	—	Control....	5 mths..	—	—	—	—	—	6	2216 Ba 2	12	8	6	14	++++	Recovered
7637	—	Control....	8 mths..	—	—	—	—	—	5	2196 Fa 5	15	12	5	8	++++	Died
7972	—	Control....	5 mths..	—	—	—	—	—	5	2198 Ba 7	8	4	6	9	++++	Died

* The reasons for the calves not reacting are given under the heading " Aetiology " and in Table 2.

TABLE 14

Observations on the Susceptibility of Calves, the Progeny of Recovered Sweating Sickness Cows

Cow No.	Calf No.	Date of Birth	Date of Infestation	Age of Animal	Infested with Adult Ticks		Period before Tick Infestation of Cow	Period after Tick Infestation of Cow	Clinical Observations				
					Gen.	Batch No.			Incubation Period	Duration of Disease	Total Period	Nature of Reaction	Result
									Days	Days	Days		
3065.....	—	—	27/10/54	83 months.	3	2142 Ca 2....	—	—	7	4	11	+++	Recovered
	7491	6/8/54	4/10/54	2 months.	3	2142 Aa 11...	23 days....	—	7	7	14	++++	Recovered
	8065	24/10/55	28/3/56	5 months.	6	2216 Ca 1....	—	17 months.	6	14	20	++++	Recovered
	8821	24/11/56	10/1/57	2 months.	6	2242 Ba 1....	—	27 months.	7	3	10	+++++	Died
3665.....	—	—	27/10/54	81 months.	3	2142 Ca 1....	—	—	7	5	12	+++	Recovered
	7487	31/7/54	2/10/54	2 months.	3	2142 Aa 10...	25 days....	—	—	—	—	—	NR
	8639	3/10/56	14/12/56	2 months.	6	2242 Aa 1....	—	26 months.	9	6	15	+++++	Died
5300.....	—	—	28/4/54	84 months.	2	2118 Ba 10...	—	—	6	16	22	+++	Recovered
	6841	30/1/54	21/4/54	3 months.	2	2118 Ba 1....	7 days....	—	5	2	7	+++	Killed
	8408	3/4/56.	30/5/56	2 months.	6	2224 Da 1...	—	25 months.	6	6	12	+++	Recovered
4254.....	—	—	3/3/54	61 months.	2	2118 Qa 2....	—	—	6	2	8	+	Recovered
	6842	31/1/54	24/2/54	24 days....	2	2118 Ra 1....	7 days....	—	8	7	15	+++	Recovered
	7700	31/3/55	13/4/55	13 days....	4	2171 Sa 1....	—	13 months.	—	—	—	—	NR
	—	—	18/5/55	48 days....	4	2148 Fa 5....	—	14 months.	—	—	—	—	NR

It has also been determined that an immunity may be produced in animals on which " infective " ticks had fed for periods of either 72 or 96 hours. In comparing the results of the experiments on cattle (Table 2) and pigs (Table 3) it is apparent that the results, though closely parallel are not identical. Three calves (No. 7642, 7643, 7805) showed no apparent or detectable reaction to the primary interrupted tick feeding but were found to have developed a solid immunity. Such inapparent reactions followed by a solid immunity were not observed in pigs but there was brought to light the phenomenon of partial immunity, which may or may not be quantitative, as exemplified by pigs No. 1429 and 1411, and to a slightly lesser extent by pig No. 1396. It is possible that this is due to the ease with which a reaction can be detected in pigs.

During the course of these studies an attempt was made to determine whether or not a transmitted immunity (colostral milk immunity) occurs in calves, the progeny of cows which had recovered from sweating sickness. It should be borne in mind that the available information on the duration of the immunity is limited. At present it is known that it persists for periods of up to 13 months but that it may have disappeared as early as 21 months after recovery (*vide supra*). The observations described below should, therefore, be considered with full knowledge that the four cows (Table 14), which recovered from sweating sickness, might have lost their immunity at the time of calving, and that they were thus incapable of excreting antibodies in the colostral milk. The results of these studies are included in this discussion as they show that in certain circumstances a transmitted immunity to the offspring does not take place.

The results of the experimental observations are listed in the appended Table 14. They show that of the four calves born before the dams had contracted sweating sickness, three (No. 7491, 6841, 6842) were fully susceptible, while one calf (No. 7487) developed an inapparent form of the disease as evidenced by its solid immunity when challenged. Tests conducted on five calves born after these cows had recovered from sweating sickness revealed that four (No. 8065, 8821, 8639, 8408) were fully susceptible, while one calf (No. 7700) developed an inapparent reaction. These results clearly indicate that calves born out of dams at intervals varying from 17 to 26 months after recovery from sweating sickness, show more or less the same degree of susceptibility as those from fully susceptible cows. It needs to be determined whether or not calves born within the known immune period of 13 months will develop a transmitted immunity.

Consideration of the above-mentioned results, in conjunction with observations on the occurrence of sweating sickness in calves, the progeny of cows born and bred in highly enzootic areas, indicates that even if a transmitted immunity should occur, it is of little significance, if any, in materially influencing the incidence of this disease in calves.

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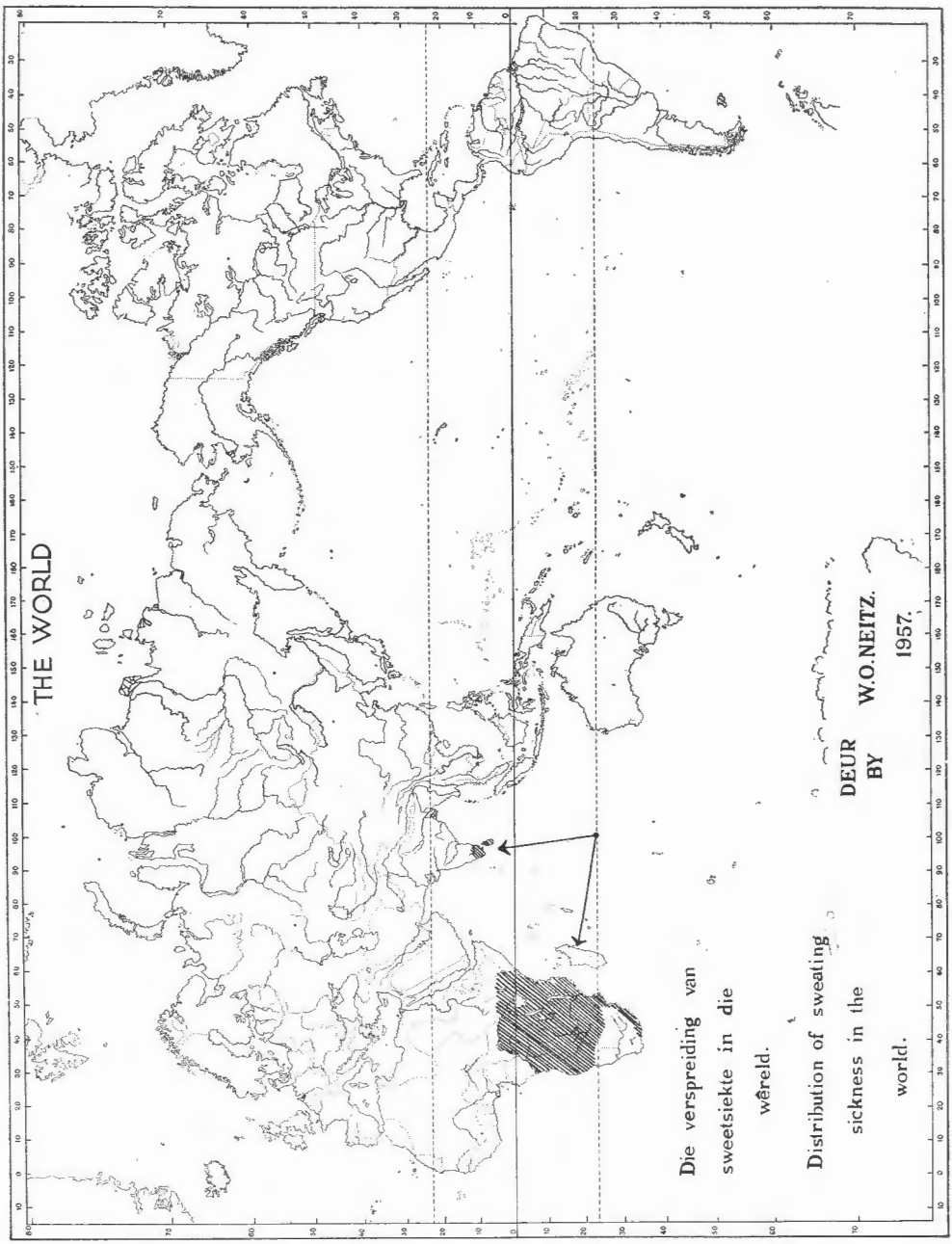
THE WORLD
Die verspreiding van sweetsiekte in die wêreld.
Distribution of sweating sickness in the world.
DEUR W.O.NEITZ. 1957.