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Edited by E. M. ROBINSON

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CONTENTS.

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NEWCASTLE DISEASE IN NATAL.

V. R. KASCHULA AND A. S. CANHAM (Pietermaritzburg),
A. M. DIESEL (Pretoria) AND J. D. W. A. COLES (Onderstepoort).

Newcastle disease was diagnosed in fowls in South Africa for the first time in May, 1945. The birds had been sent to Allerton Laboratory for examination by a farmer of Verulam.

Geographical Distribution of the Disease in Natal.

There is a certain amount of evidence to show that the infection was contracted by fowls belonging to Africans and Indians in the vicinity of the dock area of Durban about the end of September, 1944. Non-Europeans seldom submit pathological specimens to the veterinary laboratories for examination, and to this unfortunate circumstance must be ascribed the subsequent rapid spread of the disease.

There is no doubt, whatsoever, that fowls died from the disease at Mount Edgecombe in December, 1944; the sera of recovered birds neutralised known Newcastle disease virus when sent to England. Most probably the infection swept through the brickfields at Avoca at the same time.

Either because veterinary officials have seen the cases, or because accurate descriptions of the disease have been furnished by the poultry owners concerned, we know that the condition has been widespread.

Starting in the north, almost on the very banks of the Tugela river, fowls have died, often in very large numbers, at Fort Pearson, Darnall sugar mill, New Guelderland district and sugar mill, Stanger village, district and beach, Gledhow sugar mill, Melville sugar mill, Groutville, Glendale district, Mapumulo district, Kearsney tea estates and district, Maidstone sugar mill, Tongaat beach, Verulam district, Mount Edgecombe district and sugar mill, Duffs Road district, Inanda police post, Avoca, Redhill, Riverside, Umgeni, the old borough of Durban, Springfield, Sydenham, Clermont native township, Mayville, Clairwood, Clairmont, Rossburgh, Brighton Beach, Merebank, Reunion, Lamontville native township, Isipingo village, Illovo sugar mill, Umkomaas district, Esperanza sugar mill, Renishaw sugar mill, Sezela and Umtwalumi district. The disease does not seem to have spread further south.

From Durban, the infection was carried about twenty miles inland to Kloof, the intervening places being Westville, Escombe, Sea View, Malvern, Shallcross, Mariannhill monastery, Pinetown and Wyebank.

Generally speaking, Newcastle disease has been restricted to the sugar mills along the north and south coasts, and to the areas closely

populated or intensively farmed by Indians. Indeed, of the 100,000 fowls estimated to have succumbed, probably 95 per cent. were owned by Indians.

As a result of the diminished supplies of mutton due to the war, the Indians were compelled to fall back on poultry, and as most could not afford to buy birds, they took to raising them on a greater scale than before. In districts such as Clairwood and Sydenham, the Indian homes are built very close to one another and the six to twelve fowls belonging to each family roam through two or three backyards, generally roosting in the highest tree in the neighbourhood. Relatively few Indians have houses and runs for their birds, particularly as wire netting has been unprocurable since 1940, and those that do have pens let their fowls wander all day in search of garbage, insects and grass. In such circumstances, there was nothing to check the outbreak. Moreover, in contrast to what happens in the native reserves, Indians buy and sell fowls freely and transport them considerable distances.

At the sugar mills, the Indians usually live in barracks, and there is nothing to prevent the disease killing all the fowls owned by any one group of families, should it be introduced. Not infrequently a thousand birds of all ages are attached to a barracks. It has been a common experience to visit a barracks and find only ducks walking around, a grim reminder of the deadliness of the virus.

The flocks owned by Europeans are relatively well housed and fed. Indeed the conditions are as good as anywhere in the world. Where Europeans have suffered losses, they have almost invariably been due to the purchase, at tempting prices, of table poultry from Indian or African hawkers, eager to dispose of their remaining fowls. One European had 22 miserable-looking survivors of a fine flock of about 850 White Leghorns. Another lost over 600 Leghorns, every one he had, but Bantams running with these fowls for some unknown reason escaped; a dozen turkeys on the same farm also remained healthy, as have a turkey and also ducks elsewhere that have come in intimate contact with the infection. No other European seems to have lost more than 300 birds, and only three or four have been so unfortunate as this.

A few Africans have suffered losses, particularly those living in barracks at the sugar mills. It was feared that the disease would spread into the native locations surrounding the whole infected area, but so far only a few very minor outbreaks have occurred. There are reasons for this good fortune. Africans seldom buy fowls to take to their homes, and there is remarkably litle movement of poultry from kraal to kraal. When birds are moved, they are carried in baskets and crates to trading stores and railway stations for transport to Durban, where an excellent market exists. Very often, large

numbers are taken at the week-end for sale to the Indians at the sugar mills. From the locations, the poultry moves predominantly in a one-way stream to Durban and the sugar mills; there is practically no traffic in the reverse direction.

The Origin of the Outbreak.

Everything points to the infection having been introduced through the port of Durban. Since the virus can survive in carcases kept in cold storage for six months or longer, it is possible that the infection came straight from the East. The symptoms and autopsy findings, however, have borne such a close resemblance to those described by Hudson in Kenya, that is it almost certain that the disease has been brought by ship from some harbour on the East Coast of Africa. Hudson himself considered that the infection had spread even to Lindi in the south, and there are persistent rumours suggesting that the whole East Coast has become involved. Although Hudson diagnosed the disease accurately in Mombasa in 1935, it is doubtful whether these were really the first cases occurring in Africa.

In this connection, it will not be out of place to discuss briefly the alarming manner in which Newcastle disease is spreading all over the world. At one time it seemed to be confined to India, Ceylon, Burma, Indo-China, the Dutch East Indies, Malaya, the Philippines, Korea and Japan. Minor outbreaks, which were suppressed, occurred in both England and Australia.

The year 1935 was fateful, for it was then that the disease was diagnosed in Africa. On this continent it spread first across Kenya to the Congo, where it was found by Malbrant, and then south to the Union.

In 1935, too, the American continent experienced its initial outbreaks, in California, where the disease was described as a new one, under the name of avian pneumoencephalitis. The mistaken diagnosis was excusable, for the condition did not appear in its usual highly virulent form. The mortality rate was relatively low, young chickens seemed to be as susceptible as older fowls, and turkeys often sickened and died. In 1944, the exact nature of the infection was realised, but by then the virus had spread to the eastern seaboard, and outbreaks occurred in the states of New Jersey, New York and Massachusetts.

It is known, too, that the disease has swept through Italy and Palestine during the recent war, and the whole of Central Europe is menaced.

The Natural Disease in South Africa.

Only fowls have been found infected. There is a general belief, which seems to be well founded, that chickens up to two months old often show considerable resistance. Hudson made the same observation in Kenya. On many occasions ducks, particularly Muscovies, have

been exposed to the disease, but none has sickened. Turkeys have been said to be naturally susceptible to the Californian strain of the virus, but the few birds exposed naturally on two occasions here were resistant, and there is experimental evidence to show that the local virus is indeed innocuous to them.

On the whole the symptoms conformed to the usual pattern. Temperatures seldom exceeded 109°F. About half the cases had dyspnœa, extending their heads and necks during inspiration and retracting them when exhaling. Bubbling, gurgling, choking and squawking sounds often accompanied respiration and the noise was frequently so loud that the disease could be suspected in a yard before the fowls were actually seen. Another symptom was sneezing. In many instances there was diphtheritic laryngitis and small cheesy deposits were fairly common on the congested mucosa of the pharynx. Occasionally the tip of the tongue became necrosed for a distance of about 5 mms. and fell off — a symptom that does not appear to have been noticed elsewhere. There was always an excess of mucus in the mouth, and occasionally a thick ropy mass of it hung from the end of the beak.

The face was usually somewhat puffy. Slight nasal discharge was nearly always seen. Very often the cornea had a decidedly dull appearance. Frequently the eye had a vacant stare. As the disease progressed, the comb darkened.

The majority of fowls developed diarrhœa, sometimes greenish and at others whitish-yellow. Most birds, too, had nervous symptoms. The head might be pulled back or twisted. One or both wings often drooped. Leg weakness was common and then the fowl frequently lay on its side. Many birds crouched, and shuffled along on bended legs when roused.

Death occurred usually one to five or six days after the onset of symptoms and about 95 per cent. of cases succumbed. Some recovered birds were seen that still exhibited different forms of paralysis of the wings, legs or neck.

The morbidity rate varied considerably. On many farms all fowls sickened. On others, a third or even a half escaped infection, but then it was noticed that the birds were ranging over a comparatively large area. It is doubtful if the virus in most cases will persist in the soil for more than three days, so the fowls may easily escape ingesting it if their camps are sufficiently large. Furthermore, shedding of the virus is limited, for carriers have never been found; the infection dies out in the body during the process of recovery.

Some of the mouth and other lesions discernible at autopsy have already been described. The resemblance of our findings to those of Hudson in Kenya was noteworthy, particularly in so far as the laryngeal and tracheal lesions were concerned. The floor of the

larynx often showed mild to marked inflammatory changes, with the formation of a caseous plug, and the upper half of the trachea was also not infrequently involved. Hæmorrhage into the trachea has been seen. In one case an abscess 7 mms. in diameter was found in the submucosa behind the tongue. There was generally a catarrhal rhinitis. The lungs were usually unaffected, but some were slightly reddened. Occasionally the bronchi were blocked with aspirated tracheal exudate or blood.

The crop generally contained a fair amount of food, and sometimes a greyish sour-smelling fluid. More often than not the mucosa of the proventriculus showed petechiæ and ecchymoses, particularly near the junctions with the œsophagus and gizzard. Often there were red rings round the glandular openings. Gizzard lesions, when present, took the form of small hæmorrhages under the horny lining. Some degree of catarrhal enteritis was nearly always present. The duodenal mucosa often revealed petechiæ. Rarely the enteritis was hæmorrhagic in type and plaques might be seen as described by Malbrant in the Congo. Typhlitis was common. The mucosa of the large intestine between the cæca and the vent was occasionally intensely reddened, and even revealed diphtheritic lesions such as were seen in the pharynx.

A few petechiæ were often noticed in the abdominal and pericardial fat and even on the epicardium. The kidneys were often slightly swollen and greyish, due to degeneration. A low-grade fibrinous peritonitis characterised some cases.

Blood smears were negative. So also were cultures on ordinary and brilliant green agar and in ordinary and brilliant green broth.

Diagnosis and Differential Diagnosis.

The combination of respiratory, nervous and intestinal symptoms at once suggested Newcastle disease. The production of a fatal paralysis in pigeons by the subcutaneous injection of the infective agent more or less clinched the diagnosis. However, various experiments to be described later, were done to remove any remaining doubt.

The differential diagnosis involved a consideration of the following diseases:—

(1) *Infectious laryngotracheitis.* The respiratory symptoms and the marked laryngeal and tracheal lesions were highly suggestive of this disease. Laryngotracheitis, however, could be excluded because it is not pathogenic for pigeons, it is not reproduced by swabbing the cloaca of a fowl with the virus, and because typical intranuclear inclusions are often observed in the tracheal epithelium. In Newcastle disease, no such inclusions can be found.

(2) *Spirochætosis.* In the early stages of this disease nervous symptoms are common. Also there is diarrhœa. Examination of a blood smear will reveal *Spirochæta anserina.*

(3) *Fowl Plague.* There are more extensive hæmorrhages. Respiratory symptoms are absent. The intracerebral inoculation of white mice is fatal, but the injection of pigeons is not.

(4) *Roup.* Laryngitis, tracheitis, pharyngitis, sinusitis, rhinitis and conjunctivitis may all be seen. There is often dyspnœa, and frequently diarrhœa, but there are no nervous symptoms. The cause is *Hæmophilus gallinarum,* and this bacterium cannot produce coryza when injected subcutaneously or into the cloaca. The pigeon is not susceptible.

(5) *Fowl Typhoid.* There are no respiratory difficulties and the pharynx and larynx are not involved. The spleen is swollen. The liver is enlarged and light reddish brown or bronze in colour. *Salmonella gallinarum* is easily isolated on brilliant green agar.

(6) *Visceral Gout.* Large numbers of fowls may sicken and die. There are no respiratory or nervous symptoms. There is usually a catastrophic drop in general egg production. The disease has not been transmitted as yet to other fowls or experimental animals. In subacute and chronic cases, extensive greyish deposits of uric acid salts may be seen on the surface of the epicardium, liver and intestinal serosa.

(7) *Fowl Cholera.* There is no marked dyspnœa. Nervous symptoms are not pronounced. Rabbits usually die within 48 to 72 hours when injected with the bacterium, whereas they are insusceptible to Newcastle disease. *Pasteurella aviseptica* is usually visible in blood smears, and its growth is good on serum agar.

(8) *Neurolymphomatosis.* This is a chronic disease and though the nervous symptoms are marked, there is seldom dyspnœa or diarrhœa. The confusion arises only when one is confronted by paralysis in a bird that has recovered from Newcastle disease. An experienced diagnostician will generally appreciate the difference, but a careful examination of the nerves should be made.

Animal Inoculations.

FOWLS:

(1) Two hens (Nos. 1 and 2 on temperature chart) were injected subcutaneously on 15.5.45 with a normal saline suspension of tracheal scrapings from a natural case from Verulam. Both died on 20.5.45.

(2) One hen (No. 3 on temperature chart) was infected intranasally and dosed per os with the same tracheal scrapings in saline on 15.5.45 and died on 21.5.45.

(3) Two hens (Nos. 4 and 5 on temperature chart) had the upper end of the trachea swabbed on 16.5.45 with tracheal material from a dead fowl. These died on 21 and 22.5.45 respectively.

(4) Three hens had the cloaca swabbed with virus on 16.5.45 and died on 21.5.45.

(5) Two fowls inoculated intramuscularly on 25.5.45 with a suspension of infected fowl brain in saline died on 1 and 2.6.45 of the typical disease.

(6) Two hens injected intravenously on 25.5.45 with the pooled blood of two sick fowls died on 1 and 2.6.45 respectively.

(7) Two fowls were put in a small pen, on 25.5.45, that had just been vacated by sick fowls. They succumbed on 2 and 3.6.45 respectively.

(8) Mucus was collected from the trachea and pharynx of a sick fowl on 22.5.45 and it was mixed with saline and filtered through a Seitz EK special asbestos pad. No bacterial growth developed in liver broth seeded with the filtrate. On 22.5.45 the larynx of each of two fowls was swabbed with the filtrate, but no disease developed. After two weeks these same fowls were injected subcutaneously with virulent material and died in five days.

(9) One hen was injected subcutaneously on 18.10.45 with pigeon brain material (the virus had been passaged through 13 generations of pigeons infected intracerebrally), sickened on 22.10.45 and died on 24.10.45.

The following temperatures were recorded for five fowls artificially infected on the first day:—

Fowl.	1st Day.	2nd Day.	3rd Day.	4th Day.	5th Day.	6th Day.	7th Day.
	a.m p.m.	a.m. p.m.	a.m. p.m.	a.m. p.m.	a.m. p.m.	a.m. p.m.	a.m.
1	NT/107	107.2/NT	108.4/107.2	110 /109.4	108.4/NT	✠	
2	NT/106.8	107 /NT	107.4/108	108.5/109	107 /NT	✠	
3	NT/107	106.8/NT	107 /106.6	107 /107.4	108 /NT	108.2/NT	✠
4	107 /NT	107.5/106.8	108.8/108.2	109.4/NT	108.4/NT	103.8/✠	
5	106.8/NT	106.6/106.8	108 /108	109.2/NT	108 /NT	107.6/108.4	✠

NT = Not Taken

These readings tend to confirm the observations made in other countries that very high temperatures, or even moderately raised temperatures persisting for two or three days, do not characterise Newcastle disease.

TURKEYS:

(10) Two adult American Bronze turkeys were injected intracerebrally on 27.5.45 with mashed fowl brain material in saline, that had been stored in the refrigerator for 40 hours. No symptoms developed.

(11) Two adult turkeys remained healthy after being injected intramuscularly on 27.5.45 with the same inoculum used in experiment 10.

(12) Three adult turkeys were infected, both per os and subcutaneously, with the same material used in experiment 9 and remained well.

DUCKS:

(13) Two Pekin ducks did not sicken after being injected subcutaneously with a mixture of fowl tracheal scrapings and saline on 22.5.45.

(14) Two ducks were inoculated intramuscularly on 25.5.45 with fowl brain material suspended in saline. The results were negative.

(15) Two ducks were injected intracerebrally on 25.5.45 with a saline suspension of the mashed brains of two fowls. On 31.5.45 one duck showed inco-ordination of movement and the symptoms got worse until death supervened on 3.6.45. The other duck showed mild nervous symptoms from 2.6.45 to 5.6.45, but then recovered.

PIGEONS:

(16) Two pigeons were injected subcutaneously on 22.5.45 with fowl tracheal exudate mixed with saline. One showed drooping of the wings on 28.5.45 and the other on the following day. Leg weakness and paralysis developed and the birds died on 30.5.45 and 1.6.45 respectively.

(17) Two pigeons dosed per os on 22.5.45 with the same virulent material used in experiment 16 remained well.

(18) Two pigeons were infected intracerebrally on 25.5.45 with fowl brain mash mixed with saline. Nervous symptoms involving the neck, wings and legs developed on 29 and 30.5.45 rspectively, followed by death within 24 hours.

(19) Two pigeons were injected subcutaneously on 16.6.45 with the mouth washings of two pigeons which had just died of the artificial disease. The nervous symptoms characteristic of Newcastle disease were manifest in 10 and 12 days respectively, and death supervened on the 13th and 14th days.

(20) The virus was passaged intracerebrally through 13 generations of pigeons. Brain material from a seventh generation pigeon was injected subcutaneously into a fowl and killed it. An infected brain of a thirteenth generation pigeon was stored in the refrigerator for 74 days and was then used to infect more pigeons by the subcutaneous route; the pigeons died. Most pigeons died within two to three days after being infected intracerebrally.

(21) Mucus collected from the trachea and pharynx of a sick fowl was mixed with saline and immediately passed through a Berkefeld medium porosity candle on 8.6.45. Bacterial cultures made in liver broth and on liver agar before filtration were positive, but cultures of the filtrate were negative. Two pigeons were injected subcutaneously with unfiltered material. The filtrate was injected subcutaneously into two pigeons, intraperitoneally into one, and intravenously into one. All the pigeons developed paretic symptoms on 12.6.45 and died on the 14th and 15th.

Thus the causal agent passed through a Berkefeld medium porosity candle.

(22) On 5.6.45 tracheal and pharyngeal mucus of a sick fowl was mixed with saline and passed through a Seitz EK special asbestos pad. The unfiltered material was heavily charged with bacteria and killed two pigeons, injected subcutaneously, in six days after they had shown the usual symptoms of paralysis. Cultures of the filtrate in liver broth remained negative. The filtrate was used, immediately after being prepared, to infect two pigeons subcutaneously and one intraperitoneally and one intravenously, but all remained normal.

Besides showing nervous symptoms the pigeons often developed a greenish diarrhœa. At autopsy, very few lesions have been noted in pigeons, but petechiæ and even extensive ecchymoses have been encountered in the mucosa of the proventriculus, and catarrhal enteritis has been a common feature.

WHITE MICE:

(23) Eight white mice injected intracerebrally on 25.5.45 with fowl brain mash in saline failed to develop symptoms.

GUINEA-PIGS:

(24) Four guinea-pigs were inoculated subcutaneously on 22.5.45 with fowl tracheal exudate mixed with saline. They remained normal.

RABBITS:

(25) Three rabbits were injected subcutaneously on 22.5.45 with a mixture of blood and liver suspended in saline, but did not sicken.

Experiments at Weybridge, England.

Through the courtesy of the Director of the Veterinary Research Laboratories at Weybridge, Mr. N. Dobson kindly examined nine samples of serum collected from recovered fowls (some of which showed various forms of paralysis) on two farms at Verulam and one at Mount Edgecombe. Fowls survived that were inoculated with mixtures of 0·1 cc serum and 0·001 cc, 0·01 cc and even 0·1 cc virus. Other birds died that were injected with mixtures of serum and fowl plague virus.

Viability of the Virus.

The ability of the causal agent to survive on infected premises was investigated. A pen in which a number of diseased fowls had been kept, and in which direct contact experiments had been successfully conducted, was used for the purpose. The pen had not been cleaned out while previously in use, and even the food and water left after the last two fowls died on 2.6.45 remained. The pen was not exposed to direct sunlight and the atmosphere remained cool while it was vacant. On 16.6.45 two healthy Rhode Island Red hens were transferred to the pen, but were still normal three weeks later.

Apparently the virus died within the fortnight the pen was vacant.

All investigators, except one, have observed the inability of the virus to persist for more than about ten days under ordinary natural conditions, such as in water or soil or the decomposing carcase. Generally, the virus is dead in three to seven days. The exception was Dobson, who reported that chicks were said to have contracted the disease when put in battery brooders which had housed sick birds seven weeks previously .

General Remarks.

In this description of Newcastle disease in Natal, it will be as well to mention a few other facts that have been determined by other investigators. A virus is the etiological factor and the size of the particle has been estimated at 80 to 120 $\mu\mu$. Fowls are the main sufferers, but the natural disease has been diagnosed in turkeys in California, and once in pigeons in India. The rôle of wild birds in the epizootology is still unknown. Carriers of the virus have never been found and no insect has been shown to be a vector.

All the evidence points to the vast majority of cases being due to the ingestion of the virus. The virus can, of course, gain entrance through the nostrils or the conjunctiva. The birds sicken so rapidly that it is most doubtful if many cases can be ascribed to coitus, even though this is possible.

The disease is most contagious, but it is remarkable how it fails to spread from house to house when the fowls are kept under restraint. For instance, we had fowls dying in pens only five or six feet away from many other hens in cages, yet the latter remained healthy. Of course, care was taken to have different attendants feed and water them, but no wall interposed between the two groups.

DISCUSSION.

One is struck immediately by the resemblance of this condition to that described in fowls in Kenya by Hudson, and subsequently proved to be Newcastle disease at Weybridge in England. It is true that Hudson stated that an outbreak in turkeys coinciding with one in fowls had been reported, but he apparently did not experiment on turkeys, and the condition may well have been something else, such as fowl typhoid. We have found turkeys insusceptible both to the natural and artificial disease. Obviously the Natal strain of the virus differs from the Californian, which not only kills turkeys, but is less virulent for fowls.

The diagnosis of Newcastle disease was based on the following facts:—

(1) Fowls were extremely susceptible, and both the morbidity and mortality rates were very high.

(2) Respiratory, nervous and intestinal symptoms were all present.

(3) Some fowls had ropy mucus hanging from the beak.

(4) Ducks escaped the disease.

(5) Pigeons injected subcutaneously developed the typical nervous symptoms and diarrhœa.

(6) White mice, rabbits and guinea-pigs were resistant to infection.

(7) The causal agent was apparently a virus that could pass through a filter capable of holding back bacteria.

(8) The agent survived for less than 14 days in an infected pen, but lived for even 74 days in the refrigerator.

(9) The sera of fowls, alleged to have recovered from the infection, neutralised a known strain of Newcastle disease virus (Dobson's experiments).

Control Measures.

When the disease was diagnosed, an immediate survey was made of the Natal coastal belt to ascertain how far the infection had spread. A meeting of poultrymen in Durban was very well attended, and the position was explained to them. The assistance was sought, and obtained, of the Police, the South African Railways, the African, Indian and European press, the Durban Corporation, and various bodies such as the Indian Bus Owners' Association. The sale of poultry was prohibited on the Native Market, and on the Indian and Squatters Markets.

An emergency measure, Government Notice 1056 of 22.6.45 was soon replaced by Government Notice 1328 of 27.7.45 and the text of this is given here:—

NEWCASTLE DISEASE.

"It is hereby notified for general information that the Minister of Agricuture and Forestry has, under the powers vested in him by section *nine* of the Diseases of Stock Act, 1911 (Act No. 14 of 1911), as amended, declared the area defined in clause 1 of the Schedule hereto, and referred to as the infected area, an area infected with Newcastle disease, and has further under the powers vested in him by section *sixteen* of the said Act made the orders and imposed the prohibitions specified in the Schedule hereto.

Schedule.

(1) In this notice, unless the context otherwise indicates—

"dressed poultry" means the carcass of any poultry or of any of the non-domestic varieties of guinea-fowl, ducks or geese, from which the feathers, head, legs and internal organs have been removed;

"infected area" means the area comprising the Magisterial Districts of Durban, Inanda, Lower Tugela, Pinetown and Umzinto, in the Province of Natal;

"poultry" includes domestic poultry, turkeys, domestic guinea-fowl, ducks, geese or pigeons and the chickens of all these birds;

"prohibited area" means the area comprising the Magisterial Districts of Camperdown, Eshowe, Mapumulo, Mtunzini, Ndwede, Port Shepstone, Richmond and Umlazi, in the Province of Natal.

(2) No person shall move or cause to be moved any poultry into, out of or from one place to another within the infected area except under the authority of a written permit issued by a Government Veterinary Officer and subject to such conditions as he may specify therein; provided that this prohibition shall not apply in the case where pountry is despatched direct to the Market Master, c/o Municipal Abattoir, Siding 527, Berea Road, Durban, or to the abattoir of the Federated South African Meat Industries, Maydon Wharf, Durban, for slaughtering purposes.

(3) No person shall move or cause to be moved any poultry carcass or portion thereof or any poultry manure or feathers (except feathers contained in manufactured articles) into, out of or from any one place to any other place within the infected area, except under authority of a written permit issued by a Government Veterinary Officer and subject to such conditions as he may specify therein; provided that this prohibition shall not apply in the case where dressed poultry is moved into or from one place to any other place within the infected area.

(4) No person shall move or cause to be moved from the infected area any crates or boxes in which poultry has been kept, except with the written permission of a Government Veterinary Officer, and subject to such conditions as he may prescribe.

(5) All owners or persons in charge of premises or property in any area within the infected and prohibited area and upon which there have been or are poultry infected with Newcastle disease shall immediately disinfect all poultry houses, crates, boxes, food and water containers, bags and other articles with which such infected poultry has been in contact. Such disinfection shall be carried out by total immersion for half an hour in a solution containing 5 per cent. carbolic acid (Cresol) or Hycol or by thorough spraying with a solution containing 10 per cent. carbolic acid or Hycol or by total immersion in water kept boiling for 10 minutes.

(6) No person shall move or cause to be moved from the prohibited area any poultry, any poultry carcass or portion thereof, any poultry manure or feathers (except feathers contained in manufactured articles) or any poultry crates and boxes in which poultry are or have been kept, except under authority of a written permit issued by a Government Veterinary Officer and subject to the conditions specified therein; provided that this prohibition shall not apply in the case where poultry is despatched direct to the Market Master, c/o Municipal Abattoir, Siding 527, Berea Road, Durban, or the abattoir of the Federated South African Meat Industries, Maydon Wharf, Durban, for slaughtering purposes, or, in the case where dressed poultry is moved into the infected area.

(7) Government Notice No. 1056 of the 22nd June, 1945, is hereby repealed.

NOTE. — For the purpose of this Government Notice "place" shall mean —

(1) a farm, subdivision of a farm or that portion of a farm or subdivision to which poultry have been confined by a Government Veterinary Officer; and

(2) in the case of a native location or native reserve, the area defined for the purpose of dipping cattle or an area within the said native area to which poultry have been confined by a Government Veterinary Officer."

Owing to the lack of wire netting due to the war, and to many other difficulties, it was impossible to order all poultry to be kept in pens or to adopt a slaughter policy. But with the ready co-operation of all sections of the community, the disease was eradicated. Only 527 birds were actually killed, by order of the Government, and this was done to put an end to the last smouldering focus of infection in Lamontville Native Township. Of the 100,000 fowls estimated to have succumbed, only about 3,000 died after the first steps were taken to combat the disease.

SUMMARY.

Newcastle disease broke out in Durban towards the end of 1944. The diagnosis was confirmed by serum-virus neutralisation tests.

The infection was probably introduced by ship from some East African port.

The disease was confined almost entirely to the sugar-cane belt of Natal and comparatively few fowls not belonging to Indians were lost.

Ducks and turkeys remained healthy when exposed to the natural disease. Pigeons injected subcutaneously showed nervous symptoms and diarrhœa and then died.

The infection has apparently been completely eradicated from Natal. Practically all that was done was to prohibit all movements of poultry and poultry products, except under permit. The widespread co-operation of all sections of the public in the districts involved enabled these simple measures to prove highly effective. The disease just burnt itself out on the premises where it existed.

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A METHOD OF GRADING RAW MILK, WITH SPECIAL REFERENCE TO THE GRADING OF MILK FOR SOUTH AFRICA.

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(*Continued from December,* 1945, *issue, Vol. XVI. No.* 4.)
(*Tables* 14, 15, 16 *and* 17 *appear at end of article.*)

CHAPTER 8.

GENERAL DISCUSSION OF RESULTS:

PART I: PRINCIPLES OF GRADING.

The object of this report has been to present for consideration a practical method of grading milk and to apply this method to data collected in South Africa in order to assess the applicability of the test to South African conditions and also to make available a large volume of routine data which has been collected. In discussing the method which is being suggested, it is of vital importance to consider certain basic principles involved in grading, since the ignoring of these principles in South Africa has led to entirely false impressions being built up regarding milk quality.

Paragraph A: Important factors in sampling and testing.

It has been urged, and evidence has been presented in support of the contention, that the keeping quality of milk supplied by any farmer cannot be judged by the testing of occasional random samples. The fairest system for all concerned is to test daily or if that is an unattainable ideal, at least to aim at testing about five times a week. A far clearer picture of "dairy hygiene" or "keeping quality" will be obtained from an approximate test done frequently than from a very accurate test which can only be done occasionally because of its complexity. This point is stressed because in South Africa the tendency is to regard the Breed count and methylene blue reductase tests as infinitely less accurate than the total plate count. Whether this attitude is justifiable or not can best be judged by a study of the report of Wilson *et alia* (1935), but even if the plate count is by far the most reliable test, it can not be contended that this test could be applied daily to large numbers of samples, without having a tremendously unwieldly laboratory organisation to do the work.

It has also been urged that tests should be made wherever possible upon composite samples of the whole bulk and not upon samples representing a mere fraction of the total output. In the case of producer-distributors, who bottle their milk as fast as it is withdrawn, the collection of composite samples is virtually impossible, but in the case of most other classes of milk producer it is generally possible to collect something approaching a bulk sample.

The question of the " Latent Defects " of apparently clean milk has again been raised and the need for holding milk at atmospheric temperature for a specified minimum lapse of time is once again urged. Milk should be held at atmospheric temperature for at least 8 hours before it is subjected to laboratory test, but truer results will be obtained if this storage period is longer. The methylene blue reductase test will reveal " Latent Defects " after a shorter storage time than is required by any bacterial counting method. In general 8 hours may be a long enough storage period for the reductase test done at 40°C., but for the reductase test done at atmospheric temperature or for the Breed count 12 to 18 hours storage is needed.

Paragraph B: Choice of grading test.

Quite obviously this subject can only be discussed upon the basis of already well-tried tests and it must be clearly recognised that some new test may be devised which will entirely alter the whole picture. Existing tests which might be applicable to grading consist of either assessing the number of bacteria present, or else of assessing the metabolic activity of those bacteria.

The total plate count and the Breed count fall into the first category and the relative merits of these two tests have already been referred to. Whatever advantage the plate count has to offer in the line of greater accuracy (and this is a debatable point), the test is too cumbersome and expensive to carry out upon a large scale, results only become available two days after sampling, the test gives little or no information regarding bacterial types, results can be ruined by surface spreaders and finally the test gives no information regarding mastitis contamination and pus contamination. The Breed count, though often alleged to be less accurate than the plate count is simple to perform, and can be applied daily to many samples. The test takes cognisance of all those bacteria which grow in the plate test and a whole lot of other bacteria as well. The problem of bacterial clumps applies equally to both tests and the solution in each case is the same. Breed test results can be obtained in a matter of minutes and priceless information can be obtained regarding contamination with mastitis milk and with pus cells.

Tests of metabolic activity of bacteria are best exemplified by the methylene blue reductase test, though others like the rezazurin test are also used. Like the Breed count, the reductase test is eminently suited to the testing of many samples at a time, and results are available on

the same day. Being a test of bacterial activity it probably offers a truer reflection of the keeping quality of milk but it gives no information regarding bacterial types and no information regarding mastitis and pus.

For the prosecution of a country-wide grading scheme the test is unique in that the reading comprises a clear-cut colour change from bluish to colourless. This reasonably clear-cut end point eliminates to a large degree the human factor in the reading of reductase results. Certain objections are raised periodically against this test, but they are trivial in nature compared with the advantages, which have been discussed. There has been no opportunity of carrying out extensive comparative work using the reductase test incubated at atmospheric temperature similar to that which has been adopted in Scotland.

Reviewing the foregoing, it is clear that the methylene blue reductase test is the best suited for a grading scheme, but it should not be used alone but should be supplemented by the Breed count wherever a producer is giving unexpected results or where mastitis control is contemplated. This being the case it is unfortunate that this report is largely concerned with the Breed count. By far the majority of serial results of milk testing of commercial supplies have been collected by the army inspectorate. This inspectorate began working during wartime in makeshift laboratories and using makeshift apparatus. Under such circumstances it was only possible to do a very limited amount of reductase testing and the majority of reductase results reported were collected by the Johannesburg Municipal milk-testing laboratory.

Paragraph C: Analysis of results by Good: Bad ratio method.

Whatever milk test is used, the testing of all producers four or five times a week results in the collection of unintelligable masses of figures which have to be submitted to some form of analysis before they can be used. Examples have been quoted in Table 1 to show the striking fluctuations in results achieved even by careful and enthusiastic producers. The occurrence of such fluctuations is admitted by most testing authorities and they make allowance for such fluctuations on a discretionary basis. When a recognised good producer shows a bad result he is warned and is retested. In the case of a bad producer the bad result is just another added to the long list of black marks. For satisfactory and fair grading, however, it is necessary to develop a much more uniform way of dealing with fluctuations in result, so that every result plays a proportionate part in determining the ultimate grade earned. It cannot be too strongly emphasised that to lay down a strict numerical standard and then to apply that standard upon a discretionary basis spells disaster to both the value of the standard itself and to any grading scheme.

In an attempt to make allowance for certain fluctuations in bacterial count or keeping quality, and at the same time to lay down an unequi-

vocal method of deciding what constitutes reasonable and what unreasonable fluctuations, a method has been devised of recording the proportion of good and bad results in any series of tests. The details of the method have been fully explained in Chapters 3 and 4 and need not be repeated in detail. It is suggested that an arbitrary division between good and bad should be a Breed count of 1,000,000 and a reductase time of 3¾ hours. Any test result better than these figures is classed as good and any worse than this as bad, and a series of results is recorded as the proportion of good results to bad ones or in other words the Good: Bad ratio.

One obvious practical advantage of a system such as this is that the reading of results is greatly simplified. With the Breed test it is only necessary to ascertain if the count is below or above 1,000,000 per cc. Many Breed smears can be read at a glance and only borderline cases need to be carefully counted. With the reductase test only a single reading needs to be made, and the administrative problems of reading a test which has to be read at intervals for five hours is eliminated.

Paragraph D: Choice of Good:Bad ratio as a Breed count standard.

Good: Bad ratios of many producers Breed count series have been examined to find a reasonable basic standard for fluid milk. From a Good: Bad ratio scatter graph (see Table 4) it would appear that the highest standards likely to be maintained are:—

Summer (September to March inclusive) Good: Bad ratio 2: 1.
Winter (April to August inclusive) Good: Bad ratio .... 5: 1.

In terms of the Breed count this would mean that a producer must register two counts below 1,000,000 in summer and 5 counts below 1,000,000 in winter for each count which falls above that level.

On casual inspection these basal seasonal standards seem absurdly low but when the matter is examined in the light of available figures very few producers can conform to these standards throughout the year. Out of 13 producer-distributors situated in Johannesburg 11 had no difficulty in maintaining standard, one failed during one month and one failed completely to maintain standard. Producer-retailer milk unfortunately comprises only one-thirteenth of Johannesburg's total supply. Of farmers supplying milk to raw-milk-shops and pasteurising depots the figures in Table 7 show that at Umlaas Road, Natal, from 50 to 85 per cent. of farmers consistently maintain standard, whilst at Pietermaritzburg the percentages are slightly lower. At East London the figure varied from 50 to 100 per cent. These are the only large centres where satisfactory results have been registered. At both Cape Town and Durban the figures fell below 50 per cent., whilst the high-veld centres gave results that indicated all too clearly the inability of farmers to maintain the suggested standards consistently. Details of these finding are not recapitulated here as they are set out fully in Table 7.

From the foregoing review it is therefore apparent that although very low basal standards were chosen, dairy farmers as a whole are not at present competent to maintain these standards. If farmers cannot maintain a low standard which allows for a proportion of grossly unsatisfactory results, how much less are they likely to be able to maintain some definite numerical standard such as 150,000 bacteria per cc., if that standard is to be applied upon a strict and not upon a discretionary basis.

Paragraph E: Validity of Good:Bad ratios as a method of analysing results.

The most obvious objection to the Good: Bad method of analysis, lies in the arbitrary choice of a dividing line between good and bad. It can be argued that a producer who regularly gives a count of 990,000 is classed as always good whilst another producer giving a count of 1,020,000 is always bad. This objection is admitted, but it is one which cannot be escaped in grading. Whatever grade or figure is chosen the producer who just exceeds that figure is invariably unfairly treated as compared to those who just achieve the grade. As an objection, however ,it is purely theoretical in character because in practise producers never do give results consistently just above or just below any given level. The best that a producer can achieve is to show results fluctuating around the chosen dividing line and the Good: Bad ratio system shows whether the fluctuations predominate on the good or the bad side of that line.

Another objection is that the ratio system tends to exaggerate the true state of affairs, thus a producer giving 20 good results and 1 bad one has a ratio of 20: 1 whereas an extra failure would drop his ratio to 10: 1. This objection is also admitted but it is an unimportant one. A producer is still excellent whether his ratio is 20: 1 or 10: 1 and this exaggeration affects all producers equally provided all have been tested a reasonable number of times. Actually the exaggeration is only noticeable at the extremes whereas around the basal dividing line the ratio is a very accurate reflection of the merits of the producers.

The only other point worth discussing at this juncture is whether there is justification for considering any standard which on the face of things appears so much lower than overseas standards. The only basis of comparison available is the methylene blue reductase test. Acceptable reductase standards overseas are 5 hours for winter and 4½ for summer and elsewhere the times are 4¼ and 4 hours. The standard proposed here is:—

1. *Seven-month-summer-standard.* Two counts below 1,000,000 to one above, or two reductase tests lasting 3¾ hours to one reducing in that time.

2. *Five-month-winter standard.* Five counts below 1,000,000 to one above, five reductase tests surviving 3¾ hours to one reducing in that time.

However unpalatable it may be, the fact remains that even with these low suggested standards a large proportion of our farmers cannot maintain them, nor for that matter is it entirely clear to what extent they could be maintained by overseas producers. It is only when results are available, of several years of regular testing done four or five times a week that one can decide whether or not the suggested standard can be maintained.

Paragraph F: Application of Good: Bad ratio to the Methylene Blue Reductase Test and to the Total Plate Count.

In Chapter 7 attention has been given to the practical application of this method of analysis to methylene blue reductase results. When the suggested criteria were applied to several series of tests it became clear that the reductase standards were appreciably harder to maintain than those of the Breed count. It seemed necessary to inquire more closely into this point and for that purpose a further series of correlations were worked out comparable to those recorded in Table 3. This correlation series has been worked out from the comparison of 6,764 parallel Breed counts and reductase tests, but for the present purpose attention must be focussed upon those Breed counts falling below the million level. Of the 6,764 parallel counts 5,022 fell below this level and are listed in Table 13B in which the monthly variation in percentages is indicated. The figures in this correlation table are extremely interesting and give material for much speculation. In the first year June, 1943, to May, 1944, there was very close correlation between a low Breed count and a long reduction time. Incidentally a comparison of the two lines of totals at the bottom of the table indicate that the results were on the whole good, which means that the bulk of the producers were trying to maintain a reasonable level of dairy hygiene. In the second year, however, more producers were included in the survey and as result an entirely different picture materialised. In the cold winter months the close correlation between a low count and a reduction time of 4 hours persisted, but in the spring, summer and autumn months of August to April this close correlation disappeared entirely. In these months there was a striking increase in the percentage of low count milks which reduced in 3½ hours and an appreciable number even reduced in very much shorter times. The only explanation of this is that the producers brought freshly into the survey in the second year were of a different calibre compared to the first group, and their milk frequently showed some degree of Latent Defect which remained entirely cloaked in winter but in summer weather became uncloaked by the reductase but not by the Breed test. This uncloaking of latent defects in 8-hour milk by either test, depends entirely upon the degree to which the milk has become contaminated. The only certain way of showing up this fault is by storing the milk 12 to 18 hours at atmospheric temperature between withdrawal and testing.

This discrepancy between cold weather and warm weather results, with a decrease in close correlation between the two tests during warm weather, accounts for the apparent inability of producers to maintain the suggested reductase standard for summer months. It does not affect the usefulness of the test however, but rather it serves to re-emphasise the danger of ignoring the ' Latent Defect ' factor attempting to grade milk which is too freshly drawn or has been refrigerated and held at a low temperature until tested.

No work at all has been done upon the application of this form of analysis to the total plate count, but obviously it could be applied in exactly the same way as it is applied to the Breed count. It would, however, be necessary to collect large numbers of plate counts made upon random samples of milk in order to decide upon a workable dividing line between good and bad as judged by that test. Very definitely this figure could not be based upon any of the multiplicity of figures that have been laid down as standards by overseas workers. In any case the application of this method of analysis to the plate count is not a matter of importance because it is highly improbable that milk supplies could ever be graded daily by any test as cumbersome, expensive and difficult to perform as the plate count.

Paragraph G: Application of the Good: Bad ratio to high grade milk.

One of the points made by Wilson *et alia* (1935) in their report on grading is that the Breed count is of no value for classifying high grade milk, and they, together with American workers, are inclined to revert to the plate count, for assessing the bacterial content of such milk. Since no high grade of milk has as yet been established in South Africa and since no opportunity has been available during the present enquiry, to carry out extensive plate count work, data is not at hand upon which a clear-cut expression of opinion can be based. Nevertheless the results of the testing of producers listed in Table 8 have been reanalysed with a view to investigating the feasibility of classifying high grade milk by means of a Breed count Good: Bad ratio calculated as Count Group A: B+C+D+. In other words Good counts must be 300,000 or less.

In Table 14 is listed the classification of the 13 producer-distributors (originally listed in Table 9) where the Good: Bad ratio is taken as A+B: C+D+E, and the tentative standard Good: Bad ratios of 2: 1 in summer and 5: 1 in winter are reapplied. Only 5 of the 13 suppliers maintained standard during summer and during winter. It would appear therefore that this method of analysis does offer some promise of being a satisfactory method for grading high-grade milk, but much more investigation is necessary before any decision could be reached. Furthermore, comparable reductase results running a full five hour span are also required for comparison purposes, whilst the question of coliform content should also be considered as a possible test for the grading of high-grade milk.

Part II.

GRADING OF SOUTH AFRICAN MILK SUPPLIES.

The application of a very indifferent Good: Bad ratio standard to South African milk supplies (see Table 7) shows all too clearly how erratic is the general standard of keeping quality of milk in this country, for it was only in certain areas that producers even attempted to maintain the suggested standards. Apart from this general irregularity of quality, which itself indicates haphazard and erratic methods of milk production and handling, two major points emerge from the foregoing investigations.

Paragraph A: Difference of the average quality of milk in various areas.

The best examples of this are the results shown by farmers in the Umlaas Road and Pietermaritzburg areas on the one hand and in the Witwatersrand and Pretoria areas on the other. It is far from easy to explain why such differences should exist, and certainly no single factor can be entirely responsible for them. Those factors which probably play a major part are listed below.

(a) *Length of carry.* This term is used to indicate the distance milk is transported to market. In the case of Umlaas Road and Pietermaritzburg about the longest distance that milk has to be transported is 30 miles, the majority of producers falling within this radius. Moreover all grading of farmers in those areas has been done upon milk withdrawn the same morning and a maximum of 8 hours old when tested. In Johannesburg, however, many of the suppliers listed in Tables 6 and 7 rail milk from 50 to 250 miles to market, the milk being 18 hours old or very much older when it arrives (see also Chapter 6, paragraph C, section a). Naturally this is an important point in the differing results obtained from these two regions but it is not the full answer because the first 42 Johannesburg producers listed in Table 6 are relatively nearby producers who were graded upon an 8-hour basis, and a similar group of producers are listed for Pretoria in Table 7. Neither of these groups of farmers compared with those in Natal. Actually if an attempt is made to grade producers by some handicap system whereby an allowance is made for the length of carry one is forced to the conclusion that on the whole "long-distance" farmers are better dairymen than the nearby ones. In Chapter 6, paragraph C, section (a) farmers Nos. 194 and 198 whose milk may be as much as 64 hours old before reaching the market have been quoted. Whilst these are extreme cases they are not by any means the only 'long-distance' producers who put up a good performance if allowance is made for transportation problems. This same contention was raised by Pullinger (1944) in connection with souring losses (see Tables 8 and 12 of the article quoted). In one case at least it is not even necessary to make allowance for the distance travelled. A producer in the

Port Elizabeth area is situated 50 miles from town and has a retail delivery round of 20 miles, consequently the last milk to be delivered travels about 70 miles. Deliveries are only made three times a week i.e. on Monday, Wednesday and Friday. Consequently milk withdrawn on Friday is stored on the farm until the Monday delivery, and the consumer continues to use that milk until the Wednesday delivery arrives.

The obvious interpretation of the foregoing is that some of the most hygenically produced milk comes from far-distant parts, because it is only by meticulous attention to the principles of hygienic dairying that such milk can arrive in a marketable state. This is an argument which is used to sustain the traffic of long-distance fluid milk, and is a cogent one. The fact remains, however, that as long as milk is shuttled about the country in small cans, completely outside of the control of both the producer and the distributor, long-distance milk will arrive at market nearly if not quite sour. This contention is evidenced by the figures given in Tables 4 – 7. Clearly if 'long-distance' milk is to be drawn upon in the future, and with the high capitalisation of land around big cities this seems inevitable, such milk will have to be better protected during transit. The following section contains a discussion of the temperature factor which also covers this aspect of the problem.

(b) *Temperature of milk.* All too frequently milk is improperly cooled on the farm, an aspect which was considered fully by Pullinger (1944). Additional to this no adequate facilities at present exist for protecting milk from atmospheric conditions during transit. In table 15, figures are given to show the striking effect temperature changes can have on milk quality.

The right-hand section of the table shows the temperature charts for Witbank, Standerton, and Bloemfontein during May, 1944, these meteorological stations representing radial points encompassing the area within which most of the milk was produced. Actually temperature records were also obtained from Aliwal North, Vereeniging, Germiston, and Nigel but they have been omitted to avoid making the graph over complicated.

Following a period of mild autumn weather with temperatures ranging around 75°F a sudden cold spell developed, starting in the South West and spreading North East. Between May 9th and 10th temperature dropped 10 degrees at Aliwal North and 9 degrees at Bloemfontein whilst at that stage the Transvaal temperatures only fell 2 to 3 degrees. On the following day the Transvaal temperatures fell 6 to 8 degrees and by May 12th the maximum temperatures recorded were Aliwal North 45° and Witbank 64°. The following day the Witbank figure was as low as 51°F. Thereafter the weather warmed slightly, but remained comparatively cold until the end of the month.

On the left-hand portion of Table 15 a record is given of the distribution of Good: Bad ratios (on a percentage basis) from May the 1st to 11th and from the 12th to 30th. That is to say different ratios have been worked out from the results obtained in the first and second halves of the month. In the first section 48·2 per cent. of producers gave ratios of 1 : 1 or worse and thus were thoroughly unsatisfactory, whilst 34·5 per cent. fell into the ratio group 5 : 1 to 2 : 1 and only 17·3 per cent. were above this figure. In the second section of the month 69 per cent. gave results which were "All Good" and only 2·4 per cent. of producers showed ratios of 1 : 1 or lower. The interpretation to be put on these findings is that the methods of handling fluid milk in South Africa are so dependent upon uncontrollable weather changes that a large group of producers were changed from a grading of poor to very good as a result of a sudden drop of temperature. Conversely a sudden rise in temperature throws the whole milk industry out of gear on account of excessive spoilage (see Pullinger, 1944, Table 13).

Very clearly our dairy industry will not achieve a satisfactory keeping quality footing until proper cooling is practised upon the farms and until adequate measures are taken to protect milk from heating up during transit.

So far temperature has been discussed in general terms, but quite obviously farmers like those at Umlaas Road whose morning milk is tested on an 8-hour basis will be less affected by temperature than farmers whose milk is considerably older when tested.

(c) *Class of farmer.* This is a delicate question to bring under discussion, and it is one likely to give rise to misunderstanding whatever may be said. In spite of that it is an aspect of the problem that cannot be entirely ignored. Part of the success of the Umlaas Road and Pietermaritzburg farmers must be due to their own personal effort and keenness. Whilst it is impossible to define clearly those characteristics which go to make a successful dairy farmer, it is frequently possible to recognise persons who will not make a success of dairying. In a very broad way the satisfactory dairy farmer and dairyman requires to be industrious, clean, honest, and intelligent, these characters being listed in order of importance. The dairyman must be continually controlling the work in the stable, milk-room or shop and he must control it every day and every night. Dairying cannot be controlled from the stoep, nor can control be considered to exclude night and week-end duty, public holidays and an annual holiday at the coast. In other words dairying is an unending grind which can only be relieved by a satisfactory partnership, in which case the business has to support two partners.

Cleanliness is second only to the need of being constantly on parade, and hygienic dairy-farming is very largely a question of organising and maintaining cleanliness through force of example. It is useless to try and maintain a clean dairy in the midst of dirty surroundings

and a farmer's success or failure will depend not only on his efforts to maintain a clean milkroom but through his efforts to keep the whole farm yard spick and span. In fact the matter goes even deeper inasmuch as a farmer who tolerates bad ploughing, weed-infested lands, or broken fences will soon tolerate a dirty and muck-bespattered stable and milk-room. Experienced dairy inspectors are well aware of the importance of this personal factor, but under existing regulations the personality of the prospective dairy farmer is not allowed to count provided his buildings achieve a certain minimum architectural standard.

The need for honesty hardly requires discussion. Under present conditions in the milk industry, the opportunities for dishonesty (including the moral dishonesties of neglect, carelessness, and inexcusable ignorance) are so plentiful and the rewards of dishonesty so generous that it calls for a high degree of moral stability for farmers and dairymen to resist these temptations. It is perfectly true that methods of testing could be devised to trap the wrong-doer, but the fact remains that much more will be achieved if laboratories can be used to educate and assist the decent farmer and dairyman.

(d) *Class of dairy farm.* For convenience the farmer and the farm are discussed independently though in practice the farming is largely dependent upon the character of the farmer. The point to be made is that dairying should be the first priority on the farm and not, as it sometimes is, a rather lucrative Cinderella of all farming activities. When harvesting time comes round, dairy hygiene frequently goes by the board. This is in a vague way excusable if it is a question of harvesting crops intended to feed the cows through the winter. When, however, dairy hygiene is entirely shelved because the wheat, wine, or cane crop is being harvested this neglect cannot be condoned because it is merely a question of saving wage charges on one crop by allowing the dairying to deteriorate.

(e) *Surplus milk problem.* Probably one of the most important reasons for the differences in the quality of the milk marketed at different centres is wrapped up with the question of milk surpluses.

In the case of Umlaas Road, for instance, the co-operative society has a certain outlet for fluid milk and any milk produced in excess is manufactured. The co-operative members receive an average price between the full and factory price figures, and because the diversion of surplus milk to factory use is automatic, producers there are only graded on that proportion of their milk going to the fluid market, namely the morning milk. This naturally places the members in a favourable position as regards grading.

At other centres, and Pretoria is a good example, there is as a rule only sufficient milk for the needs of the local market. When milk is in short supply, the shortfall has to be made up by importations from other centres, whilst only in the flood periods do small surpluses tend

to accumulate. The producers in such an area are well placed in that they can get full price for most of their product for most of the year. This fact places their dairying on a sound economic footing and producers have little reason to adopt expense-cutting expedients such as understaffing. Moreover the receiving depots having been designed to handle approximately the gallonage that actually arrives daily, milk can be handled expeditiously and producers benefit from this expeditious handling by their milk being graded before it has time to spoil. On the other hand the Pretoria consumer always has to accept temporary milk rationing whilst unexpected shortfalls are being balanced by importations.

In contrast to Pretoria there are other centres, and Johannesburg is a notable example, which serve as pooling and balancing centres for large areas. Johannesburg has its own roster of dairy farmers capable of supplying the city's demands, whilst its milk depots and railway handling facilities have been designed to handle these normal requirements. The practice, however, has gradually developed of using the Johannesburg market as a pooling and balancing centre for all surplus fluid milk produced in the Transvaal and Northern Free State. This activity has been sponsored by the Transvaal Fresh Milk Producers Association (see Pullinger, 1944), an organisation embracing within its membership a very large percentage of dairy farmers interested in the fluid milk trade. The method of operation is to collect all surplus fluid milk (i.e. milk produced under conditions such as entitle it to be sold for fluid consumption inside a municipal area) in a central pool and sell such milk to best advantage. If the pool becomes overflooded, milk is diverted direct from farm to factories, thus saving needless transportation and handling. By paying an average price for all "pool" milk the producer who is instructed to divert his milk to a factory is not penalised financially. This "pool" has played an invaluable role in guaranteeing large milk contracts such as those for big military camps; it has given producers a profitable return upon all milk which could not be sold to milk shops; it has prevented chaos developing in times of milk flood; it has eased the strain on rail transportation by its "diversion" policy; and it has saved Johannesburg and neighbouring towns from serious milk shortage. It will therefore be appreciated that the milk industry and country as a whole has benefited greatly from the operations of the surplus milk pool, but quite unavoidable disadvantages have also developed. It must be appreciated that this organisation is a purely voluntary one, enjoying but little official recognition and faced with the problem of disposing of surplus milk without having any say in the control of over-production. On one occasion the Pool executive endeavoured to limit seasonal over-production and at the same time to save much-needed cow feed. This effort was met by a tremendous and utterly ill-informed public outcry which brought their efforts to nought.

The problems that have arisen as result of the pool operations are:—

1. Abnormal quantities of milk have been drawn to Johannesburg where the railway facilities have been inadequate to deal with this volume of incoming milk, together with the return of empty cans.
2. This veritable flood of milk has been directed to pasteurising depots designed to handle about one-third of the actual supply.
3. The high price paid by the Pool of recent years has attracted new people into dairy farming. Over the war years many more heifers have been brought into production, and many herds are to-day in the care of people who know little or nothing about dairy farming. The outcome of all this is firstly an increase in dairy feed consumption because more cows are in milk; a relatively poor milk return for the feed actually consumed because of ignorance of animal management; rapid spread of diseases such as mastitis because of ignorance and excessive trafficking in dairy stock; bad dairy hygiene and resulting poor quality milk.
4. Quite unwittingly the Pool has encouraged milk smuggling. By this is meant the introduction into municipal areas of milk which has been produced under conditions not in conformity with municipal regulations. Because of the high price paid by the "Pool" for surplus milk in almost unlimited quantities, farmers are tempted to augment their supplies by purchasing milk cheaply from neighbours who hold no permit to sell milk within a municipal area. Elsewhere farmers form combines where a group of unlicensed producers introduce milk under cover of one or two members who hold the necessary permit. It may be mentioned here that under the present organisation of the milk industry, in which cow owners are not registered and dairy cows cannot be identified, it is virtually impossible to detect milk smuggling. Even when smuggling is known to occur, satisfactory proof of the act can rarely, if ever, be obtained.

In the foregoing section some phases of the surplus milk problem have been discussed. From what has been said it will be clear that at any place where surplus milk is handled in large quantities there are delays in handling at every stage because of over-congestion. This reacts upon every producer who delivers milk by the transport system or to the depots handling the surplus milk because their product is exposed to unnecessary opportunity for spoilage.

Paragraph B: Progressive depreciation in the keeping quality of milk.

Several times in the foregoing text mention has been made of the fact that there has been a steady lowering of keeping quality of milk during the last few years, though this depreciation has not been universal and in certain centres improvement has been shown (vide Pietermaritzburg and Pietersburg, Table 7). At the large supply centres

of Witwatersrand and Pretoria, however, grave deterioration has occurred during 1941 – 45. Whilst the cause of this is by no means clear the following factors have probably played an important part:—

1. Relaxation of municipal control of incoming supplies. Relaxation of farm inspection has occurred because of wartime depletion of staff, transportation difficulties, and increase in the number of farms to be inspected, and an ever-widening zone within which inspection has to be maintained. Laboratory control has been relaxed because of staffing difficulties; moreover the war has increased the national demand for milk and the application of strict grading would have caused a serious milk shortage.
2. Due to wartime shortages the dairy industry has had to resort to improvisation in regard to every class of equipment. Changes moreover have had to be introduced in regard to milk transportation which lay milk open to increased opportunity of spoilage.
3. Increased demand and a satisfactory price for surplus milk has encouraged farmers to step up production and has tempted new farmers into production. This has occurred at a time when equipment to deal with increased production has not been available. Moreover, increased production calls for increased supervision on the farm, but there has been a dearth of efficient dairy foremen.
4. Too many learners have been tempted into milk production. Worse still, successful business men have been embarking upon dairying as a lucrative hobby or in order to bury wartime profits. With a shortage of suitable managers and foremen many such ventures are proving disastrous to the dairy industry. Moreover the value of land, farm improvements, and dairy stock has become so enhanced that genuine dairy farmers have been tempted to sell out and in this way much milk production is slipping out of the hands of the experienced farmer and into the hands of the beginner.
5. All those factors discussed under the " Surplus Milk Problem " see the preceding paragraph) also apply here.

Whilst there is no doubt that factors other than those mentioned above play a part, it is considered that these are the major ones. Deterioration in milk quality has been most marked at those centres where the war has given rise to increased milk consumption. It may be urged that Durban has had an increased demand for milk as great as any up-country town, and yet shows improvement rather than depreciation. It must be appreciated, however, that the increased requirements of Durban have to a large extent been met by bulk importation of pasteurised milk from outlying depots. Such milk naturally did not feature in the grading survey except in regard to Umlaas Road, which has already been discussed. It cannot, however, be assumed that the other depots are of the same calibre as Umlaas Road. Although

Breed count records are not available to substantiate the fact, inspectors all report that the other depots feeding Durban operate more or less on a creamery standard (this does not apply to milk railed from Pietermaritzburg). Certainly such milk cannot be considered as safe milk, if efficiency of pasteurisation is to be regarded as a criterion of safety (vide Pullinger, 1945, Table 1, Depots G. and H.).

Part III.

METHODS OF ATTACKING THE SOUTH AFRICAN PROBLEM.

In this report it has been shown that milk in South Africa as a rule features unfavourably when subjected to extensive bacteriological examination, and by the time that it is marketed, the average keeping quality is of a low order. Moreover the data presented here shows all too clearly that few producers can consistently maintain any arbitarily chosen standard of bacterial content such as 100,000 or 200,000 organisms per cc. Even when a method of grading is applied which makes allowance for a proportion of bad results, the bulk of high-veld farmers and a large number of those in the coastal belt still fail to maintain the tentative standards which have been suggested. In fact it is no exaggeration to say that any standard which could be maintained *consistently* by 75 per cent. of the fluid milk producers would be so low as to be utterly worthless. Furthermore, attention has already been drawn in a previous report to the gradual depreciation in the chemical quality of the fluid milk and to the failure to pasteurise factory milk properly prior to the manufacture of butter and cheese (Pullinger, 1944). In another report by Pullinger (1945) the unsatisfactory state of fluid milk pasteurisation has been discussed. Taking all these points into consideration it must be admitted that this is a precarious state for the milk industry to be in at a time when attempts are being made to expand dairy trade and increase the *per capita* consumption of milk and milk products. Two methods are available for improving the quality of milk.

Paragraph A: The coercive method of improving milk quality.

This method consists of setting an arbitary standard of quality and prosecuting those farmers or dairymen whose products fail to comply with this standard. As a method, it has been tried time and time again and has met with very little success. As has been shown, even good dairy farmers produce occasional faulty samples, which have to be ignored by the testing authorities. The moment the testing authority is allowed discretionary powers in deciding whether or not to prosecute, the whole foundation of grading and standards becomes undermined.

Another disadvantage of the coercive method is that it operates more effectively against the poor man than against the rich. The poor man has to accept the controlling authorities' laboratory findings as correct, and has to face conviction before a magistrate. The rich man can afford to have control tests done, and by employing skilled legal representation and if necessary by appealing to a higher court he may well escape conviction through some legal quibble. Ultimately the controlling authority ceases to prosecute such an individual because it is a waste of time. Magistrates, however, realizing the inherent weakness of this type of prosecution tend to impose trivial fines which are out of all proportion to the cost that the defendant would have to face if he were to trouble to improve his dairying methods.

Anyone who objectively considers the coercive method in all its aspects must be forced to the conclusion that it can never be very effective. As a method it offers no encouragement to the producer to improve above the bare minimum standard demanded by law, whilst the producer of rich milk receives no return for his efforts and is tempted to adulterate his product to the legal minimum and so benefit through marketing an increased gallonage.

The normal routine of efficient agricultural or commercial marketing is to price the product according to its quality, and it is difficult to see why milk should be placed in a different category in this respect.

Paragraph B: Improvement by pricing milk on a quality basis.

The more promising way of tackling the problem of improving farm milk supplies is to price milk according to its quality. This gives a strong urge to farmers to improve their herds and their methods of production, and it removes much of the need for legal intervention. The foundation of a system of grading would be to create a basic minimum grade for milk which in chemical composition is on the adulteration line and in regard to keeping quality is just "off" sour. For this grade a low price would be paid, the price in fact being little higher than the butter-fat plus skim-milk value. This basic price could be manipulated to meet changes in cost of production. Thereafter a sliding scale of bonuses could be created for milk superior to the basic minimum.

(a) *Sliding scale of bonus-earning grades.* In view of the inferior quality of the milk at present available in South Africa it is suggested that all bonus-earning standards should be established on a sliding scale as outlined in Table 16. From this table it will be seen that the basic minimum of cleanliness would be determined upon a total acidity test, the maximum allowable acidity rising from 0·20 per cent at the beginning to an ultimate level of 0·18. This final level would be achieved by raising the maximum acidity standard by 0·05 per cent. every two years. The bonus-earning standards of cleanliness could be based upon either the Breed count or preferably upon the methylene blue

reductase test, and the Bonus Grades No. 1 and 2 would be Good: Bad ratios increasing in severity every two years. The High Grade Bonus No. 3 would be comparable to the overseas "Certified" grade. Milk to be classed as High Grade would have to comply with No. 3 standards in respect of cleanliness, butter-fat and solids-not-fat. Furthermore it would have to come from herds free from tuberculosis, contagious abortion and paratyphoid infection, and having a restricted allowable percentage of mastitis infected cows. This allowable percentage of chronic mastitis carriers might be temporarily established between 5 and 10 per cent. but the position would probably have to be reviewed later. The Good: Bad ratio for high grade milk would be taken as the ratio A: B+C+D+E instead of the normal ratio A+B: C+D+E. The chemical standards for butter-fat and solids-not-fat would also be applied upon the sliding scale principle.

The reason for proposing a scale of grades gradually increasing in severity is to give producers an opportunity gradually to improve their dairying operations. This is particularly necessary in connection with the butter-fat and solids-not-fat grades, because improvement in these directions is dependent upon breeding and disease eradication and is unavoidably a slow process. Improvement of hygiene can be effected much more quickly, and it is suggested that a comparatively high standard could be reached in eight years. It might, however, prove feasible to lift the grades yet another step during the ensuing two years.

(b) *Tentative monetary bonuses.* The actual prices to be paid as bonuses would naturally have to be settled after very careful consideration but tentative suggestions are made in Table 17 to serve as a basis for discussion. In this table, in addition to the bonuses already discussed, further bonuses for "level production" (i.e. uniform daily volume output during all seasons) are included. Under present conditions the basic price might well be settled at 11d. per gallon in which case a farmer producing milk complying with existing legal standards would earn:—

Basic price	11d.
3·5 per cent. butter-fat bonus	3d.
8·5 per cent. solids-not-fat- bonus	3d.
Cleanliness bonus No. 2	3d.
Level production bonus No. 1	1d.
Total	21d. per gallon.

On the sliding scale system this producer would have two years in which to improve the quality of his herd and his methods of production in order to earn the same monetary bonuses during the ensuing two years.

Economic Changes.

1. The payment of producers according to the quality of their product.
2. The elimination of wastage through transporting milk and cream long distances in small cans.

3. The elimination of unnecessary wastage through faulty handling of the market.
4. The planning and introduction of schemes to solve the surplus milk problem.
5. Reduction in the high cost of fresh milk resulting from faulty production methods, faulty marketing and uneconomically competitive distribution methods.

Veterinary and public health considerations.

1. Practical schemes for improving the general health of dairy herds with the eradication of the major diseases of tuberculosis, contagious abortion and paratyphoid infection; a more effective control of mastitis, contagious vaginitis, and tick infestation; an improvement in the winter state of nutrition, fertility, milk quality and calf rearing methods.
2. The development of a more effective control of the health of milk handlers particularly on farms. Whilst there has been some attempt to control the health of milk handlers within municipal boundaries such control has been confined chiefly to 'Vi' testing, whilst infectious gastro-entritis of various types as well as scarlet fever and diphtheria have been ignored. On farms outside municipal boundaries a small amount of 'Vi' testing has been done by a few local authorities, but in general it is no exaggeration to say that control of the health of milk-handlers on distant farms is non-existent. Nor is there any likelihood of such control developing whilst Public Health and Milk control is organized as at present.
3. The introduction of practical methods of eliminating the smuggling of milk on a wholesale scale.
4. Improvement in the efficiency of all fluid-milk pasteurization and the extension of such efficient pasteurization to all areas where there is an appreciable local market for fluid milk.
5. Improvement to the same high level of efficiency of pre-manufacture pasteurization of all milk and cream designed for the preparation of edible milk-products.
6. An increase in the *per capita* consumption of milk and milk products. This can only be achieved if larger supplies of safe rich milk are made available at a cost considerably lower than that at present in vogue.

Clearly one of the main solutions to the whole milk problem is the introduction of an efficient system of grading milk, but no grading system can possibly be introduced and rendered effective until all milk is purchased by a single organization comparable with but not necessarily similar to the British and Scottish Milk Marketing Boards, which purchase and redistribute all fluid milk in Britain. In South Africa some

such organization will have to be built up, but in deciding upon the precise nature of the organization it will be necessary to profit from the mistakes made elsewhere.

The underlying weakness of the British system is that the Board is only truly representative of the producer's point of view.

This is an unsatisfactory arrangement since it places the pricing of a basic food entirely in the hands of the primary producer, and with competitive price fixing eliminated. Under such circumstances there is a tendency for prices to be fixed at "the highest the consumer can possibly pay without causing a reduction in turnover" whereas all basic commodities should be priced upon the principle of "the lowest possible price to consumers which offers a reasonable return (a) to the primary producer and (b) on any handling and processing essential to the marketing of the product."

A further weakness of the British scheme is that being primarily a scheme to protect the financial interests of the producer, the problems of safety and quality of milk are inevitably subordinated to the question of financial expedience. Moreover though the Board is responsible for the production end of the industry, control of pasteurization, manufacture and distribution of milk and milk products remains in the hands of a multiplicity of other authorities. In other words the British Board would not be in a position to implement more than a fraction of the reorganizations listed at the beginning of this section.

It seems that the needs of the South African milk industry will be best served by the unification of control of all phases from the production of milk to the preparation of milk and milk products for retail distribution. Municipalization of milk cannot be considered as unification because hundreds of municipalities will pull in opposite directions. The most that municipalization could achieve would be to develop some form of zonal control. It is possible to visualize a jointly run municipal organization controlling the supplies of the Witwatersrand, Pretoria, Vereeniging and Heidelberg with a similar combine in the Cape Peninsula and yet another for Durban and Pietermaritzburg. Such organizations would very definitely represent a forward step, but it cannot be claimed that such organizations would be in any way capable of materially altering conditions on the farms situated many miles from their boundaries. They could not tackle animal disease, breeding and feeding problems, nor the problem of human ill-health on the dairy farm. Many Butter and Cheese factories are situated outside all municipal zones so that some other authority would have to supervise that phase of the industry.

The two most promising alternative organizations for effecting full control are a country-wide public utility corporation or a joint producers' and consumers' co-operative society. In either case the organization would have to be constituted under a strict charter to prevent any abuse

of the milk monopoly vested in its charge. Such a corporation would purchase all milk and cream from primary producers and would resell it to distributors and manufacturers. It would be responsible for all grading, transportation, pasteurization and bottling etc., and would have to ensure that all milk and milk products are handled safely and efficiently by manufacturers and by wholesale and retail distributors.

To fulfil these functions the organization would have to establish collecting depots at strategic points in milk producing areas and processing depots, laboratories, and administrative offices in the towns. Farmers would deliver their milk and cream to the nearest depot and empty cans washed, sterilized and dried would be returned on the same transport that brought in the full ones. On arrival at the depot milk and cream would be weighed, graded, refrigerated, and pooled. Milk would no longer be classified as factory and fluid milk on the basis of the architecture of stabling, but it would be purchased on a grade basis and would be diverted to factory or fluid use on the basis of its suitability. Milk would be despatched from collecting depots in towns in insulated tankers to eliminate wastage and spoilage during transit. As far as possible, collecting depots would be situated adjacent to butter, cheese, or powdered milk factories to simplify the handling of milk surplus to the fluid milk demands. Milk for the fluid trade reaching towns in tankers would be pasteurized and bottled in the organization's own plant and made available to existing commercial distributors.

The organization would establish competent grading staffs at all depots as well as veterinary, animal husbandry and dairy inspectors to guide and assist producers. Working in collaboration with the regional public health authority (whether an M.O.H. or District Surgeon) the senior regional officer of the Corporation would be able to effect better supervision of human health on the farm, and this supervision would become really effective if the provisions of the Gluckman National Health Report are implemented. Schemes for control of animal disease could be inaugurated by collaboration between the Corporation and the Division of Veterinary Services, as the Corporation would have or could create the machinery and supply the personnel necessary to implement the eradication schemes laid down by the Division. Strict laboratory control of all subsequent phases of processing and handling, including factory processing, could be exerted by the Corporation's central laboratory, operating if necessary in conjunction with the Department of Public Health. The central laboratory would be responsible for keeping a close check upon the accuracy of all regional grading and would also carry out research upon all outstanding milk problems.

It is not suggested that the organization should undertake retail distribution of milk, as this service can best be supplied by private enterprise. There would, however, be need for a drastic reorganization of existing distribution methods aimed chiefly at the elimination of uneconomic competition and avoidable milk spoilage.

Obviously the financing of such an organization as has been outlined would be no small matter and most certainly it would have to be self-supporting and not dependent upon some form of government subsidy. In general, income could be derived from three sources.

(a) The Corporation would pay farmers less than they are paid under the present system. In this connection, however, the average farmer would suffer no financial loss because this lower price would be more than offset by a diminution in the costs of transportation, can replacements, wastage during transit and losses from theft and souring. Farmers producing low grade milk, however, would be losers under this or any other grading scheme.

(b) The reselling price to distributors would be considerably higher than the present price paid by distributors to farmers. This difference in allowable handling charges would be offset by a decrease in the risks and expenses distributors have to meet in buying milk from farmers, and also a decrease in building and equipment requirements and in running and staffing costs because distributors would daily collect their exact requirements ready processed and bottled by the Corporation. It must, however, be stressed that handling costs allowed under such a scheme as is outlined here would not cover the expenses involved in maintaining a daily delivery of a mile or two for the sake of selling a couple of pints of milk.

(c) A levy imposed upon manufactured milk products in return for grading and control services. This levy would be of a trifling nature, amounting to a fraction of a penny a pound because manufactured milk products cannot afford to carry any heavy levy, although there would be considerable control required to improve the manufacturing aspect of the industry. Luxury products such as sweet and sour cream for the luxury trade, cottage cheese etc., could carry a heavy levy.

Chapter 9.

SUMMARY.

1. The various methods of collecting milk samples are discussed and the use of the sampling tube (i.e. milk thief) is favoured.

2. The need for frequently repeated testing is stressed. It is urged that the aim should be to test every supplier five times a week, covering both day and night milkings in the process, and the sample representing the composite output.

3. The mistake of submitting milk to bacteriological analysis when too fresh is stressed. It is emphasized that samples should be held

under atmospheric conditions for at least eight, but preferably for twelve hours or more after milking before submitting them to test. If this is not done the test results may not be a true reflection of the keeping quality of the milk.

4. The difficulty of interpreting or analysing a fluctuating series of test results is indicated. Even the best producers record occasional unsatisfactory results and the problem lies in deciding what degree of fluctuation below a prescribed standard can be allowed. A method of grading is proposed whereby a record is made of the proportion of good test results to bad test results obtained by a farmer during the month. This proportion is called the Good: Bad ratio. The dividing line between a good and a bad test result is taken as a Breed count of 1,000 per cc. or a methylene blue reductase time of 3¾ hours.

5. An attempt has been made to ascertain a median Good: Bad ratio for milk delivered to Johannesburg. From an analysis of all tests done (i.e. 103,138 Breed counts) it seemed probable that a reasonable number of nearby farmers and a few long-distance farmers should be able to maintain a Good: Bad ratio of 2: 1 from September to March and 5: 1 for the remaining winter months. These ratios mean that a farmer must produce milk with counts below 1,000,000 per cc. on two occasions or on five occasions for every sample the count of which rises above that level. Furthermore these ratios of good samples to bad samples would have to be maintained consistently month by month. On the face of things these standards appear to be absurdly low, and so the results produced by many farmers scattered throughout the Union of South Africa have been tested against these suggested ratio standards. The attempt has been made to sample every producer five times a week; this has not been achieved, but any month where less than eight results were available for a farmer his record has been omitted.

6. The outcome of applying these suggested standards to individual farmers efforts has shown:—

(a) That producer-retailers as a whole should have no difficulty in maintaining them, but producer-retailer milk only constitutes about one-tenth of the total supply of any large consuming centre.

(b) Of high-veld dairy farmers other than producer-retailers, only a very small fraction consistently maintained the suggested standards.

(c) Of farmers in the coastal belt, other than producer distributors, a considerably larger proportion maintained the suggested standards, but in view of the extremely low standards used the position even in the coastal belt cannot be regarded as being satisfactory.

7. The validity of the Good: Bad ratio as a method of analysing test results is discussed.

8. The application of this method of analysis to methylene blue reductase results is considered. Though comparatively little data is available, serial tests of fifty-five producers and the results of over 5,000 parallel Breed count and reductase tests all indicate that if the reductase dividing line is taken as $3\frac{3}{4}$ hours and the ratios of 2 : 1 and 5 : 1 are adhered to, producers find it harder to maintain standard when tested by the reductase than by the Breed method. This is due to the fact that unless the milk is stored 12 hours the Breed method fails to reveal latent defects that may be uncloaked by the reductase test. All the comparative tests available were done on 8-hour old milk samples.

9. Suggestions are made, backed by a small volume of experimental data, as to how Breed or reductase results of very clean milk might be analysed as Good : Bad ratios suitable for the classification of a high grade of milk. Further investigation is required along these lines.

10. The probable reasons are discussed as to why the percentage of farmers maintaining standard in different areas should vary so remarkably.

11. The major reasons for the striking depreciation in keeping quality that has become apparent in certain areas during the war are discussed.

12. It is concluded that at present it would be quite impossible to lay down a worthwhile set of milk standards which a reasonable proportion of South African dairy farmers could maintain. A method is therefore suggested of establishing a system of purchasing milk from farmers on a basis of grade so that the efficient dairy farmer could obtain a monetary reward for his efforts. By establishing the various grades on a sliding scale basis, increasing in severity every two years, it would be possible gradually to raise the general quality of milk production throughout the country. The type of administration necessary to implement such a grading scheme is discussed.

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TABLE 14.

Breed Count Good : Bad Ratios applied to potential producers of High-Grade Milk. Good : Bad Ratio is calculated as Group A : B + C + D + E.

Producer No.	GOOD : BAD RATIO A : B + C + D + E												No. of months Substandard ★	
	1943 : July	Aug.	Sep.	Oct.	Nov.	Dec.	1944 : Jan.	Feb.	Mar.	Apr.	May	June	Summer	Winter
505	7:0	7:1	4:0	6:1	8:1	6:1	1:8	3.5:1	1:1	.	.	.	2	0
507	.	.	.	.	8:1	6:0	9:0	3:1	8:0	6:0	8:0	8:1	0	0
539	7:0	8:0	4:0	7:0	1:2	8:0	8:1	9:0	8:0	6:0	8:0	9:0	1	0
540	7:0	3:1	1:1	1.7:1	3.5	8:0	1:3.5	1:2	1:1	5:1	8:0	3.5:1	4	2
541	7:0	3:1	1:1	8:0	8:1	8:0	9:0	9:0	1:1.7	5:1	8.0	8:1	2	1
542	7:0	7:1	3:1	1:1.7	2:1	7.0	8:1	1:1	3:1	2.5:1	8:0	9:0	2	1
548	.	.	.	2:0	7:1	6:0	9:0	8:1	8:0	4:1	8:0	10:0	0	1
549	.	.	.	.	2:1	6:1	3:1	3:1	1:1	5:1	7:1	9:1	1	0
550	.	.	.	.	3.5:1	6:0	3.5:1	8:1	7:1	6:0	7:1	5:0	0	0
551	.	.	.	.	7:0	6:1	2:1	7:1	3:1	6:0	8:0	10:0	0	0
563	.	.	.	.	.	.	0:3	0:8	0:7	0:5	1:4	.	3	2
564	.	.	.	.	.	.	1:1	3.5:1	1.7:1	5:1	8:0	9:0	1	0
565	.	.	.	.	.	.	4:0	3.5:1	8:0	5:1	8.0	10:0	0	0

N.B. — ★ Tentative SUMMER Standard Good: Bad ratio 2: 1. WINTER Standard Good: Bad ratio 5: 1.
Good: Bad ratio calculated as Class A: B+C+D+E.

TABLE 15.

TABLE AND TEMPERATURE CHART SHOWING THE INFLUENCE OF WEATHER CHANGES UPON THE GOOD : BAD RATIOS OF 230 FARMERS DISPATCHING MILK 20 TO 250 MILES TO MARKET.

GOOD : BAD RATIO GROUPS.	SCATTER OF GOOD : BAD RATIOS (PERCENTAGES) MAY 1944	
	1st to 11th	12th to 30th.
ALL GOOD	8·4	69·0
22:1 to 16:1		
15:1 to 11:1		
10:1 to 6:1	8·9	19·2
5:1 to 2:1	34·5	9·4
1:1	16·7	1·2
1:2 to 1:5	18·7	0·4
1·6 to 1:10	5·3	0·4
1:11 to 1:15		
1:16 to 1:22		
ALL BAD.	7·5	0·4

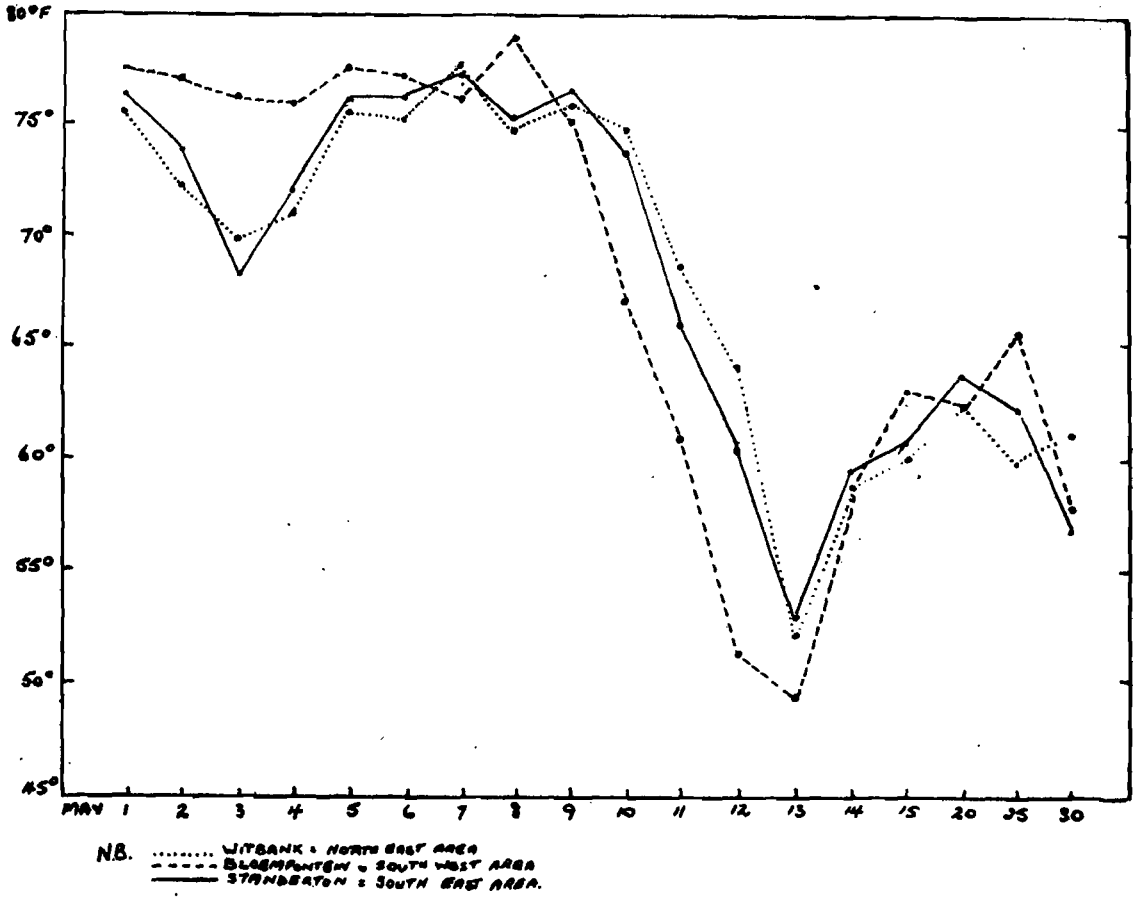

TABLE 16

BASIC MINIMUM AND BONUS EARNING MILK STANDARDS.

SCHEDULE OF BONUS IN YEARS	NUTRITIVE VALUE BONUS								CLEAN MILK BONUS.													
	BUTTER FAT %				SOLIDS-NOT-FATS %					MINIMUM GOOD : BAD RATIOS.												
		IN 80% OF MONTHLY TESTS				IN 80% OF MONTHLY TESTS				BREED						REDUCTASE.						
										SUMMER			WINTER			SUMMER			WINTER		
	MINIMUM ACCEPTABLE STANDARD BONUS NIL	BONUS NO 1.	BONUS NO 2.	BONUS NO 3. HIGH GRADE MILK	MINIMUM ACCEPTABLE STANDARD BONUS NIL.	BONUS. NO 1	BONUS. NO 2.	BONUS NO 3. HIGH GRADE MILK.	MINIMUM STANDARD BONUS NIL HIGHEST ACCEPTABLE % LACTIC ACID	BONUS NO 1. RATIO A+B : C+D+E	BONUS NO 2. RATIO A+B : C+D+E	BONUS NO 3. HIGH GRADE MILK RATIO A : B+C+D+E	BONUS NO 1 RATIO A+B : C+D+E	BONUS NO 2 RATIO A+B : C+D+E	BONUS NO 3. HIGH GRADE MILK RATIO A : B+C+D+E.	BONUS NO 1 RATIO A+B:C+D+E	BONUS NO 2 RATIO A+B : C+D+E	BONUS NO 3 HIGH GRADE MILK RATIO A+B : C+D+E	BONUS NO 1 RATIO A+B : C+D+E	BONUS NO 2 RATIO A+B : C+D+E	BONUS NO 3 HIGH GRADE MILK RATIO A+B : C+D+E.
1ST AND 2ND	2·7	3·0	3·5	4·0	7·80	8·00	8·25	8·50	0·200	1:10	1:1	2:1	1:5	2:1	2:1	1:12	1:4	2:1	1:6	1:1	2:1
3RD AND 4TH	2·8	3·1	3·6	4·1	7·82	8·05	8·30	8·55	0·195	1:5	2:1	5:1	1:1	5:1	5:1	1:8	1:1	5:1	1:4	3:1	5:1
5TH AND 6TH	2·9	3·2	3·7	4·2	7·85	8·10	8·35	8·60	0·190	1:1	5:1	11:1	2:1	11:1	11:1	1:4	2:1	11:1	1:1	4:1	11:1
7TH AND 8TH	3·0	3·3	3·8	4·3	7·90	8·15	8·40	8·65	0·185	2:1	11:1	ALL GOOD	5:1	12:1	ALL GOOD	1:1	4:1	ALL GOOD	2:1	9:1	ALL GOOD
9TH AND 10TH	3·1	3·4	3·9	4·4	7·95	8·20	8·45	8·70	0·180												
FINAL STANDARD.	3·2	3·5	4·0	4·5	9·00	8·25	8·50	8·75	0·130												

IT IS SUGGESTED THAT THE BONUSES LISTED ABOVE WILL BE AWARDED ON A MONTHLY BASIS AND THAT THE LEVEL PRODUCTION BONUS (SEE TABLE 17) BE AWARDED ON AN ANNUAL BASIS.

TABLE 17.

Tentative Monetary Milk Bonuses.

Basic Price per gallon of minimum Standard Milk	Butter Fat.			Solids-not-Fats.			Clean Milk.			Production Level	
	Bonus No. 1.	Bonus No. 2.	Bonus No. 3.	Bonus No. 1.	Bonus No. 2.	Bonus No. 3.	Bonus No. 1.	Bonus No. 2.	Bonus No. 3.	10% Fluctuation Bonus ★★	5% Fluctuation Bonus ★★
? ★	2d.	3d.	4d.	2d.	3d.	4d.	2d.	3d.	4d.	1d.	2d.

★ The basic price of milk would be varied to meet changes in the cost of production throughout the country or within any specified zone.

★★ The allowable degree of fluctuation in consistent daily output maintained throughout the year to qualify for the **level production bonuses** shown.

The records drawn upon in this report represent the unremitting work of Officers and N.C.O.'s of the Milk and Meat Inspection Section of the S.A. Veterinary Corps, of certain Government Veterinary Officers and their staffs, of the staff of the Johannesburg Milk-testing Laboratory and of a number of Municipal dairy inspectors. For upwards of four years this monotonous daily task of testing milk has been carried out with meticulous care, and very great credit is due to all those workers who have banded together into an unofficial team endeavouring to improve the milk supplied not only to the U.D.F., but also to the public in general.

(CONCLUDED).

"SPERMNESTS" IN THE OVIDUCT OF THE DOMESTIC HEN.

G. C. VAN DRIMMELEN,
Bloemfontein.

Quinlan, Maré and Roux [1932 (a) and (b), 1933] found that spermatozoa after coitus in sheep, survived considerably longer in the cervix uteri than in any other part of the genital tract. They state that under favourable conditions the cervix acts as a reservoir from which there is a constant issue of active sperm cells to the cranial division of the tract. This finding, which agrees with the observations of Giles (1919) in women, recalls examples of long periods of survival of spermatozoa in lower animals. In the queen bee, for instance, the spermathecum may contain functionally normal sperm cells for seven years after coitus (Bishop, 1920), and many invertebrates have similar spermatheca. Some fishes, in which the eggs are fertilized internally, retain live sperm in the female for long periods. V. Oordt (1928). Dulzetto (1937) found special cup-shaped depressions in the follicles of *Gambusia holbrooki,* in which the sperm are stored until the egg is mature, when the entry of spermatozoa is permitted through an aperture, the zygote developing in the follicle and the remaining sperms undergoing spermatolysis. Folk (1940) recorded cases of the 90-day storage of effective spermatozoa in female bats, and Mathews (1937) found sperm cells in the British Horseshoe bat to be retained in the vagina as well as in the uterus during hibernation. In birds the continued production of fertile eggs by the females of some species (turkey, fowl) for some weeks after separation from the male is well known.

Van Drimmelen [1945 (a)] found normal active spermatozoa in the infundibulum of the fowl's oviduct up to 14 days after insemination. Many investigators, and notably Walton and Wetham (1933), had suspected the sperm to be present, as they could not subscribe to either of the two following views recorded in the literature:—

(1) Payne (1914) and Warren and Kilpatrick (1929) thought that the tailless spermheads which were seen in the oviduct from 2 to 56 days after separation from the cock were capable of effecting fertilization.

(2) Ivanov (1924) and Vermeulen (1925) believed that spermatozoa in the fowl, after coitus, entered the ripe and unripe follicles, because fertility was not terminated by irrigation of the oviduct and peritoneal cavity with spermatolytic fluids. Although Walton and Wetham confirmed Ivanov's results, they maintained that the sperm

might be retained in the crevices of the oviduct and contemplated experiments to solve the problem.

The motility of the spermatozoa seen in the infundibulum as mentioned by Van Drimmelen [1945(b)] suggests that the sperm had been concentrated and protected during the period of storage (Milovanov, 1940; Hammond, 1940), as in all known cases of long survival the dense concentration of the sperm-suspension is characteristic (Walton and Wetham, 1933).

The object of the present communication is to describe the discovery of "Sperm-nests" or multiple concentrations of spermatozoa in the infundibular mucous membranes of fertile domestic hens. The nature and distribution of these structures is being investigated. The evidence already available shows that where active spermatozoa were discovered in the ostium abdominale of the oviducts of some fertile hens, the mucosa of the infundibulum contained numerous "sperm-nests" in the region of the transition between the predominantly ciliated epithelium of the funnel and the glandular epithelium of the chalaziferous region.

Histology and Physiology.

According to Surface (1912) the infundibulum of the fowl's oviduct consists of a cranial part, the "mouth," and a caudal tubular portion, both having a wall made up of six layers:—

(1) the serosa;
(2) the outer (longitudinal) muscle layer;
(3) the outer connective tissue layer;
(4) the inner (circular) muscle layer;
(5) the inner connective tissue layer;
(6) the ciliated and glandular epithelium.

The infundibular wall is very thin compared with that of other parts of the oviduct and the muscular tissue is only to be found in scattered bundles. The inner surface has primary longitudinal ridges with secondary epithelial folds. This author described three types of glands in the infundibulum: (a) Unicellular glands occurring only in the caudal part. As the cells of these glands have their nuclei near the basement membrane, whereas ciliated cells have their nuclei nearer to the surface, their presence in the caudal part was recognisable by the double row of nuclei in the epithelium. (b) Glandular grooves, not seen in the extreme caudal part of the infundibulum. (c) Tubular glands in the caudal part near the albumen portion of the oviduct.

Bradley (1928) mentioned the distinction between the goblet and non-goblet cells of the oviduct. In the posterior infundibulum he found small goblet cells recognised by metachromatic staining with thionin. The non-goblet glandular cells were found to be cuboidal in the glandular grooves and *glandular ducts. These ducts were*

formed by invaginations of the grooves. In the rudimentary glands (tubular glands) in the caudal parts of the infundibulum, these cells were deeper; their cytoplasm was granular and their nuclei were situated towards the basement membrane as in the case of goblet cells. On account of staining reactions observed, Bradley concluded that the secretions of both the goblet cells and the non-goblet glandular in the infundibulum differed from secretions of similar cells in other parts of the oviduct.

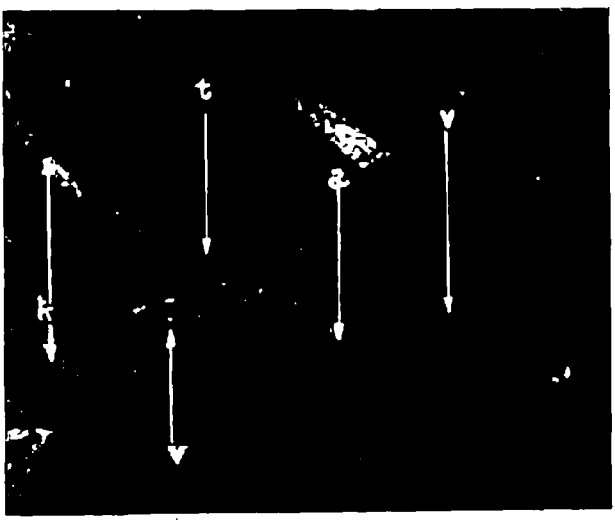

FIG. 1: *"Sperm-nest" (a) in the epithelium of the middle chalaziferous region in the oviduct of a fertile pullet eight days after intraperitoneal insemination. The sperm mass, though slightly disturbed by the microtome, shows the regular undulating bundle of tails (t) extending up the duct (k). Note the bloodvessels (v) near the end of the crypt.*
Section: 5 μ; Stain: Giemso's azur-eosin; Magnification: × 1080. Microphotograph by Dr. A. T. Neser.

Textbooks do not agree on the nomenclature of the parts of the hen's oviduct. According to Ellenberger and Baum (1939), the "infundibulum" includes all the parts cranial to the isthmus. Richardson (1935) gave a table describing as "infundibulum" the parts cranial to the albumen-secreting region, viz., the funnel proper and the chalaziferous region between the base of the funnel and the commencement of the albumen glands. His terminology is thus in complete agreement with that of Surface (1912) and Bradley (1928). Warren and Scott (1935) suggested the name "magnum" for the whole region between the base of the funnel and the demarcation line at the cranial end of the isthmus. 'Infundibulum" would then indicate the funnel only and the chalaziferous region would form the cranial

part of the "magnum". In the present communication the term "infundibulum" will be used for the region of the oviduct cranial to the albumen-secreting glands, thus following the usage of Surface (1912), Bradley (1928) and Richardson (1935).

Richardson found the chalaziferous region to be 8 cm. long in a 50-cm.-long oviduct. According to this author, the chief interest of this part is that it is the source of the heavy chalaziferous secretion which forms a thin layer around the mature ovum internal to the dense albumen. He found the folding of the mucosa to resemble that of the mammalian fallopian tube. In this region the glandular grooves which could be found in the funnel merged into the tubular glands, the so-called chalaziferous glands, which were quite distinct from the glands found in the albumen region. The mucous cells here could only be demonstrated in oblique sections near the lips of the folds, whereas in the caudal end of the funnel, mucous cells *occurred in the uniciliated grooves* and alternated with ciliated cells on the folds.

Warren and Scott (1935) found the ovum to be liberated into the body cavity in a very loosely enclosed state, so that the yolk assumed the shape of the cavity into which it fell. The very active fimbriae of the oviduct then engulfed the ovum, a process which occupied 3 to 35 minutes (mean 13 minutes). Once the ovum was enclosed in the oviduct, stronger muscles same into play increasing the rate of passage and producing an extreme elongation of the yolk mass.

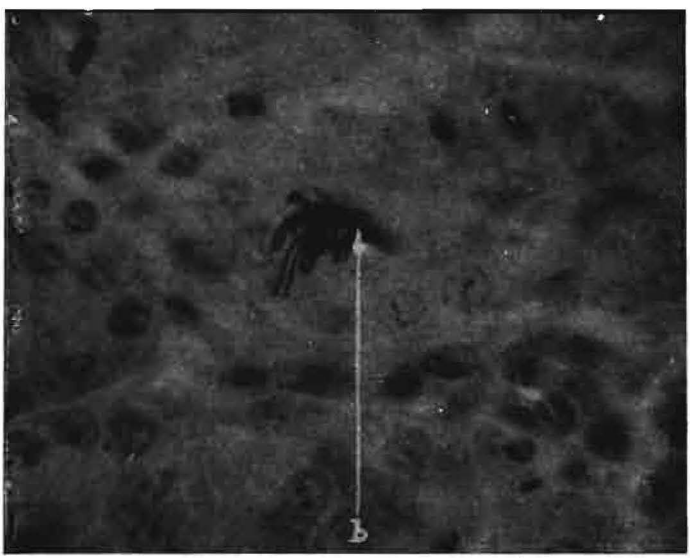

FIG. 11: *"Sperm-nest" (b) from same material as in fig. 1. Section across end of duct. Sperm nuclei slightly disturbed by microtome.*

Section: 5μ. Stain: hæmalum-eosin. Magnification: × 1080. Microphotograph by Dr. A. T. Neser.

Passage through the funnel occupied an average of 17·8 minutes. McNally (1942) showed that on coming into contact with the mucosa of the oviduct, the vitelline membrane immediately became strengthened by a layer of mucin. Parker (1931) found the cilia in the bird's oviduct to work in an ab-ovarian direction, except for those on a pro-ovarian band along the dorsal wall of that part of the genital tract cranial to the uterus.

Technique.

Hens were fertilized by artificial insemination or by natural mating of 45 White Leghorn pullets which had been kept in an intensive laying house on the Onderstepoort poultry plant. These pullets had previously been separated from males and some were mated singly with a cock in cages from which the cock was removed when required, whilst others were inseminated artificially and then marked and liberated in the poultry house. The eggs were collected by the trap-nest method every two hours and set in a still air incubator on the evening of collection. Candling was done when the opportunity occurred, but the eggs were turned every day. At various intervals after the separation or insemination, selected birds were anæsthetised and the genital organs were examined for the presence of spermatozoa by the technique described in a previous publication [Van Drimmelen, 1945(b)].

In four cases a series of one-cm.-long pieces of the cranial end of the oviduct, including parts of the funnel, the chalaziferous region and the albumen region, were clamped off with artery forceps, fixed in Helly's fluid and embedded in wax for subsequent microscopical examination of sections for the presence of spermatozoa.

Experimental Findings.

Case One. — Specimens were collected from White Leghorn pullet number 905 on 30.6.45. Eggs were collected and tested for fertility from the day on which she was inseminated with results indicated in table 1.

On examination of stained sections made from three pieces of the oviduct, no spermatozoa were found although several cell fragments having the appearance of portions of fowl sperm were seen.

Case Two. — Specimens were collected from White Leghorn pullet number 902, eight days after insemination when she had been fully three weeks in lay. Details are set out in table 2. On examination of sections made from three pieces of the cranial end of the oviduct, stained with hæmalum-eosin, iron-hæmatoxylin and Giemsa's stain, very few sperm nuclei were seen singly in the shallow crypts at the junction of the chalaziferous and albumen regions, but a large number, including many showing acrosomes and parts of middle-pieces and tails were seen concentrated around a piece of denser mucus in the lumen of the albumen-secreting region.

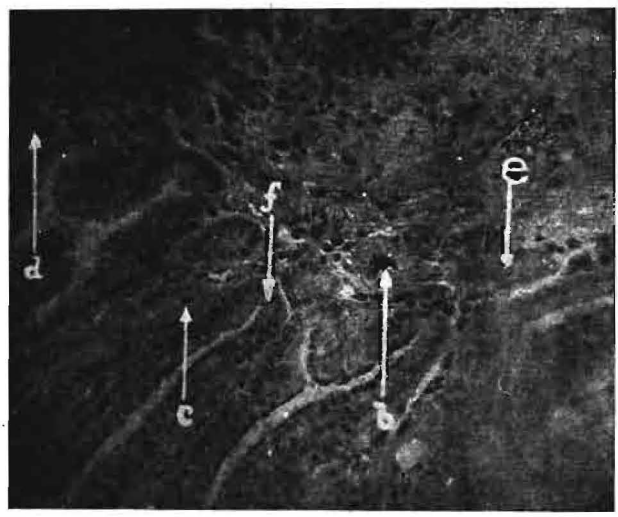

FIG. III: *Same section as in fig.* 1 *magnified* × 240, *showing "sperm-nests" (b), (c), (d) and (e). Note position of nuclei near base white membrane in the ends of the crypts (f) and vascularity of the tissues.* Section: 5 μ. Stain: hæmalum-eosin. Magnification × 240. Microphotograph by Dr. A. T. Neser.

Large numbers of similar shallow crypts were seen in the caudal portion of the chalaziferous region. Some sperm nuclei were found in these crypts and their presence encouraged more detailed search. Following the course of these crypts in serial sections concentrations of spermatozoa or "sperm-nests" were found in the fundus of every one in which the single nuclei had been found. The "sperm-nests" were most frequent in the caudal half of the chalaziferous region, and more than one hundred "sperm-nests" have been examined in serial sections from these specimens. All "sperm-nests" appeared to be associated with the crypts mentioned, which seemed to form the transition between the ciliated glandular grooves and glandular ducts (Bradley, 1928), and the tubular glands of the chalaziferous region of the oviduct. The lining of the short ducts to these crypts was consistently found to be ciliated, with the nuclei of most of the cells near the lumen; but the lining epithelium of the cup-shaped fundus was formed by non-ciliated cells with the nuclei near the basement membrane. The majority of the sperm-heads seen were arranged parallel to each other in the fundus with the tails extending in an undulating bundle in the ciliated part of the duct. The ducts of some of the crypts that were examined had a length of 250 μ to 350 μ; and some "sperm-nests" contained as many as 50 to 80 spermatozoa. In many empty crypts a mass of globules could be seen, suggestive

of material secreted by the non-ciliated cells. In many cases arterioles were found very close to the " sperm-nests " and near the fundus of each crypt. (See figures I to IV.)

Case Three. — Specimens were collected from a White Leghorn pullet number 912 four days after separation from the cock with which she had been confined in a cage from 10·00 hours on 14.7.45 to 10·00 hours on Monday, 16.7.45. Her laying record and the incubation results of her eggs are set out in table III.

On examination of sections made from the caudal parts of the chalaziferous region, " sperm-nests " were found containing one or

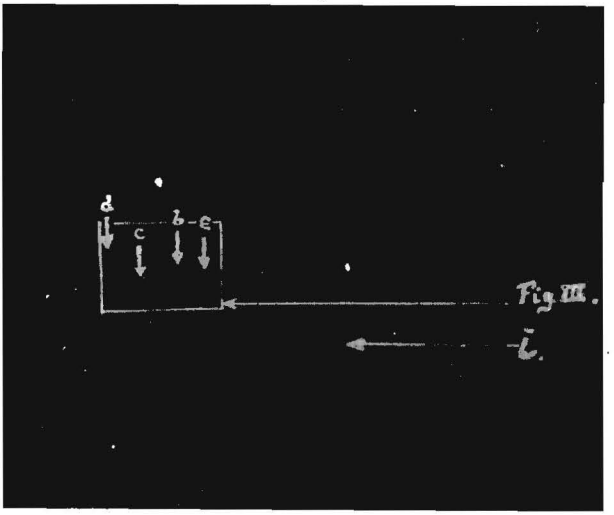

FIG. IV.: *Same section as in fig* II *magnificd* × 48, *showing site of " sperm-nests " (b), (c), (d) and (e) in a fold of the mucosa in the fowl's oviduct. Note the thin wall of the infundibulum at the middle chalaziferous region and the highly vascular inner connective tissue layer (i).*
Section: 5 μ. Stain: hæmalum-eosin. Microphotograph by Dr. A. T. Nøser.

two sperm-heads. Large concentrations of spermatozoa in the " sperm-nests " have not yet been found in naturally fertilized hens, but the impression gained from the few sperm-nuclei seen in the typical position noted in the specimens from case two, is that suitable material will probably show " sperm-nests " with a greater number of spermatozoa.

DISCUSSION.

The maintenance of fertility for long periods after the last coitus in the turkey and hen is a phenomenon that has attracted much attention, yet an arrangement comparable to the spermatheca of lower animals has not been found to exist in birds. The discovery of the

" sperm-nests " in a hen eight days after intraperitoneal insemination, during which period four fertile eggs were laid, suggests an explan- to construct the process of fertilization of an ovum liberated in a hen to construct the process of fertilization of a ovum liberated in a hen eight days after separation from the cock, by visualising the effect that the passage of a flexible yolk mass through the cranial part of the oviduct would have on the " sperm-nests " observed in the mucosa. In the first place the muscular wall would undergo active peristaltic contractions (Warren and Scott, 1935). Secondly, the lumen would be greatly distended, thereby decreasing the depth of the crypts forming the " sperm-nests ". Thirdly, the activity of the secretory cells would be markedly stimulated. All these factors tend to contribute to the dislodging of the contents of the " sperm-nests " and would therefore provide free sperm in the lumen of the duct. The activity of the spermatozoa, obviously curtailed by concentration in the " sperm-nests ", would be enhanced by liberation and " dilution " in the liquids surrounding the ovum. This view would be supported by the observation that the active live sperm were found only in oviducts through which an ovum had passed within twenty-four hours before examination. Similar explanations can be suggested for several other baffling phenomena, such as the fact that the spermatozoa of a previous coitus, stored *in vivo* in a fertile hen, are rapidly superseded or replaced by fresh sperm if the hen is mated to a different cock and the fact that conditioning of the oviduct for egg production has a favourable influence on the fertility of hens [Crew (1926), Warren and Kilpatrick (1929), Warren and Gish (1943), and Lamoreux (1940)].

The present work provides a problem for several clear-cut lines of investigation in avian sex-physiology and histology. In addition, it suggests that useful information might be obtained from sections of the cervix of the ewe collected 30 to 48 hours after copulation, because there may be a connection between some elements in the mucosa and the longer survival of live spermatozoa in this part.

In the disinfection of organs with an epithelial lining which contains crypts, or ducts, it will be well to remember that both Ivanov and Walton and Wetham used irritants highly fatal to sperm suspensions *in vitro* and yet failed to destroy the male cells in the organs of the hen.

ACKNOWLEDGMENT.

Thanks are due to the Director of Veterinary Services, Onderstepoort, for permission to do the work and for the facilities provided. Drs. C. Jackson, M. de Lange and H. P. A. de Boom as well as the staff of the pathological section were responsible for the preparations made and the material was obtained from the poultry section by the general assistance of Dr. J. W. D. A. Coles.

TABLE I.

Insemination, laying and incubation record and the intra-abdominal examination results of case one, bird number 905.

DATA ON EGGS LAID.				TREATMENT OF BIRD.	
Date collected.	*Date set.*	*Date candled.*	*Result of candling.*	*Date of treatment.*	*Treatment.*
30.6.45	Not set				
2.7.45	Not set				
3.7.45	Not set				
4.7.45	Not set				
5.7.45	5.7.45	11.7.45	Infertile	5.7.45	Inseminated [See footnote (a)]
6.7.45	6.7.45	11.7.45	Infertile	9.7.45	Operated [See footnote (b)]

FOOTNOTE (a). Inseminated at 10·00 hours on 5.7.45 by the intraperitoneal method with 0·3 cc. pure fresh semen of cock No. 47 and with 0·3 cc. pure fresh semen of cock No. 53 by means of a glass syringe fitted with a modified "holborn" inseminator applied through a canula.

FOOTNOTE (b). Anæsthetised at 11·00 hours on 9.7.45 with 0·75 cc. Nembutal intravenously and searched for active spermatozoa in the oviduct and round the ovary by the method previously reported. Doubtful specimens were found.

TABLE II.

Laying, incubation and insemination record of case two, hen number 902, and the results of intra-abdominal examination of this bard eight days after insemination.

DATA ON EGGS LAID.				TREATMENT OF BIRD.	
Date collected.	*Date set.*	*Date candled.*	*Result of candling.*	*Date of treatment.*	*Treatment.*
3.7.45	Not set				
8.7.45	Not set				
9.7.45	Not set				
11.7.45	Not set				
12.7.45	Not set				
14.7.45	Not set				
15.7.45	Not set				
16.7.45	16.7.45	21.7.45	Infertile	16.7.45	Inseminated [See footnote (c)]
18.7.45	18.7.45	21.7.45	Fertile		
		30.7.45	Dead embryo		
21.7.45	21.7.45	24.7.45	Fertile		
		30.7.45	Strong embryo		
22.7.45	22.7.45	24.7.45	Fertile		
		30.7.45	Strong embry		
24.7.45	24.7.45	30.7.45	Infertile	24.7.45	Operated [See footnote (d)]

FOOTNOTE (c). Inseminated at 12·00 hours on 16.7.45 by the intraperitoneal method with 0·5 cc. of pure fresh semen from three-year-old White Leghorn cock number 6, by means of an all-glass syringe and a stout 8-cm. long wax-coated metal needle.

FOOTNOTE (d). Anæsthetised at 10·00 hours on 24.7.45 with 0·8 cc. Nembutal intravenous and successfully searched for active spermatozoa which were demonstrated in the ostium abdominale.

TABLE III.

Egg-production, incubation and candling record of case three, bird number 912, and the dates of mating and operation.

DATA ON EGGS LAID.				TREATMENT OF BIRD.	
Date collected.	*Date set.*	*Date candled.*	*Result of candling.*	*Date of treatment.*	*Treatment.*
8.7.45	Not set				
10.7.45	Not set				
13.7.45	14.7.45	18.7.45	Infertile		
14.7.45	14.7.45	18.7.45	Infertile	14.7.45	With male in cage
				16.7.45	Separated alone in cage
17.7.45	17.7.45	18.7.45	Fertile		
		24.7.45	Strong embryo		
18.7.45	18.7.45	21.7.45	Fertile		
		24.7.45	Strong embryo		
				20.7.45	Operated [See footnote (e)]

FOOTNOTE (e). Anæsthetised at 10·00 hours on 20.7.45 and examined for live sperm in the genital organs. A few spermatozoa-like bodies were found, but no motile spermatozoa were seen in the wet preparations.

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THE USE OF GONADOTROPHIC SUBSTANCES IN THE TREATMENT OF REPRODUCTIVE DISORDERS IN ANIMALS.

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1. — INTRODUCTION.

In considering the problems related to sexual development and function in all their divergent phases of normality and abnormality, one is immediately confronted by one of the most interesting, most important and certainly one of the most complicated of biological phenomena. Moreover the reproductive system constitutes the last of the organ systems to be subjected to intensive detailed study, and already there is ample evidence that knowingly or unknowingly biologists had shelved one of their most difficult tasks. As if to make up for lost time and despite the encumbrances imposed by the long years of war, a prodigious amount of research work, much of which is of a truly brilliant character, has nevertheless been conducted on the intracacies of reproductive function, within very recent times. Although much of the relevant Continental literature has yet to be reviewed, it can be stated that from British and American sources alone, many hundreds of articles have been published annually during the war years and our knowledge concerning these problems correspondingly widened.

As several detailed reviews are available, it is not the intention in this short paper to discuss the voluminous literature already in existence, but rather to emphasize a few of the outstanding facts definitely known about the control of sexual function and to indicate what lines are being followed in order to rectify some of the commonest forms of sexual dysfunction in our animals.

2. — THE CONTROL OF SEXUAL FUNCTION.

It is an established fact that the various phenomena of reproduction are intimately associated with the endocrine balance maintained within

the body, while the development and the function of the gonads in particular, i.e. the ovaries and the testes, are carefully controlled by gonadotrophic hormones elaborated in the anterior lobe of the pituitary. Moreover, recent evidence has shown that the pituitary, through its connection to the base of the brain, is closely associated with the region of the hypothalamus and that as a result of the intervention of nervous influences it is more correct to regard the pituitary and the hypothalamus functionally as a neuro-endocrine unit.

Although the pituitary has been referred to as the conductor of the endocrine orchestra as a result of its dominating action, it is nevertheless subject to a wide variety of influences arising from both the nervous system and the endocrine organs in the rest of the body. Consequently, a highly complicated and delicately adjusted endocrine balance is normally maintained within the body which, through its association with growth, metabolism, and reproduction, is intimately concerned with the maintenance of the internal environment within physiological limits. Of special significance in this respect are the factors governing the characteristic rhythmicity of the female reproductive cycle and the altered metabolism of pregnancy.

While the general pattern of the inherent mechanism controlling reproduction is essentially the same in all animals and man, it is nevertheless strongly influenced by such factors as species, age, sex, nutritional status and general health. Moreover, the external environment as determined by food conditions, infection, locality, climate and more specifically by light, temperature, altitude and season are all capable of affecting the internal conditions of the body and therefore the functional relationships of all its organ systems.

In considering reproduction as but a single function of the body, it is essential to realize beforehand that it is constantly being subjected to a wide variety of factors both from within and without the body.

3. — The Pituitary – Gonad Relationship.

Apart from elaborating a series of hormones controlling body growth, metabolism and the function of the other endocrine organs, the *anterior pituitary* normally secretes several closely related hormone fractions responsible both for the initiation and maintenance of gonad activity and the reproductive potential of the individual. These pituitary gonadotrophins, as they are known, can, according to our present knowledge, be grouped as follows:—

I. — *Follicle-stimulating hormone* (*F.S.H.*) responsible for the primary growth of the follicles in the ovary and the seminiferous tubules in the testicle.

II. — *Luteinizing hormone* (*L.H.*) which has been shown to be identical with the *interstitial cell-stimulating hormone* (I.C.S.H.), causes preovulatory swelling, ovulation, and the formation of the corpus luteum in the female.

There is an extremely close relationship between these two hormones, both of which are essential for the maturation of the follicles. The luteinizing hormone probably increases very rapidly at the time of ovulation, thereby effectively suppressing further action of the follicle-stimulating hormone. Moreover, these two hormones are responsible for the production of the *sex hormones,* i.e. the oestrogen formed in the ovary and the androgen in the testicle.

III. — *Luteotrophic hormone.* Although the luteinizing hormone is essential for the formation of the corpus luteum, it requires a third pituitary factory, viz. the luteotrophic hormone, to maintain the secretion of progesterone from the corpus luteum. There is reason to believe that this factor may be identical with the lactogenic hormone (prolactin) which is responsible for the actual secretion of milk after the building up of the mammary glands under the influence of the oestrogen-progesterone complex.

IV. — *Placental gonadotrophins.* In addition to the above pituitary hormones it is of interest to note that in certain species during pregnancy, the placenta, which assumes the role of a temporary endocrine organ, elaborates hormones very similar to those normally produced in the pituitary and gonads. Thus in the woman right from the onset of pregnancy, gonadotrophin arising from chorionic tissue is secreted in the urine which through its presence constitutes the basis for the Zondek-Ascheim pregnancy test. This hormone is mainly luteinizing in character and simulates the L.H. (or I.C.S.H.) of the pituitary. In the mare, on the other hand, there is a marked concentration of follicle-stimulating hormone resembling F.S.H., which is detectable in the serum from approximately the 40th to the 170th day of pregnancy. The appearance of this hormone coincides with the nidation of the embryo and is in all probability formed within the endometrial cups of the equine uterus. Why these hormones have thus-far only been demonstrated in primates and equidæ and not in any of our other domesticated animals remains a matter as yet to be explained. Conceivably pregnancy can be maintained in cattle, sheep, pigs and dogs without the intervention of these additional gonadotrophins.

4. — The Sex Hormones.

Whereas the chemical composition of the various gonadotrophins has not as yet been fully established, there is reason to believe that they are of complex protein nature. Chorionic gonadotrophin from human pregnancy urine has in fact been identified as a glycoprotein. Much more, however, is known about the nature and composition of the sex hormones which are all sterol derivatives, being closely associated with the general sterol metabolism of the body.

Oestrogen, representing the female sex hormone, is elaborated within the maturing follicle as *oestradiol* and is excreted in the urine in a less active form as *oestrone.* A group of closely related oestro-

gens have also been identified in the urine of mares. Physiologically oestrogens are responsible for the development of all the sexual characteristics of the female including that of heat or oestrus, which coincides with ovulation and the initial vascularization and swelling of the uterine wall preparatory to conception. In addition to production in the ovary, oestrogen can also be formed in other parts of the body, and during pregnancy relatively large amounts are produced in the placenta and subsequently eliminated in the urine. There is reason to believe that oestrogen acts as a growth stimulant for many body cells, and it is also the primary factor concerned in the budding of the mammary duct system.

Progesterone. Closely related to oestrogen both chemically and functionally, progesterone secreted from the corpus luteum exerts an important trophic action on the uterus and is essential for the maintenance of pregnancy especially during the first half. Moreover, it is closely associated with the building up of the mammary gland, especially the secretory alveolar tissue.

Androgen or male sex hormone is elaborated primarily in the testicle in the form of testosterone and is excreted in the urine in a slightly modified form as *androsterone.* Male and female sex hormones are very closely related to each other, and both are excreted in the urine of females as well as of males, the only difference being that in the male androgen predominates and in the female oestrogen. The adrenal cortex is intimately concerned in the maintenance of the normal balance between male and female hormones in the two sexes. One of its active principles corticosterone is a closely related steroid with progesterone-like functions. There is evidence to suggest that the adrenal cortex is largely responsible for the regulation of sterol metabolism throughout the body.

From the above brief description is should be clear that normal reproduction is governed by the constant interplay of a variety of endocrine factors which themselves are continuously being subjected to both endogenous and exogenous influences.

5. — Hormone Therapy in Reproductive Dysfunction.

Based on our knowledge of the physiology of reproduction, much of which has been obtained from studies on laboratory animals under widely varying conditions, numerous attempts have been made either to stimulate or to inhibit reproduction or to correct the various forms of reproductive dysfunction in man and animals through the use of hormone therapy. Although success has undoubtedly been achieved along certain lines, it cannot be denied that with an exceedingly complicated problem such as this and the incompleteness of our knowledge, much of the work has yielded erratic and frequently disappointing results. These difficulties, however, can be largely explained by full appreciation of the fact that the principal reactions in the reproductive

organs, as pointed out by Friedman, are not confined to any one time scale and that we are confronted with phenomena which take weeks or months for completion and which involve intermediate steps in a progression from one magnitude of velocity to another. Apart from all other important considerations such as species, breed, age, sex, nutrition and environment, the choice and subsequent administering of hormones in true physiological manner remains one of our greatest difficulties. Further development of the implantation technique of hormone pellets under the skin, offers hope for a slower absorption and more physiological action of the hormone as compared with the results induced by rapid injection into the body.

As pointed out before, the normal rhythm of the female cycle is conditioned by a series of accurately timed stimulation-inhibition reactions arising from the mutual interaction of gonadotrophic and ovarian hormones, whereby a characteristic shuttle service between the pituitary and the gonads is brought into existence. Successful hormone-therapy demands the re-establishment and subsequent maintenance of these relations.

Based on the above physiological considerations, it should be quite clear that hormone therapy can on no account be considered as a panacea for the numerous forms of reproductive disturbances in our farm animals and that treatment of this type should not be entrusted to any unqualified person. Moreover, it is essential that all animals be carefully examined before hormone treatment is recommended. Where the presence of malnutrition, organic disease or other obvious cause can be established, hormone therapy is in any case definitely contra-indicated. Due to widespread malnutrition amongst farm animals, which is frequently of an insidious nature, correction of the daily diet should be the first matter attended to.

From the numerous publications, mainly American, British and Russian, which have appeared recently on the results of hormone treatment on the reproduction of farm animals, it is clear that the data are still very divergent and frequently of a contradictory nature. This is no doubt due to the marked differences of the conditions under which experimental work is conducted as well as to differences in the hormones used and the methods of their administration.

The main hormones used thus far include: (a) *Gonadotrophins* derived either from the pituitary itself, from human pregnancy urine or from mare serum, and (b) *Oestrogen*, either as the natural product formed in the body and frequently used in the form of oestradiol benzoate or as one of the various synthetic oestrogens, of which stilboestrol is the commonest; (c) *Androgen*, usually in the form of testosterone, obtained from bull's testicles.

In *cows* showing functional infertility as a result of hypogenitalism attended by anoestrus, the injection of moderate doses of pregnant

mare serum at intervals of ± 19 days has been found to initiate normal cycles associated with oestrus, ovulation and ending in conception usually after the second or third oestrous cycle. The results, however, may vary widely depending upon a multiplicity of factors. Thus with the presence of a retained corpus luteum, hormone therapy is usually of no avail, while manual expression of the corpus luteum as a rule leads to prompt reappearance of oestrus within a few days and normal re-establishment of the cycle. Excessive doses of pregnant mare serum may result in multiple ovulation in cows with the development of two or more foetuses frequently terminating in their death.

The use of oestrogen, commonly administered as sythentically prepared stilboestrol, likewise yields unpredictable results when used on anoestrous cows. In some animals stimulation of the pituitary by oestrogen therapy may lead to normal oestrus and ovulation, while in others nothing definite is achieved. In any case, the use of massive doses of stilboestrol is contra-indicated, as these inhibit gonadotrophic activity in the pituitary. While the implantation of stilboestrol tablets into virgin heifers and goats stimulates the production of an apparently normal milk flow, the use of this oestrogen in lactating cows and parturient women promptly decreases the milk flow. Regarding the treatment of cystic ovaries and nymphomania in cows it is claimed that this can be successfully relieved by the injection of crude pituitary extract. It is obvious, however, that far more data of a comparable nature are required before they can be fully assessed.

In ewes the main object with hormone treatment thus far has been to study its effect on the characteristic seasonal anoestrus shown during the initial summer months and if possible to shorten this period so as to expedite the following lamb crop. Again, however, the results of work conducted in different countries are widely at variance. In this connection differences associated with breed, nutrition, climate and stage of anoestrus all constitute important considerations in evaluating the data.

From our own observations in sheep in South Africa in confirmation of what has been noted by workers overseas, ewes are commonly subject to the phenomenon of "silent heat," i.e. ovulation may take place without any concurrent signs of oestrus, the result being that such ewes are not detected by the ram. To what extent this takes place throughout the seasonal anoestrus period has not been fully established, although there is evidence to indicate that ovulation without heat frequently occurs both during the initial phase of anoestrus and again towards its end. Consequently it can be expected that the effects of hormone therapy will be determined by the stage of anoestrus at which it is instituted. Moreover, nutrition appears to be a factor of primary consideration in determining the duration of the anoestrus period in sheep. The fact that these points are not always fully

reckoned with may explain, at least in part, the wide divergence of the data which have been presented in the literature.

According to our own results as well as those of many other workers, the use of either oestrogen (stilboestrol) in single doses or gonadotrophin (pregnant mare serum) on ewes causes prompt ovulation in a large percentage of cases, although again it may not be detected, seeing that it is frequently unaccompanied by any signs of oestrus. An excessive single dose of p.m.s. frequently causes marked proliferation in a shower of follicles which, instead of ovulating, tend to become cystic. Injection of smaller doses of p.m.s. repeated at 16-day intervals, according to reports from different sources, leads to initiation of normal cycles in a good percentage of cases attended by oestrus and fertile ovulation. Again, however, more comparable data are required in order to assess the full value of hormone treatment.

In *mares, sows* and *bitches* the results to date are insufficient to allow of any conclusive deductions to be drawn regarding the efficacy of hormone therapy.

In conclusion it may be stated that despite the prospects held out by the use of hormones in correcting reproductive disorders amongst our farm animals, methods are as yet by no means standardized. With the availability of increased hormone supplies at much cheaper rates it can, however, be predicted that more conclusive information may be expected within the near future. In this regard it is trusted that our own veterinarians will actively contribute to the growing pool of knowledge, especially in so far as this is affected by conditions peculiar to South Africa.

CASE REPORT.

BONES IN THE OESOPHAGUS.

P. ROBERTSON,
Cape Town.

A four-months-old Irish Terrier Cross pup was admitted to hospital with the history that he had swallowed a bone two days before. He was very hungry, ate well, but vomited food as a sausage-shaped mass shortly after. Liquids were not vomited.

The pup appeared quite well and lively. I gave him a solid feed, which he vomited two minutes later; the form of the vomit satisfied me that there was some obstruction in the oesophagus. X-ray revealed an obstruction at the entrance to the stomach. The animal weighed 13 lbs. He was given an intravenous injection of 2 cc. of Nembutal, which brought about complete anæsthesia and relaxation. The neck was then stretched out well, a long forceps inserted into the oesophagus,

the bone was grasped, and, taking time, it was gently dislodged and removed. On completing the operation I noticed that the animal had stopped breathing and appeared to be dead. I remarked to my assistant that we had successfully removed the bone, but had killed the patient. We then proceeded to examine the bone and discussed the operation. Quite some minutes afterwards — for the want of something better to do — I gave the limp body a good-bye pat and in so doing I noticed that pressing the air out of the lungs was followed by inhalation; I continued this artificial respiration for some time, just assisting him to get rid of the air. I found by immobilising any chest movement by grasping the costal area with my hands, that the abdominal muscles came into play and did the breathing action quite well. I called for a 3-inch bandage which I rolled tightly around the chest, immobilising any chest movement as my hands had done. The dog was now breathing comfortably as described. I put him away for the night in a box with a hot-water bottle. Next morning he was allowed out, the bandage removed, and he was found to have recovered completely and that the breathing had become costal again.

I have written this case up because I had a similar one following it, but in the second case the anæsthetic used was chloroform, the bone was at the top of the oesophagus and was only removed after great effort on my part. This animal reacted to the method used in the first case, only this one could not eat for a few days owing to the soreness of the throat, which was somewhat lacerated as a result of the force used to extract the bone.

It may be suggested that the paralysis was brought about by injury to the vagus nerve, but I think it is more likely to have been caused by the pulling and stretching of the oesophagus in the long direction.

CASE REPORT.

P. L. UYS,
Dundee.

The following is a short report on a case of acute prussic acid poisoning which was encountered in the Helpmekaar District, Natal:—

On the 8th November, 1945, at about 11 a.m., I was called out to a farm to investigate the sudden deaths of eleven cows. The only information I could gather telephonically was that fourteen animals had taken ill soon after they had started feeding on the young leaves of a "Kaffer-wag-'n-bietjie" tree which had been chopped down by the owner, for the purpose of supplying some form of green feed to his cows. It was also stated that eleven cows died within an hour after they had commenced feeding, and that one ox, a cow and a heifer were still sick.

On arriving at the farm at about 1 p.m., I found the eleven cows lying dead on a small area of ground next to a huge "Kafferwag-'n-bietjie" tree which had been chopped down. This tree stood on a rocky hill with hardly any grazing on it. On being questioned, the owner informed he that at about 9 o'clock that morning he took his animals, viz. twelve milking cows, one ox and a heifer, up the hill to feed them the leaves of this tree. He also informed me that his animals were accustomed to eating the leaves of the "wag-'n-bietjie" and also those of species of Acacia, especially the "Soetdoring." He had been regularly chopping down these trees for his cows, the leaves of which they eat greedily.

He further informed me that as soon as the tree was chopped down the animals rushed up and started eating the leaves. According to his information he was busy for about five to ten minutes chopping off smaller branches and pulling them away to give the animals better access to the leaves, when he noticed one cow showing signs of muscular inco-ordination, distress, shivering, and anxiety. While he was more closely examining this animal, a second one started showing similar symptoms, and within about 15 to 20 minutes' time all the animals were affected, showing the same symptoms. In the meantime some of the affected animals fell down and could not get up again, showing signs of respiratory distress and "drunkenness". On noticing this he went home quickly, his home being about 600 yards from the scene, to fetch some lard with which to dose the animals. On returning he found that three cows were already dead, eight others were lying down, while one cow, a heifer and the ox were still standing. He managed to dose some of the animals with the lard, but they all died shortly afterwards, except the three animals mentioned. These had not shown such acute symptoms.

I made a diagnosis of prussic acid poisoning. Stomach contents and leaves from the tree were afterwards tested for prussic acid, with positive results.

The three remaining animals were each given a quantity of a 20 per cent. solution of sodium hyposulphite intravenously. When the solution was administered the cow and the heifer were lying down, but got up in about 15 minutes' time and started walking about. The ox was still standing, but it also showed an improvement after the injection.

The post-mortem appearances of five of these cows were those of acute prussic acid poisoning, viz. cyanohæmoglobinæmia, engorgement of veins, distension of right ventricle of heart, hoven, hyperæmia of lungs, liver and parts of the intestine, hæmorrhages in the epicardium and other serous membranes. A fair amount of the leaves of the tree was found in the rumen.

The day on which the animals were poisoned can be described as cool and cloudy with no sunshine. The previous two days had been

very hot and windy, and in my opinion the large amount of prussic acid formed by the tree in its leaves can be attributed to this sudden change in the temperature. The affected animals were all in poor condition.

SUMMARY.

Fatal cases of acute prussic acid poisoning are described in cattle due to the ingestion of young leaves of the "Kaffer-wag-'n-bietjie" tree.

CASE REPORT.

A CASE OF DEPRAVED APPETITE AND APPARENT TOLERANCE TO INGESTION OF TOBACCO IN A DOG.

E. C. S. DAWE,
Lobatsi, Bechuanaland Protectorate.

The subject is a Smooth-haired Fox Terrier bitch aged nine years and weighing 28 lbs. Except for obesity of probably hereditary and perhaps also of endocrine origin, the animal appears to be normal and in good health.

Some six years ago this bitch developed the habit of swallowing lighted cigarette ends, and the writer has frequently seen her swallow in a few minutes as many as six such butts of usual size, viz. from one-quarter to one-third of a cigarette. Although the bitch was kept under observation, on no occasion could any toxic effect be detected.

Virginia tobacco is said to contain about 2 per cent. of nicotine and it is estimated that a stub would contain about 6 milligrammes, but without recourse to direct analysis it is difficult to compute the actual quantity of nicotine consumed, as it is believed that a cigarette end contains more nicotine than a portion of unlit cigarette of similar size. The writer estimates, however, that the consumption of six ends would entail an intake of little short of a minim of nicotine. H. Kirk records definite toxic symptoms in a cat which had swallowed one-third of a cigarette, and quotes Finlay Dun as stating that a single drop of nicotine destroys small dogs and rabbits in five minutes, producing convulsions and paralysis. It seems likely therefore that the subject possesses some degree of tolerance to the drug.

The cause and origin of the habit are obscure. It is indulged in freely if encouraged, but encouragement is not essential, as the animal has been known to snatch a lighted end from an unsuspecting hand. More usually, however, the bitch approaches a live butt which has been thrown on the ground, sniffs at it, licks her lips several times,

and with a quick movement licks it up, chews it for a moment with a smacking noise and swallows it.

Of the explanations considered, dietetic deficiency is discounted by the fact that raw meats of various kinds and milk are regularly and plentifully included in the diet. Metabolic disorder, possibly associated with the endocrine glands, is suggested as a possible factor, as the animal has been known to lick cigarette ash from the floor. But, as the subject evinces no interest in unlit cigarettes or cold butts, the writer inclines to the view that the habit is of psychopathic origin associated with a fear of fire and developed as a parlour trick.

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THE MANUFACTURE, SALE, KEEPING, ETC., OF POISONS, HABIT-FORMING DRUGS AND BIOLOGICS.

B. S. PARKIN,
Onderstepoort.

The authorities in charge of the regulations governing the control of *poisons, habit-forming drugs,* and *biologics* have recently become more active in the enforcement of the regulations, and it is considered advisable to draw the attention of all veterinarians to the regulations governing the importation, manufacture, dispensing, etc., of the above-mentioned substances. In certain cases there appears to be some doubt as to the rights of veterinarians, and various contentious points may have to be decided by the Courts. This summary represents the personal opinion of the writer.

The Medical, Dental and Pharmacy Act (Act No. 13 of 1928) regulates the keeping, the sale and the use of poisons and habit-forming drugs, and the Diseases of Stock Act (Act No. 14 of 1911) the importation, use, etc., of biologics for the diagnostic and the treatment of stock.

Poisons. — A poison for the purpose of Act 13 of 1928 means one of those drugs listed under Divisions I and II of the 4th Schedule. The list is a fairly comprehensive one and a copy of it should be in the possession of every veterinarian.

(a) *Manufacture of preparations containing poisons.* — Provision is made in Act 13 for the manufacture for human use by a competent person (other than a chemist and druggist) of remedies containing poisons. A legitimate assumption is that remedies containing poisons may be manufactured for animal use by veterinarians. A permit issued by the Minister of Public Health is apparently essential.

(b) *Keeping of poisons.* — If a veterinarian keeps poisons for the purpose of sale, manufacture or dispensing he must comply with the requirements of Act 13 as to labelling, storage, etc., and must record in a "Poison Book" various details such as quantities, nature, etc. His premises and his poison book are open for inspection by a properly authorized person.

(c) *Sale of poisons.* — Provision is made for the sale of poisons and preparations containing poisons under a certificate issued by a magistrate. A veterinarian may, however, supply poisons in the course of his practice for the treatment of animals under his care. The sale of poisons in bulk or by wholesale for subsequent retailing is also permitted under certificate, but a certain amount of doubt exists whether a veterinarian would be permitted to sell directly to a consumer such preparations for the treatment of animals not under his care.

(d) *Dispensing of poisons.* — Act 13 states quite clearly that a veterinarian may dispense preparations containing poisons for the treatment of animals under his care.

Habit-forming drugs. — These drugs are listed in the 5th schedule of Act 13 of 1928. Additions to the list are made by proclamation from time to time. It is the right of a veterinarian to import, acquire, use, supply, etc., habit-forming drugs in the course of his practice for the treatment of animals under his care. He is, however, required to fulfil the requirements which include the keeping of a "Habit-forming Drug Book." The control of such drugs is set out in great detail in Act 13, and the penalties for infringement of the requirements are severe.

A veterinarian, as mentioned above, is an authorized veterinarian, i.e. he holds a certificate, current at the time being, issued to him by the Minister of Public Health. The possession of such a certificate is essential for the prescribing of poisons and habit-forming drugs.

Biologics. — These include vaccines, sera, antigens, etc. The importation and keeping of biologics for use in stock are controlled under the Diseases of Stock Act (Act No. 14 of 1911). A permit issued by the Minister of Agriculture is necessary for the importation, manufacture, keeping, use, etc., of biologics gazetted as such and capable of causing any infectious disease in stock. More recent regulations govern both the use of biologics for treatment and for diagnostic purposes. Under the latter head are included mallein and tubervulin. The Principal Veterinary Officer has to approve of the use by veterinarians of these diagnostic agents of Government Notices 638 1915 and 1256 of 1923.

In view of the provisions of the Veterinary Act (Act No. 16, 1933) the Principal Veterinary Officer is not prepared to authorize the issue of mallein and tuberculin to persons whose names do not appear in the Register referred to in this Act.

OBITUARY.

RICHARD JAMES WHITE.

The death of Richard James White, better known as "Jock" White, on Sunday, 20th January, 1946, will be regretted by all who knew him, and particularly by the veterinary profession in South Africa. Born in Sutherlandshire some 70 years ago, Jock came to the Cape during the South African war as a soldier in the Seaforth Highlanders. At the cessation of hostilities, he joined the staff of Sir Arnold Theiler at Daspoort, and later proceeded with Sir Arnold to Onderstepoort. Until 1931 he was employed as a technical assistant at Onderstepoort, where he worked in many different sections and became one of the most experienced of the senior men. In 1931 he retired, and after a brief venture in dairy farming, he joined the staff of the South African Institute for Medical Research, Johannesburg. There, until his death, he was employed as horsekeeper, first in Johannesburg and for the last four years at the Serum Laboratories at Rietfontein.

All who knew White will testify to his cheerfulness, his willingness to help, and his ability to turn his hand to anything. His type is becoming rare nowadays.

To his widow and children, the profession offers its sincere sympathy.

J.H.M.

MOFFAT ON ANTHRAX.*

"He (Peclu, a young Bechuana Chief) died of what is called kuatsi, a disease that appears to be endemial, which assumes the form of a carbuncle, and carries off many cattle; and as the natives will on no account abstain from eating the dead meat, they are often attacked by it. If it happens to be near a vital part, as in the case of Peclu, it is very frequently fatal; if internal and not suppurating outwardly, it is always so. The meat of goats which have died of this disease is particularly noxious, and I have known persons cut off in five days after having eaten it. It is always accompanied by considerable swelling, attended with great stupor, though with comparatively little pain. I write from experience, having had one on my right eyebrow, which gave my constitution a severe shock; and from its position my recovery was considered very doubtful. From long observation, I have found it important to give aperient medicines, scarify the pustules, and get some one to suck it, either with an instrument or the mouth, and to apply any kind of cataplasm to promote a discharge; it is also important as much as possible to prevent the individual from being exposed to the cold air."

J.H.M.

* "Missionary Labours and Scenes in South Africa," by Robert Moffat. London; John Snow, Paternoster-Row, 1842.

LADY ANNE BARNARD ON VACCINATION.*

"In one of the last ships (few there are who honor the poor Cape with a call) there is come a Doctor Tytler,..... a man of infinite Greek, Latin, Hebrew, &c., &c.,....." "I understand he is to inoculate here the *cowpox,* lately discovered in England, to be a preventative against the *smallpox* ever being caught afterwards. I suppose it would be thought a very high honor that of being inoculated from *a cow* in Bengal, and a child of a Brahmin only coud aspire to do it; but I own the idea of being inoculated from an animal makes me shudder. Don't tell, as I should be laughed at if it is the fashion in England. Here a very few successes will render it general, the idea is already caught up with avidity by the Dutch....."

J.H.M.

* From a letter from Lady Anne Barnard at Vineyard, Cape of Good Hope, December 31st, 1800, to the Earl of Macartney. "Lady Anne Barnard at the Cape of Good Hope, 1797 - 1802," by Dorothea Fairbridge.

BOOK REVIEWS.

The "Manual of Veterinary Clinical Pathology,"* by Dr. Davis L. Coffin, a publication of the Comstock Series in Veterinary Medicine, was originally published in 1944 as an enlargement of the laboratory notes used in the combined course of Clinical Pathology and Laboratory Diagnosis in the Pennsylvania School of Veterinary Medicine. New material added in the present edition makes it a very readable and useful reference manual. It is useful particularly to the laboratory diagnostician and to the veterinarian who wishes to augment his findings, obtained by physical examination.

Many of the techniques described are applicable without intricate and costly equipment, and do not presuppose any particular experience in laboratory work. Useful hints are given on how to avoid certain pitfalls that one who is not familiar with or trained to the technique may encounter.

The scope of the book includes chapters on the collection and packing of specimens for laboratory diagnosis, parasitological examinations, examination of urine and interpretation of findings, diagnostic methods in bacterial, mycotic, protozoan and virus diseases, fertility examinations, poultry post mortems, and, finally, a chapter of formulæ of all solutions and reagents used in the techniques described in the book.

Scattered through the book are very useful tables which facilitate quick reference and are thoroughly practical. The value of the book is further enhanced by the lists of references after most chapters. The illustrations are very much to the point.

Criticisms are mainly on minor points. To my mind, too brief a reference is made to the use of the Van den Bergh test on serum in

* *Manual of Veterinary Clinical Pathology,* by David L. Coffin, V.M.D., School of Veterinary Medicine, University of Pennsylvania; pp. viii + 263. Figs. 66. Ithaca, New York: Comstock Publishing Company, Inc. 1945. Price $4.00.

order to differentiate between "direct" or hepatic bilirubin and "indirect" or hæmolytic bilirubin. Some of the methods recommended for submission of material to laboratories seem somewhat laborious. In the case of anthrax, for instance, it is stated that laboratories like to receive a few drops of blood between two slides, a small sample in a test tube, or a ligated ear, whereas in this country an ordinary dried bloodsmear has always been found to be sufficient for all ordinary diagnostic purposes. Refrigeration in transit of samples for brucellosis seems hardly necessary when we consider the satisfactory results obtained in this country by adding a few cc. of 5 per cent. boric acid solution to blood samples. Refrigeration may also be ideal for rabies-suspected material, but all the South African diagnostic work is done on brain material transmitted in 10 per cent. formalin (for histopathological examination) and 50 per cent. glycerine (for biological tests).

The book is well arranged and of a convenient format, and should find ready application as a complement to good physical examination technique.

W. D. MALHERBE.

* * *

A second edition of Kirk's "Index of Diagnosis"* has recently appeared, within five years of the original publication, under conditions of acute wartime paper shortage. It appears to be smaller, but actually contains more reading matter.

There is no doubt that the popularity of the book is in the main due to the method of presentation of the subject matter. The arrangement is encyclopædic, symptoms being given alphabetically and each one being treated as a differential diagnostic problem.

Treatments are also given, but these could with profit be made more concise, and in many cases modernised. One might even suggest that the book as a whole be condensed to manual form, giving only selected modern treatments. This could also apply to the descriptive matter.

The vexed question of Stuttgart Disease, which is treated at great length, is, as the writer points out, very confused, and one is left with the impression that the vast majority of so-called Stuttgart cases are nothing but the syndrome of uræmia. Most of these would be the result of acute renal insufficiency due to acute or chronic interstitial nephritis. In the opinion of the reviewer, the term Stuttgart Disease should be limited to that caused by Leptospira canicola.

And talking of nephritis, various parenchymatous forms are described which are known not to occur in dogs although they are the usual ones in man. The rather vague use of the term "kidney disease" or "renal disease" should perhaps be avoided.

* *Index of Diagnosis for Canine and Feline Surgeons*, by Hamilton Kirk, M.R.C.V.S, Major R.A.V.C., Member British Institute of Radiology, Lecturer on Animal Management, L.C.C. Pp. 587. Figs. 302. London: Bailliére, Tindall & Cox. 1945. Publ. price £1.15.0.

Random sampling reveals some misconceptions, e.g. the suggestion on p. 161 that coccidiosis in dogs results from close contact with rabbits and poultry; diagnosis of rabies, *inter alia,* by saliva smears for detection of the virus; and the reddish-brown colour of the urine as an important diagnostic aid in canine piroplasmosis.

The author has had considerable experience of radiography and gives a wealth of information on this subject as applied in the veterinary clinic. The radiographs are widely representative, but suffer somewhat in clarity no doubt on account of wartime printing difficulties.

The book's continued popularity is assured and it is a valuable contribution to the literature of veterinary science. The reviewer, however, as indicated above, feels that its value would be greatly enhanced if it could be carefully edited and condensed before being printed as a third edition.

W. D. MALHERBE.

ANTISEPSIS

An authoritative statement

'The most reliable procedure for the complete elimination of streptococci from the naked hands is as follows: Wash for one to two minutes in a pint of warm water, using plenty of yellow bar soap and a nailbrush to the nail sulci; then pour into the palm of one hand a teaspoonful of neat Dettol, or a thin tragacanth paste containing 30 per cent. Dettol, and work into the skin of the hands till dry (one to two minutes).*

An antiseptic with a high Rideal-Walker coefficient whose bactericidal activity is well maintained in the presence of blood, pus or other organic matter; lethal to a great diversity of bacteria, including hæmolytic streptococci; non-poisonous even at full strength and applicable, without causing pain or injury, to raw wounds and surfaces; which does not inhibit the natural processes of repair; which is stable at all clinically desirable temperatures and at all dilutions; which is non-staining, agreeable in use and pleasant to smell.

This list of qualities might well describe the theoretically ideal antiseptic. In fact, it describes 'Dettol' — which in ten years has become the CHOSEN ANTISEPTIC, for the protection of patients and staff alike, in nearly every hospital in the British Empire.

* *Colebrook, L. (1933), "Brit. Med. J.," 2,725.*

THE TRANSPORT OF ANIMALS BY SEA BETWEEN SOUTH AFRICA AND INDIA DURING WORLD WAR II.

R. K. LOVEDAY,
Capt., S.A.V.C.

1. Introduction.

In May, 1942, an Indian Remount Purchasing Commission commenced buying animals in South Africa for shipment to India, and it became the work of the S.A. Veterinary Corps to conduct these animals from Durban to Karachi. During the period August, 1942, to September, 1945, 58 shipments were made, all except two of which were in charge of S.A. personnel. In all, 22,016 mules, 10,022 donkeys, 3,527 horses, 323 bovines, 2,259 pigs, 1 zebra and 3 Angora goats were shipped, a total of 38,151 animals. In March, 1943, one ship was, unfortunately, torpedoed with 737 animals on board, but no further losses from enemy action occurred.

2. Preparation for Shipment.

The animals were prepared for shipment at Durban and Pinetown, and everything possible was done to fit them for the long sea voyage ahead. It was inevitable that animals from inland should lose condition for the first few weeks in the damp Natal coastal climate, but thereafter they improved rapidly. In the Embarkation Depot, horses and mules received the following ration, which aimed at accustoming them to the ration they would receive on board and ensuring that they were embarked with their bowels in a loose condition, a very important factor in avoiding subsequent digestive upsets at sea:—

8 lbs. teff hay.
4 lbs. grain (crushed oats and/or maize).
6 lbs. chaff (4 lbs. lucerne hay, 2 lbs. oat hay).
1 lb. bran.
¼ lb. linseed.
2 ozs. coarse salt.

The short feed was damped down with molasses diluted ten times with water. In season, green sugar cane tops were chaffed and fed in addition to the normal feed, and were greatly relished. In wet and cold weather the ration was increased to avoid condition being lost, and the limited grazing available was utilised. The animals were also exercised daily on the Durban beach to harden them as much as possible. At one time, before increasing numbers made this

impossible, horses and mules were led over a large ramp built in the Embarkation Depot, to accustom them to being loaded.

All mules and donkeys despatched during the hot monsoon season, between 15th April and 15th September, were clipped out except heads and legs. It was found expedient not to clip the tails of donkeys owing to the unfortunate habit these animals have of leaning back against the back bail of their stalls and chafing their tails. Each consignment was carefully selected for shipment by a veterinary officer, to ensure that, as far as possible, only good conditioned animals were shipped, and no cases of contagious disease or obviously sick animals were embarked. The average duration of the forward voyage was about 20 days, but forage for 24 days was always put aboard. It is essential that forage for animal transports be very securely baled, as it is subjected to much rough handling. Forage should also obviously not be loaded during wet weather, as it soon goes mouldy in the ship's hold.

The Animal Embarkation Depot at Durban is situated about 3½ miles from the docks, and the practice was to lead horses and mules, 4 - 5 animals to each man, and drive donkeys to the docks. This resulted in a large number of coronary treads being suffered by the animals, and later they were driven to the docks with a mounted leader and five outriders, with better results.

3. Ships' Fittings.

All ships were fitted with the usual Board of Trade fittings athwart the ship, a pen of four stalls for four large horses or mules having a breadth of 9′ 10″ and a length of 7 - 8′, and fitted with a platform on which the animals stood. Four parting boards, sliding in grooves could be utilised to partition off individual stalls or to alter the size of the pens. The breast rails fitted into brackets on the front stanchions and were removable. Each front stanchion was fitted with two halter rings, one above and one below the breast rail, and also a staple for hanging the hay net. A breadth of not less than 5′ was allowed as a gangway between two rows of stalls. It was found by experience that animals could be loaded in the following proportions:—

5 large horses or mules to four stalls.
5 ponies or small mules to four stalls.
11 donkeys to eight stalls.
1 bovine to two stalls.

The capacity of each ship was estimated on this basis. Pigs were usually shipped in crates and only during cool weather. Before the animals were embarked heavy coir mats were laid on all decks and alleyways to afford foothold and were well secured to prevent slipping. Parting boards were removed to allow free access to all stalls, and hay put out, so that the animals commenced to feed as soon as they were loaded and thus settled down more quickly. All brows leading

from one deck to another were fitted with 6′ sparred sides and hardwood battens, were padded at sharp corners and provided with padded head guards where necessary. Animals were shipped unshod, and it was found that no undue wearing away of the horn occurred during the voyage, provided excessive moisture underfoot was avoided.

4. Embarkation.

Horses and mules were led up the gangway singly and then to their stalls and secured. Later, a lead pony only was used and 10 - 12 mules were allowed to follow it into the ship. This method was very successful, and considerably shortened the loading time. Litter strewn at the entrance to the gangway helped to give the animals confidence. Donkeys proved the most difficult class of animal to load, and their persistent refusal to proceed in any direction except backwards was

Donkey being slung ashore at Karachi.

a source of much annoyance and back-breaking labour to the loading personnel. All sorts of ruses in loading were tried, such as blindfolding, backing, tying the ears together and using a lead animal, but usually in vain, resulting in a rope being passed round the quarters and each donkey being forcibly hauled on board, with unavoidable injury of many animals. Later, a suitable box was used to sling donkeys aboard four at a time and worked quite successfully. Disembarkation at Karachi followed much the same pattern, except that donkeys were sometimes put ashore in slings.

Each conducting party was commanded by a veterinary officer, and personnel were allocated on the basis of roughly 1 European and 10 Cape Coloureds to each 100 animals carried. As the work on a mule ship is very strenuous, it is essential that all such personnel are absolutely physically fit.

5. Ventilation.

The top deck was naturally the best deck as regards the comfort of the animals, and the tween decks were generally regarded as the hottest and also the worst drained, which gave them the highest humidity also. The parts of each deck next to the bulkhead were always the most airless, especially against the engine-room bulkheads, where the heat was often excessive. All animal decks were fitted with thermometers and temperatures of 90 – 100°F and even higher were not at all uncommon, especially on the tween decks.

Ample means of ventilation, especially for lower decks and holds, are of prime importance in animal transports, especially when these are sailing through the tropics, and were accomplished by various means. Firstly, all hatchways and portholes were kept open whenever possible, and iron windscoops fitted to the portholes to deflect air inwards. Permanent air trunks are fitted to each deck in pairs, furnishing an inlet and an outlet for air, and care must be taken that the cowl of the inlet is kept turned into the wind and that of the outlet away from the wind. Canvas windsails were arranged from the rigging with their mouths to the wind, and functioned as air inlets only, the air coming down the long funnel into the required deck. These windsails are very liable to become blocked or collapsed, and must be frequently inspected. Electric extractor fans were usually also fitted in the corners of each deck, and if large and powerful enough, served to keep the air circulating. These fans should be fitted with adequate guards.

By far the best means of ventilation is with an electrically operated blowing machine, which directs numerous jets of fresh air into the deck, and ensures that a fresh atmosphere is constantly created everywhere. Mechanical ventilation, by means of fans and blowers is undoubtedly the most efficacious, and should, if possible, be fitted to all lower decks. It is of special value in the calm hot weather often encountered round the equator and in the Arabian Sea, when all other means of ventilation are rendered practically useless. The fitting of mechanical ventilation throughout the ship was very often limited by the force of the ship's dynamo, which should be powerful enough to carry the extra burden of the fans and blowers as well as the normal ship's lighting. As a total black-out prevails at sea during wartime, lighting of animal decks is naturally very meagre, and may consist of one or two heavily screened blue lights fitted on the further corners, so that a very dim illumination is provided which, nevertheless, allows of inspections being carried out at night.

6. Water and Drainage.

Fresh water was pumped by the ship to tanks on each deck, and from there was run into a wooden tub and conveyed with buckets to the metal mangers used for watering and feeding the short feed.

These mangers should be absolutely watertight, and hook on to the breast rail in front of each animal. The average water consumption of equines was between 6 and 12 gallons daily, depending on the weather. Drainage is always a difficult problem in mule ships, as the pipes leading from the scuppers to the ship's bilges, although

Embarkation of mules with a lead pony. Note funnel-shaped extension of gangway, built with bales of forage.

protected by a perforated cover, usually become blocked, so that urine has to be raised by hand in buckets and thrown overboard. This represents considerable labour and must be done frequently during the day to keep down humidity and purify the atmosphere of the unpleasantly strong ammoniacal fumes of equine urine. All dung is raised by hand in baskets or sacks and stored on the top deck until it can be thrown overboard at nightfall.

7. Feeding.

For the first two to three days at sea all equines were kept tied up and fed hay only. It is a fact that most of the animals seemed to be "sea-sick" at first, and would eat very little. Donkeys especially took a long time to settle down in their new surroundings, and had often to be shown where the water was before they would drink. After this, some officers loosened the animals in their pens if the weather was not too rough, and a gradual commencement was made with the feeding of short feed. This was mixed in the wooden feed boxes provided for each deck, and each horse or mule received the following ration:—

3 lbs. compressed oat fodder
1 lb. bran
¼ lb. linseed
2 ozs. coarse salt,

the whole ration being damped down with molasses diluted ten times with water. The linseed was soaked overnight in water before being mixed with the remainder of the feed. This short feed was fed in the metal mangers already mentioned, and after the feed the mangers were washed out and again filled with water.

Teff hay (12 lbs. for mules and horses; 8 lbs. for donkeys) was fed at either two or three subsequent feeds during the day, the largest amount being given in the evening. The use of hay nets was not found to be very satisfactory, for various reasons. Mules especially were inclined to gnaw pieces out of them, so that they soon became unserviceable, the animals often pushed them into the water-troughs, and the hay often got into the animals' eyes. Hay nets also take a lot of time to fill. It was found that by feeding the hay off the deck these disadvantages were overcome, the animals did not fight so much over the hay and more time was available for the all-important task of keeping the ship clean. Feeding hay off the deck was generally practised later, although it has the disadvantage of being a more wasteful method of feeding.

8. Routine.

Some account can now be given of the routine adopted on board during the actual voyage:—

Mucking-out of the whole ship was performed daily where

possible, the animals being moved out of each pen to the deck, while the platforms were lifted and scraped, and all dung and urine removed. Although no ships had any spare space for the exercising of the animals, the fact that they were in pens and could thus move about a little, and were also moved out every day while mucking-out was in progress, seemed to provide sufficient exercise to prevent any ill effects from the voyage. The feeding of bran also did much to reduce the incidence of oedema.

The following is a specimen of the type of routine followed at sea, and indicates how the animals were managed:—

A.m.: 0500 hours, reveille; 0515 hours, roll call; 0525 hours, empty water troughs, clean and prepare for short feed; 0700 hours, short feed; 0730 hours, breakfast; 0830 hours, clean troughs and fill with water; 0845 hours, raising forage from hold; 0900 hours, mucking-out, cleaning decks, inspection of animals; 1200 hours, long feed; 1215 hours, dinner; p.m.: 1300 hours, water; 1345 hours, stables, grooming O/C's. inspection of general health and condition of animals, inspection of teeth and feet, etc., continuation of mucking-out where necessary; 1630 hours, water; 1700 hours, long feed; 1730 hours, break; 1800 hours, supper; 1830 hours, picquets posted; 1900 hours, fill water troughs for night; 1930 hours, O/C's. final inspection.

The condition of the animals depends greatly on the care given to watering, and it will be noticed what an important place this has been given in the daily routine. The ship must be kept as clean as possible, and every effort made to keep the humidity down. As every animal deck has some parts which are hotter than the remainder of the deck, the animals should be re-arranged frequently, so that the same animals do not stand in the hottest parts, e.g. against the engine-room bulkhead, during the whole voyage. A strict lookout was kept for signs of distress, and such animals were immediately moved to a cooler part. In this way much can be done to combat heatstroke and heat exhaustion. The salt fed also helps in this respect, and also stops the animals from licking the lime wash from the stalls. The feeding of linseed was often discontinued round the equator as being rather heating. It was found that mules gnawed the woodwork a great deal, especially at night, and that donkeys are prone to lean back on the back bail of their pens and chafe their tails. For this reason the back bail of stalls intended for donkeys should always be padded. The voyage back to South Africa was occupied in cleaning out the ship thoroughly, and in lime-washing the fittings.

9. Veterinary.

All ships were well equipped with veterinary instruments, dressings and drugs, usually housed in a special veterinary pharmacy. Constant observation of the animals was necessary, especially for commencing cases of heat exhaustion and also colic, where speedy attention was

required. The administration of oxygen was tried as a treatment for heatstroke, but not, to the writer's knowledge, with good results. Nasal catarrh and strangles were commonly encountered, especially in horses of course, but in spite of inadequate space for proper isolation measures, did not spread as much as was to be expected. The Board of Trade stipulates that 5% of spare stalls are to be fitted for the isolation and treatment of veterinary cases, but in wartime it is rarely possible to observe this most desirable precaution.

The largest number of casualties was caused by minor wounds and injuries such as kicks, bites, bruises and chafes. As has been mentioned, donkeys were the greatest sufferers from chafes. A surprisingly large number of eye cases, chiefly conjunctivitis, were experienced, caused mainly by irritation from the teff hay.

The one zebra which was shipped was so wild that it was unfortunately injured during leading, and had to be destroyed later.

Table 1 shows the conditions encountered, as far as these were recorded, and gives a good idea of the main causes of casualty which may be expected in work of this nature. Table 2 gives the causes of death, and is exclusive of the 737 animals lost by enemy action. As was to be expected, the chief causes of death were heatstroke, digestive disturbances and pneumonia, in that order. The percentages do not reveal any significant differences in the susceptibility of the various species to shipping conditions.

10. Horse-sickness.

It will be noticed from Table 1 that five mules and one horse are shown as having suffered from horse-sickness and recovered, and from Table 2, seven horses and two donkeys are shown as having died from horse-sickness.

These diagnoses should be taken with some reserve as no confirmation by subinoculation of the blood into a known susceptible horse was carried out, except in one case when the blood of an animal which was supposed to have died of horse-sickness on board ship was sent by air from Karachi to Onderstepoort, but was found to be negative on subinoculation.

Appreciation of the difficulty of making an accurate diagnosis of horse-sickness on board ship of immunised — or for that matter — non-immunised animals, from clinical symptoms, makes it a necessity for diagnosis to be confirmed by a biological test, a procedure which is in no way a criticism of the veterinary officer in charge.

A brief summary of the procedure adopted in the Union Depots in respect of horse-sickness inoculation of animals to be exported would not be out of place.

During 1942 and 1943 all animals were embarked without previously being inoculated officially, although it must be accepted that some

animals must have been inoculated prior to purchase.

In April, 1944, a very severe outbreak of horse-sickness occurred at the Mooiplaas Remount Depot, so it was decided to inoculate all the Indian Government horses held in the Union at that time.

The inoculations at Mooiplaas were in the nature of a "block inoculation" experiment carried out by Dr. Alexander, of Onderstepoort, to arrest the mortality. From the experimental point of view it was unfortunate that the somewhat early beginning of winter introduced a complicating factor which effectively prevented a statistical evaluation of the results. It is, however, felt that the decision to immunise all the animals was a factor which prevented the number of deaths reaching alarming numbers.

It might be mentioned that although over 70 mules and horses died at Mooiplaas between 1st April and 15th May out of 2,050, not a single donkey contracted the disease, although the latter were running with the mules and horses, nor were any cases of horse-sickness in donkeys at any time reported from the other Depots (10,000 donkeys passed through the Depots).

No further inoculations took place until December, 1944, when it was decided to inoculate all the mules and horses which had been bought after the April inoculation and not yet exported, as well as all subsequent purchases. The Director of Remounts, India, also instructed that all horses and mules should be retained in the Union 28 days after inoculation.

From the middle of December, 1944, further instructions were received from India to the effect that all donkeys had also to be inoculated and retained for 28 days before embarkation. Thus from December, 1944, the practice of inoculation and retention for 28 days before embarkation became the routine procedure.

In view of the fact that about 40,000 equines were exported to India, some fully susceptible to horse-sickness, and others having varying degrees of immunity, it would be reasonable to assume that if any danger of the introduction of horse-sickness into India by the export of equines exists at all, it is not a great one and should therefore not be a deterrent in the export of equines to that country in peace time.

11. Conclusion.

In general, the health of animals transported by sea will depend on the attention paid to the factors of ventilation, watering, feeding, exercise, cleanliness and pre-embarkation preparation. Neglect of any one of these may prove disastrous, especially in war time, when animals have to be landed as fit for operational use as possible.

I wish to record my indebtedness to Captain S. W. Findlay, S.A.V.C., O.C. Animal Embarkation Depot, Durban, for much valuable

information supplied, and to the D.D.V. & R.S. for placing the official records at my disposal and permitting publication of this article.

SUMMARY.

A brief account is given of the preparation and shipment of 38,151 animals from South Africa to India during the period August, 1942, to September, 1945, of World War II.

LITERATURE.

1. Animal Management, 1933 (His Majesty's Stationery Office), pp. 253 - 276.
2. Manual of Horse-mastering, Equitation and Animal Transport, 1937. (His Majesty's Stationery Office), pp. 216 - 220.

TABLE 1: CASUALTIES.

	MINOR INJURIES.	COLIC.	NASAL CATARRH.	STRANGLES.	EYE CASES.	HEATSTROKE.	PNEUMONIA.	BRONCHITIS.	FRACTURES, SPRAINS AND LUXATIONS.	OEDEMA.	RINGWORM.	LAMINITIS.	HORSE-SICKNESS.	ARSENICAL POISONING.	ABORTION.	MINOR DENTAL AND DIGESTIVE TROUBLES.	* MISCELLANEOUS.	SUPPURATIVE PODODERMATITIS AND FOOTROT.	BILIARY FEVER.	TOTAL.	% MORBIDITY.
Mules	395	149	227	194	96	87	13	5	25	51	22	—	£	3	—	34	9	5	3	1323	6·3
Donkeys . . .	1060	25	67	34	49	5	6	3	2	9	14	—	—	5	5	10	62	9	4	1369	13·7
Horses	106	25	67	34	49	5	6	3	2	9	6	3	1	4	2	3	9	—	—	551	15·6
Bovines . . .	4	—	—	—	12	13	1	—	—	—	6	—	—	—	—	4	—	7	—	47	21·6
TOTAL	1565	209	330	461	230	119	25	19	29	68	48	3	6	12	7	51	80	21	7	3290	.

* Under the classification "miscellaneous" are included such conditions as small abscesses, fistulae, tumours, and ear canker.

TABLE 2: DEATHS.

	PROLAPSED UTERUS.	RUPTURED BOWEL.	VOLVULUS.	INTERNAL HAEMORRHAGE.	CACHEXIA.	HEATSTROKE.	HORSE-SICKNESS.	PNEUMONIA.	FRACTURE.	UNKNOWN.	RUPTURED SPLEEN.	ARSENICAL POISONING.	STRANGULATED HERNIA.	ENTERITIS.	RUPTURED ANEURISM.	BILIARY FEVER.	SEPTICAEMIA.	HEPATIC DEGENERATION.	ACUTE NEPHRITIS.	STRANGLES PERITONITIS.	TOTAL.	% MORTALITY.
Mules	1	7	3	5	1	24	7	2	2	7	4	1	2	4	1	2	—	—	—	—	74	0.33
Donkeys . . .	—	3	3	5	—	1	2	9	2	3	—	2	—	8	—	—	5	2	1	—	46	0.46
Horses	—	2	3	—	—	—	—	1	1	—	—	2	—	1	—	—	—	—	—	2	12	0.35
Bovines . . .	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	0.31
TOTAL	1	12	9	10	1	26	9	12	5	10	4	5	2	13	1	2	5	2	1	2	133	

AN EXAMPLE OF LOW BUTTER-FAT PRODUCTION BY A HIGH-GRADE FRIESLAND HERD.

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The problem of deterioration in the chemical quality of South African milk supplies was raised by Pullinger (1944), reference being made to the result of testing samples of bulked milk. In the present report, figures are given of the butter-fat production of an individual herd, which serve to illustrate the gravity of the situation that will develop if active steps are not taken to reverse this trend of deterioration.

The herd under consideration is situated on the Transvaal highveld, and it has been built up during the last five years, mainly by the introduction of high-grade Friesland cows purchased through a dealer from the Malmesbury–Darling area of the Cape Western Province.

In appearance, the herd is an excellent one, the cows being of a large type, very sleek and, if anything, in an over-fat condition. At the time of test, 80 lactating cows were producing 240 gallons daily, this average of 3 gallons per head being maintained by some cows giving up to 8 gallons. The diet at the time was confined to good quality teff grass supplied *ad libitum*, and a ration of commercial dairy meal restricted to 2 lbs. of meal per gallon of milk, whereas the manufacturers were recommending 3 lbs. per gallon.

RESULTS OF TESTS.

Butter-fat percentages were estimated by the Gerber method, the tester collecting samples in the stables at both the a.m. and p.m. milkings. Every care was taken to strip each udder properly, and all the milk was properly mixed before samples were taken. The a.m. and p.m. samples were tested separately, and the records are listed in this way. Facilities were not available for weighing the milk of each cow, and it is therefore impossible to give weighted average butter-fat percentages. Individual cows were not tested for non-fatty solids, but pooled can samples were tested for total solids by the gravimetric method.

Results of Testing Pooled Can Supplies.

These results are recorded in Table 1. The butter-fat percentages were extremely low, whilst the non-fatty solids percentages were on

the whole high for the time of the year, namely, October. These results agree closely with those which had been obtained when consignments were tested at the city receiving depot, results which had led to this detailed investigation being undertaken.

In Table 2, a record is given of the a.m. and p.m. butter-fat percentages of the 80 individual cows.

Discussion.

If it is remembered that the minimum standard for butter-fat is 8·5%, there is no need to comment upon the gravity of the position outlined here. The farmers' solution to the problem involves changing the feed, culling, introducing high butter-fat producers, discarding first milk, and even adding cream. The dairy industry, however, is not concerned with such palliatives, but rather with the circumstances which give rise to such a state of affairs. Possible explanations include:—

1. *Bad Breeding.* These cows did not come from a single breeder, but were collected at random in the Western Province. If bad breeding is to blame, the fault must be all too prevalent in that area.

2. *Bad Cows.* It can be suggested that farmers select their bad butter-fat producers for sale. If true, this means that the Transvaal is gradually being flooded with rubbish culled from Cape herds. In any case, the question still arises of the breeding system that produces 3 to 6 gallon cows giving only 2% of butter-fat.

3. *Faulty Feeding.* Dairy authorities are generally agreed that butter-fat percentages cannot be appreciably raised by feeding methods. This subject is discussed by Eckles, Combs and Murray (1943), and need not be elaborated upon here. It is also accepted that, if production is forced too much, the output of milk may exceed the capacity of the udder to secrete butter-fat, in which case, the percentage content of butter-fat falls by simple dilution. A further point of importance is that the dictum that butter-fat cannot be influenced by feeding has been developed on the basis of investigations done on normal cows receiving reasonably well-balanced rations. On the high-veld, however, at certain seasons even high-class herds may be subjected either to general semi-starvation, or at least to existence upon ill-balanced rations. In the case of the herd under consideration, it can hardly be claimed that teff grass and a restricted ration of commercial dairy meal constitutes an ideal balanced ration for heavy-producing dairy cows. However well balanced the dairy meal might be, in itself a moot point in view of concentrate shortage, theoretically one would like to see an

addition of silage, green barley or lucerne to the diet. In actual practice, the danger exists that such supplements would further increase the flood of milk and so dilute the butter-fat still more. In any case, it is justifiable to criticise a diet which at a comparatively low cost keeps cows in prime condition and producing large gallonages of milk. Theoretically, it might be possible to incorporate in a commercial meal some unspecified constituent capable of increasing udder productivity 33 per cent. above its natural maximum level of output, in which case butter-fat percentages would decrease by dilution. Whilst gallon-production can be increased, it is very doubtful if it could reach this high level. Alternatively an ill-balanced ration might produce a pathological state in which the udder is unable to synthesise fat to the normal degree.

Reviewing this discussion, it seems likely that the fundamental trouble is due to a general tendency on the part of breeders to aim too much at high gallonage producers, whilst paying but scanty attention to the nutritive constituents of the milk. It is, however, hard to escape the thought that some dietary factor has been superimposed upon bad breeding to produce the extraordinary results recorded here. This herd shows up in an exaggerated form the trouble that is brewing throughout the South African dairy industry.

Summary.

Records are given of very low butter-fat production in a high-grade Friesland dairy herd, and the manner in which this situation has developed is discussed.

Acknowledgments.

Thanks are due to the Officer Commanding the S.A. Veterinary Corps for permission to publish this article, and to N.C.O.'s of the S.A.V.C. Fod Inspection Section for the collection and testing of samples.

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TABLE 1.

Results of Testing Pooled Can Samples.

CAN NO.	BUTTER-FAT.	NON-FATTY SOLIDS.
1	1·9%	8·550%
2	2·2%	8·485%
3	2·1%	8·840%
4	2·5%	8·395%
5	2·2%	8·935%
6	2·4%	8·600%
7	2·1%	8·790%

TABLE 2.

Results of Testing Individual Cows' Milk for Butter-fat Content.

Cow No.	Butter-fat % a.m.	Butter-fat % p.m.	Cow No.	Butter-fat % a.m.	Butter-fat % p.m.	Cow No.	Butter-fat % a.m.	Butter-fat % p.m.	Cow No.	Butter-fat % a.m.	Butter-fat % p.m.
1	1·4	2·3	21	1·5	2·0	41	1·7	2·2	61	1·4	1·3
2	2·7	3·5	22	—	2·0	42	3·0	3·0	62	2·4	3·1
3	*Not	dry	23	2·2	2·8	43	1·1	—	63	3·1	3·2
4	2·7	2·8	24	1·7	2·0	44	1·2	2·1	64	1·5	1·6
5	3·1	**—	25	—	2·4	45	2·6	2·7	65	1·9	2·0
6	1·8	2·1	26	2·1	2·7	46	2·5	3·3	66	2·3	2·7
7	2·1	2·5	27	2·6	3·0	47	3·0	2·5	67	1·5	1·6
8	2·7	2·8	28	2·0	2·5	48	3·0	2·5	68	1·6	1·8
9	—	3·6	29	2·0	3·0	49	4·0	4·0	69	1·8	1·7
10	1·9	—	30	3·8	2·3	50	—	3·1	70	2·3	2·1
11	—	2·1	31	2·7	2·4	51	3·8	3·4	71	2·3	—
12	3·0	2·6	32	1·4	1·7	52	Colostrum		72	1·7	3·5
13	2·3	2·0	33	1·7	1·6	53	1·8	2·1	73	3·0	3·0
14	2·8	2·0	34	—	4·0	54	—	2·4	74	2·6	2·5
15	2·8	3·8	35	—	2·3	55	2·1	2·3	75	2·3	2·4
16	2·7	2·7	36	1·9	—	56	2·8	2·8	76	2·5	2·5
17	1·6	1·7	37	2·4	2·6	57	3·5	3·4	77	1·7	1·9
18	—	2·0	38	2·2	3·0	58	3·1	3·3	78	2·1	2·9
19	1·5	2·1	39	2·5	2·8	59	3·3	2·8	79	3·0	2·8
20	2·3	2·6	40	—	1·9	60	2·7	2·8	80	2·4	2·7

* Cow No. 3 could not be dried off though due to calve again.

** Blank means sample missed or spoiled.

PERSISTENCE OF BOTULINUS TOXIN IN CARCASE MATERIAL WITH SPECIAL REFERENCES TO THAT OF TORTOISES.

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Little experiment work appears to have been carried out to determine the persistence of botulinus toxin in carcase material, and few references in this connection could be traced in the literature. Theiler, A., Viljoen, P. R., Green, H.H., du Toit, P. J., Meier, H. and Robinson, E. M. (1927) stated that carcase material may develop high toxicity in few days, retain it for long periods, but finally lose it through 'weathering and the ultimate aerobic changes". According to their records the oldest material used which still killed cattle was bone material from a carcase which had been exposed in the open for 34 days. These workers also found that the toxin was easily destroyed at temperatures over 70 C. but exposure to low temperatures, sunlight and desiccation had no effect on it. Material kept under sterile conditions for two months showed no appreciable diminution in toxicity, but after six months there was a decline.

Although Theiler and others considered the cadavers of small wild animals such as meercats, hares, squirrels, small game birds and *even lizards and tortoises as minor sources of danger,* experience in the field leads one to believe that the carcases of these animals are of great importance in the epidemiology of lamsiekte, and are to-day the greatest source of infective material. In some areas, e.g. the Kaap Plateau in Bechuanaland, tortoises seem to be responsible for almost 80 per cent. of the cases of lamsiekte. Theoretically the carcase of a tortoise by virtue of its enclosing horny and bony shell would appear to constitute the best natural medium for the strictly anaerobic *Clostridium botulinus,* and would therefore favour the development of a high degree of toxicity. The porous bony shell is bound to absorb a large amount of toxin and at the same time serves as a protection against loss of toxin through leaching. Factors which also appear to favour the preservation of the toxin are the relatively strong protective shell which delays disintegration, small size and hollow inside, which ensures quick drying.

In the opinion of the writer, many cases of mortality in cattle in the lamsiekte area, ascribed by farmers as due to geilsiekte from wilted grass on burnt veld are actually cases of acute lamsiekte caused by carcase material of small animals, especially tortoises, which succumbed in the fire.

Investigations at the Veterinary Research Laboratory, Vryburg, into the duration of toxicity of carcase material in general and with special attention to that of small animals yielded information which is recorded below.

Method of Testing For Toxicity.

Fifty grams of the material to be tested was ground or minced and extracted for about an hour with 100 c.c. normal saline solution. Guinea pigs under general ether anaesthesia were drenched through a stomach tube with 10 and 5 c.c. of the extract. Controls were anaesthetised and drenched with the same volume of water or saline. For this purpose ether was found to be the most satisfactory anaesthetic and losses through drenching into the lungs were negligible. The results of these trials are given in the following table :

Toxicity Tests on Carcase Material.

Type of Carcase Material.	*Conditions under which stored.*	*Period after which still toxic: days.*	*Remarks.*
Core of bovine horn (in shell)	In open	365	No subsequent test
Iguana	In open	90	No subsequent test
Meercat	In open	138	No subsequent test
Hare	In open	54	No subsequent test
Springhare	In open	44	No subsequent test
Tortoise	Dried in open and stored in laboratory	198	No subsequent test
Tortoise	Dried in open, ground and stored in laboratory	605	0·1 gm. material lethal to a guinea-pig at last test. No subsequent test made
Tortoise	In open, protected only from rain	352	No subsequent test made
Tortoise (17·02in. rain) Nov. '44 to Nov. '45	In open	365	No subsequent test made
Tortoise (3·19in. rain) April '45 to Oct. '45	In open	224	Negative after 252 and 294 days

Discussion :

From the above experiments it is evident that the cadavers of small animals may retain toxicity for long periods even when exposed to conditions in the open. Under such circumstances tortoise material

has been found to retain toxicity from 224 to 351 days. Under laboratory conditions dried toxic carcase material, will retain its toxicity for very long periods and tortoise material kept in a box in the laboratory was still highly toxic after nearly a year and 8 months. In the experiments with tortoise material it was found that toxin was developed in almost all carcases. After being kept for six months in the open, but protected from rain, half of the remains of one small tortoise caused fatal lamsiekte two days after being dosed to a year old bullock. Many cadavers of hares, meercats, springhares and other small animals were tested, and most of them appear to lose their toxicity after 4 to 6 weeks. Although leaching is probably the biggest single factor responsible for the loss of toxin in the open, even after heavy rainfall over several months, much toxin may be retained in tortoise material. A probable explanation for this may be the fact that the horny shell often retains a large amount of fat which would tend to prevent leaching.

ACKNOWLEDGMENT.

Thanks are due to Mr. E. A. Deacon, Technical Assistant at Armoedsvlakte, who carried out most of the drenching of the guinea-pigs.

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AN OUTBREAK OF TENDO-VAGINITIS AND BURSITIS DUE TO SALMONELLA ABORTUS-EQUI.

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Pretoria. Durban.

The outbreak described occurred among a number of horses and mules in a remount depot at Pinetown, Natal, during 1945. The animals were kept under the supervision of the South African Veterinary Corps in an area of approximately 200 acres in extent. They were purchased in South Africa by the Indian Army Remount Purchasing Commission (S.A.) and were destined for operational duties in the Far East. The animals were obtained from different parts of South Africa and were detained at Pinetown while being prepared and conditioned for the sea voyage to India. The clinical observations were carried out by one of us (B.M.M.) with the permission of the Deputy Director of Veterinary and Remount Services.

Consignments of equines were continually arriving and departing, and the average number of animals in the depot varied from 1,000 to 1,800, each animal being detained for aproximately three months before embarkation. The horses were mostly of the Basuto pony type, of about 14 hands; the mules were of a less uniform type.

During the period July – November, 1944, an outbreak of infectious equine abortion occurred amongst the horse and donkey mares, resulting in about 60 abortions. *Salmonella abortus-equi* was isolated from most of the foetal organs and after-births examined; and the sera of mares, whose foetuses were infected with *S. abortus-equi,* collected approximately 10 days after abortion, gave a positive agglutination reaction with this organism; sera obtained prior to or immediately after abortion were negative, but when it was not possible to recover *S. abortus-equi* from the foetal organs the serum of the mare was negative (Henning, 1945).

During 1944 only three horse foals and one donkey foal were born alive. Of the horse foals two developed and eventually succumbed to joint-ill in which *S. abortus-equi* was implicated. In addition to a purulent arthritis there were a number of abscesses in the lungs of both foals. *S. abortus-equi* was isolated from the pus obtained from both the lungs and the joints. Henning (1945) produced joint-ill in a foal whose mother was injected during its pregnancy with a bacteria-free filtrate of foetal organs submitted from the Pinetown outbreak in 1944; *S. abortus-equi* was also isolated from the pus of the joints.

No cases of purulent arthritis, bursitis, or tendo-vaginitis were recorded in adult equines during 1944, but from April 10th to September 30th, 1945, when 593 horses, 2,979 mules and 25 donkeys were handled, 27 cases, involving 12 horses and 15 mules, were diagnosed; of these, 17 were considered incurable and were destroyed and 7 were discharged as cured. (See Table I.) In addition, a few cases occurred in animals transferred from Pinetown to another remount depot at Durban, and some of the equines developed the disease during the voyage to India; there were also a few cases in which a diagnosis could not be made with certainty. The morbidity was therefore at least 0·75 per cent.

Susceptibility. The disease affected animals of all ages and sexes and breeds alike, but animals that were underfed or recovering from a debilitating disease were found to be highly susceptible to infection. In the Pinetown outbreak a number of animals that developed the disease were suffering from strangles. There are apparently other predisposing factors which also play an important part in the pathogenesis of the disease — one of the most important of these being the prevalence of infectious equine abortion on the premises. According to Henning (1945) *S. abortus-equi* apparently acts as a synergist with a filterable infective agent in infectious equine abortion. When this latter disease makes its appearance there are always a variable number of young or adult equines suffering from a condition associated with suppurative changes of the joints, tendon sheaths or internal organs.

Symptoms and Lesions. There is swelling of the joints, tendon sheaths or bursae of one or more limbs, resulting in a more or less severe lameness of the affected extremity. The swelling is usually hot and tender, and the slightest movement or handling of the affected part may provoke the most intense pain. When the lungs are involved the respirations are hurried and there may be a purulent nasal discharge. At the same time there may be abscessation in other parts of the body, like the gluteal regions, the flanks and the limbs. The nutrition of the animal generally suffers and its condition is lost fairly rapidly. The affected joint capsule, tendon sheath or bursa is distended with a turbid, blood-stained, flocculent or purulent fluid, usually rich in *S. abortus-equi.* Sometimes there is a variable amount of jelly-like pus resembling semi-coagulated egg albumen. There may be an arthritis, a poly-arthritis, tendo-vaginitis or bursitis depending on the part affected. This is frequently associated with erosion of the synovial membrane, articular cartilage, adjacent tendon or muscle; and even the underlying bone may be affected, giving rise to a periostitis.

In some cases there is a localised necrotic broncho-pneumonia associated with a number of lung abscesses.

Of the 27 cases studied seven were affected with arthritis, eleven with bursitis, eight with tendo-vaginitis and two with lung abscesses

in addition to suppurative changes elsewhere. Ten of these animals recovered or apparently recovered, and seventeen were destroyed. The symptoms manifested by some of the equines were so mild that had it not been for the agglutination test the presence of the disease would probably not have been recognised.

Treatment. Surgical procedure was adopted, but not with any great success. Rest, hot water irrigation and the application of concentrated magnesium sulphate packs appeared to help. Sulphonamide therapy was not of any avail.

Repeated inoculations of a saline suspension of *S. abortus-equi,* killed by the addition of 0·1 per cent. formalin, were also tried. Although the vaccine was used only on a few cases (seven) there was definite evidence that the animals derived some benefit. The prophylactic use of the vaccine is, however, recommended whenever there is an indication of *S. abortus-equi* infection.

Results of Agglutination Tests and Bacteriological Examination of Pus.

An "O" agglutination in a dilution of more than 1 in 80 is regarded as positive, and not much significance is attached to an "H" agglutination unless the titre is very high (Henning, 1945). The sera from 15 of the affected equines were examined and all gave positiev reactions; the pus from three of these animals was examined bacteriologically and all three yielded *S. abortus-equi.* The pus from six other clinically affected animals was also examined, but *S. abortus-equi* was isolated from only two, the other four samples being apparently sterile at the time of the examination. The sera of eight of the nine control animals selected at random from the apparently healthy equines in the depot gave negative agglutination reactions; the serum of one of the controls was positive. It is suggested that this animal had recently recovered from a mild or unnoticed infection.

Summary.

1. An outbreak of arthritis, bursitis and tendo-vaginitis in remount horses and mules has been described. *Salmonella abortus-equi* has been implicated both bacteriologically and serologically.

2. This outbreak was preceded by an outbreak of infectious equine abortion in which *S. abortus-equi* was partly implicated.

3. Although only 27 equines out of a total of 3,597 were affected the disease was nevertheless serious, and a number of animals had to be destroyed.

4. An "O" agglutination appears to be a reliable method of detecting the disease, but the bacteriological examination of the pus is also of value.

5. The use of a saline emulsion of the organism killed by the addition of 0·1 per cent. formalin is advised both prophylactically and curatively.

TABLE 1.

Agglutination reactions, results of the bacteriological examination of pus, the main lesions presented by the various animals and the terminations of the disease.

Type of Animal.	*No. of Animal.*	*Date of Bleeding.*	*Agglutination reaction with abortus-equi*		*Result of Bacteriological examination of pus.*	*Type of lesions presented.*	*Termination.*
			O	*H*			
HORSE	F.37	24.7.45	320	160	Positive	Arthritis and bursitis	Destroyed
HORSE	T.2522	,,	160	40	Positive	,,	,,
MULE	1584	,,	640	640	Positive	tendo-vaginitis	,,
HORSE	F.21	,,	640	640	—	bursitis	inoculated and recov.*
HORSE	F.110	,,	640	160	—	,,	,,
HORSE	F.1	,,	640	10	—	Arthritis	,,
MULE	V.5528	,,	1280	60	—	bursitis	,,
MULE	V.6609	,,	640	80	—	tendo-vaginitis	,,
HORSE	400	,,	160	10	—	Arthritis	apparently recovered*
MULE	6658	,,	640	80	—	bursitis	recovered*
MULE	V.6302	17.8.45	160	320	—	tendo-vaginitis	inoculated and recov.*
MULE	V.5588	,,	1280	640	—	bursitis	,,
MULE	1974	,,	2560	40	—	,,	,,
HORSE	F.4	,,	160	20	—	Arthritis	destroyed
MULE	V.5859	,,	2560	2560	—	,,	,,
HORSE	F.95	—	—	—	Negative	abscesses in lungs and forearm	,,
HORSE	F.104	—	—	—	Positive	Arthritis and purulent pneumonia	,,
MULE	1737	—	—	—	Negative	tendo-vaginitis	,,
MULE	V.4399	—	—	—	Negative	Arthritis and tendo-vaginitis	,,
MULE	V.6468	—	—	—	Negative	tendo-vaginitis	,,
MULE	V.6645	—	—	—	Positive	,,	,,
MULE	1726	—	—	—	—	,,	,,
HORSE	376	—	—	—	—	Arthritis	,,
HORSE	T.2613	—	—	—	—	,,	,,
MULE	1113	—	—	—	—	tendo-vaginitis	,,
HORSE	T.2597	—	—	—	—	Arthritis	,,
MULE	V.4592	—	—	—	—	bursitis	,,
HORSE	T.2465	10.9.45	0	160	—	Control	—
HORSE	T.2602	,,	40	160	—	,,	—
HORSE	T.2491	,,	160	320	—	,,	—
MULE	V.7044	,,	0	80	—	,,	—
MULE	V.7355	,,	10	40	—	,,	—
MULE	V.8828	,,	0	10	—	,,	—
MULE	V.7395	,,	0	40	—	,,	—
MULE	V.6873	,,	0	20	—	,,	—
MULE	V.4842	,,	0	80	—	,,	—

* Recovery was either complete or apparently complete.

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STUDIES ON THE AETIOLOGY OF EAST COAST FEVER.

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INTRODUCTION.

In a recent publication (Neitz, 1943) attention was drawn to several features of East Coast fever, which seem to indicate that the disease cannot be considered purely as a protozoal disease. The discussion was chiefly based upon the results of the immunization process, conducted on a very large number of cattle under laboratory and field conditions by Theiler, Kirkpatrick, Goodall and Chambers (1912) and Wölfel (1912), and the studies on the nature of the immunity in this disease by Du Toit (1928). The theory was advanced by the writer that East Coast fever is in fact a combined infection of at least two factors:—

(a) a protozoal parasite *Theileria parva* and

(b) probably a "virus" and that the sterile immunity in this disease is due to the latter.

Such a combination of infectious agents appears to be possible if one considers the fact that a number of diseases have been described, where two infectious agents are essential for the production of a disease entity. The symbiotic action of vira with bacteria has been established in hog cholera and swine influenza. In the former disease there is a joint interrelated activity of two infectious agents, swine fever virus and *Bacillus suipestifer,* and in the latter, that of swine influenza virus and *Haemophilus suis.* Each of the mentioned infectious agents produces a specific disease, but the characteristic syndrome of either hog cholera or swine influenza develops only from the complex symbiosis of the virus and bacterium. Symbiotic relationships are not restricted to combinations of a virus and a bacterium. In several diseases of plants it has been established that there is a joint interrelated activity of two vira, and that the infection by both is essential for the production of the disease entity.

The recognition of such a biological manifestation was only made possible once (1) the respective rôles of each of the vira responsible for the disease entity of plants, and of the virus and the bacillus in animal diseases was established, and (2) after the biological and the epizootological characters of the infectious agents were carefully considered. The object of this article is to look for an explanation for the sterile immunity that develops in cattle which have recovered from

the two protozoal diseases, *Theileria parva* and *Theileria dispar* infections. The short account of the symbiotic action of two infectious agents suggests that a symbiotic relationship may also exist between *Theileria* spp. and vira. Relapses of malaria in man during and after an attack of virus diseases, i.e. dengue, pappataci or yellow fever are an additional indication that such a relationship is not impossible.

In order to understand the problems which confront us to-day in the study of the theileriases, it is essential to review briefly a number of biological phenomena exhibited by the Protozoa. Before discussing these manifestations, a brief account of the results of the immunization process recorded by Theiler (1912) will be given. Generally speaking, the results obtained at the laboratory were similar to those in the field. Since better facilities for detailed investigations were available at the laboratory, only these records will be considered in this review. In addition the attempts of the artificial transmission of East Coast fever, with either blood or emulsions prepared from partially engorged ticks by Theiler and Du Toit (1928 and 1929) will also be mentioned.

The analysis of the results of the immunization process, which was conducted on 286 susceptible cattle, is detailed in Table 1 and presented schematically on Chart 1. The results show that the course and the nature of the reactions following an artificial infection with emulsified spleen and gland emulsions was markedly different from that observed in cattle which acquired the disease naturally. The different types of reactions in the artificially infected cattle may be briefly summarized as follows:—

1. Typical reactions with a fatal termination were observed in 24·3 per cent. of the cattle. (*Th. parva* in the erythrocytes and Koch's bodies could be demonstrated.)
2. Typical reactions followed by recovery were noticed in 22·3 per cent. of the animals. (*Th. parva* in the erythrocytes and Koch's bodies could be demonstrated.)
3. Mild reactions developed in 13 per cent of the cattle which recovered. (*Th. parva* in the erythrocytes and Koch's bodies could not be demonstrated.)
4. Irregular reactions from which the animals recovered occurred in 19·2 per cent. of cattle. (*Th. parva* in the erythrocytes and Koch's bodies could not be demonstrated.)
5. In 21 per cent. of the cattle no reactions were observed at all.

The animals which survived the artificial infection, were all subjected to an immunity test. This was done by infecting the animals with known infected ticks. In many instances the immunity test was applied for a second time and in a large number of cases the animals were exposed to a natural infection as well, for periods up to three years. In a fair number of animals the exposure to a natural infection only was used for testing the immunity. The method adopted for the

TABLE 1.

The Result of the Immunization Process and the Immunity Test

NATURE OF THE REACTIONS FOLLOWING THE ARTIFICIAL INFECTION.	THE RESULT OF THE ARTIFICIAL INFECTION.	NATURE OF THE REACTIONS FOLLOWING THE IMMUNITY TESTS.				
		R ✠	R.P.R.	R.R.	I.R.	N.R.
Reacted and died = R ✠ .... Protozoal parasite present.	70=24·5%	—	—	—	—	—
Reacted and recovered = R.P.R. ... . Protozoal parasite present.	64=22·3%	4= 6·2%	12=18·7%	7=11·0%	8=12·5%	33=51·6%
Mild reaction and recovered = R.R. ... Protozoal parasite could not be demonstrated.	37=13·0%	5=13·6%	4=10·8%	5=13·6%	6=16·0%	17=46·0%
Irregular reaction and recovered = I.R. .... Protozoal parasite could not be demonstrated.	55=19·2%	20=36·4%	5= 9·1%	6=10·9%	3= 5·4%	21=38·2%
No reaction = N.R.	60=21·0%	32=53·3%	5= 8·3%	4= 6·7%	2= 3·3%	17=28·4%
TOTAL	286=100%	61=21·2%	26= 8·4%	22= 7·8%	19= 6·6%	88=30·5%

Protozoal parasite = Koch's bodies and endoglobular parasites of *Theileria parva*.

presentation of the data does not make it necessary to deal with each group separately. Generally speaking, it can be stated that the pattern of the different types of reactions in each group, following the immunity tests, had some resemblance to that exhibited by the susceptible cattle after the artificial infection. Deaths occurred in all the groups irrespective of whether typical, atypical or no reactions were noticed previously. What is even more striking is the fact that as many as 46·7 per cent. of the cattle which had not reacted at all to the artificial infection were found to be either partially or even solidly immune when the immunity test was applied.

The marked difference between the reactions following a natural and an artificial infection was also observed in the experiments of Theiler and Du Toit (1928 and 1929), when they attempted to transmit East Coast fever with partially engorged emulsified ticks or infective blood.

The observations made on the transmission experiments with emulsions prepared from partially engorged infected *Rhipicephalus appendiculatus* nymphæ and adults, which had been allowed to feed for periods varying from 72 to 240 hours, are mentioned below. The emulsions were administered intravenously. The results may be summarized as follows:—

A. The nine controls infested with infected ticks all contracted East Coast fever and died.

B. The 25 animals which were injected with tick emulsions responded in the following way:—

1. Typical reactions which terminated fatally occurred in six animals. (*Th. parva* in the erythrocytes and Koch's bodies could be demonstrated.)
2. A typical reaction was observed in one animal, which recovered. (*Th. parva* in the erythrocytes and Koch's bodies could be demonstrated.)
3. Mild reactions developed in two animals, which recovered. (In one animal *Th. parva* in the erythrocytes and Koch's bodies could not be demonstrated, and in the other one only small piroplasms of the *Th. mutans* type appeared.) On testing their immunity both were found to be solidly immune.
4. Irregular reactions were not observed.
5. No reactions were observed in 16 animals. On testing the immunity of 12* animals by infesting them with infected ticks, all reacted and 11 died. The immunity of three of the remaining animals was tested by administering infective blood. Typical East Coast fever reactions developed in two cattle, of which one recovered. The third animal developed an indefinite reaction and recovered.

* The ox that recovered had also been injected with infective blood.

The results of the experiments on the attempts to transmit East Coast fever with blood, which was taken at different intervals varying from 6 to 22 days after the commencement of the reaction are mentioned below. In the majority of the recipients the blood was administered subcutaneously and intradermally, whilst in others a combination of several methods was employed. The results may be briefly summarized as follows:—

A. The 10 cattle infested with East Coast fever ticks all reacted and died.

B. The 33 cattle which received infective blood responded in the following way:—

1. Typical reactions with a fatal termination occurred in 6 cattle. (*Th. parva* in the erythrocytes and Koch's bodies could be demonstrated.)
2. Typical reactions followed by recovery were noticed in 4 cattle. (*Th. parva* in the erythrocytes and Koch's bodies could be demonstrated.) On testing the immunity of 2 of them they were found to be solidly immune.
3. Mild reactions developed in 2 cattle, which recovered. (*Th. parva* in the erythrocytes and Koch's bodies could not be demonstrated.) On testing the immunity of one animal on 7 occasions and that of the other 9 times, they were both found to be solidly immune.
4. Irregular reactions developed in 7 animals which all recovered. (*Th. parva* in the erythrocytes and Koch's bodies could not be demonstrated.) On testing the immunity of two of them, both reacted and died.
5. In 14 animals no reactions were observed at all. On testing the immunity of two with known infected ticks, both reacted and one* recovered. The immunity of 2 other animals was tested by exposing them on the East Coast fever infected farm Kindergoed. One reacted and died, whilst the other one remained perfectly healthy for a period of three years. The immunity of the remaining 10 cattle was not tested.

The consideration of these investigations shows that although the results of the two series of transmission experiments did not follow the identical pattern exhibited by the cattle subjected to the immunization process, there was nevertheless some similarity. Recoveries following typical reactions were observed in 5 animals. A solid immunity developed in 4 cattle in which the East Coast fever parasites could not be demonstrated. A partial and a complete immunity was observed in 2 animals, which had not shown any reaction after the artificial infection. The 19 control animals infested with infected ticks all died.

* The ox had also been injected with an emulsion of partially engorged ticks.

Some Biological and Morphological Characteristics of Protozoa.

Before approaching the main problem in connection with the possible association of vira with some species of the Theileridae, it is advisable to provide a short resumé of the current views on the symbionts and parasites harboured by protozoa, and also the influence of the environment as well as that of stimuli on the protozoa. These phenomena will be discussed under separate headings.

1. *The symbiosis of heterotrophic Protozoa with autotrophic Micro-organisms.*

An excellent review of such a symbiosis is given by Doflein and Reichenow (1929). From the observations made so far it would appear that this association is advantageous for both parties. The metabolism of the host and symbiont in these circumstances is the same as in mixotrophic organisms. Numerous Protozoa belonging to the Rhizopoda and Ciliata are known to harbour symbionts, generically known as Zoochorella and Zooxanthella in their protoplasm. For these hospitable Protozoa the name *Xeniozoa is proposed. It is suggested that this term should also be applied to Protozoa harbouring vira in the form of symbionts should such a phenomenon be established in future.

Many of the larger Protozoa belonging to the Amoebina, Testacea, Heliozoa and Ciliata, living in fresh water, appear green. Each such individual protozoon includes a number of individuals in its cytoplasm. Such associated Algae are called Zoochlorella. They belong to the Protococcaceae and are capable of leading an independent life. The Xeniozoa which shelter them, with few exceptions, are believed not to be capable of living continuously without these symbionts. When the Xeniozoa are cultivated in the dark the symbionts die. Re-invasion takes place when such Xeniozoa are exposed to the Zoochlorella.

Symbiosis has also been observed in a large number of marine Protozoa. They contain yellow or brown chromatophores, generically known as Zooxanthella. The Xeniozoa in this case belong to the Radiolaria, Foraminifera and Ciliata. The Zooxanthella, on the other hand, are chiefly members of the Cryptomonadina, but it has also been observed that several species of the Dinoflagellata, Chrysomonadina and Phytomonadina belong to this group of symbionts. At present it is not quite clear whether all the Xeniozoa and Zooxanthella can lead an independent life for any length of time. Some of the Xeniozoa appear to be facultative hosts of the Zooxanthella. It has also been observed that the developmental forms (gametes of the Foraminifera and free forms of the Radiolaria), do not harbour any symptoms, but that the invasion of Zooxanthella takes place when they reach maturity.

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*Xeniozoa: Greek xenios = hospitable, and zoon = animal.

Before this occurs the Cryptomonadina and other members of the Mastigophora lead an independent life, which is apparently of short duration.

The number of intracellular symbionts — Chlorella and Xanthella — in an individual protozoon varies a great deal. The number present, naturally depends chiefly upon the relative size of the host and the symbiont. In some ciliates only 6 to 8 *Spastostyla* sp. (Foraminifera) may be present, whereas in the case of *Peneroplis* sp. (Foraminifera) as many as 100,000 *Chrysidella* sp. (Cryptomonadida) occur. When the Xeniozoa divide the symbionts separate into two groups, which migrate into the daughter cells. In the case of *Actinophaerium* sp. the Zoochlorella usually die when cysts are formed.

The changes that take place in Xeniozoa harbouring symbionts are not restricted to the metabolism only. Morphological changes may occur as well. *Mesodinium rubrum* (Ciliata) possesses a distinct cytostome, when the symbionts are absent. When an invasion of Zooxanthella occurs a total degeneration of the cytostome is brought about. This morphological change causes a complete interference of the ingestion of solid particles.

2. *The parasites of Protozoa.*

Very frequently Protozoa are parasitized by organisms, which may be either protozoa, fungi, bacteria or micro-organisms of unknown nature. The cytoplasmic inclusions have sometimes been mistaken for developmental stages of the protozoa. The hosts are either free-living protozoa or parasites of vertebrates or invertebrates. Parasitized protozoa may live for a long time and sometimes even after extensive destruction of the nucleus. They usually die unless they succeed in surrounding the parasite with a vacuole in which they are digested.

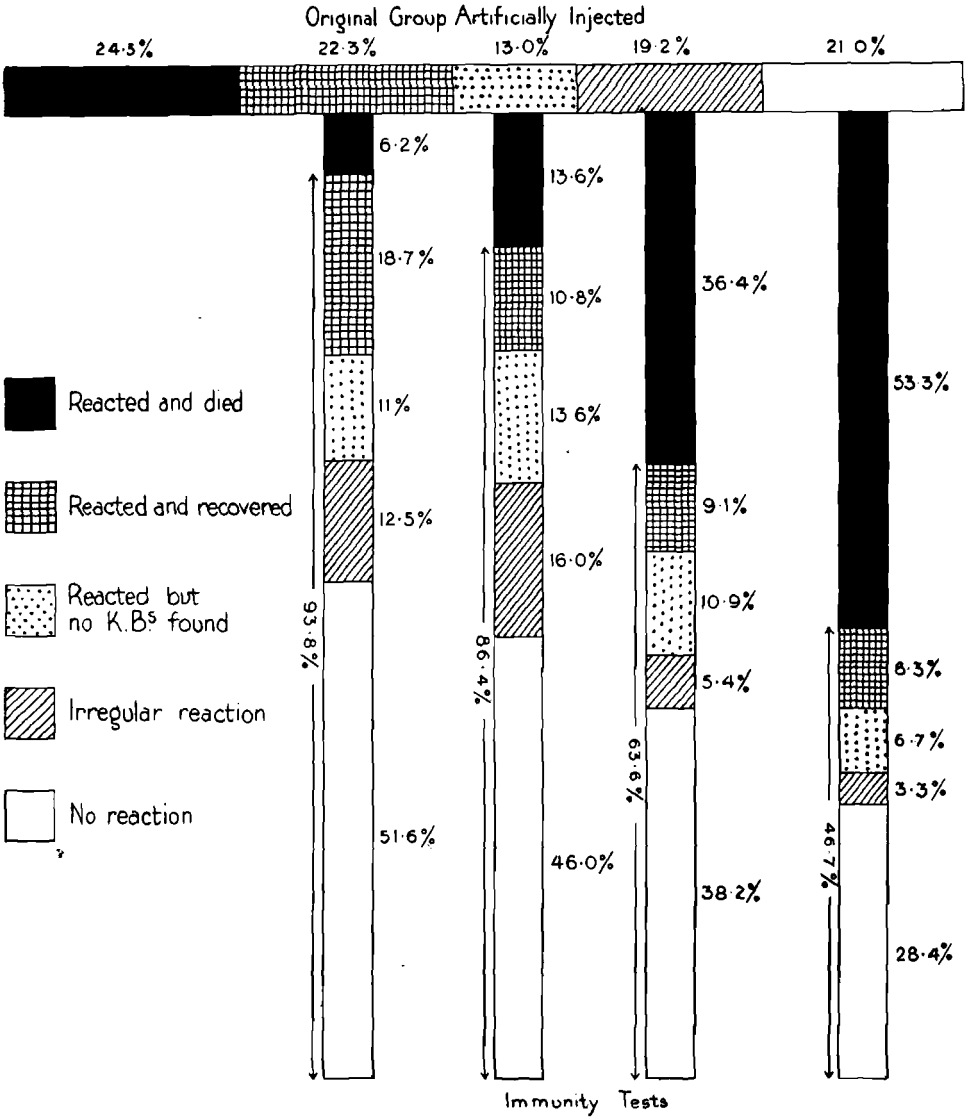
During the process of ingesting solid food several Protozoa ingest micro-organisms, which are in many instances killed and digested. This is, however, not always the case. In *Stentor coeruleus* the parasite *Mastigamoeba* sp. multiply rapidly and eventually fill the cytoplasm of the host. Before death, the host is still capable of moving about actively, despite the severe parasitism.

Parasitism has also been observed in ciliates, which occur chiefly in the colon of horses. The suctorian *Allantosoma intestinalis*, which occurs in the same locality is parasitic on these ciliates. The suctorian *Sphaerophrya* sp. is a parasite of the free-living ciliates, *Paramaecium*, *Stentor*, and *Stylonichia*. Several *Nosema* spp. are known to parasitize members of the Myxosporidia, Gregarinidae and Ciliata, which in their turn are parasites of fish, crabs, etc.

Fungi belonging to the Chytridiacea parasitize several members of the Amoebae, Testacea, Heliozoa, and Flagellata, particularly the Euglenoidea. The fungi are usually present in the cytoplasm

CHART I.

Transmission of East Coast Fever with emulsions prepared from the spleen and gland



(*Sphaerita* sp.) and in some instances in the nucleus (*Nucleophaga* sp.). These fungi do not only parasitize free-living protozoa, but have also been encountered in pathogenic and non-pathogenic intestinal protozoa of man, i.e. *Entamoeba histolytica, Entamoeba coli, Endolimax nana, Jodamoeba bütschlii* and *Trichomonas* sp.

Bacteria which may be present in large colonies, and which eventually destroy the macro- and micro-nucleus of the host, have been demonstrated in the Ciliata, especially in *Paramaecium, Stentor* and *Stylonichia.*

A peculiar organism *Drepanospira mülleri,* Petschenko 1911, has been observed on several occasions in *Paramaecium.* This parasite destroys the macro- and micro-nucleus of the host. It resembles the bacteria in some respects, but its true nature has not yet been established.

The consideration of the various types of parasites of the protozoon hosts, naturally raises the question whether they are restricted to the mentioned groups of micro-organisms only. Such a possibility must be considered, because a variety of bacteriophages are known to occur in nature. Protozoophages have as yet not been isolated, but the possible existence of such vira must not be lost sight of. Furthermore, investigations may show that protozoa, which can be easily cultivated, may serve as an excellent medium for the growth of some types of vira.

3. *The environmental influences.*

The environment in which protozoa find themselves has a decided influence on their morphology and their development. In the case of malaria only the asexual and the early stage of the sexual phase are encountered in the vertebrate host. In the vector, on the other hand, the vegetative forms die, whereas the gametocytes continue their development. Different developmental stages of *Trypanosoma* have been described in the *Glossina* spp. whilst in the vertebrate host only the trypanosome form is present. *Leishmania donovani* undergoes such morphological changes when cultivated on artificial media, that the forms have no resemblance whatsoever to the parasites in the vertebrate host. Over-nourishment has also a decided influence on the morphology. *Trichomonas vaginalis* is frequently much larger than *Trichomonas hominis* of the intestine. If, however, both these organisms are cultivated on the same artificial media, it is noticed that the forms soon become indistinguishable from each other.

Only a few examples have been quoted in order to show that the modification in the developmental phase in the vertebrate host, is not restricted to the *Theileria* spp.

4. *Reaction to stimuli.*

Practically all Protozoa react to stimuli, whether mechanical, chemical, thermal, electric or photic. The response to stimuli has been observed in both the parasitic and free-living protozoa. The influence of chemicals is very interesting. Some compounds have been synthesized which have a specific action on some pathogenic protozoa, and a provocative influence on others. Trypan blue which has a specific action on the *Piroplasma* spp., may be responsible for relapses

of malaria in monkeys and anaplasmosis in cattle. The temporary provocative influence of some of the antimony compounds, which precedes their specific action on *Trypanosoma congolense* infection of cattle and *Leishmania donovani* infection of man has been described by several workers. During the course of the chemotherapeutic studies on East Coast fever, the author found that uleron sodium had a provocative influence on *Th. parva*. Practically every erythrocyte harboured parasites, varying from 1 to 12 in number. Immature and mature schizonts were extremely frequent. The lymphocytic hyperplasia in the kidneys was more pronounced than in untreated cases of East Coast fever.

The influence of inter-current infections on protozoa is perhaps the most interesting. During the course of the investigations on *Rickettsia canis* infection of dogs, Neitz and Thomas (1938) found that this organism was responsible for the relapse of piroplasmosis. The interrelated activity of these pathogenic organisms must be taken into account in areas where the mentioned diseases occur enzootically in order to prescribe the correct treament. Relapses of malaria which are brought about by intercurrent infections of virus diseases have been mentioned earlier in the article. In the case of theileriasis Lestoquard (1929) demonstdated that the infection of several sheep and a goat harbouring a latent infection of *Theileria ovis* (=*Gonderia ovis*), with cultures of Paratyphoid B, resulted in severe reactions. Numerous erythrocytic parasites in the blood and a small number of Koch's bodies in the lymphatic glands could be demonstrated. A similar observation was made by Du Toit (1930) in a calf artificially infected with *Theileria mutans* and *Anaplasma marginale*. He states that Koch's bodies were produced under the stress of the *A. marginale* reaction.

The Biological and Morphological Characteristics of the *Theileria* Spp. of Cattle.

The morphological comparison of the *Theileria* spp. has given very little aid in the classification of these micro-organisms. The classification by Du Toit (1918) is based chiefly upon the biological characteristics. In this connection the type of reactions following the natural transmission, the nature of the immunity, the available results of the cross-immunity tests and the various vectors responsible for the transmission were considered. Du Toit (1930) emphasises that it is questionable whether the four species of *Theileria* of cattle are distinct species. In his review he clearly shows that there is a gradual transition from the most virulent species *Th. parva* to the far less virulent species *Th. mutans*. Furthermore, he stresses that no good purposes would be served by changing this classification and recommends that at present it is far better to accept provisionally the four species named. The consideration of (1) the biological characteristics, (2) the possible

reason for the sterile immunity after the recovery from *Th. parva* and *Th. dispar* infections and (3) the modification of the developmental cycle of the parasites brought about by the serial passage in the vertebrate host make it evident that the recommendation by Du Toit (1930) was sound.

The marked difference between the nature of the reactions following the natural and the artificial infections in the four *Theileria* infections, may be conveniently summarized in tabular form as presented in Table II.

The consideration of the various manifestations recorded in Table II shows that there are several differences, which are not only of general interest, but which may lead to the solution of the complex problem associated with the aetiology of the diseases produced by the *Theileria* spp. With the possible exception of *Th. annulata* infection, it is evident that there is a marked difference between the reactions produced by a natural and those following an artificial infection. The biological transmission is always followed by typical reactions. The artificial mode of transmission, on the other hand, produces (1) typical, atypical or no reactions at all in the case of East Coast fever, (2) typical but a far less fatal disease in *Th. dispar* infection and (3) an extremely mild reaction in the *Th. mutans* infection.

According to Cordier, Mènager and Delorme (1936) the serial passage of the "Kouba" strain of *Th. dispar* produced a remarkable change in the developmental cycle of this parasite. After several generations only Koch's bodies appeared in the vertebrate host, but no parasites in the erythrocytes. Sergent, Donatien, Parrot and Lestoquard (1932) state that all attempts to infect the vector with the vaccine strain of *Th. dispar* have failed. From this observation they conclude that this immunization process can be safely used, without the danger of creating reservoirs for the infection of the vectors. In the case of the serial passage of *Th. mutans* the very opposite takes place. Only the erythrocytic parasites, but no schizonts are demonstrable in the experimental animals. Since no attempts have yet been made to infect ticks with *Th. mutans* maintained in cattle by serial passage, it cannot be stated whether a similar biological modification as in the case of *Th. dispar* takes place. Barzilai, Vivaldi and Kauders (1924) state that a strain of malaria, which had been passaged in man became gametocyteless. They failed to secure any transmission by laboratory-bred *Anopheles maculipennis,* which were repeatedly fed upon patients.

The different types of reactions which may produce a partial or a complete immunity in East Coast fever have already been discussed. In the case of *Th. dispar* infection a solid and sterile immunity develops in artificially infected cattle in which only Koch's bodies are demonstrable. In *Th. mutans,* on the other hand, no immunity develops

TABLE II.

The Biological Characteristics and Modification in the Development Cycle of the Theileridae.

OBSERVATIONS ON	EAST COAST FEVER *Theileria parva.*	NORTH AFRICAN THEILERIASIS *Theileria dispar.*	THEILERIASIS OF MIDDLE EAST AND FAR EAST ASIA *Theileria annulata.*	PSEUDO-EAST COAST FEVER *Theileria mutans.*
A. Natural transmission.	Typical reactions; Blood parasites and Koch's bodies always demonstrable; disappear completely after recovery; Mortality 90 - 100%, but in enzootic regions less.	Typical reactions; Blood parasites and Koch's bodies always demonstrable; may disappear completely after recovery; Mortality 20 - 80%.	Acute, mild or chronic reactions; Koch's bodies only demonstrable during an acute attack; blood parasites always demonstrable; labile infection after recovery; Mortality 5 - 35%.	Acute or mild reactions; Koch's bodies nearly always demonstrable; Blood parasites sometimes completely absent; labile infection after recovery; Mortality as a rule absent, but may under certain conditions be as high as 20%.
B. Artificial transmission with blood, spleen and gland emulsions.	1. Typical reactions and fatal in ±25%. 2. Typical reactions and recovery in ±22%. 3. Mild reactions and recovery in ±13%. (Blood parasites and Koch's bodies not demonstrable.) 4. Irregular reactions and recovery in ±19%. (Blood parasites and Koch's bodies not demonstrable.) 5. No reactions at all in ± 21%.	Typical reactions; Blood parasites and Koch's bodies always demonstrable; Mortality very much lower than in natural transmission.	The same as in natural transmission.	Very mild reactions: only *Theileria mutans* demonstrable, in the erythrocytes. No mortality.
C. Serial passage with blood.	Only passaged for 3 generations. Generally speaking same type of reactions as in B. Not known whether ticks can infect themselves on artifficially infected cattle.	Passaged for many generations; After a fair number of generations Koch's bodies only demonstrable; *Th. dispar* in erythrocytes absent. Ticks unable to infect themselves on these animals.	Only passaged for a few generations. Generally speaking the same type of reactions as in B. Not known whether ticks can infect themselves on these animals.	Passaged for at least 100 generations; only *Th. mutans* demonstrable in erythrocytes. Not known whether ticks can infect themselves on these animals.

TABLE II — (Continued).

The Biological Characteristics and Modification in the Development Cycle of the Theileridae.

OBSERVATIONS ON	EAST COAST FEVER *Theileria parva.*	NORTH AFRICAN THEILERIASIS *Theileria dispar.*	THEILERIASIS OF MIDDLE EAST AND FAR EAST ASIA *Theileria annulata.*	PSEUDO-EAST COAST FEVER *Theileria mutans.*
D. Nature of immunity that develops in naturally infected cattle.	Solid and sterile; relapses do not occur; exposure to a natural infection may result in a breakdown in a very small number of cattle.	No immunity, but a state of premunition; relapses may occur; exposure to a natural infection may result in a breakdown in a very small number of cattle.	No immunity, but state of premunition; relapses associated with alarming symptoms may occur; exposure to a natural infection may result in a breakdown in a small number of cattle.	No immunity but a state of premunition; relapses but not associated with symptoms or the development of Koch's bodies may occur; exposure to a natural infection may result in a breakdown in a small number of cattle; very often Koch's bodies only are demonstrable.
E. Nature of immunity that develops in artificially infected cattle.	A complete, partial, or no immunity develops (same pattern of reactions as exhibited by artifficially infected cattle as mentioned under B, see Chart I); Koch's bodies and blood parasites only observed in cattle showing typical reactions; in atypical reactions small piroplasms only of unknown nature may appear. (Experimental work conducted on *Th. mutans* premune cattle.)	Solid and sterile; exposure to a natural infection may result in a breakdown in a very small number of cattle; Koch's bodies and blood parasites demonstrable.	The same as mentioned under D.	No immunity, but state of premunition; relapses but not associated with symptoms or the development of Koch's bodies may occur; exposure to a natural infection often results in a breakdown; Koch's bodies and blood parasites demonstrable in most cases.
F. Cross-immunity tests.	Produces no immunity against *Th. dispar*; no definite information about the other species of *Theileria.*	Produces no immunity against the other species of *Theileria,* with the possible exception of *Th. annulata* under certain conditions.	No definite information; may produce an immunity against *Th. dispar* under certain conditions.	Produces no immunity against either species of *Theileria.*
G. Splenectomy.	East Coast fever parasites do not appear.	*Theileria dispar* does appear after a natural infection, but not always after an artificial infection.	Relapses do occur; Koch's bodies and blood parasites demonstrable.	Only *Th. mutans* in erythrocytes appear. In one case recently recovered from a natural infection, Koch's bodies and *Th. mutans* in the erythrocytes could be demonstrated.

to a natural infection after an artificial infection despite the fact that the animal harbours the protozoal parasite. Breakdowns in the immunity have also been observed in cattle, which have contracted the disease naturally, after having been exposed to a natural infection in another locality.

The observations that have been made on the immunity in protozoal diseases are frequently very difficult to interpret. It would be beyond the scope of this review to give a full description of the available information on the immunity in all the protozoal infections. The discussion will, therefore, be confined to the order Haemosporidia to which the family Theileridae belongs. Generally speaking, it can be stated that the recovery from the various diseases produced by the members of the Haemosporidia results in a "*labile infection*" or "*immunitas non sterilisans.*" The best example of this "*immunity phenomenon*" is presented by the various types of malaria. In these diseases one notices that the demonstrable stages of the life cycle — schizonts, merozoites and gametocytes — in the vertebrate host always appear, with few exceptions [Barzilai, Vivaldi and Kauders (1924), and Kopeloff (1930)] irrespective of whether the disease results from a biological or an artificial transmission. The severity of the reactions following either mode of infection is the same. In the theileriases, on the other hand, it is always possible to demonstrate the stages of the life cycle — schizonts, merozoites and blood forms — in naturally infected susceptible cattle, except in some cases of *Th. mutans* infections. Then again the exposure of naturally *Th. mutans* premunized cattle to a severe tick infestation may result in acute and even fatal reactions. The careful examination of blood, gland and spleen smears in several instances only revealed the presence of the schizogonous phase of the life cycle of *Th. mutans.* The artificial infection, however, is subject to a number of variations mentioned in Table II. These records show that schizonts only or blood parasites only may appear, and in the case of East Coast fever the protozoal parasite may be completely absent. Besides these manifestations the nature of the immunity in *Th. parva* or *Th. dispar* recovered animals does not conform with that produced by the other members of the Haemosporidia.

These extraordinary variations naturally give rise to the following questions:—

1. What is the nature of the Koch's bodies in *Th. dispar* and *Th. mutans* infections? Are they true schizonts? If so, why do they fail to produce merozoites under certain conditions?
2. Is the environment in the vertebrate host responsible for the change in the developmental cycle?
3. How can an immunity develop in a protozoal disease in the absence of the protozoal parasite?

4. Can the theory of the symbiotic action of a virus with a protozoon elucidate these complex problems?

Before attempting to answer these questions it will be necessary to consider the biological phenomena exhibited by and the habitat of the free-living and parasitic protozoa. The symbiosis of the various micro-organisms with the Xeniozoa and the parasitism of the protozoal hosts can go on undisturbed in their natural environment. Here the microbial successions and associations are chiefly controlled by the food supply. The habitat of the pathogenic protozoa and vira may either be the vertebrate host or the arthropod vector. In the animal the microbial succession and association under natural conditions is dependent upon the prevalence of infected vectors and the defensive mechanism of the body. A close association of protozoa and vira is as a rule of comparatively short duration. A change in the environment is sooner or later brought about by the virus, which stimulates the production of immune bodies, which undoubtedly interfere with the contact between the protozoon and the virus. It stands to reason that the period of symbiotic activity, should this occur, is limited. It is, however, not known in what way the immune bodies would interfere with the association of a virus and a protozoon, should the latter act as a Xeniozoon of the virus. It is possible that the free contact between the mentioned pathogenic microbe and sub-microbe may be longer under these conditions. This may possibly explain why schizonts appeared in a splenectomized calf, which had recently recovered from a natural infection of *Th. mutans* (c.f. observations in Table II). The serial passage of the infectious agents, living in an environment, where the normal contact has been interfered with may be followed by a change in the life cycle of the protozoon (c.f. remarks on the influence of the environment on *Leishmania donovani,* etc.). These observations may explain why the Koch's bodies in the vaccine strain of *Th. dispar* fail to produce merozoites. The modification in the developmental cycle of the Koch's bodies of *Th. mutans* in animals that recovered from this disease on a previous occasion, may also be ascribed to the change in the environment produced after the primary infection. These arguments, of course, are based on the assumption that the Koch's bodies of these two diseases are true schizonts and not inclusion bodies of a virus.

A free association of a protozoon and a virus can occur in the vector due to the absence of immune bodies. In some vectors the stage to stage and in others the generation to generation transmission of protozoa and vira occur. The observations (1) that have been made in connection with the symbiosis and the parasitism of free-living protozoa, (2) the fact that several vira are not specific for one host, or one tissue in the host, and (3) the provocative influence of intercurrent infections on *Th. ovis* and *Th. mutans,* suggest that the following manifestations may take place in the intermediary host:—

1. The virus and the protozoon may lead an independent life.
2. The virus may parasitize the protozoon.
3. The association may result in a form of symbiosis, and under certain conditions similar to that presented by the Xeniozoa.

From the accounts of Regendanz and Reichenow (1933) and Shortt (1936) on the life cycle of *Babesia canis* in the arthropod vector, it is evident that the results of a possible association of a protozoon and a virus cannot be determined histologically. In view of this the action of various vira on the body cells and the results of the biological transmissions will be considered before drawing conclusions on the outcome of such an association.

1. One may safely assume that the transmission of a protozoon and a virus, which had led an independent life in one vector, will produce the same clinical symptoms as those resulting from two vectors each of which harboured one infectious agent.

2. Remarks have been made in the literature about the failure of the biological transmission of protozoal diseases by some arthropods, which are known vectors. It is suggested that in some cases the vector might have accidentally harboured a virus that parasitized and destroyed the protozoon which it would have transmitted under normal conditions.

3. Should the protozoa *Theileria spp.* harbour a virus the following manifestations may occur. It is a well-known fact that in some virus diseases the interaction with cells capable of multiplication, is rather slow, and that the outcome of such an association is a hyperplasia. In the case of East Coast fever both the protozoal parasite and the lymphocytes multiply very actively. Before the infected vector feeds, *Th. parva* and the possibly associated virus are dormant. Engorgement is followed by a rapid multiplication of the protozoon (Reichenow, 1937). It is believed that the provocative influence of the virus is such that all the pre-sporozoitic parasites in the salivary gland develop into the true sporozoites, which are not able to maintain themselves in the salivary gland. This results in the cleansing of the tick. From the pathological changes in East Coast fever, it appears that the virus has an affinity for both the protozoal parasites and the lymphocytes in which the schizonts develop. The resultant effect is seen in the nature of the lesions produced and the large number of Koch's bodies and blood parasites in the vertebrate host. It would appear that the ideal symbiotic interaction between the protozoon and the virus is sooner or later disturbed in the host, so much so, that the artificial transmission is followed by various types of reactions described above.

How long the suggested symbiotic relationship is maintained by the *Theileria* spp. in the vertebrate host is difficult to state. In the case of East Coast fever the splenectomy of a heifer and a bull, which

had recovered respectively two and three months previously, was not followed by a relapse to this disease (Du Toit, 1931). Donatien* and Lestoquard (1930) state that *Th. dispar* does not always disappear from the blood after recovery. The artificial infection with the "*virus fixe*" strain results in a sterile immunity. On the other hand, the transmission of the "*virus latent*" is followed by a premunition. In the case of *Th. annulata* it was established by Brumpt (1923, 1924) that in some infected individuals the infection may suddenly become acute. The period which elapsed between infection and appearance of an acute attack, varied from three-and-a-half to seven months. The splenectomy of cattle, irrespective of whether they contracted the *Th. mutans* infection naturally or artificially, is followed by a heavy infection of the endoglobular parasites only. An exception to this rule was observed in a calf, which was splenectomized approximately three months after recovery from a natural infection. The result of this operation was that both schizonts and endoglobular parasites appeared (De Kock, Van Heerden, Du Toit and Neitz, 1937). The consideration of these results suggests that the period of symbiotic relationship varies a great deal, and that in the case of *Th. annulata* the association may be permanent. It would be interesting to ascertain the length of the period that would have to elapse, after a natural infection of *Th. dispar*, before a complete dissociation of this protozoon and the virus is brought about.

Conclusion.

It will be noticed from the discussion that it has been suggested that the interrelated activity of a protozoon and a virus may be responsible for the production of East Coast fever and the related diseases. The sterile immunity resulting from a protozoal infection prompted these studies in order to find a satisfactory explanation for such an extraordinary phenomenon. This naturally involved tracing the infectious agents through the vertebrate host and the arthropod vector. In doing so, the biological phenomena exhibited by various protozoa and the influence of the environment and of stimuli mentioned earlier in the article were taken into account. It was established that the development cycle of the *Theileria* spp. in the vertebrate host depended upon the method employed for the infection. Doubt was expressed whether there is any true cycle of solely protozoal activity.

The production of a pure culture is always the aim of the laboratory microbiologist. In the atempts to do so by artificial transmission, various workers on this group of diseases found that the true life cycle became greatly modified, so much so that in East Coast fever a solid immunity developed in cattle which had not shown any sign of a reaction whatsoever. Although the various arguments brought

* Quoted by Du Toit (1930).

forward are purely theoretical, it is nevertheless believed that the investigations on the symbiotic action of a virus and a protozoon may lead to the solution of the complex problem associated with the actiology of the theileriases. Furthermore, it is suggested that a joint interrelated activity of a virus and a protozoon or other infectious agents may also occur in the various diseases transmitted by arthropod vectors.

Summary.

1. A short resumé of the current views on the symbionts and parasites harboured by protozoa is given.

2. The name Xeniozoa is proposed for the protozoa that harbour the symbionts, Zoochlorella and Zooxanthella.

3. It is suggested that protozoa may harbour vira either as symbionts or as parasites.

4. There are marked differences between the reactions following the natural and the artificial infections in the theileriases.

5. The modification in the life cycle of the *Theileria spp.* with the possible exception of *Theileria annulata* maintained by serial passage in the vertebrate host is attributed to the partial or complete inactivation of the associated vira.

6. It is suggested that probably all *Theileria* spp. live in close association with vira in their normal environment, the arthropod vectors.

7. The milder reactions following the artificial infection is attributed to the changes that take place in the association between the protozoon and virus.

8. Attention is drawn to the work in Algiers, where it has been established that ticks do not infect themselves when allowed to feed on cattle immunized with the vaccine strain of *Theileria dispar.*

9. No attempts have yet been made to ascertain whether a similar biological modification can occur in the other members of the Theileridae.

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KORT AANTEKENING OP DIE GEBRUIK VAN PENICILLIN BY STREPTOKOKKUS-BESMETTING BY 'N HOND.

T. B. NEL,
Vryheid, Natal.

'n Doberman is ingebring met 'n temperatuur van 103, kwyl het in stringe by die bek uitgeloop, die kondisie was in die algemeen swak. Op inspeksie het die bek 'n gingivitis gewys, en agter op die farinks en mangels was swere waaruit bloed gesyfer het. Geen tekens van uremie was in die asem te ruik nie. 'n Smeer van die slym agter in die keel is ondersoek en streptokokkus was oorwegend. Penicillin-behandeling is begin, 100,000 eenhede is intramuskulêr ingespuit as volg: 30,000 eenhede as begin en vier uur later 20,000 eenhede, daarna elke vier uur 10,000 eenhede. Na die derde inspuiting was die temperatuur af na 100·2. Die volgende dag was geen kwyl teenwoordig nie, en die hond het vir die eerste keer na vier dae kon sluk. Hy is 'n verdere drie dae onder obserwasie gehou en toe huis-toe gestuur.

A REMARKABLE FOETAL POSITION IN A COW.

C. F. B. HOFMEYR,
Pretoria.

The author was summoned one night to attend a case of dystokia in a cow. The client stated that he had been away from home for a few days and that he had been informed by the natives on arrival the same evening of the condition of the cow, which had been in labour for approximately three days.

The patient was a multiparous cross-bred Afrikaner cow, about 8 - 9 years of age and in good condition. Four previous pregnancies had terminated normally. During the general examination particular care was taken to ascertain whether there had been any lay interference, but this could definitely be excluded. As it was immediately evident that the foetus was emphysematous, 7 cc. 2% planocain solution was injected intrathecally in anticipation of a prolonged delivery.

Vaginal exploration revealed four feet, which belonged to the same foetus. With further investigation it was discovered that the foetus itself was normal in conformation, but was lying with its head on the abdominal floor and had its sacrum in contact with the maternal sacrum — therefore abdominal presentation, sacro-sacral position.

As the cow remained standing throughout the operation, matters were greatly facilitated. The hindlegs were secured with cords and

the fore limbs repelled. In spite of the bloated, decomposed state of the foetus, delivery was fairly easy as the calf was small and the maternal pelvis roomy. Appropriate treatment was given after the birth, and, contrary to the usual experience in this condition, the cow made an uninterrupted recovery, proving it by bringing a healthy heifer into the world a year afterwards.

This case is recorded as inquiries from colleagues or into the literature failed to reveal a similar experience. No comment can be offered as to the cause of this most unusual foetal position.

OBITUARY.

CECIL MOLESWORTH SHARPE.

Cecil Sharpe passed away suddenly at his home in Pietermaritzburg on 24th December, 1945, and was buried on the morning of Christmas Day.

He graduated at the Royal Veterinary College, London, on 13th July, 1899, and had as college friends with him there Amos and Woollatt.

Mr. Sharpe came to South Africa as a lieutenant during the Boer War in the old Army Veterinary Department. He served until the termination of hostilities, and then resigned his commission to join the Civil Veterinary Department of Natal, working as a District Veterinary Officer until 1907, when he set up in private practice in Pietermaritzburg.

"Pop" Sharpe was a great sportsman from his school and college days, and I can still remember the look of pride on his face when he produced two Middelsex rugby caps with the dates 1896 and and 1897. He smiled when I told him 1897 was the year I was born. He was a good shot, rowed well, played a good game of squash and was one of nature's gentlemen.

He served as a Veterinary Officer through several native rebellions in Natal, and saw service in South West Africa and East Africa during the 1914 - 1918 campaign, ending up as a Lieut.-Colonel of the South African Veterinary Corps (R. of O.).

During this war his Thursday night club will be well remembered by many overseas army and naval officers, whom he loved to entertain.

His health began to fail about two years ago, and if it had not been for his strength he would have died much sooner.

He leaves a wife and two daughters by a previous marriage.

With his passing the veterinary profession has lost a lovable character, who always endeared himself to those who knew him. We are the poorer. Vale.

A.S.C.

SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION.

BALANCE SHEET AS AT 31st MARCH, 1946.

LIABILITIES.			ASSETS.			
Benevolent Fund as at 31st March, 1945 ..	£863 9 0		**Investments** at Purchase Price plus Interest:			
Add: Subscriptions Collected	95 10 0		Union Govt., 3½% Stock, 1952-57	£405 0 0		
Commission on Insurance Premiums	38 10 5		Union Govt., 3¼% Stock, 1954	202 14 2		
Donations	11 11 0		Union Loan Certificates	2066 0 8		
Interest Accrued	34 10 4		United Building Soc. Pref. Shares	101 6 8		
	1043 10 9				£2775 1 6	
Less: Assistance Payments	96 0 0		**Interest** (U.L.C.) Suspense Account		88 19 4	
		947 10 9	**Loans** plus Accrued Interest:			
Prize Fund as at 31st March, 1945	200 0 11		To Member	50 14 10		
Add: Interest Accrued	10 0 3		,, Students (two)	194 18 10		
Profit on Book Fund Transactions	151 6 0		,, Deceased Member's Widow	188 8 6		
	361 7 2				434 2 2	
Less: Awards	20 0 0		**Stock of Books** at Purchase Price		60 15 8	
		341 7 2	**Sundry Debtors** for Journal Advertisements		29 3 0	
Natal Branch as at 31st March, 1945	13 2 0		**Due by Members:**			
Add: Subscriptions Collected	0 5 0		For Books Sold	£193 10 3		
£-for-£ Contribution	0 5 0		**Less:** Paid in Advance	7 4 9		
Donation	1 1 0			186 5 6		
	14 13 0		Membership subs.	181 3 6		
Less: Wreath	1 2 9		**Less:** Subs. paid in Adv.	140 15 0		
		13 10 3		40 8 6		
Sundry Creditors:			Insurance Premiums	0 8 8		
Caxton Printing Works	119 4 9				227 2 8	
Baillière, Tindall & Cox	315 9 9		**Cash** at Bank		271 12 6	
Central News Agency, Ltd.	8 2 3					
Voortrekker Pers, Bep.	1 17 10					
		444 14 7				
Subscriptions Reserve Account		10 10 0				
General Fund as at 31st March, 1945	2106 12 8					
Add: Excess of Income over Expenditure	22 11 5					
		2129 4 1				
		£3886 16 10			£3886 16 10	

SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION.

INCOME AND EXPENDITURE ACCOUNT, 1945 - 46.

1944 - 45.					1945 - 46		1944 - 45				1945 - 46	
£	s.	d.			£	s. d.	£	s.	d.		£	s. d.
4	6	7	To	Wreaths, etc.	3	4 9	293	11	0	By Subscriptions Accrued	316	9 6
2	10	0	,,	Branch Subsidies	3	7 6	71	6	11	,, Interest	76	13 8
				Witwatersrand Branch £3 2 6			57	6	10	,, Excess of Expenditure over Income	–	– –
				Natal Branch 0 5 0								
				3 7 6								
4	14	10	,,	Bank Charges	6	16 10						
26	0	3	,,	Cost of Meetings	23	15 11						
10	14	8	,,	Adjustments	16	4 0						
				Subscription Reserve 10 10 0								
				Other 5 14 0								
				16 4 0								
19	6	6	,,	Stationery	22	16 9						
81	7	4	,,	Miscellaneous Expenses (including Honorarium £15 15/-)	31	14 4						
56	12	0	,,	Clerical Assistance and Typing	51	0 0						
216	12	7	,,	Nett Cost of Printing and Distributing Journal	211	11 8						
				Gross Costs 329 11 9								
				Less: Advertisements, etc. 118 0 1								
				211 11 8								
–	–	–	,,	Excess of Income over Expenditure	22	11 5						
£422	4	9			£393	3 2	£422	4	9		£393	3 2

A PRELIMINARY REPORT ON THE GRADING OF PASTEURISED MILK.

MAJOR E. J. PULLINGER,
S.A. Veterinary Corps.

Efficient pasteurisation of milk depends upon three interdependent factors, namely:—

(a) The efficiency of heat-treatment.
(b) The quality of the incoming raw milk.
(c) The international pasteurising-plant hygiene, and the post-pasteurisation hygiene.

Of these factors the questions of efficient heat-treatment and the grading of raw milk supplies have already been considered in earlier reports — *vide* Pullinger 1945(a) and 1945(b). In any scheme for the grading of pasteurised milk cognisance must be taken of these factors, but in addition the problem of milk-spoilage during and after pasteurisation must also be taken into account. The objects of the present report are:—

1. To ascertain the extent to which the bacterial quality of commercially pasteurised milk is dependent upon the quality of the incoming raw milk. While it is normally accepted that a first-grade product cannot be made from third-grade raw material, yet in the dairy industry the pasteuriser is expected to turn inferior quality raw milk into a satisfactory pasteurised product.
2. To study the Breed count and the methylene blue reductase test as applied to the grading of pasteurised milk.
3. To show the extent to which pasteurised milk can be recontaminated during and after processing, and to study the value of the presumptive coliform test as an index of plant hygiene.
4. To suggest ways and means of grading pasteurised milk.

In the report on the grading of raw milk, to which reference has already been made, stress was laid on the fact that milk varies markedly from day to day in its bacterial quality, and that no grading scheme can with justice be based upon the testing of occasional random samples. In the case of pasteurised milk, heat-treatment is a continuous process which is carried on day and night, and at any time the process may go wrong. Breakdowns of this type may or may not be detectable by automatic recording instruments, and the only certain way of detecting developing faults is by subjecting all milk to laboratory tests. In the case of small plants in which the milk is bottled and distributed

as fast as it is processed, it is extremely difficult to get representative samples and recourse has to be made to collecting random samples from the „dispatch" at frequent intervals. In big plants, on the other hand, the pasteurised milk is cooled and stored in insulated storage tanks of 500 to 3,000 gallons capacity, in which paddles are continually mixing the contents. Samples collected from such tanks are fairly representative of the total contents.

Choice of Tests.

Samples of all pasteurised milk should be tested to check up upon the efficiency of heat-treatment, and for this purpose the phosphatase test is available as a simple and reliable one. The real difficulty in connection with the phosphatase test is to ensure that samples have been collected which really represent the whole output of pasteurised milk. Similarly, simple tests are available for estimating the chemical or nutritive quality of milk. When, however, attention is turned to the question of the bacterial content of pasteurised milk, difficulties with regard to the choice of test are immediately encountered.

Most of the work that has been done upon the grading of pasteurised milk has been based upon the total plate count, and many numerical standards have been established on this basis, but it cannot be claimed that the standard plate count has proved satisfactory for this purpose. Grading tests should serve:—

1. To indicate the density of the bacterial flora that has survived pasteurisation.
2. To ascertain the degree of post-pasteurisation recontamination
   (a) as result of growth during pasteurisation of bacteria which have survived the heating process,
   (b) by recontamination of milk during processing.
3. To ascertain the keeping quality of the milk irrespective of bacterial content.

Quite clearly, no single laboratory test is likely to give information upon all these factors, and it is necessary to consider how each factor may be examined and whether the information obtained from any test justifies the expense and labour involved in such testing. At this point it is desirable to stress once again the fact that a single daily test of a plant is useless and that efficient testing should involve examining:—

(a) A sample from every tank of milk filled in 24 hours. If milk is continuously being fed into and withdrawn from the storage tank, then a sample from the tank should be tested every 30 to 60 minutes, depending upon the rate of flow.

(b) Random samples should be collected from cans, bottles and cartons issued for distribution, to check the hygiene of various washing and filling machines.

(c) Random samples should be collected from all trade returns to check upon mishandling during distribution.

(d) Check samples should be collected wherever necessary to locate and correct faults revealed by the results of routine tests.

THE DENSITY OF THE BACTERIAL FLORA SURVIVING PASTEURISATION.

Spore-bearing and many non-sporulating varieties of bacteria survive pasteurisation. A comprehensive bibliography of this subject has been arranged by Hammer (1938), and no useful purpose would be served by recapitulation. None of the known non-sporulating pathogenic bacteria survive pasteurisation. Bacterial standards for pasteurised milk have in the past been based upon the total plate count carried out in the standard way upon occasional random samples. This procedure, which Wilson *et alia* (1935) do not favour particularly, is open to the objection that only a portion of the organisms to be found in pasteurised milk will grow in standard agar when incubated at about 37° C. Thermophilic bacteria for instance, the presence of which indicates faulty hygiene on the farm or in the depot, are not detected by this test. To overcome this particular shortcoming Anderson and Meanwell (1933) as well as the other workers have advocated running duplicate plate counts incubated at 37°C and at 56° C. The information obtained from such comparative counts is most valuable, but the duplication of this already cumbersome test begins to render the whole technique too expensive and laborious for large-scale routine use.

The only other test of bacterial density is the Breed count. The value of this test in the grading of raw milk is generally accepted and a system for incorporating this test into the grading of raw milk has already been outlined [*vide* Pullinger 1945(b)]. When this test is applied to pasteurised milk, however, its value is less apparent and certain factors have to be considered:—

(a) The Breed count shows up all bacteria that have retained their staining properties, which is advantageous since anærobic and thermophilic bacteria as well as types such as mastitis streptococci are seen. The test does not, however, show whether bacteria are alive or dead. Chains of slightly pleomorphic streptococci are sometimes seen in Breed smears of mastitis milk that has been pasteurised. Laboratory evidence indicates that pasteurisation destroys all pathogenic streptococci, but under practical commercial conditions it seems doubtful whether all bovine strains are destroyed. Pullinger (1935) found that commercial pasteurisation at 145° F for 30 minutes destroyed all hæmolytic types of bovine mastitis streptococci, but facilities were not available for testing for non-hæmolytic strains. Zeller *et alia* (1928), on the other hand, found that all mastitic streptococci

were not destroyed. These varying findings were possibly due to differences in the efficiency of pasteurisation, since the phosphatase test was not available as a control at that time. Even less is known about the effect of modern short-time high-temperature pasteurisation upon the viability of mastitis streptococci. Taking the above factors into consideration it seems desirable to consider the presence of any stainable bacteria in pasteurised milk as being significant, and this course is followed in the Breed test. Apropos of this it should be mentioned that death does not necessarily render bacteria harmless. Unless the heating process is sufficient to render the enzymes inactive, non-viable bacteria may retain some ability to spoil the milk. Similarly bacterial toxins are not destroyed by pasteurisation, so that the presence of innumerable Salmonella bacteria may still be important even though they have been killed by pasteurisation.

(b) In view of the inaccuracies and approximations involved in the Breed technique, and the fact that the bacteria are not necessarily alive, it would be absurd to use this test for drawing delicate comparisons between counts unless tenfold differences are under consideration.

Reviewing what has been written in this section it is obvious that no suitable test is available for judging pasteurised milk on a basis of the density of the bacterial flora. The Breed count is, however, quickly and easily performed and gives immediate results, so that while it could not be used for grading milk against some definite numerical standard it can nevertheless give considerable information on the following points:—

(a) The density of the bacterial flora irrespective of whether the bacteria are dead or alive.

(b) The types of bacteria present and consequently the probable sources of these organisms.

(c) Leakage of pus cells through the filters.

When correlated, this information has a bearing upon the quality of pasteurised milk and so is of value in grading. For this reason the test has been studied in detail and the data that have been collected are presented in the subsequent text.

Testing for Post-pasteurisation Recontamination.

Contamination of milk during processing can occur before, during or after the application of the heating process. Before heating, contamination is not of great significance unless the contaminants are thermophilic bacteria. Such bacteria grow at pasteurisation temperature, and if "holder" pasteurisation is prolonged as result of a breakdown, they may multiply to an enormous extent. Thermophilic

organisms do not necessarily come from the plant and contamination may occur on the farm (*vide* Anderson and Meanwell, 1933). Such organisms are best detected by doing comparative plate counts incubated at 37° and 56°C, but if they are multiplying excessively their presence is immediately suspected from the examination of Breed smears.

The much more serious form of recontamination is that which occurs after the heating process is completed, since this is the stage where contamination with human pathogens is likely to be dangerous. For the purpose of controlling plant management Wilson *et alia* (1935) suggested that the presumptive coliform test might be of value, and Barkworth presented evidence in support of this view. Preliminary work, done in South Africa before Barkworth's report had been seen, supported his views so closely that it was decided to try out the presumptive coliform test on as large a scale as possible. The result of this survey will be recorded.

Testing for Keeping Quality of Pasteurised Milk.

The methylene blue reductase test is the most useful one at present available for testing the keeping quality of raw milk, but some differences of opinion seem to exist as to whether the test should be applied to pasteurised milk. The argument that is used against it is that the test depends upon the interaction of a series of complex oxidation-reduction systems, some of which are destroyed by pasteurisation. The most comprehensive study of these reactions that has been carried out was done by Twigg (Wilson *et alia* 1935), and in the resulting report the general statement was made that the reductase test should be useful for grading pasteurised milk, providing a fairly stringent time-limit for reduction is set. In the absence of any more suitable test for keeping quality it seemed desirable to explore the possibilities of using the standard reductase test for the grading of pasteurised milk.

Results of Survey.

Influence of Raw Milk Quality upon the Pasteurised Product.

In a previous report on the grading of raw milk [Pullinger, 1945(b)] a careful analysis was made of the bacterial quality of farm milk supplies, the quality being judged by the Breed method. These data have again been drawn upon in the present report, and in Table I there are recorded the monthly percentages of farmers who delivered dirty* milk to two large pasteurising depots. Correlated with these figures is the Good : Bad ratio of the final pasteurised product in the depot storage tanks. The Good : Bad method of grading milk was described fully in the above-mentioned report, but stated briefly

* **In this report "clean milk" means milk having a low bacterial count and with high keeping quality. "Dirty milk" is the reverse.**

TABLE I.

Correlation between the Bacterial Quality of Incoming Raw Milk and that of the Final Pasteurised Product.

	1943: Jan.	Feb.	Mar.	Apr.	May	Jun.	Jul.	Aug.	Sep.	Oct.	Nov.	Dec.
Percentage of producers delivering milk having a high bacterial count *	72	60	65	51	18.2	5.7	8.4	8.8	61.2	79	80	80
Depot No. 1. Ratio of Good: Bad tank samples. **	1:2.1	1:1.4	2.2:1	1.4:1	2.1:1	2.4:1	1:1	1:1	1:1.8	1:1	2:1	3.9:1
Depot No. 2. Ratio of Good: Bad tank samples. **	1:3	1:2.2	1.9:1	1:2.1	1:2.4	1:1.7	1:3.5	1:10.5	1:5.9	1:4.9	1:1.4	1:2.1
	1944: Jan.	Feb.	Mar.	Apr.	May	Jun.	Jul.	Aug.	Sep.	Oct.	Nov.	Dec.
Percentage of producers delivering milk having a high bacterial count *	98	79	60	35.5	4.6	2.5	1.5	33 5	32.2	51.5	99	88.5
Depot No. 1. Ratio of Good: Bad tank samples. **	1:2.6	1.4:1	4.7:1	1.8:1	5.5:1	9.4:1	10:1	1:1.3	1:1.3	1:6.2	1:4.1	1:1.2
Depot No. 2. Ratio of Good: Bad tank samples. **	1:3.1	4:1	5.4:1	7:1	6.1:1	6.9:1	9.2:1	1.3:1	4.9:1	1:3.5	1:6	1:1.2
	1945: Jan.	Feb.	Mar.	Apr.	May	Jun.						
Percentage of producers delivering milk having a high bacterial count *	88	88.5	69	47.5	16.2	38						
Depot No. 1. Ratio of Good: Bad tank samples. **	2:1.1	2:1	1.5:1	1.4:1	1.8:1	2.4:1						
Depot No. 2. Ratio of Good: Bad tank samples. **	1.6:1	2.4:1	2.9:1	1.3:1	4:1	7.6:1						

* Producers showing a proportion of two or more Breed counts above 1,000,000 to one count below that level are here classed as having a high bacterial count. No strict division of producers could be maintained, as the milk from these farmers was delivered to both depots.

** Good:Bad ratio for pasteurised milk is taken as the proportion of samples with Breed counts or 300,000 per cc. or under to those showing Breed counts above 300,000 per cc.

a Breed count of 1,000,000 per cc. is taken as an arbitrary line between milk that is predominantly good and that which is predominantly bad (or dirty). In the case of pasteurised milk the dividing line is taken as a count of 300,000. The Good:Bad ratio is the proportion of good counts against bad ones returned during the month. Thus a Good:Bad ratio of 1:4 for pasteurised milk means that of all samples tested during the month only one showed a Breed count of 300,000 or less for every four samples that gave counts above that figure.

In Table I the quality of raw milk has been expressed as the percentage of producers who fell below a certain fixed standard, namely a Good:Bad ratio of 1:2. (This ratio means that in any month a producer who returned only one count under 1,000,000 per cc. to every two counts above that level was classed as delivering satisfactory milk.) Actually this constitutes an absurdly low level, but in spite of that it will be seen from Table I that only in the depths of winter were less than 50% of producers delivering unsatisfactory milk. In hot weather upwards of 100% of producers were unsatisfactory.

The actual correlation between the quality of the incoming raw milk and that of the final pasteurised product is best seen in Graphs Nos. 1 and 2. In these graphs the raw milk quality is expressed as pillars and the pasteurised milk quality as a curve. During 1943, while the raw milk improved in the winter all pasteurised milk remained consistently unsatisfactory, because at this stage outside control was just developing and plant operators know very little about the cleaning and sterilizing of their plant. In the subsequent years there was a close correlation between the quality of the raw and pasteurised products. Naturally, correlations of this type can only be general in nature because at any time good raw milk can be spoiled by bad depot hygiene. The two outstanding features of these graphs are:—

1 Low count pasteurised milk could never be produced when high count raw milk was fed to the plant.
2. Low count raw milk could be seriously spoilt by faulty handling during pasteurisation.

Another way of examining this problem is by comparing the quality of the incoming raw milk with the percentage of samples of pasteurised milk containing coliform organisms. The use of the presumptive coliform test will be discussed more fully later, but at this point it is of value to examine the figures in Table II, in which a comparison is drawn between the quality of the raw milk and the monthly percentage of pasteurised milk samples free from bacilli on 1 cc.

To exclude cleaning failures as far as possible the pasteurised samples were drawn at the "cooler exit" sampling tap. It will be seen that when only a small percentage of farmers were delivering

TABLE II.

Percentage of Pasteurised Milk Samples showing coliforms absent in 1 cc. compared with the percentage of farmers delivering milk with a low bacterial count.

Month and Year	% Producers delivering Low Count Milk *	% of Pasteurised Milk Samples from cooler exit with coliform absent in 1 cc.	
		Depot No. 1.	Depot No. 2.
Jan., 1943	28·0	82·0	50·0
Feb.	40·0	50·0	6·0
Mar.	35·0	43·0	57·0
Apr.	49·0	93·0	100·0
May	81·8	100·0	100·0
Jun.	94·3	100·0	100·0
Jul.	91·6	84·0	100·0
Aug.	91·2	72·0	95·0
Sep.	38·8	95·0	100·0
Oct.	21·0	75·0	78·0
Nov.	20·0	91·0	84·0
Dec.	20·0	100·0	95·0
Jan., 1944	2·0	90·0	84·0
Feb.	21·0	67·0	72·0
Mar.	40·0	96·0	41·0
Apr.	66·5	100·0	28·0
May	95·4	100·0	95·0
Jun.	97·5	88·0	90·0
Jul.	98·5	90·0	94·0
Aug.	66·5	96·0	100·0
Sep.	67·8	95·0	90·0
Oct.	48·5	95·0	58·0
Nov.	1·0	77·0	85·0
Dec.	11·5	63·0	74·0
Jan., 1945	12·0	81·0	81·0
Feb.	11·5	40·0	72·0
Mar.	31·0	48·0	69·0
Apr.	52·5	76·0	80·0
May	83·8	74·0	79·0
Jun.	62·0	95·0	94·0

* **Producers showing a proportion of less than two Breed counts above 1,000,000 to one count below that level.**

low-count milk the percentage of pasteurised samples containing coliform bacilli in 1 cc. was correspondingly high. The full implications of these findings will be discussed later, but the suggestion is obvious that pasteurisation may not succeed in killing all coliform bacteria if these organisms are present in enormous numbers in the original raw milk.

The use of the Breed count and the reductase test in the grading of pasteurised milk.

To study the comparative value of the methylene blue reductase test and the Breed count for the grading of pasteurised milk, parallel tests have been applied to 1,463 samples of milk pasteurised at seven different depots, samples being tested twice weekly for two years. Records are given in Table III of the monthly distribution of all reductase and Breed counts, while in Table IV these results are summarised and expressed as percentages.

Firstly, there is no apparent correlation between the two tests such as may be found in the case of raw milk (*vide* Pullinger, Davidson and Hogg, 1944, Tables III – V). This result was anticipated, because the bacteria chiefly to be seen in pasteurised milk are sporulating ærobes and anærobes, mastitis streptococci thermophilic and thermoduric bacteria, none of which have any marked effect upon the reductase test.

Secondly, the scattered distribution of the Breed counts suggests that the actual count has very little bearing upon the keeping quality of milk and cannot be used as an index of keeping quality. The Breed examination does, however, reveal:—

(a) Excessive growth of thermophilic bacteria. The organisms cannot be recognised morphologically, but the Breed examination may rapidly indicate that a detailed search for thermophiles should be made.

(b) The probable source of bacterial contamination.

(c) The leakage of pus through the filter cloths.

These advantages all combine to make the Breed examination a useful adjunct to any grading scheme.

Thirdly, if the reductase results are considered alone, in 62% of samples of pasteurised milk the blue colour persisted for more than four hours and this constitutes a justification for choosing a period of four hours for the dividing line between good and bad pasteurised milk. Pasteurised milk in which the colour persists for over four hours is therefore classed as predominantly good, and that which decolourises within four hours as predominantly bad. In Table V the reductase results are analysed as monthly Good : Bad ratios based upon the four-hour dividing line. It will be seen that except in October and December, Depot No. 3 always showed high Good : Bad ratios; Depot No. 7 did badly one May and June; Depot No. 4 gave

TABLE III.

Result of Parallel Methylene Blue Reductase Tests and Breed Counts of 1463 Samples of Pasteurised Milk collected from Seven Depots.

MONTHLY DISTRIBUTION OF METHYLENE BLUE REDUCTASE RESULTS INTO GROUPS A TO E* (GROSS NUMBERS OF TESTS)

Monthly Distribution of Breed counts into Groups 1–5 **		July					August					September					October					November					December				
		A	B	C	D	E	A	B	C	D	E	A	B	C	D	E	A	B	C	D	E	A	B	C	D	E	A	B	C	D	E
1943–1944	1	19	4	–	1	–	22	–	–	–	–	19	3	1	–	–	10	–	–	–	–	14	1	–	–	–	12	–	1	–	–
	2	6	2	–	4	–	16	2	–	–	–	15	6	–	–	–	16	14	–	–	–	20	4	2	1	–	33	1	1	1	–
	3	1	–	–	–	1	2	1	–	1	–	8	6	–	1	–	3	12	1	–	1	13	4	–	–	–	7	–	3	–	–
	4	2	–	1	1	–	2	–	1	–	–	3	5	6	3	–	3	10	3	1	–	12	5	2	1	–	12	4	3	1	1
	5	–	–	–	3	1	9	–	2	5	4	–	7	4	17	3	2	10	7	10	4	20	6	9	9	16	7	3	3	3	2
1944–1945	1	22	–	–	–	–	11	1	–	–	–	24	–	2	1	–	13	1	–	–	–	18	2	1	–	–	6	–	–	–	–
	2	11	–	1	–	–	4	1	–	–	–	3	1	–	–	–	6	–	3	–	–	8	1	2	–	–	3	–	–	–	–
	3	6	–	4	–	–	2	1	4	2	–	3	1	2	1	–	–	5	3	–	1	6	3	1	1	–	–	1	–	–	–
	4	1	–	1	–	–	–	–	2	1	–	2	–	–	–	–	–	2	2	1	–	1	1	1	2	–	–	–	–	1	–
	5	1	1	1	–	2	1	–	–	–	1	1	–	1	–	2	–	1	1	3	1	–	–	1	1	–	–	–	–	–	–

Monthly Distribution of Breed counts into Groups 1–5 **		January					February					March					April					May					June				
		A	B	C	D	E	A	B	C	D	E	A	B	C	D	E	A	B	C	D	E	A	B	C	D	E	A	B	C	D	E
1943–1944	1	15	–	–	–	–	4	–	–	–	–	8	1	–	–	–	6	1	1	–	–	13	–	–	–	–	25	–	–	–	–
	2	15	1	–	2	–	7	3	1	–	–	12	–	–	–	–	5	–	–	–	–	11	2	–	–	–	10	2	1	1	–
	3	9	–	–	3	–	5	–	1	1	–	4	–	3	–	–	6	1	–	–	–	5	3	1	–	–	2	–	1	2	–
	4	27	6	–	5	–	12	2	3	2	–	3	–	4	–	–	1	–	–	2	–	2	2	–	–	–	3	1	1	1	–
	5	8	4	19	18	5	2	1	4	12	3	–	–	1	9	2	–	–	1	8	1	1	–	2	2	1	–	–	1	–	1
1944–1945	1	21	1	–	–	–	18	3	–	–	–	35	3	3	–	–	45	3	–	–	1	27	–	1	–	–	30	1	1	–	–
	2	3	3	1	2	–	5	1	1	2	–	7	5	1	1	–	4	1	2	1	–	8	1	1	–	–	9	–	1	–	–
	3	1	–	1	4	2	1	–	1	2	–	1	3	1	1	–	–	1	–	–	–	3	–	–	1	–	2	–	–	–	–
	4	–	–	–	–	–	–	2	–	1	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	5	–	1	1	–
	5	–	–	–	1	3	–	1	–	2	4	1	–	–	2	–	–	–	–	–	1	1	–	–	–	–	–	–	1	–	–

* *Key to methylene blue reductase grouping*

A=Over 4 hours.
B=4 hours.
C=2½ to 3½ hours inclusive
D=1 to 2 hours inclusive.
E=½ hour or less.

** *Key to Breed count grouping.*

1=Up to 300,000 per cc.
2=300,000 to 900,000 per cc.
3=900,000 to 2,100,000 per cc.
4=2,100,000 to 4,200,000 per cc.
5=Too numerous to count.

N.B.—Tests only read at ½-hour intervals.

TABLE IV.

Summary of Results of Parallel Reductase Tests and Breed counts in Table III expressed as percentages.

	Annual distribution of results as percentages.										Summary of the distribution of all results as percentages of 1463 parallel tests.				
	1943 – 1944					1944 – 1945									
*	*A	B	C	D	E	A	B	C	D	E	A	B	C	D	E
1	17·9	1·0	0·2	0	0	50·4	2·8	1·5	0·2	0·2	29.85	1·7	0·8	0·1	0·1
2	17·8	4·0	0·5	0·8	0	13·3	2·6	2·4	1·1	0	16·2	3·5	1·2	0·9	0
3	7·0	2·9	1·1	0·7	0·2	4·7	2·8	3·1	2·2	0·6	6·2	2·7	1·8	1·3	0·3
4	8·8	3·7	2·6	1·7	0·1	1·9	0·9	1·3	1·3	0	6·3	2·7	2·1	1·6	0·05
5	5·3	3·3	5·7	10·1	4·5	0·9	0·6	0·9	1·7	2·6	3·7	2·3	3·9	6·9	3·8

* For key refer to Table III.

poor results in the early and late summer. The other depots were fairly consistently unsatisfactory, though some like depot No. 1 put up a satisfactory performance in winter, but not in summer.

Deterioration of Milk in the Pasteurising Depot.

While it was very obvious that serious deterioration of milk can take place if bad hygiene is practised at the pasteurising depots, it is by no means easy to show definitely that such deterioration has occurred. Probably the surest method of demonstration would be to perform bacterial counts on the incoming raw milk after it has been bulked and then to compare these counts with others made on the pasteurised products. Similar samples could be subjected to the reductase test. It is sometimes possible to carry out such comparisons at small milk-handling depots, and an example such as this was quoted in a previous report [*vide* Pullinger, 1944(b), paragraph 3, section B(2)]. In practice, however, and particularly at pasteurising depots it is rarely possible to obtain genuine bulk samples of the incoming raw farm milk unless milk is bulked in the farming districts and delivered to market in large tankers. (This practice has not yet been adopted in South Africa.)

Occasionally where an extreme degree of spoilage is occurring, by comparing the quality of individual raw supplies with that of the heated product, it is possible to show that deterioration has occurred. Examples of this extreme form of spoilage are to be found in the 1943 sections of Graphs Nos. 1 and 2. Generally speaking, however, such extreme cases are not often seen, and in consequence, tests for bacterial density are as a rule of little value in assessing the efficiency of plant hygiene. The extent to which variations in bacterial count can be used is indicated by the figures in Table VI, which show the Breed count Good:Bad ratios of samples collected from storage tanks and from cans and bottles ready for distribution. The general trend of these figures shows that considerable deterioration takes place between the tank and the small receptable. Occasionally the figures suggest that improvement has taken place between canning or bottling, but this is an illusion arising from the fact that the consolidated tank average was in some months pulled down by certain tanks, the milk from which was not used for normal distribution and so was not tested when in cans. Sometimes really grave deterioration takes place, as was the case in June and July, 1944. At this stage, high-quality tank milk was being ruined by being put into dirty cans, while in June the bottles, too, were dirty, but by July the bottle-washing machine was working well once more.

Another attempt was made to use bacterial density as an index of deterioration by collecting samples from the cooler-exit of the heat-exchanger and also from the carton-filling machine. Samples were

TABLE V.

Monthly and Annual Record of Reductase Test Good:Bad ratios for seven Pasteurising Depots incorporating 1463 tests.*

DEPOT	MONTHLY AND AVERAGE GOOD:BAD REDUCTASE RATIO*												
MONTH	July	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	Mar.	Apr.	May	June	Annual
De- 43–44	3.8:1	39:1	1.4:1	1.2:1	5.5:1	4.4:1	1.3:1	1:1.6	3:1	5:1	6:1	9:1	2.8:1
pot 44–45	3:1	1:4	2:1	1:1	5:1	2:1	0:7	0:7	1:2	1.7:1	6:0	7:0	1.4:1
1. Average	3.6:1	8:1	1.5:1	1.2:1	5.3:1	4:1	1:1.1	1:3.2	1.1:1	2.5:1	12:1	16:0	2.3:1
De- 43–44	1:5	1:3	1:8.3	1:31	1:1.2	1:1	1:6.2	1:5.5	1:7	0:6	1:3	1:1.2	1:3.3
pot 44–45	1:3.5	2:1	9:0	1:2.5	1:2.5	1:1	1:2.5	1:1.5	1.7:1	2:1	4:0	2:1	1.2:1
2. Average	1:4	1:1.3	1:2	1:12	1:1.3	1:1	1:5.1	1:3.5	1:1.7	1:1.8	1:1	1.3:1	1:2.2
De- 43–44	—	—	—	—	—	—	—	7:1	8:0	4:1	7:1	9:0	11.7:1
pot 44–45	9:0	4:1	7:0	1.2:1	3.5:1	2:0	8:0	6:1	12:1	7:1	12:0	14:0	9.1:1
3. Average	—	—	—	—	—	—	—	6.5:1	20:1	6:1	19:1	23:0	9.6:1
De- 43–44	6:0	8:0	1:2	1:1.2	1:1.1	8.3:1	4.1:1	1.4:1	2:1	1:1	3:1	8:1	2.2:1
pot 44–45	8:0	1.5:1	6:1	1.2:1	6:1	2:0	1:5	1:1.5	1.5:1	7:0	2:1	8:0	2.6:1
4. Average	14:0	5.5:1	1:1.1	1:1	1.2:1	9:1	2.5:1	1.1:1	1.7:1	3.3:1	2.5:1	16:1	2.4:1
De- 43–44	1:5	0:8	0:9	0:11	1:1.3	1.7:1	2.2:1	1:1.4	0:7	1:4	1:1	1:3	1:1.6
pot 44–45	8:1	1:1.5	1:7	1:5	1:2	2:0	6:1	1:1.5	1:1.7	7:1	6:0	5:1	1.4:1
5. Average	1.5:1	1:5.5	1:16	1:16	1:1.4	2:1	2.6:1	1:1.4	1:4	1.6:1	2.5:1	1:1	1:1.2
De- 43–44	—	—	—	—	—	—	—	—	—	—	—	—	—
pot 44–45	—	—	—	—	—	—	4:0	6:1	6:1	6:1	5:0	6:0	11:1
6. Average	—	—	—	—	—	—	—	—	—	—	—	—	—
De- 43–44	—	—	—	—	—	—	0:1	4:1	6:1	5:0	7:0	8:0	10:1
pot 44–45	8:0	4:1	4:0	—	—	—	4:0	6:1	9:0	6:1	1.5:1	1:1.5	5.8:1
7. Average	—	—	—	—	—	—	4:1	5:1	15:1	11:1	5:1	3.3:1	6.9:1
Mon. 43-44	1.8:1	3.3:1	1.3:1	1:2.2	1.3:1	2.6:1	1.2:1	1.1:1	1.4:1	1.2:1	2.5:1	3.3:1	1.3:1
sum. 44-45	4.1:1	2.2:1	3:1	1.3:1	1.9:1	4.5:1	1.4:1	1.2:1	2.2:1	4.9:1	10:1	7.7:1	2.5:1
(7 deps.) Av.	2.7:1	2.7:1	1.3:1	1:1.8	1.5:1	2.8:1	1.2:1	1:1	1.8:1	2.7:1	4.2:1	4.8:1	1.6:1

* Good:Bad ratio represents the proportion of tests in which colour persisted over 4 hours to those which decolourised within 4 hours.

TABLE. 6

DETERIORATION OF PASTEURISED MILK WITHIN TWO PASTEURISING DEPOTS, EXPRESSED AS MONTHLY GOOD: BAD RATIOS *

GOOD: BAD RATIO *	1943 JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP.	OCT	NOV	DEC	1944 JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC	1945 JAN	FEB	MAR	APR	MAY	JUN	TOTAL SAMPLES TESTED
CONSOLIDATED GOOD: BAD RATIOS FOR ALL TANK SAMPLES FROM DEPOT 1. AND DEPOT 2.	1/3·2	1/1·6	2·1/1	1/1·2	1/1·1	1/1	1/2	1/2·3	1/3·1	1/1·8	1·2/1	2·2/1	1/2·8	2·2/1	6/1	4/1	5·8/1	8/1	10/1	1·8/1	1/1	1/4·2	1/8·5	1/1·2	1·8/1	2·2/1	1·9/1	1·4/1	2·8/1	3·7/1	3674
GOOD: BAD RATIOS FOR 10-GALLON CAN SAMPLES DEPOT 1.	–	1/8·5	1/1·2	1/1·9	2·8/1	3/1	1·2/1	1/2	1/2·7	1/3	1/1·8	1·3/1	1/2·8	1/1·3	1·9/1	3·5/1	2·3/1	2·1/1	2/1	1/1·6	1/2·5	1/4	1/6·3	1/8·5	1/2·1	1/1·7	1/1·2	1/1	3·8/1	1/1·2	596
GOOD: BAD RATIOS FOR 10-GALLON CAN SAMPLES DEPOT 2.	–	1/2·8	–	1/1·8	1/3·5	1/2	1/1·6	1/9	1/1·7	1/7·5	1/2·5	1/1·6	1/2·5	1·1/1	1·3/1	1·1/1	3/1	3·4/1	1·1/1	1/1·4	1/10	1/6	1/6·3	1/1	1/1·2	1·1/1	2·3/1	1/1	1·8/1	1·3/1	529
CONSOLIDATED GOOD: BAD. RATIOS FOR ALL 10-GALLON CAN SAMPLES DEPOT 1 AND 2	–	1/4·7	1/1·2	1/1·8	1/1·1	1·1/1	1/1·1	1/3·6	1/4·7	1/4·3	1/2·2	1/1·1	1/1·7	1/1·1	1·6/1	1·9/1	2·6/1	2·7/1	1·5/1	1/1·4	1/3·7	1/4·9	1/6·3	1/2·9	1/1·6	1/1·3	1·4/1	1/1	2·4/1	1·4/1	1125
GOOD: BAD RATIOS FOR PINT BOTTLE SAMPLES DEPOT 1.	–	–	1/2·3	1/3·8	1/1·7	1/2·5	1/4·5	1/5·7	1/2·1	1/3·2	1/1	2·8/1	1/3	2·5/1	2·3/1	1·8/1	5·3/1	1/1·2	9·5/1	3·7/1	1·4/1	1/4·2	1/10	1·1/1	1/1·7	1·7/1	5/1	3·3/1	1·2/1	4/1	540

* GOOD: BAD RATIO FOR PASTEURISED MILK IS TAKEN AS THE PROPORTION OF SAMPLES WITH BREED COUNTS OF 300,000 per cc. OR UNDER TO THOSE SHOWING BREED COUNTS ABOVE 300,000 per cc.

collected from the flowing milk in quick succession into sterilized sample bottles, trade bottles washed by the routine method, and cardboard cartons (South African manufacture). These samples were allowed to incubate at room temperature for about four hours before Breed counts were prepared. The result of this survey is recorded in Table VII in summary form, from which it will be seen that there is comparatively little evidence of deterioration arising directly from these small receptacles. In the case of the cartons occasional batches were found to be badly contaminated, but on the whole the cartons themselves did not cause serious spoilage, though the carton-filling machines themselves are a constant source of bacterial contamination.

TABLE VII.

Investigation into the Deterioration of Pasteurised Milk in Trade Bottles and Cartons.

PERCENTAGES OF SAMPLES SHOWING BREED COUNT VARIATIONS BETWEEN SPECIALLY STERILISED BOTTLES AND TRADE RECEPTACLES (BOTTLES 1158 TESTS, CARTONS 1166 TESTS)

Increase fivefold or less		Increase fivefold to tenfold		Increase over tenfold		Trade receptacle showing count of at least five times lower than that from sterile bottle (i.e. index of faulty sampling)	
Trade Bottle	Carton	Trade Bottle	Carton	Trade Bottle	Carton	Trade Bottle	Carton
80·3	80·2	8·9	9·4	6·3	7·0	4·5	3·4

Lack of any better test for deterioration in pasteurised milk has led to the presumptive coliform test being tried out. The use of this test is based upon the assumption that pasteurisation destroys all coliform bacilli and that if such organisms reappear the reason must be recontamination. For this particular purpose the presumptive coliform test is sufficient since the point at issue is simply one of recontamination regardless of whether fæcal or non-fæcal strains of *Bact. coli.* are involved. Obviously the whole value of the test hinges upon the question of whether or not coliform bacilli are always destroyed by pasteurisation (whether by holder or short-time high-temperature methods). Unfortunately no definite conclusion has been reached with regard to this point. Available laboratory evidence indicates that they are probably killed and evidence such as that presented in Table VIII with regard to Depot No. 3 supports the view. Figures for Depots Nos. 1 and 2, however, throw some doubt on this point as do the correlation figures recorded in Table II, and this problem is being subjected to more detailed investigation.

In Table VIII monthly results are recorded of the regular testing of samples for coliform organisms. The cooler-exit sample can be

TABLE VIII.

Percentage of Samples from various points in three Pasteurising Depots showing coliform organisms absent in 1 cc.

Year and Month		DEPOT 1				DEPOT 2			DEPOT 3	
		Cooler Exit	Tanks	Can	Bottle	Cooler Exit	Tanks	Can	Cooler Exit	Tanks
	Jan. . .	82	71	12	70	50	—	15	—	—
	Feb. . .	50	50	11	16	6	54	40	—	—
	Mar. . .	43	100	—	—	57	67	—	—	—
	Apr. . .	93	100	65	94	100	84	84	—	—
1	May . .	100	100	83	94	100	97	82	—	—
9	Jun. . .	100	100	79	90	100	87	81	—	—
4	Jul. . .	84.8	79	59	68	100	76	95	—	—
3	Aug. . .	72	70	95	43	95	91	84	—	—
	Sep. . .	75	86	33	75	100	50	33	—	—
	Oct. . .	75	84	25	50	78	22	6	—	—
	Nov. . .	91	86	44	63	84	41	9	—	—
	Dec. . .	100	97	5	81	95	35	13	—	—
	Jan. . .	90	87	78	79	84	35	13	100	23
	Feb. . .	67	76	9	70	72	56	11	100	68
	Mar. . .	96	81	26	82	41	36	5	95	93
	Apr. . .	100	100	43	75	28	79	6	100	87
1	May . .	100	100	40	95	95	89	55	100	85
9	Jun. . .	88	89	78	0	90	89	79	100	100
4	Jul. . .	90	97	71	100	94	92	86	—	—
4	Aug. . .	96	91	36	79	100	94	58	100	68
	Sep. . .	95	88	19	37	90	77	52	100	43
	Oct. . .	95	91	90	19	58	36	14	100	58
	Nov. . .	77	79	40	36	85	39	9	100	98
	Dec. . .	63	67	0	9	74	33	0	100	93
	Jan. . .	81	63	0	0	81	29	11	100	93
1	Feb. . .	40	25	0	13	72	26	7	100	89
9	Mar. . .	48	33	0	0	69	68	47	95	77
4	Apr. . .	76	63	5	14	80	60	11	100	85
5	May . .	74	63	37	44	79	47	42	100	91
	Jun. . .	95	89	35	81	94	58	70	100	94

N.B.—All coliform tests were based upon their presence or absence in 1 cc. amounts.

taken as the criterion of whether or not coliform organisms are being destroyed by the heat. This assumption is not really justified as will be explained later, but presuming it is true there is still evidence of serious deterioration of milk between the cooler exit and the cans and bottles available for distribution. Actually the interpretation of coliform test results is by no means straightforward and considerable discretion has to be used in evaluating results. In Table IX a monthly circuit control record is given as an illustration. On the fifth day a tank, the can-filling tap and the can sample all contained coliform bacilli in 1 cc. The rest of the circuit was clear, indicating that a storage tank was probably dirty. The various interpretations are shown in the remarks column, but attention should be drawn to those instances where the cooler-exit sample is contaminated. On the 16th this was probably a simple sampling error. On the 22nd, however, the whole circuit was contaminated while there was already partial contamination on the previous day. It is almost certain that that coliform contamination from the heat-exchanger began to develop on the 21st either as a result of a dirty heat-exchanger, internal leaks between the pasteurised milk and either the raw milk or the cooling water, or else as a result of failure of the heat to kill the coliform organisms present in the raw milk. The final answer can only be obtained by a process of elimination.

From Table VIII it will be seen that while Depot 3 usually gave 100% of cooler-exit samples free from coliform organisms, the other two depots showed contamination, sometimes in 50% of samples. Four explanations of this are offered:—

1. Faulty cleaning at Depots Nos. 1 and 2. This is not convincing, because the rest of the circuit cleaning at these Depots was better than at No. 3.
2. Faulty sampling at Depots Nos. 1 and 2. This is also unconvincing, because the rest of the sampling was as good as at No. 3.
3. Leakages in the heat-exchangers at Depots Nos. 1 and 2. Such leaks between the pasteurised milk and the cooling water definitely did develop in both plants. This matter has been discussed more fully already [*vide* Pullinger, 1944(a)].
4. There is a strong suggestion that if the coliform contamination of the raw milk is very severe as during summer, some coliform organisms may survive the heating process, just as some streptococci may survive (*vide* Zeller, 1928).

Quite obviously, unsatisfactory heat-exchanger results must be reflected in the results of tank samples. In Table X a summary is given of the percentage of samples from each sampling point that was free from coliform bacilli. It will be seen that while Depot No. 3 gave the best cooler-exit results, indifferent plant hygiene was

TABLE IX.

Example of a table of Monthly Circuit Control Tests for Coliform Contamination in a Pasteurising Plant.

Date	Cooler Exit	Storage Tank No. 1	Storage Tank No. 2	Can-filling Tap	10-gallon Can	Pint Bottle	REMARKS.
1	—	—	—	—	—	—	
2	—	—	—	—	—	—	
5	—	—	+	+	+	—	Contaminated tank.
6	—	—	—	—	—	—	
7	—	—	—	—	—	—	
8	—	—	—	+	+	—	Contaminated discharge pipe.
9	—	—	—	—	+	—	Contaminated can.
12	—	—	—	—	—	—	
13	—	—	—	—	—	+	Contaminated bottle.
14	—	—	—	—	—	—	
15	—	—	—	—	—	—	
16	+	—	—	—	—	—	Probably faulty sampling.
19	—	—	—	—	—	—	
20	—	—	—	—	—	—	
21	—	—	+	+	+	—	Contaminated tank.
22	+	+	+	+	+	+	Contamination throughout circuit. Suspected breakdown in heat exchanger. Alternatively heat-resisting coliforms present.
23	+	+	+	+	+	+	Persistence of coliforms in cooler exit indicates fault in heat exchanger.
26	—	—	+	—	—	+	Heat exchanger rectified. Circuit not yet clean.
27	—	—	—	—	—	—	
28	—	—	+	—	—	—	Probably faulty sampling.
29	—	—	—	+	—	—	Faulty sampling or contaminated discharge pipe.
30	—	—	—	—	—	—	

practised, and far greater recontamination of the milk occurred at Depot No. 3 as compared with Depot No. 1. The average tank percentage for No. 1 was 81 and for No. 3 was 77%. In Depot Nos. 1 and 2 very serious recontamination took place in the circuit used for filling cans and also in the cans themselves.

Discussion.

Evidence has been presented to show that there is a definite relationship between the quality of raw milk and the same milk when pasteurised. It might be thought that this is a case of proving something that is already obvious, but it is all too apparent that in South Africa many people believe that any semi-sour milk is suitable for pasteurisation. A more serious aspect is that local health authorities lay down a standard of maximum allowable density of bacterial flora to which pasteurised milk is expected to conform without giving the pasteurising firm the protection of ensuring that clean farm milk is introduced on to the market. In other words the onus is placed upon the pasteurising firm of turning a third-rate article into one of first-rate quality. In earlier reports the need for a more strict control or grading of incoming farm milk was stressed [*vide* Pullinger, 1944(b) and 1945(b)], and the opportunity is again taken of re-emphasising the essentiality of regular chemical and bacteriological grading of all farm supplies. Until such a grading system is in operation it will be impossible to enforce any severe standard with regard either to pasteurised milk or to raw milk distributed by milk shops.

Both the Breed count and the methylene blue reductase test have been used in attempts to grade pasteurised milk, because these tests are easily and cheaply performed and may readily be applied to the daily testing of many samples. In this respect these tests differ from the duplicate plate count incubated at low and high temperature (*vide* Anderson and Meanwell, 1933), which is a most cumbersome procedure. When parallel Breed and reductase tests were done, no correlation between results was found to exist. This finding was only to be expected, since those organisms most likely to survive heat-treatment do not spoil milk rapidly. Only in extreme cases was the quantitative Breed examination of value in judging pasteurised milk. Thus if the average raw milk Breed count is in the hundred-thousand zone while the pasteurised milk count is over five million, it is obvious that something is wrong with the plant hygiene, but such tenfold differences are not often seen. An instance of the use of the quantitative Breed test in showing up gross failures is to be found in the 1943 sections of graphs Nos. 1 and 2 and again in Table VII. From a qualitative point of view, however, the Breed test can frequently be of use because it can give a clue to the identity of organisms present and hence their probable source. It can moreover show whether excessive amounts of pus are leaking through the filters.

TABLE X.

Average percentage of samples from various points in circuit with coliforms negative in 1 cc (whole period of survey).

DEPOT	Cooler Exit	Tank No. 1.	Tank No. 2.	Tank No. 3.	Tank No. 4.	Tank No. 5.	Tank No. 6.	Tank Average.	Can-filling Tap.	Can.	Bottle.
No. 1	83·8 (580)	81·0 (485)	81·0 (353)	79·0 (171)	——	——	——	81·0 (1009)	61·5 (571)	33·3 (572)	54·0 (507)
No. 2	78·2 (521)	82·7 (213)	65·0 (345)	54·2 (301)	56·0 (148)	61·5 (104)	45·8 (133)	63·0 (1244)	21·7 (426)	39·5 (515)	——
No. 3	98·7 (359)	59·0 (108)	84·0 (192)	82·0 (143)	76·0 (93)	——	——	77·0 (536)	——	——	——

N.B. — Figures in parenthesis represent total number of tests done.

The methylene blue reductase test has been applied only to a limited number of samples and caution must be exercised in interpreting the findings. Clearly it is reasonable to set the period of four hours as the dividing line between Good and Bad pasteurised milk. That is to say, milk decolourising in four hours or less is predominantly bad and that taking 4½ hours or more to decolourise is good. If a standard is applied that in summer two samples should be good to every one that is bad and in winter that five should be good to one bad one (i.e. Good:Bad ratios of 2:1 and 5:) then it will be seen that Depots Nos. 3, 6 and 7 generally conformed to these standards while the others generally failed to do so (*vide* Table V). Provisionally, therefore, and as long as no grading restriction is placed upon the quality of the incoming raw milk, the most stringent demand that could be made for pasteurised milk would be:—

SUMMER — Good:Bad ratio of 2:1,

WINTER — Good:Bad ratio of 5:1,

taking a good sample as any one in which the colour persists for over four hours and taking summer as including the months September to March. Quite obviously when the quality of the incoming raw supplies is improved this pasteurised standard would have to be altered.

Deterioration of milk during processing consists in part of the growth of thermophilic bacteria during holder pasteurisation. Such growth would be revealed by the Breed smear, if excessive, or by comparative plate counts incubated at high and low temperature. It is not, however, suggested that any strict numerical standard of allowable bacterial density should be laid down, and it is considered sufficient to use the Breed smear as an indication of whether thermophilic bacteria are growing, in which case a closer investigation of the plant is warranted. A much more serious form of recontamination during processing results from bad washing and sterilisation of apparatus, because in this case contaminants appear which spoil milk rapidly. Moreover at this stage recontamination with pathogenic bacteria becomes an important factor. The big problem is to choose a test which will serve as a reliable index of this recontamination. Of those available, the presumptive coliform test has so far proved to be most helpful. It is easily and cheaply performed and calls for no undue technical skill in application. There are, however, serious drawbacks to the use of this test, the one being that the result is often not available for 48 hours or even longer. Moreover the value of the test depends entirely upon the assumption that pasteurisation always kills all coliform organisms. Actually there is much reason to doubt whether all coliform bacteria are killed by holder or by short-time high-temperature pasteurisation, if the initial contamination of the raw supplies is very heavy. An isolated test for coliform bacilli

done on a random sample is perfectly worthless and this test must be used by applying it to a series of samples taken daily from suitable points in the pasteurising and distributive circuits. In the first place it is imperative to get coliform negative results* in the sample collected immediately after the milk has passed the heating unit. If this sample is positive it means:—

(a) That the sampling was badly done;
(b) That the heat-exchanger is dirty;
(c) That the heat-exchanger has an internal leak; or
(d) That coliform organisms are not being destroyed entirely.

Any of the foregoing causes may apply and only detailed investigation will reveal which factor is operative on any particular occasion. If, however, it happens to be the last factor that is responsible, then the presence of coliform bacilli in the final pasteurised product cannot be classed as the responsibility of the pasteurising firm. It is for this reason that it is necessary to have constant serial tests of samples taken at suitable points throughout the circuit if the coliform test is to be used and interpreted intelligently.

The percentages given in Table X indicate the best level that it has been possible to maintain in South Africa by firms kept under constant laboratory control. Averages of from 63 to 81% of tank samples gave negative coliform results. Of ten-gallon can samples, 33 to 39% of samples were negative and in one depot 54% of bottle samples were negative. The degree of contamination which occurs between the storage tank and the distributive container is frightening, because this is the stage where contamination by milk-handlers can take place, but even the storage tank figures are appallingly unsatisfactory when compared with figures quoted by Barkworth of three pasteurising depots in England in which trade bottle samples showed from only 1 to 5·6 per cent. of positive coliform results.

The presumptive coliform test is an arbitrary one in that great significance is attached to the presence of coliform bacilli in one cubic centimetre, while their presence in a density of one per two cubic centimetres is ignored. This difficulty can be overcome by using the incubated coliform test in which case the result reads as coliform organisms present in or absent from the total sample (*vide* Barkworth). Under South African conditions, however, much progress in coliform control will have to be effected before it becomes necessary to use the incubated test as a routine procedure.

Obviously from the point of view of effective plant control by the presumptive coliform test, the primary effort must be directed to obtaining cooler-exit samples negative to this test. This result was practically always achieved at Depot No. 3 (*vide* Table VIII), and

* **Throughout this report positive or negative coliform results means the presence or absence of coliform organisms in 1 cc.**

in theory similar results should also be achieved in any plant in which the heat-exchanger rubbers and plates are sound, where the phosphatase test records 99% heat-treatment efficiency, and where the incoming raw milk is reasonably clean. In the case of Depots Nos. 1 and 2 positive cooler-exit results have frequently led to a search for leaky plates or rubbers and in fact this test has come to be used as the first indication of the presence of internal leaks between the raw and pasteurised milk or between the cooling water and the pasteurised milk [*vide* Pullinger, 1944(a)].

The presence or absence of coliform organisms in the rest of the circuit obviously depends upon the cooler-exit results, since leakage of coliform organisms at this point is bound to contaminate the whole circuit, but if cooler-exit samples are generally negative then the appearance of these organisms in the subsequent circuit is due to recontamination. From the results actually obtained with controlled plants (see Table X) it is reasonable to demand a proportion of four tank samples negative to each sample giving a positive coliform result, because this level has already been achieved by Depot No. 1 and nearly so by Depot No. 3. Actually if the cooler-exit samples could be maintained in the region of 100% negative — as was done in the case of Depot No. 3 — then tank samples should show a proportion of at least five negative to one positive.

Clearly, until efficient can-washing and sterilising machines are available it would be useless to set too severe a standard for milk-can samples, and a proportion of one positive to one negative sample is the highest level that could be demanded at present. A much more severe standard would have to be established when efficient can-washing facilities become available.

Good bottle-washing, sterilising, filling and capping machines are already available and a comparatively severe standard could therefore be demanded. In the only available set of figures, 54% of the bottles were negative, but months are on record when this figure rose to 80% and higher. It is suggested therefore that a ratio of four negative samples to one positive one would be a reasonable standard to demand, always assuming that the storage tank samples were being maintained at standard too.

In reviewing the question of grading pasteurised milk, certain factors have to be borne in mind because they are of fundamental importance:—

1. Grading of incoming raw farm supplies is an inescapable prerequisite to the grading of raw or pasteurised milk available for distribution to the consumer.
2. Pasteurised milk should always be filled by automatic means into sealed containers for continuous storage under refrigeration conditions. Pasteurised milk should never be filled by hand

into standard ten-gallon milk cans for shipment in open lorries or railway trucks under hot sunshine.

3. Since the organisation of the South African dairy industry is such that pasteurised milk must be mishandled in bulk (e.g. in supplying hospitals, hotels, military camps and schools, some far distant from the Depot) then it becomes quite futile to expect milk so misused to line up to standards which can only just be maintained at the depot.
4. Grading of pasteurised milk can only be done at the depot and during delivery. The final grading must be based upon the correlated results of all the samples. In fact the decision is not that a particular sample of milk is substandard, but rather that over a given period a certain factory is operating in a sub-standard manner. It cannot be too strongly stressed that an unfavourable coliform result from an isolated sample of milk collected from a ten-gallon can thirty miles away from where the can was originally filled is quite meaningless. Such random and casual sampling is at best merely a guide to the need of closer investigation, except when the phosphatase test reveals under-pasteurisation or the chemical tests reveal adulteration.

The actual choice of standards is difficult as long as no grading of farm supplies is practised and the best that can be done is to choose average figures that distributors should be able to maintain. The distributor should, however, retain the right to plead that the fault lies in his farm supplies, and machinery should be available for checking up on the quality of such supplies. It must be fully appreciated that some of the data given in this report is limited and that the standards to be suggested are not considered to be definite, but rather as a guide to some future grading scheme.

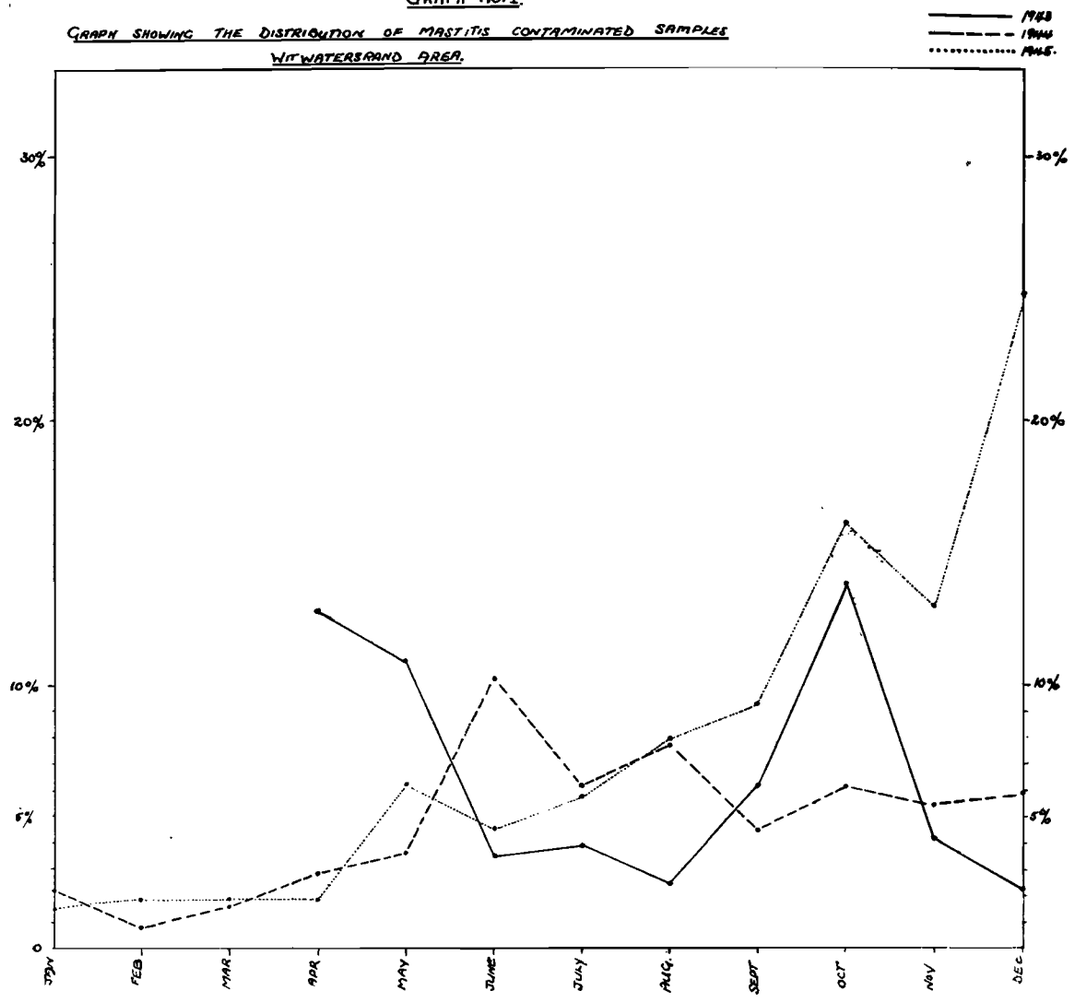
Tentative Standards for Pasteurised Milk.

Source of Sample	*Phosphatase Test*	*Butter Fat*	*Solids Not Fat*	*Reductase Good:Bad ratio*		*Presumptive coliform test negative in 1 cc.*
				Summer	*Winter*	
Storage Tank	99% of	3.25%	8.25%	Good:	Good:	90% of samples
Trade Bottle	samples	Butter	S.N.F.	Bad	Bad	80% of samples
Carton	to be	Fat in	in 80%	ratio	ratio	80% of samples
Large Cans	properly	80% of	samples	2:1	5:1	50% of samples
	pasteurised	samples				

1. A Breed count should automatically be done on every sample and an unexpected result would indicate the need for a closer investigation.
2. The presence of added preservative would class as adulteration.

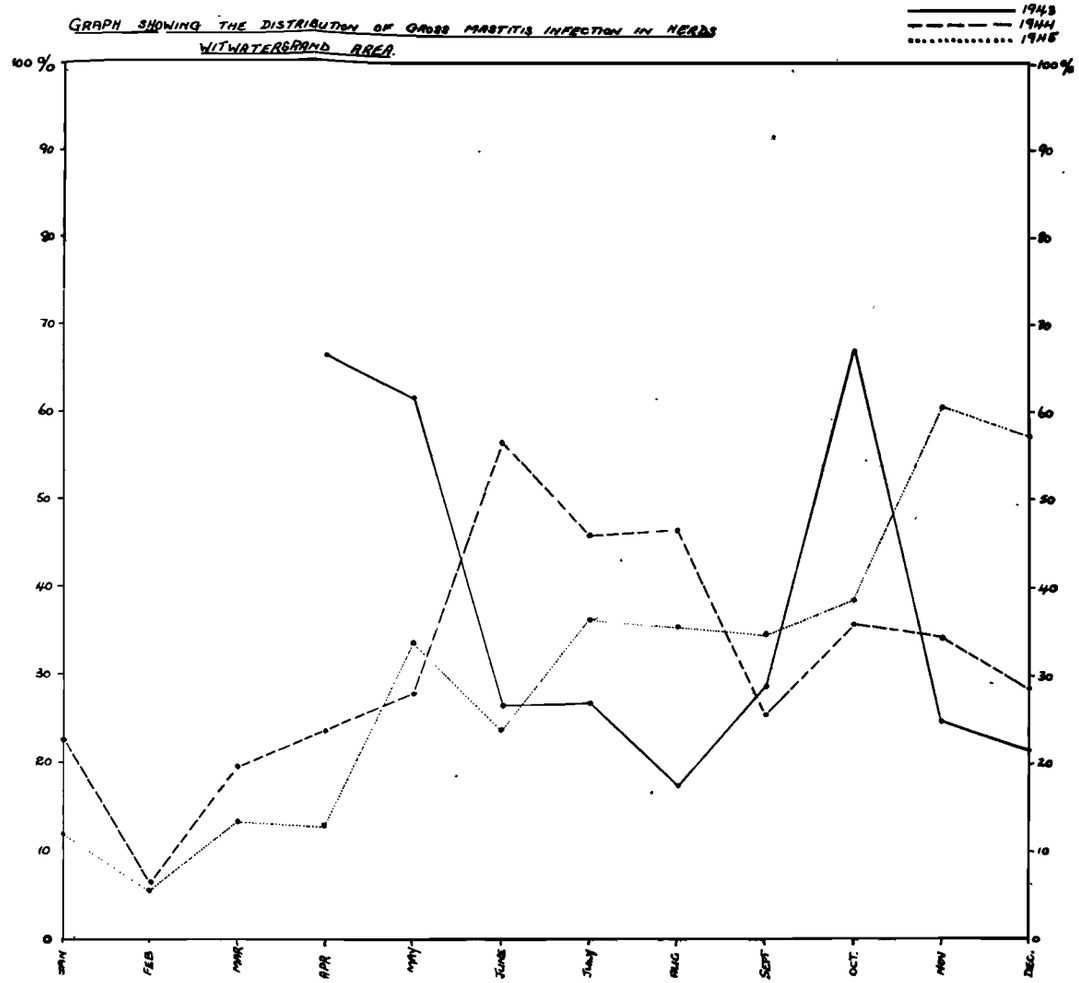
GRAPH No. 1.

GRAPH SHOWING THE DISTRIBUTION OF MASTITIS CONTAMINATED SAMPLES WITWATERSRAND AREA.



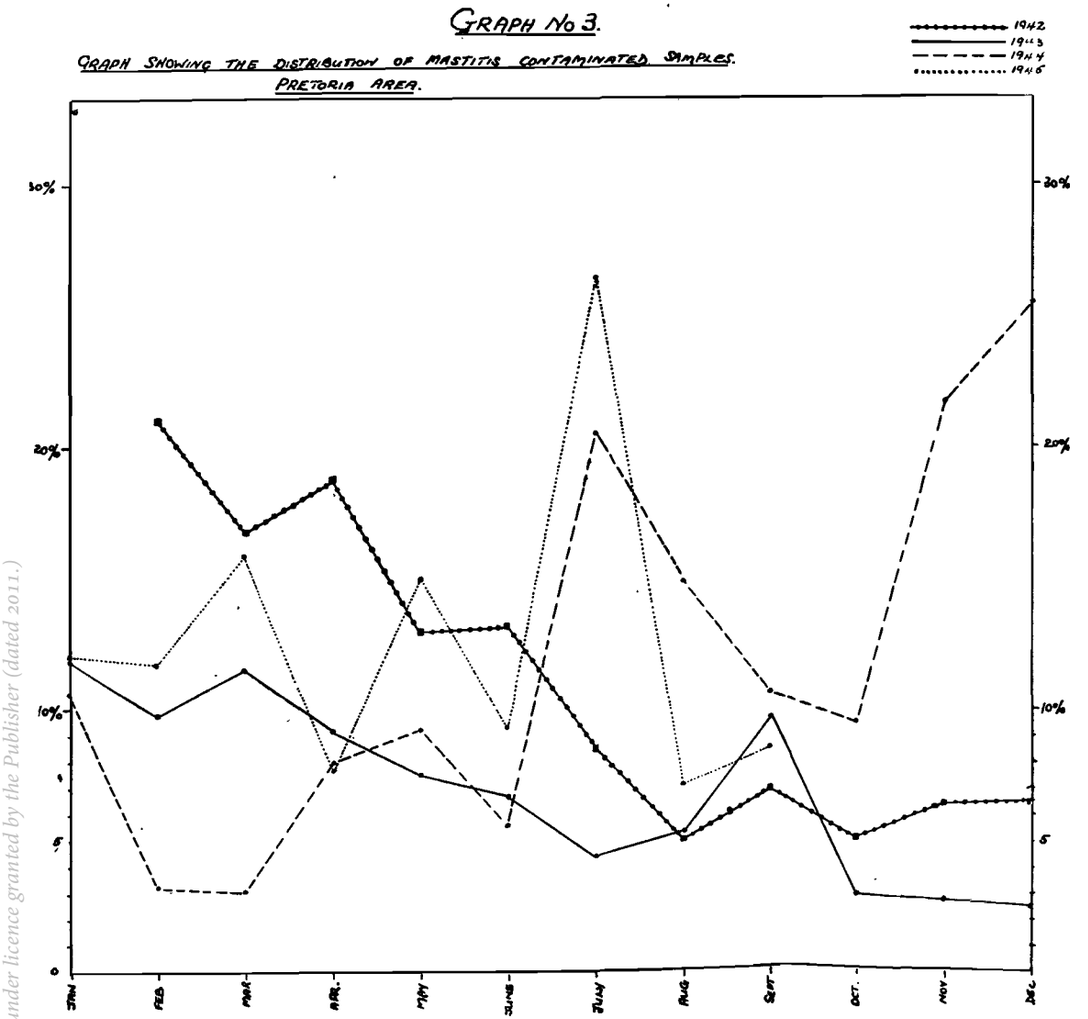
GRAPH No. 2.

GRAPH SHOWING THE DISTRIBUTION OF GROSS MASTITIS INFECTION IN HERDS WITWATERSRAND AREA.



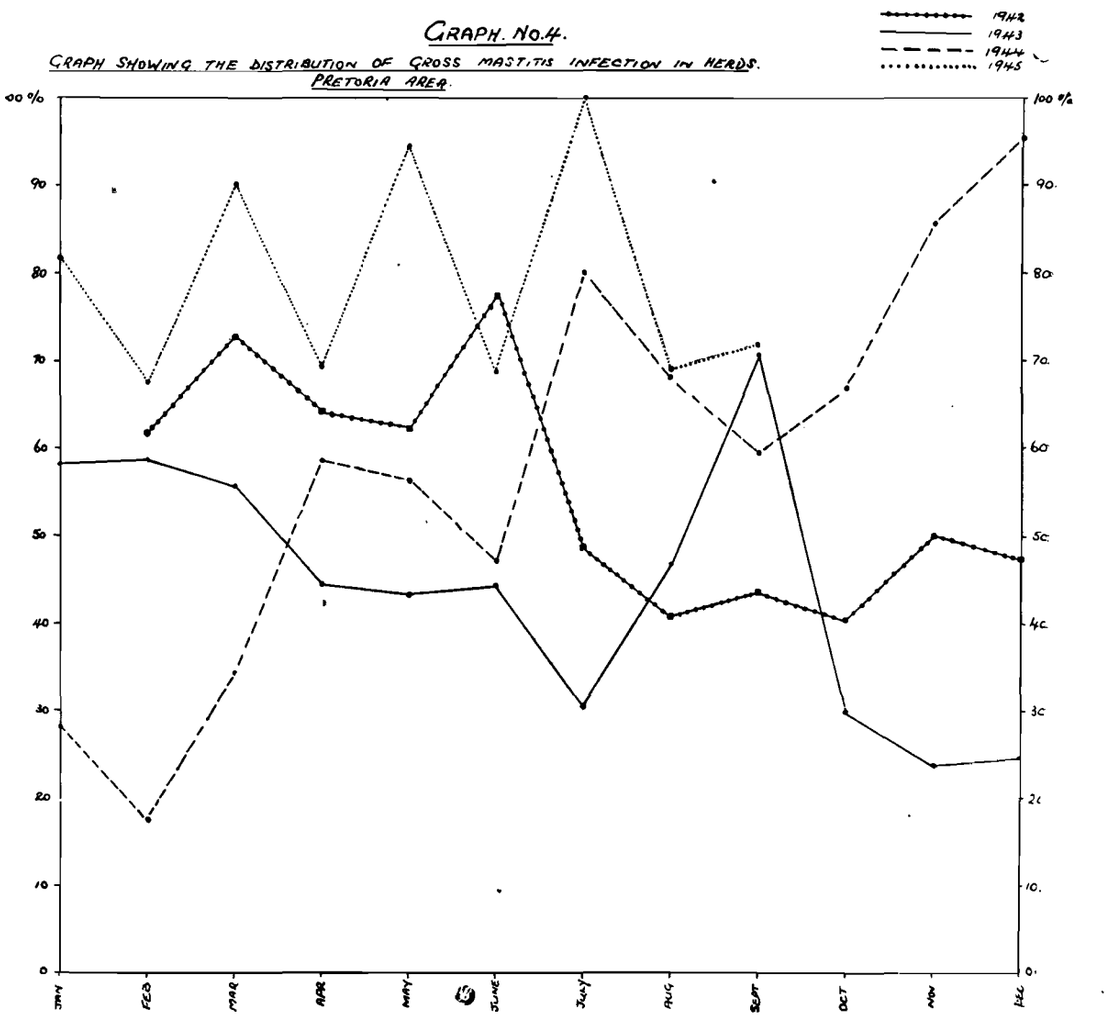
GRAPH No 3.

GRAPH SHOWING THE DISTRIBUTION OF MASTITIS CONTAMINATED SAMPLES. PRETORIA AREA.



GRAPH. No.4.

GRAPH SHOWING THE DISTRIBUTION OF GROSS MASTITIS INFECTION IN HERDS. PRETORIA AREA.



Summary.

1. It is urged that the practice of distributing pasteurised milk in bulk is highly dangerous and that such milk should be automatically bottled and sealed and stored under refrigeration.

2. Where pasteurised milk is distributed in bulk it cannot be expected to line up to any strict bacteriological standards.

3. The judging or grading of pasteurised milk on the basis of isolated tests of random samples is valueless except as a guide to the need for closer investigation. Grading cannot be based upon the results of single tests, but only upon a series of tests covering the whole processing and distributing circuits. The final decision on grading should be whether the factory is operating in a satisfactory or a substandard manner.

4. Both in respect of nutritive and bacteriological quality, pasteurised milk can reflect the quality of the original farm supplies. Therefore, a strict grading of farm milk supplies is an essential procedure to the grading of pasteurised milk. Until farm-milk grading is introduced, the pasteuriser should have the right to plead that his raw supplies are unsatisfactory and machinery should be available for checking this aspect.

5. A set of tentative pasteurised milk standards is suggested.

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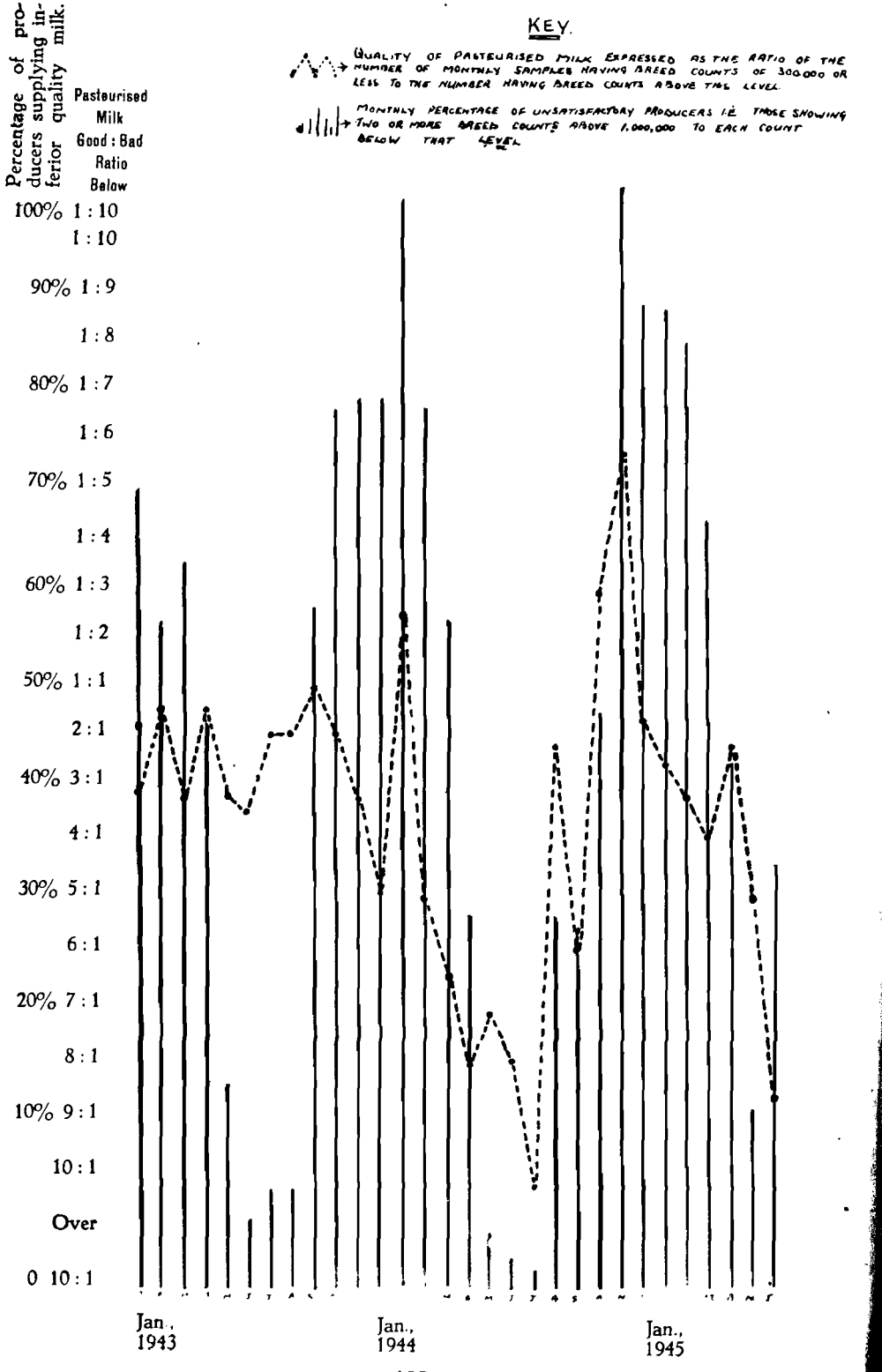
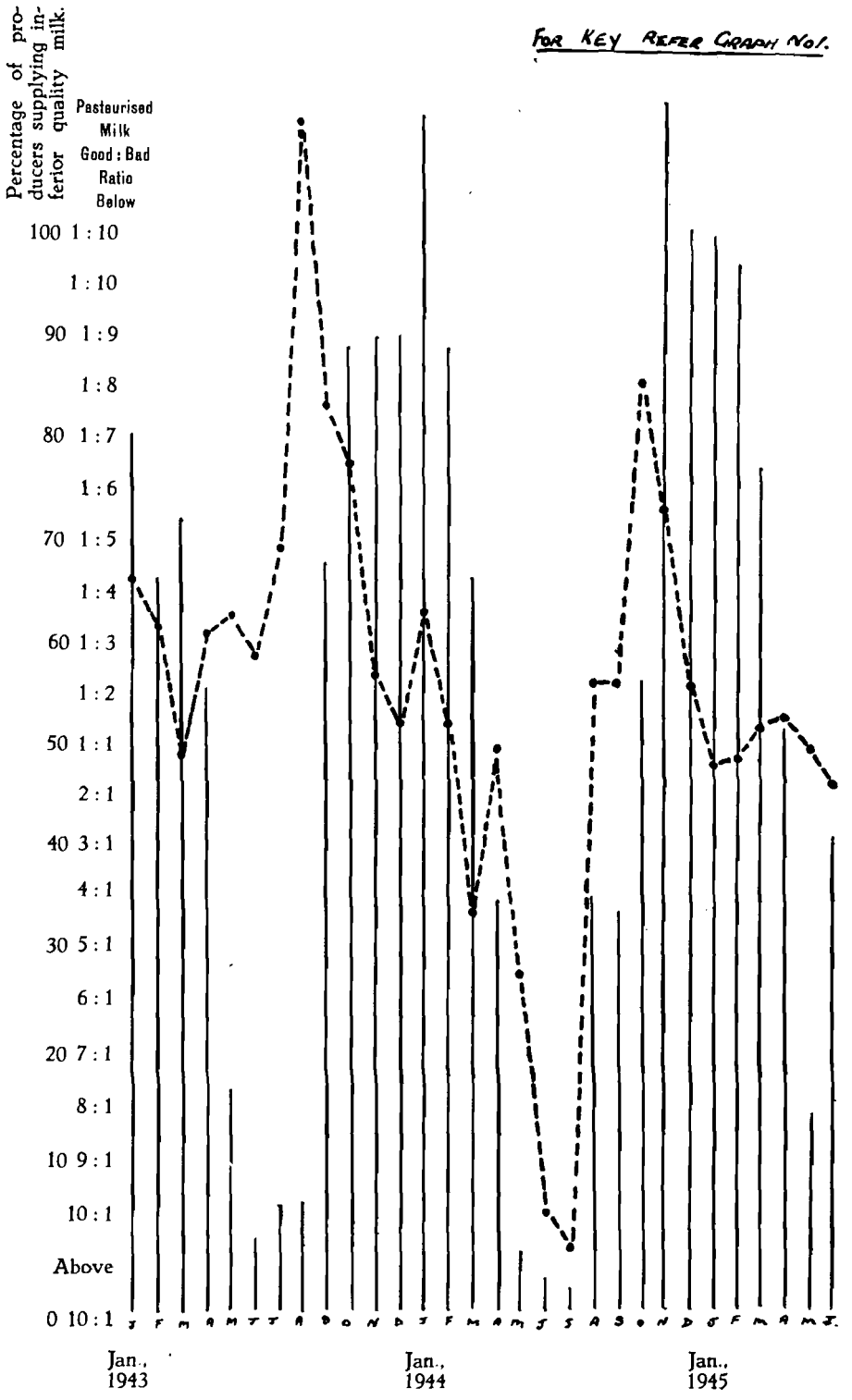
GRAPH No. 5.

Influence of the quality of the incoming raw milk upon the quality of the final product (Depot No. 2.)



GRAPH No. 6.

Influence of the quality of the incoming raw milk upon the quality of the final product (Depot No. 2.)



PARASYMPATHETIC CONTROL OF BODY FUNCTION AND ITS ROLE IN VETERINARY PRACTICE.

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Onderstepoort.

1. *Physiological Concepts.*

Although in the past it was generally accepted that the functions of different organ systems of the animal body are controlled and co-ordinated by a complex interplay of (a) the nervous system, (b) specific agents elaborated in the endocrine organs and (c) various products of cell metabolism, it was within recent years only that the brilliant researches of Loewi, Dale, Cannon and others have revealed the fact that conduction of nerve impulses themselves is dependent upon the action of specific chemical mediators. These substances are elaborated either within the central nervous system itself, various nerve ganglia or in the terminal endings of peripheral nerves as these enter the effector organs.

Concerning the autonomic nervous system, it has moreover been definitely established that two important though mutually antagonistic groups of substances referred to as *cholinergic* and *adrenergic* respectively are responsible for the effects following stimulation of this system.

Adrenergic substance is liberated as far as is known only from the post ganglionic fibres of the sympathetic system and is in all probability identical with adrenalin as formed in the adrenal medulla. Functioning in close harmony with each other, the sympathetic system and the adrenal medulla are jointly responsible therefore for the rapid adaptation of the circulation as well as of other body functions to emergency conditions.

Contrarily, in the parasympathetic system and in the ganglia served by the preganglionic fibres of the sympathetic division, an extremely active though highly unstable cholinergic substance is liberated immediately upon stimulation of the nerves concerned. This substance has been identified as *acetylcholine* which, as the acetic ester of choline, is promptly synthesised though in the minutest traces only. After momentarily exerting its characteristic action on the tissues concerned, this ester is, however, equally promptly broken down again by an enzyme *cholinesterase,* which thereby obviates any continued after-effects. Similar results can be achieved by the injection of *atropine sulphate* which effectively blocks the action of acetylcholine.

The injection of *eserine* (physostigmine), on the other hand, specifically inhibits the enzyme cholinesterase and thereby prolongs and accentuates the acetylcholine effects. Continued cholinergic action can similarly be achieved by the use of *more stable choline* compiunds, i.e. ones more refractory to the disintegrating effects of the cholinesterase. Of these, the carbamic ester of choline, known also as *carbamycholine,* has proved to be of special interest also from the clinical aspect, while another preparation, *prostigmine,* although synthesised along different lines, exhibits characteristic cholinergic effects.

In mimicking normal parasympathetic action, these cholinergic compounds evoke increased motility of plain muscle especially of the digestive tract and pelvic viscera, widespread and profuse glandular secretion, while cardiac rhythm becomes depressed and peripheral vascular tone decreased.

2. *Clinical use of cholinergic compounds.*

Apart from their physiological interest, cholinergic drugs are being used in a progressively widening array of clinical conditions both in medical and in veterinary practice. This is due to the realization of the fact that while producing prompt physiological response in various organs, this is not generally associated with drastic action nor with any untoward cumulative after-effects as may be expected with other drugs. Moreover, the administration of atropine immediately blocks any undesired cholinergic action, thereby ensuring effective control over it.

While carbamycholine chloride was previously marketed only under the trade name of *Lentin* (Merck) it is at present also being manufactured by various other firms under the name *Carbachol* and is available both in the form of tablets intended for oral administration and as a stable solution suitable for subcutaneous or intramuscular injection. The same applies to prostigmine, which is manufactured by the Swiss firm Hoffman la Roche.

Although considerable experience has been gained both in Europe and America concerning the uses of these compounds in veterinary practice, the lack of supplies as a result of war conditions has prevented any extensive trials in South Africa, Investigations at Onderstepoort, although limited mainly to physiological considerations, have, however, been in progress for some years and further interesting data obtained on the comparative action of some of these compounds.

Before referring to these results, a brief summary will be given of records from overseas literature mainly in regard to the use of carbamylcholine on animals.

Cattle: Dose 2-8 mgrs. subcutaneously or intramuscularly, repeated at intervals of four hours or longer as required.

Indications: (1) As a ruminatoric in ruminal impaction, paresis, atony, and tympany. (2) Relief of foreign body obstruction in œsophagus. (3) Indigestion and constipation, loss of appetite. (4) As a diagnostic in foreign body gastritis. (5) As a uterine stimulant during calving. (6) In uterine atony, catarrhal endometritis, pyometra, expulsion of afterbirth, removal of rinsing fluids from atonic uteri; milk fever(?).

Horses: Dose as for cattle.

Indications: (1) Indigestion associated with loss of appetite. (2) Colic due to intestinal atony and tympany. (3) Impaction of colon and cæcum with constipation. (4) Foreign body in œsophagus (choke). (5) Chronic catarrhal metritis. (6) In urinary retention.

Pigs: Dose up to 8 mgrs.

Indications: (1) Stomach engorgement (to produce vomition). (2) Indigestion and constipation. (3) Induction of labour (repeated small doses). (4) Promotion of milk flow following parturition.

Dogs and cats: Dose up to 1 mgrm. for large dogs.

Indications: Constipation, emetic, stomachic, urinary retention.

Sheep and Goats: Dose up to 1 mgrm.

Indications: Ruminal paresis, constipation.

Contra indications: In all animals — advanced debility, cardiac weakness, bronchial catarrh, emphysema, acute lung conditions, late pregnancy(?), sweating in horses, foreign body gastritis in cattle except as a diagnostic, spastic conditions in plain muscle.

Prostigmin: Although the experience with prostigmine on animals is more limited, the general indications appear to be identical with those concerning the use of carbamylcholine, except that the doses recommended are approximately 2 to 3 times greater. In human practice prostigmine has been found in addition to promote contraction in skeletal muscle and hence its recommended use in myasthenia gravis.

Atropine sulphate in therapeutic doses can be relied upon as a specific antidote to cholinergic action and is recommended therefore wherever such action is to be controlled. For similar reasons it can be used to relieve naturally occurring spasm in the gastro-intestinal and uro-genital systems.

3. *Results of investigations at Onderstepoort.*

As indicated above, this work has been conducted primarily from physiological aspects, although various observations of clinical interest have also been recorded. For this purpose healthy animals were used throughout. The results obtained thus far can be summarized briefly as follows:—

A. — Sheep with permanent ruminal fistulæ: Motility of rumen and rectum graphically recorded before and after treatment.

(1) *Acetylcholine* 100 mgrs. intramuscularly caused prompt increase in ruminal rhythm and tone as well as in size of individual excursions, similar changes being noted in the rectum. Starting within 1 to 2 minutes after injection, these effects passed off again after 5-20 minutes without any signs of purgation. Marked salivation and bronchial secretion associated with coughing appeared in 1-2 minutes. Smaller doses of acetylcholine failed to evoke any definite response, probably as a result of its rapid destruction.

(2) *Carbamylcholine chloride (Lentin or Carbachol):* 1 to 1·25 mgrs. subcutaneously caused either *prompt* or *delayed* increase in ruminal tone, rhythm and force of contraction within 5 to 20 minutes after injection, such effects lasting for a period of 3 to 4 hours. Motility and tone in the rectum were likewise markedly increased even in cases where the rumen itself showed little change. This seemed to indicate some degree of selective action of carbamylcholine on different levels of the digestive tract. Associated with the above changes, repeated defæcation with progressive softening of the pellets finally resulted in outspoken purgation though only in certain animals and usually within 3 to 4 hours. With their highly efficient water absorption from the large intestine, sheep are difficult animals to purge normally and may require repeated doses of carbamylcholine to provoke full effects. In all cases profuse salivation and frequent urination appeared within 5-10 minutes and continued intermittently for 3-4 hours. Furthermore the pulse rate was decreased and slight superficial muscular twitches noted over various part of the body. With the disappearance of the above symptoms usually after 3 to 4 hours, all animals showed good appetite and appeared clinically normal in all respects. As shown in the graph, injection of atropine sulphate immediately blocked all responses initiated by carbamylcholine.

(3) *Prostigmin:* 2-3 mgrs. intramuscularly — increase in the size of ruminal contractions but without change in tone or rhythm — rectal motility also increased — purgation as with carbamylcholine — muscular shivering over body much more pronounced — salivation less evident.

B. — Cattle — full-grown and in normal health.

(1) *Carbamylcholine* (Lentin or Carbachol) — 10 mgrs. subcutaneously or intracuscularly. Marked salivation and frequent urination in 5-10 minutes. Purgation in 1½ to 2 hours.

(2) *Prostigmin.* 25 mgrs. subcutaneously — salivation slight, but marked muscular twitches in 5-10 minutes. Purgation in ¾ to 1½ hours.

C. — Horses — full-grown and in normal health.

(1) *Carbamylcholine* (Carbachol): (a) 5 mgrs. subcutaneously. Severe and continued salivation and sweating — depression of pulse rate — no purgation. (b) 2 mgrs. subcutaneously, repeated three times at 2½-hour intervals — slight salivation, no sweating, copious urination, no purgation.

(2) *Prostigmin.* 20 mgrs. subcutaneously — slight signs of abdominal cramp within 5 minutes, frequent defæcation leading to definite purgation one hour after injection and which continued for another one hour. Two hours after injection animal normal and feeding.

D. — Dogs — full-grown, varying in size, in normal health.

(1) *Carbamylcholine* (Carbachol) from 0·2 to 0·5 mgr. (± 1 mgr. per 100 lb.) subcutaneously. Prompt and continued salivation — retching and vomition — copious urination with emptying of bladder followed by continued dribbling of urine (bladder spasm) — repeated defæcation soon leading to watery purgation associated with straining (abdominal cramp); well-marked depression of pulse rate — nervous excitement followed by slight dullness. Above symptoms passed off within 1½ hours: appetite normal again.

(2) *Prostigmin.* 2·5 mgrs. per 100 lb. subcutaneously. Prompt retching and vomition — no increased salivation — repeated defæcation soon leading to watery purgation associated with straining and passing of mucus — urination prompt and copious — muscular twitches very slight — normal liveliness maintained throughout. Appetite not affected.

Summary.

From overseas publications as well as from investigations undertaken at Onderstepoort, there is ample evidence to show that both *carbamylcholine chloride* (Lentin or Carbachol) and *prostigmin* exert a well-defined cholinergic action despite certain minor differences, e.g. on salivary flow. As stimulants of the plain muscle of the digestive tract and bladder and especially as purgatives in different species of animals, both have been found to exert prompt and efficient action except in the case of horses in which carbamylcholine failed to produce the expected purgation.

In view of the prevalence of gastro-intestinal disturbances amongst animals in South Africa, especially of ruminal atony in cattle, and also of uterine complications after parturition, the use of these cholinergic compounds may in practice be found to fill a much-felt hiatus in regard to reliable and rational treatment. Before recommending the general use of these drugs, it would, however, be advisable to obtain further information about their action on animals under local conditions.

THE TITRATION OF THE BETA FRACTION OF THE TOXIN OF CLOSTRIDUM, WELCHII, TYPE B, BY THE FLOCCULATION REACTION.

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The beta fraction of the toxin of *Clostridum welchii,* Type B (which in this communication will be referred to as "toxin") is easily, accurately and quickly titrated *in vivo.* A few lethal doses, administered intravenously, kills the mouse or the guinea-pig in from 30 to 60 minutes, and an approximation of the L+ dose (intravenous injection in mice) can be obtained in the course of a working day, although an observation period of 24 hours is necessary to obtain an accurate end-point. Formol-toxoid, used to prevent lamb dysentery, may be titrated with some accuracy by ascertaining its antitoxin-binding power. In this test the toxoid is allowed to stand in contact with dilutions of antitoxin for one hour; free (unbound) antitoxin is determined by adding a known amount of accurately titrated toxin to each toxoid-antitoxin mixture, and after the lapse of another hour, injecting the whole mixture into mice. However, the true value of the toxoid is not necessarily obtained because toxin is, as a rule, more avid than toxoid and may throw some toxoid out of combination with the antitoxin and take its place, thus giving the impression that the toxoid has a lower binding value than it really has. However, it has been shown over the course of many years that the antitoxin-binding power of Type B toxoids, determined by this method, is a good index of antigenicity.

In 1936, one of us (J.H.M.) had the privilege of seeing Miss H. E. Ross of the Wellcome Laboratories, Beckenham, Kent, carry out a number of flocculation tests with Type B toxins. The Lf values assigned to the toxins agreed fairly closely with those obtained by the L+ test in mice. To the best of our knowledge, no communication has appeared in the literature. We decided to investigate the possibility of titrating toxins and toxoids by the flocculation method in the hope that the in vitro result would be at least as accurate as that obtained in vivo.

MATERIALS AND METHODS.

Strain: The so-called "1930" variety of *Cl. welchii,* Type B, i.e. the strain that has lost the power of producing the epsilon toxic fraction, was used for producing the one dry test toxin employed throughout and all the liquid toxins. A South African "bloed-

pens" strain, able to produce both the beta and epsilon fractions, gave origin to the formoltoxoids (anacultures).

Toxins: The toxins were made in meat-particle horse-flesh broth, rendered sterile by filtration through British Berkefeld candles or through Seitz pads. The dry toxin was an ammonium sulphate precipitate of such a toxin.

Toxoids: These were supplied by Dr. J. R. Scheuber of Onderstepoort as formolized whole cultures of the organism. They were clarified by centrifugation and Seitz pad filtration.

Antitoxin: Two horses were immunized against the toxin of the "1930" strain and the sera purified and concentrated by peptic digestion and ammonium sulphate precipitation. An enzyme-purified antitoxin was used because experience, particularly with diphtheria, has shown that such an antitoxin flocculates more easily with toxin than natural antitoxic serum. The antitoxin of one horse proved unsatisfactory, because of slowness of flocculation, and was discarded; that of the other horse gave good results and was used for all purposes (flocculation, L+ titrations, and estimations of antitoxin-binding power of toxoids).

L+ of toxins and antitoxin-binding power of toxoids: These tests were carried out by standard methods.

Immunization of guinea-pigs and test of immunity: Guinea-pigs weighing 350 g. received subcutaneously 1 cc. of toxoid followed in 28 days by a further 1 cc. After a further 14 days, they were bled from the heart, the serum from each group pooled and tested for the presence of antitoxin. For this purpose, the dry, stable, accurately titrated toxin was used and mice (intravenous injection) were employed as indicators of toxicity.

When the antitoxin titres of the serum pools had been obtained, the resistance of each guinea-pig to toxin injected intracardially was ascertained by the cumulative lethal dose method (Mason, 1935). In this test, advantage is taken of the quickly-killing power of Type B toxin. Every animal receives one certain lethal dose; an hour later 3 doses are injected into the survivors, then 4 doses into survivors and finally 8 doses. In an experiment planned to show up differences in the antigenicity of different toxoids, it makes no material difference if the toxin tolerance is recorded as the sum of the fatal doses injected or as the largest single dose injected.

Flocculation tests: In order to hasten the appearance of visible floccules, the tubes containing the toxin-antitoxin or toxoid-toxin-antitoxin mixtures were rocked gently whilst being held immersed in a water-bath at 45°C. It is generally known that agitation hastens precipitation (e.g. as applied in the Kahn test for syphilis) and we have shown that flocculation is accelerated three to six-

fold when this procedure is used with both *Cl. welchii*, Type A, and *C. diphtheriæ* toxin and their respective antitoxins (unpublished work).

Tubes containing the toxin and antitoxin were stoppered, laid flat on holders which were placed on a platform immersed in a water-bath. This platform was pushed to and fro by the moving arm of a windscreen wiper, activated by a water pump.

Toxins: To 1 cc. amounts, antitoxin was added with a Trevan microsyringe in increments increasing by between 10% and 15%. Fresh toxins flocculated in from 25 to 45 minutes. The floccules were small, flat, and of a dull-grey colour. The pH of the toxin was between 6 and 6·5; at pH 7 and over, "crystals," — small, bright, irridescent specks — appeared and blurred the true flocculation.

It was not uncommon for 3 adjacent tubes to flocculate within a few minutes of one another; however, it was usually possible to detect that which flocculated first and nearly always this was the middle tube of the three.

Toxoids: Even when shaken, the toxoids tested did not flocculate in 6 hours, so that recourse was had to blending with a quickly-flocculating toxin of known in vitro value. In these circumstances, visible particulation, view through a hand lens against an illuminated black background, was seen in from 45 to 90 minutes. In practice 1 cc. amounts each of the known-value toxin and of the toxoid under test were mixed and to the mixtures antitoxin was added.

Results.

In Table I, the actual amount of antitoxin neutralising 1 cc. of toxin by the flocculation test and the calculated amount neutralizing 1 cc. of toxin by the L+ method are recorded. The neutral points by both methods are in very close agreement.

Table 1.

Comparison of Lf and L+ values of toxins.

Toxin	cc. AT. neut. 1 cc. toxin in vitro	in vivo
1	0·006	0·005
2	0·013	0·012
3	0·016	0·016
4	0·016	0·015
5	0·012	0·012

(AT. = antitoxin; neut. = neutralizing)

Table 2 records the in vitro results for toxoids blended with toxins and the in vivo figures obtained by the antitoxin-binding power test.

TABLE 2.

Comparison of antitoxin-binding values of toxoids obtained by in vitro and in vivo methods (April, May, 1946).

Toxoid	Date of Prep.	cc. AT binding 1 cc toxoid in vitro	cc toxoid in vivo
13 a	April, 1942	0·003	0·0009
40	July, 1943	0·006	0·0014
41	July, 1943	0·001	0·00065
42	July, 1943	0·004	0·0009
43	August, 1943	0·007	0·0009
52 b	March, 1944	0·004	0·0011
78 d	March, 1945	0·01	0·004
92 d	August, 1945	0·01	0·0019
97 a	May, 1946	0·01	0·0026

(AT. = antitoxin; Prep. = preparation).

Both methods place the toxoids roughly in the same order, but it is clear that the in vivo test is detecting only a small fraction of the antigen. Probably the toxin, added to the toxoid-antitoxin mixture in the binding-power test, has thrown some toxoid out of union and taken its place.

A more detailed experiment was carried out, using toxoids 40, 41, 42, 43 and 78. Six guinea-pigs were immunized with each toxoid and the pooled serum from each group titrated for antitoxin content and, in addition, the resistance of each animal to toxin injected intracardially ascertained. Table 3 summarizes the results.

TABLE 3.

In vitro and in vivo values of toxoids, antitoxin response in immunized guinea-pigs, and resistance of guinea-pigs to toxin.

Toxoid	Value* in vitro	Value* in vivo	L.D. toxin neut. by 0.2 cc. serum	Cum. L.D. withstood by G.P. 1.	2.	3.	4.	5.	6.
40	0·006	0·0014	12	1-4	1-4	1-4	8-16	8-16	8-16**
41	0·001	0·00065	2	1-4	1-4	1-4	1-4	1-4	4-8
42	0·004	0·0009	7	1-4	1-4	1-4	1-4	4-8	8-16
43	0·007	0·0009	28	1-4	1-4	8-16	8-16	>16	>16
78	0·01	0·004	100	>16	>16	>16	>16	>16	>16

(*Value — see table 2; ** 1-4, 4-8, etc. = G.P. survived 1 lethal dose, but not a further 3, etc.; neut. = neutralized; G.P. = guinea-pig; cum. L.D. = cumulative lethal doses.)

A good indication of the antigenic value of a toxoid may be obtained by titrating the pooled sera of a group of immunized animals. However, a few unresponsive or a few very responsive individuals will upset the true answer. To obtain the correct answer the serum of each animal must be titrated or, as in this experiment, the resistance of each individual must be determined. The results in table 3 show that the in vivo antitoxin-binding value test is the poorest indicator of the true immunizing value of a toxoid, taking the toxin resistance of the guinea-pigs as the correct value. By Lf and by antitoxin response, the 5 toxoids were placed in the same order of value as by the toxin tolerance test.

We appreciate that the amount of work done is not sufficient to justify a switch-over to the exclusive use of the flocculation test for titrating toxins and toxoids, but as we, ourselves, are not engaged in preparing *Cl. welchii*, Type B. prophylactic vaccines we consider the results sufficiently promising to place on record. To obtain satisfactory in vitro results, we would stress the necessity of a quickly-flocculating enzyme-purified antitoxin and the use of a rocking machine.

Conclusion.

Toxins and toxoids of *Cl. welchii*, Type B. may be titrated by the flocculation test, when quickly-flocculating enzyme-purified antitoxin is used. To speed up particulation, the toxin-antitoxin or toxin-toxoid-antitoxin mixture should be rocked. Unblended with fresh toxin, the toxoids tested did not flocculate in 6 hours. The Lf of a toxoid agrees very closely with its antigenic power in guinea-pigs.

Reference.

Mason, J. H. (1935: The production of immunity against *Cl. welchii*, Type B, Wilsdon (The "Lamb dysentery bacillus"). *Onderstepoort J.* 5. 2. 391-416.

Acknowledgments.

We have pleasure in thanking our colleagues, Dr. J. R. Scheuber, of Onderstepoort, for supplying the toxoids, and Dr. Agerholm Christensen, of this Institute, for purifying the antitoxin.

A BRIEF NOTE ON VIBRIO METCHNIKOVI AND ITS OCCURRENCE IN NATAL.

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Allerton, Pietermaritzburg.

Reports on the occurrence of Vibrio infection among animals and birds in South Africa are few in number. Snyman (1931) described the first observed outbreak of abortion in South Africa, caused by Vibrio fœtus. A number of similar cases were observed by the senior author (A.S.C.) in Natal about 1940, and since that time further outbreaks have been diagnosed. The opinion is held that Vibrionic abortion in Natal is of fairly frequent occurrence.

Topley and Wilson (1938) make mention of many vibrios, among them being Vibrio metchnikovi, which was isolated by Gamaleia (1888a) from the blood and intestinal contents of chickens dying from a cholera-like disease at Odessa. It is stated that this vibrio has been isolated from water.

In October, 1945, twelve swabs were received from the Government Veterinary Officer, Eshowe, taken from birds suspected to be suffering from Newcastle Disease in the Amatikulu area. The swabs were made from the throats. The symptoms shown by these birds were dyspnœa, catarrhal discharge from the nose, diarrhœa, and partial paralysis in some cases. The post-mortem lesions were not definite; there was reddening of the pharyngeal mucosa and a slight reddening of the intestines.

The twelve swabs were divided into batches of three, and each batch was then washed off with about 5 cc. of sterile physiological saline. One cc. of the emulsion was then injected subcutaneously into a pigeon. Of the four pigeons used, three were dead the following morning, while the fourth had a profuse watery diarrhœa, but recovered in a few days' time and remained healthy. Post-mortem examination of these dead pigeons showed a very marked enteritis, together with an enlarged spleen. Cultures were made from the heart's blood and spleen on liver agar slopes. Incubation for twenty-four hours revealed four different types of colonies. They were:—

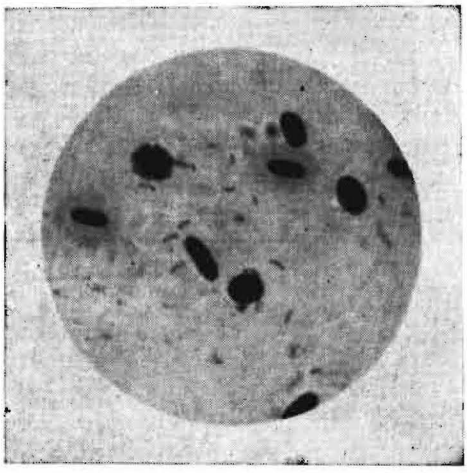
(a) a small white raised colony, organisms of which proved to be Gram positive staphylococci;

(b) a fairly large, raised greyish colony, organisms of which were long Gram positive bacilli;

(c) a slightly raised transparent colony, the separate organisms of which were Gram negative bacilli;

(d) fairly large brownish colonies. Smears made from these showed vibrio-like organisms.

Pure cultures were obtained from each of these four different types of colonies. A saline emulsion was made from each after twenty-four hours' growth, and two pigeons were utilized for each separate emulsion. They were injected by the subcutaneous route on 17th October, 1945. Within twenty-four hours only the two pigeons that received the



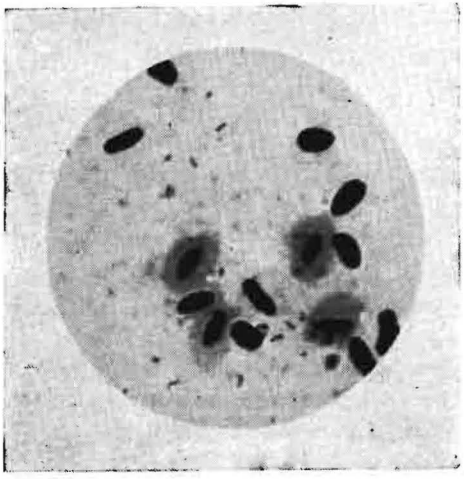
Comma forms showing bi-polar staining in blood of pigeon killed with V. metchnikovi.

vibrio-like organisms were dead, and from the heart-blood and spleen the same type of organism was regained in pure culture. This comma-shaped organism was also fairly frequent in smears made from the heart-blood.

Morphology: Slightly curved bacillus, resembling a comma, and varying in length from $2-4\mu \times 0{\cdot}2-0{\cdot}4\mu$. It is markedly pleomorphic in culture. Small, straight, rod-like forms are often present in young cultures, but spirillar forms appear more frequently in older cultures. It is a slightly larger organism than is Vibrio fœtus. The typical comma type with spirillar forms are obtained from cultures on blood agar poured plates. It is an exceedingly motile ærobe, which exhibits active darting movements. The comma and bacillary type are the most rapid forms. They can be seen to dart across the field of vision, and, if followed up, will be observed to become motionless for a moment only to dart off again. The spirillar forms move more slowly with a characteristic corkscrew movement. The movement of this organism is much more vigorous on the circumference of a drop of liquid than in the centre, where it is relatively sluggish. This is due to its oxygen requirements.

Cultural characteristics: (a) *Broth* — 24 hours at 37°C — there is a slight turbidity with a good growth. After 72 hours there is a heavy pellicle on the surface. A small portion of this pellicle, when placed on the surface of a flask of broth and incubated, covered the surface entirely in 24 hours. There is only a slight turbidity with it. The organisms, after 24 hours, show many straight or bacillary forms, but some fine comma forms were observed together with a few spirillar forms showing up to four curves. After 72 hours' growth the organisms do not stain well with methylene blue and appear to be degenerating.

(b) *Peptone water* — 24 hours at 37°C — there is a slight turbidity together with a slight pellicle. After 72 hours there is a heavy pellicle with turbidity. The organisms after 72 hours' growth show many straight or bacillary forms with some short, thick comma forms.



Comma forms showing bi-polar staining in blood of pigeon killed with V. metchnikovi.

(c) *McConkey's medium* — 24 hours at 37°C — two types of colonies were observed: (1) a small pin-point colony in which the organisms were mainly straight or bacillary in form; they were thick and some showed a slight bulge at one end; (2) a large colony, in which the organisms were mainly of the straight or bacillary form, some comma forms and some of the nature of long spirilla. Single colonies of each type were transplanted on to liver agar.

(d) *Brilliant green broth* — 24 hours at 37°C — only slight turbidity with poor growth. The organisms were largely of a fine comma type with some straight forms. After 72 hours there was a slight increase in the turbidity of the medium. The organisms had mainly assumed spirillar forms with comma and straight forms intermixed.

(e) *Brilliant green agar* — no growth for 48 hours and then only a few clear pin-point colonies.

(f) *Blood agar plates* (poured) — within 36 hours the whole plate appeared greenish due to hæmolysis. There was a continuous surface growth. Organisms from this culture were mainly comma forms, but numerous spirillar forms weer also present.

(g) *Blood agar* (streaks) — a fairly thick greyish growth after 24 hours at 37°C. Hæmolysis started from the edge of the growth after four days at room temperature. Organisms from this culture were mainly of the comma type.

(h) *Liver agar* — 24 hours at 37°C — thick, slightly brownish growth which became darker after 72 hours. Small daughter colonies grew on old cultures at room temperature. The organisms from this medium showed comma, bacillary and spirillar forms.

Staining — these organisms stained well with methylene blue and giemsa. They stained poorly with gram stain, and were gram negative.

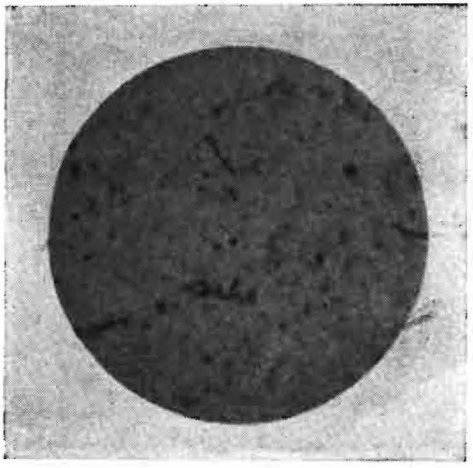
Biochemical characteristics: Fermentation tests using lactose, maltose, glucose and saccharose gave the following results:—

Lactose — negative after 10 days.

Maltose — acid, but no gas in 24 hours.

Glucose — acid and gas in 24 hours.

Saccharose — acid, but no gas after 3 days.



Spirillar, comma and bacillary forms of V. metchnikovi from cultures 72 hours old.

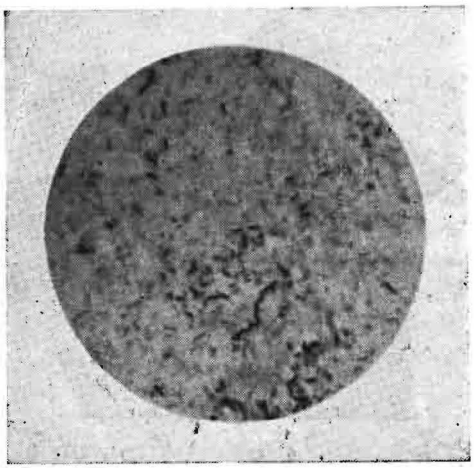
Indol reaction was positive and nitrates were reduced to nitrites.

Biological examination: The organism has both "O" and "H"

antigens. Against serum from fowls that had recovered from artificial infection the O antigen had a titre of 1 – 20 and the H antigen a titre of 1 – 40. 106 blood samples from fowls on the Amatikulu farm, from which the original swabs were collected, were tested against both O and H antigens, all with negative results. Controls were inorder.

Pathogenicity: (1) Between 17.10.45 and 28.10.45 twelve pigeons were injected subcutaneously with 1 cc. of an emulsion of the organism, the emulsion being $\frac{1}{4}$ of a loopful of culture to 3 cc. of a sterile physiological saline. They were invariably injected at 3.30 p.m., and except for two pigeons that survived, all the rest died within 15 hours of being injected. Smears from the heart-blood and spleen revealed many comma-shaped organisms — this was doubtless a septicæmia. Post-mortem examinations revealed markedly enlarged spleens, engorged livers, slightly inflamed lungs, markedly reddened mucous membrane of the anterior portion of the proventriculus, very acute catarrhal enteritis and congestion of the subcutaneous vessels of the pharyngeal region. The pectoral muscles of the injection site showed swelling, were of a light grey colour and were friable. The surviving pigeons showed a marked diarrhœa, but eventually recovered.

(2) On 23.10.45 three pigeons were given large doses of the organism per os. Two remained normal, but the third died on 26.10.45 after having shown a profuse diarrhœa. Cultures from the



Spirillar, comma and bacillary forms of V. metchnikovi from cultures 72 hours old.

heart blood of the dead pigeon yielded a pure growth of the comma organism. Post-mortem lesions resembled those previously described.

(3) On 29.10.45 an adult fowl was injected subcutaneously with

1 cc. of an emulsion of the organism. The emulsion used was ¼ loopful of culture to 3 cc. sterile physiological saline. The following day the bird showed a marked watery diarrhœa, was very weak, refused to eat and its feathers were ruffled. These symptoms were present for four days and then gradual recovery took place. On 8.11.45 the serum of this bird was tested against the ',O" antigen of the organism by the quick-slide method and proved positive.

(4) On 3.11.45 two cockerels of 3 months of age were each given a large dose per os of a suspension of the comma organism. These birds remained healthy.

(5) On 13.11.45 two adult guinea-pigs were injected subcutaneously at 3.30 p.m. with 1 cc. of a saline emulsion which was similar in concentration to that used on 29.10.45, when an adult fowl was infected. At 8.15 a.m. the next morning both showed prostration and coma. One died at 9 a.m. and the second at 10 a.m. Post-mortem examination showed a slightly enlarged spleen and enteritis. There was necrobiosis of the injection site. Slides made from the heart-blood showed numerous comma organisms. Cultures made from the heart-blood yielded a pure growth of the comma organism.

While work was being done on this organism that had been isolated from fowls from Amatikulu, a farmer from Sarnia, near Durban, sent in four Andalusian chickens of about two weeks of age for examination, suspecting B.W.D. infection. Post-mortem examination showed the feathers of the vent were soiled with a whitish excreta, the spleen appeared normal, but both the intestines and the lungs were somewhat reddened. Cultures were prepared from the heart-blood, and a comma-like organism was isolated. No salmonella organisms were present.

Fermentation tests of a pure culture of this comma-like organism yielded the same results as did the organism isolated from the Amatikulu birds with this slight difference. The Amatikulu organism took three days to acidify saccharose, while the Sarnia strain took only 24 hours.

With regard to their morphology, cultural characteristcs, staining and biological properties, this Sarnia organism was identical with the Amatikulu organism. Agglutination took place with positive serum of a bird infected with the Amatikulu strain and organisms of the Sarnia strain.

Pathogenicity: On 6th December, 1945, two young rabbits were injected subcutaneously, at 3.30 p.m., with 1 cc. of a saline emulsion of the Sarnia organism, the density of the emulsion being tube 6 and corresponded to about 6556 million organisms per cc. At the same time an adult rabbit received, subcutaneously, 1·5 ccs. of a similar emulsion of the organisms. At 8.30 a.m. on the following day both young rabbits were found stiff and cold, indicating that death had

taken place some considerable time previously. Cultures from the heart-blood yielded the organism in pure culture, and smears made from the same source revealed the presence of this comma-shaped organism. The adult rabbit showed nothing unusual and was discharged a week later, having shown no signs of disease.

Both the Amatikulu and Sarnia strains grew well and remained viable after a month, as was shown by sub-culture.

An emulsion of the Amatikulu strain was tested against positive serum from a case of Vibrio fœtus abortion with negative results.

Discussion.

To judge from the scanty references made to V. metchnikovi in veterinary literature, this organism is either of little importance or has not been encountered very frequently.

(1) Gamaleia (1888a) was the first to isolate this organism, which he found in the blood and intestinal contents of chickens dying from a cholera-like disease at Odessa.

(2) Van Heelsbergen (1929) gives an account of its pathogenicity for adult birds, young birds and guinea-pigs. These views are, for all practical purposes, similar to ours. With young rabbits, the Sarnia strain killed within 15 hours, but had no effect on adult rabbits.

(3) Topley and Wilson (1938) make mention of Deneke's Vibrio-tyrogenus, which is pathogenic for the guinea-pig and pigeon. This is distinguished from the organism under discussion by the fact that it does not give the cholera-red reaction.

(4) Reference is also made to Finkler-Prior's Vibrio proteus which resembles V. tyrogenus.

(5) Spanedda (1941) describes an unflagellated vibrio, obtained from a diseased pigeon. From its biological and pathogenic properties it was regarded as distinct from Metchnikoff's vibrio and has been named "V. columbæ".

Summary.

(1) A description has been given of a comma-shaped organism, which from its morphology, cultural characteristics, biochemical characteristics and pathogenicity, is indistinguishable from V. metchnikovi.

(2) It is pathogenic for pigeons, chickens, young rabbits and guinea-pigs.

(3) It was isolated from material obtained from two widely-separated areas in Natal.

(4) This is the first mention of V. metchnikovi in veterinary literature in South Africa.

(5) There was no cross agglutination of this organism with serum from a cow that was suffering from V. fœtus infection.

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A SURVEY OF BOVINE MASTITIS BASED UPON BREED SMEAR EXAMINATIONS.

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In a previous report (Pullinger, 1944) it was stated that, in the process of examining Breed smears made fairly regularly from the milk of 152 dairy herds over a period of 10 months, only 7·9% of herds failed to show gross signs of mastitis infection during the period of survey. This finding was so disturbing that it was decided to collect further data, with a view to:—

1. Confirming or disproving this apparent high incidence of bovine mastitis;
2. Ascertaining to what extent the Breed smear can be used for performing initial mastitis surveys;
3. Studying the seasonal fluctuations of mastitis incidence.

The data, from which the following analyses have been made, consist of Breed smear records of individual farm milk supplies coming into the Witwatersrand and Pretoria areas. When Breed smears were examined, a record was made if definite signs of mastitis were seen, and it is these results that have been analysed. In the case of the Witwatersrand series, milk samples at all times have represented the whole bulk sent in by farmers. In the case of Pretoria, the samples sometimes were representative of the whole bulk, and sometimes came from a single 10-gallon can picked at random from the whole consignment.

Definite signs of mastitis were taken as:—

(a) Typical mastitis streptococcal chains, associated with aggregations of pus cells;

(b) Enormous quantities of pus cells and pus aggregates associated with morphologically atypical streptococci, which had clearly undergone degeneration.

At no time was a diagnosis of mastitis based upon the presence of streptococci alone, though in certain stages of chronic infection very little pus formation occurs. Similarly, no diagnosis of mastitis was based upon a moderate increase in the leucocyte count. In Breed smears prepared with milk from healthy udders it is rare to find a leucocyte in every microscopic field (using the oil-immersion objective), and it is fairly certain that a leucocyte Breed count of 1,500,000 per cc. indicates the presence of udder disturbance amongst the cows producing the milk. In spite of this, in the present survey, mastitis was only diagnosed on the grounds of an innumerable leucocyte count combined with phagocytosed streptococci. Furthermore, in the examination of smears, no attempt was made to make a detailed search for signs of mastitis.

Smears giving the low Breed counts were fairly carefully examined for mastitis in the process of making the bacterial count, but in samples which gave an innumerable Breed count (a state of affairs that was all too prevalent during the survey, *vide* Pullinger, 1945), the examination for mastitis was most cursory, and confined to passing the smear rapidly across the microscopic field. It will be seen, therefore, that the mastitis figures recorded in this analysis refer only to very gross admixture with mastitis milk, and all percentages of contamination must be regarded as minimal figures.

Results.

An analysis is given in Tables 1 and 2, showing monthly the percentage of producers who showed signs of mastitis in the bulk milk, and the percentage of samples which, on examination, showed mastitis contamination. Table 1 refers to the Witwatersrand area, and Table 2 to the Pretoria area. It will be seen that the monthly incidence of producers showing mastitis varied in the Witwatersrand area between 5·5 and 66·5 per cent, whilst in the Pretoria area these figures ranged from 17·5 to 100 per cent. In other words, during July, 1945, every producer under survey in the Pretoria area showed signs of mastitis infection in his milk, whilst in April, and again in October of 1943, 66·5 per cent. of producers in the Witwatersrand showed signs of their herds being heavily infected. These figures, which have been collected from the repeated testing of 435 producers, involving the examination of over one hundred thousand smears,

amply confirm the earlier finding regarding the high instance of bovine mastitis amongst South African highveld herds.

In Tables Nos. 3 and 4, a record is given, for the two areas, of the degree to which producers showed mastitis contamination of their supplies. These tables give the percentage of farmers who showed no contamination, and the proportion of farmers whose samples showed various percentages of mastitis contamination. Thus, in the Pretoria area in 1942, 11·5 per cent. of the producers showed no mastitis contamination. In the case of 6·5 per cent. of producers, 1 per cent of their samples showed this contamination, whilst 8 per cent of all the Pretoria producers showed mastitis contamination in 21 to 30 per cent. of all their supplies. The figures in Tables 3 and 4 show that, in general, a producer comparatively infrequently shows mastitis contamination in his bulk milk, and consequently no satisfactory survey could be made of mastitis incidence on a basis of occasional Breed examinations. If, however, such examinations are repeated fairly regularly throughout the year, a fairly accurate mastitis picture can be compiled.

Out of the whole series of figures, 5·8 per cent. of 436 Witwatersrand producers and 6·8 per cent. of Pretoria producers failed to show mastitis contamination at some time. The significance of these negative findings has been examined in Table 5, from which it will be seen that, of all these cases, only one producer was tested a significant number of times. All the others were tested for less than the average number of tests per producer, and the probability is that, had they been tested more often, some at least of them would have shown mastitis signs.

As regards seasonal fluctuations of mastitis, the figures obtained for the first year suggested that this incidence might reach a peak in autumn, and again in spring. A more detailed examination of these fluctuations over several years of results is set out in graphical form. In Graphs 1 and 3, the percentages of mastitis contaminated samples are shown, whilst in Graphs 2 and 4 the percentages of producers showing mastitis are set out. From these graphs it would appear that there is no seasonal factor to account for mastitis flare-ups which were noted.

Summary.

1. It has been confirmed that there is a very high incidence of mastitis amongst dairy herds situated on the highveld. Only from 1·8 to 6·7 per cent. of producers showed no signs of mastitis during the course of several years' survey. Of such producers, only one was subjected to a reasonable number of tests.

2. The Breed smear is useful for carrying out a bovine mastitis survey, provided that Breed smears are examined regularly throughout the year. Occasional Breed smears are useless for survey purposes, because mastitis infection fluctuates in incidence from time to time.

3. The Breed smear is a reliable and rapid way of locating a sudden and gross admixture of mastitis milk to any bulk supply that has been under regular test.

4. The fluctuations in intensity of mastitis infection have been clearly demonstrated in the foregoing data, but it has not proved possible to correlate such fluctuations with climatic or seasonal factors.

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TABLE No. 1.

Monthly and yearly distribution of mastitis contaminated milk, in a survey covering 3 years, with 75,863 samples from 346 Witwatersrand suppliers.

Nature of Data.	Year	Month. Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sep.	Oct.	Nov.	Dec.
No. of producers tested	1943	—	—	—	138	141	146	141	140	135	116	137	132
	1944	131	127	242	236	244	234	233	235	241	242	217	205
	1945	235	213	190	210	205	192	163	172	212	199	221	217
AVERAGE		183	170	216	195	197	191	179	182	196	186	192	185
No. of samples examined monthly	1943	—	—	—	2382	2351	2357	2444	2184	1976	1714	1920	1879
	1944	1750	1695	4066	3176	3748	3697	3368	3496	2467	2388	2712	2202
	1945	2723	1742	2084	2239	1986	1949	1819	1409	1271	949	2761	954
AVERAGE TOTAL		2237	1719	3075	2932	2695	2668	2544	2363	1905	1684	2464	1678
Percentage of producers showing mastitis	1943	—	—	—	66.6	61.7	26.7	26.9	17.1	28.9	66.4	24.8	21.2
	1944	22.9	6.3	19.4	23.3	27.9	56.4	45.9	46.3	25.3	35.5	34.1	28.8
	1945	11.9	5.6	13.2	12.9	33.7	23.4	36.2	35.5	34.4	38.7	60.6	57.1
AVERAGE		17.4	6.0	16.3	34.3	41.1	35.5	36.3	33.0	29.5	46.9	39.8	35.7
Percentage of mastitis contaminated samples	1943	—	—	—	12.8	10.9	3.5	3.9	2.4	6.1	13.8	4.1	2.6
	1944	2.2	0.8	1.6	2.7	3.6	10.3	6.2	7.7	4.5	6.1	5.4	5.8
	1945	1.6	1.8	1.9	1.9	6.2	4.5	5.7	7.8	9.3	16.1	12.9	24.8
AVERAGE		1.9	1.3	1.8	5.8	6.9	6.1	5.3	6.0	6.6	12.0	7.5	11.1

TABLE No. 2.

Monthly and yearly distribution of mastitis contaminated milk, in a survey covering 4 years, with 34,297 samples from 89 Pretoria suppliers.

Nature of Data.	Year	Month. Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sep.	Oct.	Nov.	Dec.
No. of producers tested	1942	—	42	42	42	45	48	49	47	46	47	50	53
	1943	50	46	43	44	44	43	46	45	44	44	42	41
	1944	39	40	38	41	41	36	35	38	37	37	41	43
	1945	44	43	41	39	36	35	33	35	32	—	—	—
AVERAGE		44	43	41	41	41	40	41	41	40	43	44	46
No. of samples examined monthly	1942	—	276	881	623	764	921	961	848	889	895	937	1022
	1943	929	876	945	868	830	704	838	877	883	892	879	868
	1944	721	768	851	716	784	733	661	576	669	694	709	628
	1945	867	768	772	751	632	683	686	649	518	—	—	—
AVERAGE TOTAL		839	670	842	740	753	760	787	738	740	827	842	839
Percentage of producers showing mastitis	1942	—	61.9	72.9	64.3	62.2	77.1	48.9	40.4	43.5	40.4	50.0	47.2
	1943	58.0	58.7	55.8	44.5	43.2	44.2	30.4	46.7	70.5	29.5	23.8	24.4
	1944	28.2	17.5	34.2	58.5	56.1	47.2	80.0	68.4	59.5	67.6	85.4	95.4
	1945	81.8	67.5	90.2	69.3	94.4	68.6	100.0	68.6	71.9	—	—	—
AVERAGE		56.0	51.4	63.3	59.2	64.0	59.3	64.9	56.0	61.4	45.8	53.1	55.7
Percentage of mastisis contaminated samples	1942	—	21.0	16.7	18.8	12.9	13.1	8.4	5.1	6.9	5.0	6.4	6.5
	1943	11.8	9.8	11.5	9.2	7.5	6.7	4.4	5.5	9.6	2.9	2.7	2.6
	1944	3.9	3.3	3.1	7.9	9.3	5.6	20.4	14.9	10.6	9.5	21.6	25.8
	1945	11.9	11.7	15.9	7.8	15.0	9.2	26.4	7.1	8.5	—	—	—
AVERAGE		8.5	11.5	11.8	10.9	11.2	8.7	14.9	8.2	8.9	5.8	10.2	11.6

TABLE No. 3.

Distribution of mastitis contaminated samples amongst producers, Witwatersrand area.

YEAR	1943	1944	1945	THROUGHOUT FULL PERIOD OF SURVEY.
No. of producers tested	156	279	287	346
No. of samples tested	19217	34765	21881	75863
Proportion of samples containing mastitis milk. [Expressed as a Percentage Group]	*Percentage of producers whose samples fell into the mastitis contaminated groups as shown.*			
	1943	1944	1945	THROUGHOUT FULL PERIOD OF SURVEY.
0	7·0	15·1	19·3	5·8*
1	15·5	11·8	7·7	9·5
2	10·9	15·8	11·2	13·1
3	13·6	11·1	10·0	11·4
4	9·0	11·8	7·4	10·7
5	7·0	7·9	4·9	8·7
6	2·6	2·9	4·9	5·8
7	3·8	3·6	6·4	4·9
8	5·8	3·9	2·8	4·6
9	3·2	3·6	3·5	5·8
10	3·8	2·9	3·8	2·4
11	2·6	1·4	1·0	2·0
12	0	1·1	2·1	2·0
13	0·6	1·8	2·1	1·4
14	1·9	0·4	1·4	0·9
15	1·9	1·1	1·4	1·2
16	1·3	0·4	2·1	2·0
17	1·3	0·7	0·7	1·2
18	1·3	0·7	0·7	1·2
19	0	0	0·3	0·3
20	1·3	0·4	1·7	0
21 to 30	2·5	1·6	3·3	3·0
31 to 40	2·5	0·4	0·3	0·9
41 to 50	0	0	0·3	0·3
51 to 60	0	0	0	0
61 to 70	0·6	0	0·3	0·6
71 to 80	0	0	0·3	0·3
81 to 100	0	0	0	0

* For detailed analysis of the significance of this result see Table No. 5.

TABLE No. 4.

Distribution of mastitis contaminated samples amongst producers, Pretoria area.

YEAR.	1942.	1943.	1944.	1945.	THROUGHOUT FULL PERIOD OF SURVEY.
No. of producers tested	61	66	55	47	89
No. of samples tested	9117	10389	8464	6327	34297

Proportion of samples containing mastitis milk [Expressed as a Percentage Group]	*Percentage of producers whose samples fell into the mastitis contaminated groups as shown.*				
	1942.	1943.	1944.	1945.	THROUGHOUT FULL PERIOD OF SURVEY.
0	11·5	16·8	7·3	4·3	6·7*
1	6·5	7·6	0	0	5·6
2	13·1	9·1	5·5	2·1	2·2
3	4·9	10·6	3·6	2·1	8·0
4	4·9	7·6	9·1	2·1	8·0
5	4·9	9·1	1·8	6·4	6·7
6	6·5	7·6	7·3	2·1	3·4
7	3·4	4·5	1·8	6·4	8·0
8	8·2	3·0	0	4·3	9·0
9	1·6	3·0	7·3	8·5	6·7
10	3·4	6·1	5·5	6·4	4·5
11	4·9	1·5	3·6	14·9	3·4
12	1·6	0	5·5	2·1	3·4
13	1·6	0	5·5	4·3	5·6
14	0	1·5	5·5	4·3	2·2
15	1·6	1·5	3·6	4·3	2·2
16	3·4	0	1·8	0	2·2
17	1·6	1·5	5·5	4·3	1·1
18	3·4	0	0	4·3	1·1
19	0	0	1·8	2·1	0
20	3·4	3·0	3·6	2·1	0
21 to 30	8·0	3·0	9·0	6·3	7·8
31 to 40	0	1·5	5·4	6·3	0
41 to 50	0	1·5	0	0	1·1
51 to 60	0	0	0	0	0
61 to 70	1·6	0	0	0	1·1
71 to 100	0	0	0	0	0

* For detailed analysis of the significance of this result see Table No. 5.

TABLE No. 5.

Analysis of records of producers whose tests were entirely free of mastitis contamination.

Area.	Producer No.	No. of months under test.	No. of samples tested without showing mastitis contamination.	Standard for significance.
WITWATERSRAND AREA.	1	21	202	An average of 105 tests were examined per producer in the Witwatersrand area. Negative results from producers examined 105 or more times are classed as significant, but when tests fall below this figure, negative findings must be ignored.
	2	10	91	
	3	13	83	
	4	11	79	
	5	15	73	
	6	10	66	
	7	10	54	
	8	3	51	
	9	3	43	
	10	10	34	
	11	8	31	
	12	4	29	
	13	3	28	
	14	4	27	
	15	3	25	
	16	4	25	
	17	4	25	
	18	4	24	
	19	6	22	
	20	2	12	
PRETORIA.	21	7	131	Average of 153 tests (See above).
	22	6	72	
	23	7	68	
	24	4	65	
	25	2	10	
	26	2	5	

REMARKS.

Producer No. 1: This producer was consistently negative over a significant number of tests.

Producers No. 2 and No. 3: These producers were consistently negative in 91 and 83 tests respectively. These figures approach the average of 105 and are possibly significant.

Producers No. 4 to 20: These producers cannot be considered significant owing to insufficient tests.

Producer No. 21.: This producer was consistently negative in 131 tests. This figure approaches the average of 153 and is probably significant.

Producers No. 22 to 26: The producers cannot be considered significant owing to insufficient tests.

CANKER OF THE FOOT OF THE HORSE: A NOTE ON TREATMENT.

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A lengthy list of drugs and methods used for treating canker could be compiled, indicating that there is no specific treatment for the disease. In South Africa, statements varying from a refusal to treat any but mild cases to ability to affect a cure in from three to six weeks have been volunteered to me. I suggest that something midway between these two divergent views meets the case in practice. It is not practical to rest a horse worth £15 for six weeks and charge £10 for treatment; on the other hand it may be well worth while to persevere for even six months when the animal is valuable. When once treatment has been decided upon, dogmatism on the outcome should be avoided and a guarded prognosis offered until improvement is evident. Not infrequently a severely affected foot is cured with a minimum of treatment, whereas an apparently slight affection demands weeks of painstaking attention.

It is frequently stated that bad stable management — failure to clean out and inspect the feet daily and to keep the stable clean — is possibly or probably the cause of the trouble. Rather would I say that attention to these details would prevent the disease getting a hold. Daily foot inspection is a very desirable objective, but almost impossible of attainment in a stud where the grooms are a moving population of inexperienced Africans with little interest in horses.

It is commonly said that the coarse phlegmatic type of horse is more prone to the disease than the animal with some thoroughbred admixture. Although many experienced observers hold this view, and in spite of the apparent truth of the statement, it is just possible that the poorly-bred horse is usually owned by that type of individual who does not devote so much care to his animal as does the owner of a thoroughbred. There have been so few thoroughbred, or partly thoroughbred, horses in the 600 serum-producing animals that have passed through my hands in the past six years that I am unable to offer an opinion. But if by "phlegmatic" is meant docile, then I can say that this does not hold; the temperament of the horse appears to have no connection with proneness to the disease.

My approach in treatment differs from that of a practitioner in that the serum horses are unshod, are not worked, and I have adequate assistance and facilities, and have not to consider time and cost. Further, the value of a serum horse bears no relation to its working abilities; the value of a "crock," because of its antitoxin-producing power, may approach that of the race horse.

For the first few days, I content myself with cleaning and disinfecting the diseased area, by the cautious use of the knife and the application of acriflavine or proflavine (1:1000). The antiseptic is applied on cotton wool packed in tightly, held in position first with hessian, and then with a leather covering encasing the whole hoof. This "boot" is made from sun-dried salted horse hide; a circular portion is cut out, soaked in water to soften it, "Vs" cut out so that it will fit around the pastern and slots cut to take a riem or cord. Such a water-tight boot will last for from two to seven days.

When the affected portion of horn has lost its soggy appearance, the knife may be used more freely and an attempt made to expose the junction of the diseased and healthy horn. However, a hole should not be dug; the healthy horn should be pared right down to the quick for some distance from the cankered part. A firm paste, consisting of zinc oxide (2 parts), kaolin (2 parts), boracic acid (2 parts) and glycerine should then be applied liberally. This dressing should be renewed, at first daily, and later every two or three days, and on each occasion as much diseased horn as conveniently possible should be removed. I have not found it necessary to be radical at an early stage, as sometimes so-called "thorough" methods are contra-indicated. Blood obscures the area, and the horse becomes frightened and restive, making subsequent dressings tedious and irritating to the operator.

Progress depends in great measure upon the extent of the disease at the beginning, but if the wall is not affected, definite improvement can usually be expected in a fortnight. However, at all times, reliance should not be placed upon a cursory glance at apparently healthy new horn. It is essential to pare and probe to make certain that such horn is not underrun with spongy horn.

Only when it is apparent that cure is in sight may the drying-up process be hastened by applying a mild astringent, and for this purpose I should use picric acid, but I am not convinced that it has much advantage over the glycerine paste. Caustics, irritants, or powerful astringents such as nitric acid, formalin, and the sulphates of copper, iron and zinc are, in my experience, definitely contra-indicated. A coagulum is formed over the cheesy horn giving an appearance of firmness; the removal of this film reveals the spongy horn more inflamed than before.

If, during treatment with the glycerine paste, the horn again becomes soggy and purulent, recourse should be had to acriflavine dressings for a few days or acriflavine may be incorporated in the paste. At the present moment, I am testing the effect of acriflavine in the paste, right from the start of treatment, with, apparently, good results.

I am unable to offer an opinion on the effect of the exhibition of arsenic, because I combined this form of treatment with that just

described. However, there was no indication that arsenic hastened recovery.

On two occasions I attempted to perform the radical operation where the sole, frog and wall were affected. These animals, gifts to the Institute ,had been affected for a considerable time, and most of the diseased foot was a raw, bleeding, fungoid mass. A successful outcome was not obtained in either case, because, probably, as the autopsy showed, the wall was diseased right to the coronet, and a truly radical operation would have demanded the removal of the complete horny foot. A feature was the remarkable speed of growth of new diseased horn; there appeared to be as much of this four days after the operation as before it. With the exception of these two severely affected feet, I have been able, by mild simple treatment, to effect a cure in the 20 cases I have tackled during the last six years.

To the already long list of drugs used for treatment,I could add more, not noted in the text books I have consulted. However, I will make mention only of the following, all of which failed or were inferior to the glycerine paste. In an attempt to remove the diseased horn by non-surgical methods, a culture of the proteolytic anærobe, Clostridium sporogenes, was applied, and with the same object in view papain powder was dusted on thickly. The spongy horn was not digested by either. In a few instances silver proteinate appeared to have a beneficial effect, but this could not be maintained. Without sustained good effect were salt and acriflavine (1 : 1000), glycerine and acriflavine (1 : 1000), Lugol's iiodine solution, liq. picis carbonis, tannic acid, the triple dyes (acriflavine, brilliant green, and gentian violet), penicillin (either the crude filtrate from the mould or the mould plus the filtrate), and cod liver oil.

I consider it an advantage to treat the unshod foot because of the easy and unrestricted access to all parts. When a horse must be worked and therefore shod, the shoe should be as narrow as possible and pains should be taken to ensure that the foot is made inaccessible to moisture.

Summary.

Canker of the horse's foot may often be cured by minor surgery, cleanliness, antiseptics, and the application of a bland paste consisting of glycerine, kaolin, zinc oxide, and boracic acid. Caustics, powerful astringents and protein precipitants are contra-indicated.

Acknowledgment.

I have pleasure in thanking my Onderstepoort colleague, Dr. B. S. Parkin, for recommending the glycerine paste as a "general purposes" ointment.

CYSTICERCOSIS IN CALVES IN NATAL.

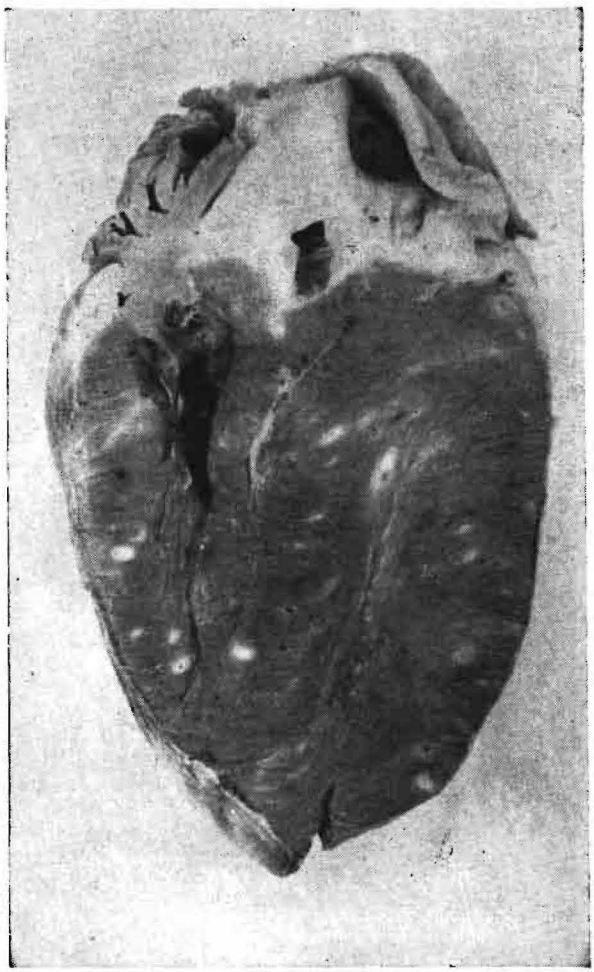
A. S. CANHAM,
Allerton.

During the last few years Cysticercosis of cattle has been repeatedly diagnosed in both adult and young cattle at the Pietermaritzburg Abattoir. Viljoen (1937) claimed that the incidence of Cysticercus bovis was not high in South Africa, but qualified it by stating that the extent of infection may, of course, be considerably higher than

External surface of heart of three-weeks-old calf showing Cysticercus cysts.

is anticipated, due mainly to the fact that calves are seldom slaughtered after six weeks of age. He then goes on to quote a letter from the Manager of the Pietermaritzburg Abattoir, who stated that one aspect of measles infection at his abattoir was the number of calves found to be infected. The following figures were then given:—

Year.	Calves slaughtered.	Number infested.	Percentage.
1931-32	559	31	5·54
1932-33	552	34	6·15
1933-34	670	37	5·52
1934-35	624	28	4·48
1935-36	673	47	6·98



Cross-section of same heart showing Cysticercus cysts deep in myocardium.

Quoting from the Annual Report of the Medical Officer of Health, Pietermaritzburg, for 1944 to 1945, there were 3,682 calves slaughtered, of which 3·69% were condemned for measles.

Recently, a number of young calves under the age of six weeks were condemned for the presence of measles, and this resulted in a heated controversy between the owners of the calves and the Abattoir Manager. Some of the owners produced Viljoen's article and other meat inspection works and, presumably, misreading the statements made by these authors, claimed that calves under six weeks could not be infected with measles.

To satisfy himself the Abattoir Manager brought specimens of organs of a three-weeks-old calf to the laboratory. The age had been readily given by the owner. A photograph was taken of the heart, showing lesions of Cysticercosis. A week or so later a newly-slaghtered calf was brought in by the manager. Dr. Kaschula carried out a post-mortem examination of this calf and stated that its organs and muscles were grossly infested with measles. Photographs were taken of the heart and lungs. This was also a three-weeks-old calf. The youngest age at which measles was diagnosed in a calf at the Pietermaritzburg Abattoir was ten days.

Sandig (1924) and Haas (1928) recorded cases of intrauterine infection in calves. Haas (1928) also described a case of generalized measles in a calf three weeks old.

This brief note is written for the purpose of drawing attention to the young age at which calves can become infested with Cysticercus bovis.

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Case Report :

DIABETES MELLITUS IN A DOG.

R. PAINE AND B. T. PAINE,
Pietermaritzburg.

Subject: Male Alsatian dog, 4½ years old. The animal was first examined on 5.6.46. The temperature was 102·4°F, slight anæmia was noted, condition poor. A blood smear was negative. The owner had treated the animal for internal parasites three weeks previously. An iron tonic was prescribed.

On 13.6.46 the dog was treated for worms and passed a few hookworms. It was not seen again until 13.8.46, when it showed a much more marked loss of condition and extreme lassitude. There was an increased thirst and excessive rumination. Treatment was again given for intestinal parasites with negative results.

As a positive reaction was obtained with the Sheftel test, the animal was destroyed and specimens from the pancreas were sent to Onderstepoort for examination. The organ was approximately 11 inches in length by 1¼ inches in width and half an inch in depth. It weighed 2 ozs. and 3 drams (62 grams). Thanks are due to Dr. K. C. A. Schulz for the following report on the specimen: ', It would appear that degenerative changes had occurred in the islands of Langhans and that the cells of the parenchyma were fairly active."

Case Report :

EMERGENCY TREATMENT OF A CASE OF MILKFEVER IN A COW.

A. J. LOUW,
Potgietersrust.

Recently I was called out to see a cow that had " gone down " about ten hours after calving. Unfortunately I was unable to attend till about 24 hours after the first symptoms had been noticed. I found the cow, a grade Friesian, in an extreme state of exhaustion, bloated and prostrate with ruminal contents oozing out from the nose and mouth.

Milk fever was diagnosed after a cursory examination, and the udder hurriedly inflated with a bicycle pump. The bloat was relieved

with a trocar and canula, the canula being left in situ while I hurried to a chemist to get the usual calcium-gluconate solution dispensed. Unfortunately, none of the two local chemists had any calcium-gluconate or a chemically pure soluble calcium salt in stock. The only calcium preparation on hand was a proprietary preparation called " Calsuba," a compound of the calcium salts of lactic and glycerophosphoric acids and usually prescribed by the medical profession to infants and pregnant mothers as an oral administration. Two ounces of " Calsuba " and eight ounces of glucose was dissolved in a litre of distilled water.

On returning, the cow appeared to be worse, with the eyelids showing hardly any reflex reaction. The whole litre of " Calsuba "-glucose mixture was slowly run into the jugular vein, there being a marked improvement noticeable while the mixture was being administered as she defæcated and took an interest in her calf. Twenty minutes after the last drop was run in she got up, urinated and drank a copious amount of water.

The usual instructions concerning the after-care was left with the owner and two days later he phoned me to say that she had made an uneventful recovery and was giving three gallons of milk a day in addition to feeding her calf.

Discussion.

I thought this case might be of interest to some colleagues, who, like myself, are stationed in a beef-breeding area and very seldom have to attend to a case of milk fever. As calcium-gluconate is very seldom prescribed, it is not uncommon for small-town chemists to be out of stocks, whereas " Calsuba " is very easy to procure and may probably be found in many a housewife's medicine chest.

A PIONEER OF VETERINARY SCIENCE IN SOUTH AFRICA: JOHN WILLIAM PHILLIPS (8th March, 1875 — 31st October, 1942).

In 1933, an article appeared in the *South African Medical Journal* (Curson, 1933) drawing attention to the services rendered to Veterinary Science by certain medical men, among whom were Robert Koch, Kohlstock, George Turner and W. Kolle. Later, a tribute was paid to Theiler for his remarkable work in combating the rinderpest epizootic of 1896-1903 (Curson, 1936).

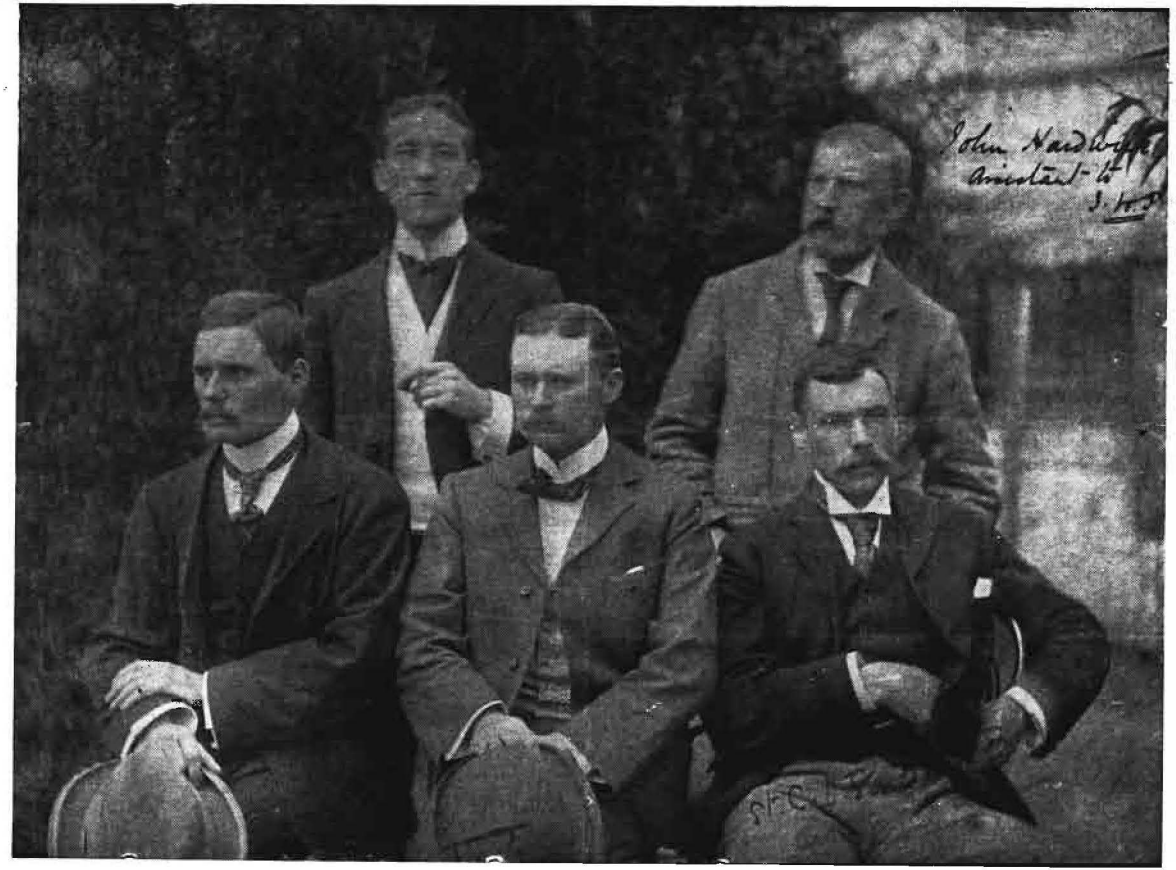
It is now fitting to add a note on the career of the technical assistant whose interest, accuracy and administrative ability contributed largely towards the advances recorded at the time, i.e. 1896 to 1903.

Phillips, the eldest son of a farmer at Wykin, Hinckley, England, arrived at Cape Town in November, 1896, soon after the Rinderpest Conference held at De Aar on 27th October, 1896. Within a few days he had received an appointment in the Cape Agricultural Department as junior assistant on the staff of Professor Koch who, with Dr. Kohlstock (and their respective wives), arrived at Cape Town on December 1st to investigate the scourge. As is generally known a laboratory was established at the Victoria Compound, Kimberley and the medical workers were assisted by Messrs. Otto Henning and J. O. O'Donoghue of the Cape Veterinary Service. Phillips assisted Koch for three months, when the former proceeded to India to study plague and Kohlstock left for "German" South West Africa. Before Koch's departure in March he placed young Phillips in charge of the laboratory, his special duty being to prepare rinderpest serum for the British South Africa Chartered Company on the lines evolved by the savant soon after his arrival.

Koch was succceeded by Dr. George Turner, Medical Officer of Health, Cape Colony. His professional assistant was Dr. W. Kolle, but the administration of the laboratory and the undertaking of all the experiments was left to Phillips. A photograph of the European staff at the time (Dr. Turner being absent) is shown in Figure 1.

In July, 1898, the Laboratory was transferred from the Cape Government to the Rhodesian authorities and Phillips was placed in entire charge. For three months' operations the profit on the sale of serum, chiefly to the Egyptian Government, was £9,000 and the officer-in-charge was rewarded with a handsome bonus.

Subsequently Phillips proceeded to Egypt, " the Egyptian Government having applied through His Excellency the Governor, for your services in connection with the establishment of a Manufactory in



GROUP OF RINDERPEST STAFF AT VICTORIA COMPOUND, KIMBERLEY, 1897.

Standing: E. STRATFORD PIPKIN; J. HARDWICK.
Sitting: J. W. PHILLIPS; DR. W. KOLLE; ST. C. O. SINCLAIR.

Egypt for the preparation of Rinderpest Serum" (letter dated 21st March, 1900, from Under Colonial Secretary — Noel Janisch — to J. W. Phillips, Bacteriological Laboratory, Grave St., Cape Town). After his work in Egypt (and incidentally the Sudan as well), the subject of our note proceeded to England via the Continent on long leave. He returned to Cape Town on January 8th, 1901.

Then occurred probably the next momentous period in his career, for at the end of May he was offered posts by both the Rhodesian and Transvaal Governments. Dr. Turner had in the meantime been appointed Medical Officer of Health for the Transvaal, and realising the value of Phillips' services, recommended to Lord Milner "that Mr. Phillips should be placed at the Leper Asylum, as lay superintendent .... (because) he is not only a very capable man of business, accustomed to farm operations and stock, but that he had a good knowledge of the manufacture of Rinderpest serum" (Turner 1902).

In the circumstances it is not surprising that Phillips accepted the post offered by his old chief. The conditions of service were a salary of £500 per annum, quarters and rations.

In January, 1903, Phillips married Aletta, daughter of Col. I. P. Ferreira, C.M.G., and in 1917 retired the Civil Service.* He settled at Port Alfred and became Secretary of the Royal Port Alfred Golf Club where he was well respected.

In conclusion, it is clear that Phillips was an outstanding character. Although a mere lad and stranger he accepted a post foreign to his training. Within a few months, by his interest, initiative, reliability and business acumen, he had gained the confidence of his seniors and established a reputation of which any man could be proud. It is gratifying to know that his son, conscious of his duty to the State as was his father, has just returned from a P.O.W. camp and is resident at Maseru.

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* Thanks are due to Major G. Tyiden, P.O. Commissie Poort, O.F.S., for material on John Phillips. Indeed the documents have been despatched to Dr. P. J. du Toit, Director of Verterinary Services, P.O. Onderstepoort, for safe-keeping in the Library of the South African Veterinary Medical Association.

THE CHEMICAL COMPOSITION OF MILK PRODUCED ON THE SOUTH AFRICAN HIGH-VELD FOR SUPPLYING THE FLUID MILK TRADE.

E. J. PULLINGER.

Municipal Abattoir and Livestock Market, Johannesburg.

In an earlier report (Pullinger, 1944) attention was drawn to the unsatisfactory quality of the Witwatersrand Milk Supplies in regard to the content of butter-fat and of nonfatty solids. An objection was raised against the validity of the conclusions drawn, on the grounds that the figures quoted had all been collected during the spring and early summer of a single year, and it was suggested that had the survey covered a whole year or a series of years, an entirely different picture would have been obtained.

Whilst it has not proved possible to carry on the regular chemical testing of the individual samples from a large number of farmers, the testing of pooled milk has been continued for three years, samples coming from an ever-growing pool, which, by the end of the survey, was receiving at least a portion of the output of 500 dairy farmers. Naturally it was never possible to obtain a single sample representing the whole bulk and in actual fact the samples tested were collected from storage tanks of 250 to 3,000 gallons capacity, up to three tanks being tested daily. Owing to the need to conserve space, it is not possible to record these daily results, but with few exceptions the individual tank samples gave very similar results to one another, each tank following the general trend of improving or deteriorating milk quality. Very occasionally a tank sample was outstandingly high or low due to some extraneous cause such as faulty mixing.

The results of this three-year survey are summarised in Tables 1 and 2, from which the following major points emerge:—

1. The annual average figure for total solids was below the minimum standard of 12.0% during 21 successive months. Thereafter the standard was maintained for a five-month spell, after which it fell below 12.0% once more.
2. In 1944 and 1945 the annual average butter-fat content just reached the minimum legal standard of 3.5%, whilst the annual average for non-fatty-solids just achieved the minimum of 8.5% in 1945. At all other times these averages were sub-standard.
3. During late summer and autumn each year, the butterfat content tended to rise appreciably, but in 1944 this rise was offset by

a comparable fall in the non-fatty-solids. In 1945, on the other hand, the non-fatty-solids figure was comparatively high in autumn and early winter.

4. There is the suggestion of a gradual improvement in the chemical quality over the three years under survey.

Discussion.

The figures that have been presented indicate that whilst seasonal fluctuations in the chemical quality of the milk do occur, the period of high quality is short, whilst even during the peak period the total-solids content often fails to reach the legal minimum level of 12.00%. For the rest of the year pooled milk is definitely sub-standard.

In the course of the survey under consideration, so many suppliers were involved that the question of adulteration can be safely excluded and the figures given are a reasonably fair picture of the quality of the milk actually produced by South African grade Frieslands living on the high-veld. Very little comfort can be gained from the slight but steady improvement noted during the three years under survey because, whilst detailed data are not yet available, all evidence indicates that in 1946 a record low level of milk quality is likely to be attained. This is generally attributed by dairymen to the indifferent quality of animal feed available, which raises the question of the interrelationship between diet and milk quality.

It is a generally accepted dictum that the chemical quality of milk is unaffected by the diet and, within reasonable limits, this must be correct because it has been confirmed by endless physiological experiments. In South Africa, however, it would appear that 'reasonable limits' do not apply, because there is an abundance of circumstantial evidence to show that the composition of the diet has a profound effect upon the milk quality.

A high butter-fat content is expected on the highveld in the late summer and autumn, and this expectation is borne out by the figures quoted in this report. This rise is probably due to the combined effect of a satisfactory diet, coupled with the fact that at this season of the year, herds carry a large percentage of cows in the late stages of lactation. For all the rest of the year, the butter fat content remains low, even during October and November, when the digestibility of the veld-grass proteins is supposed to reach the highest level. The main effect of the spring growth appears to be to stimulate the volume of milk produced and this may even have a diluting effect upon the milk-solids. There is a definite suggestion of this in the figures recorded for 1943.

In the survey under consideration the average figures for non-fatty-solids were consistently sub-standard for twenty consecutive months, the lowest level being reached in March, 1944, after which the monthly averages rose steadily to reach the legal minimum of 8.5% in March of 1945. The non-fatty-solids remained above this

level for six consecutive months and then dropped once more. No climatic or dietetic explanation can be offered for these peculiar variations, nor was it possible to show any direct or delayed correlation between these figures and those of the incidence of mastitis in the group of herds involved in this survey.

It is of considerable interest to note that during ten different months the figure for total solids fell below 12.00%, although the butter-fat was in each instance up to or above standard. Results of this type are so commonly encountered in South Africa that many dairymen accept as inevitable the fact that a high butter-fat must be offset by a correspondingly low figure for non-fatty-solids. There is, however, no justification whatsoever for adopting such a view and the indifferent chemical quality of South African milk must be attributed to three major causes:—

1. Diseased udders,
2. Inadequate or unbalanced feeding,
3. Unsuccessful breeding methods.

1. *Diseased Udders:* It is generally accepted that chronic streptococcal mastitis can have a very deteriorating effect upon the chemical quality of milk, particularly in regard to non-fatty-solids. In earlier reports, Pullinger (1944 and 1946) has shown that streptococcal mastitis is all too prevalent in the average South African dairy herd. In the course of a prolonged survey covering the milk of 200 producers, only 6.0% of these producers never showed signs of mastitis in their bulk supplies. Within the individual milking herd upwards of 60% of the cows might be infected at any one time. Obviously, therefore, until serious efforts are made to control mastitis, there is little hope of greatly improving the quality of South African milk.

2. *Inadequate or unbalanced feeding:* Some reference has already been made to the generally held belief amongst South African dairymen that feed quality can influence not only the productivity of the cow but also the richness of the milk. This aspect of the problem has already been considered in a report by Pullinger (1946), in which year "an example of low butter records are given of the butter-fat output of a high-class grade Friesland herd. The butter-fat content of pooled milk from this herd fluctuated between 1.9% and 2.5%. Out of 80 cows only three gave butter-fat of 3.5%, whilst many cows tested below 2.0%. Other herds similar to this one have since been detected.

From evidence of this type and also from the cumulative evidence collected during many adulteration investigations, it would appear that on the South African high-veld milk quality can be seriously affected by diet, because the cows exist for periods of the year under conditions of semi-starvation or because rations are hopelessly unbalanced.

3. *Unsuccessful breeding methods:* Probably the greatest criticism that can be levelled against the breeding of dairy stock in South Africa is the fact that in the past the non-fatty-solids records have been so

consistently ignored and in consequence Friesland breeding strains have been perpetuated, which are very weak in regard to this constituent. This attitude has been further exaggerated by the purchase of milk for manufacturing purposes, upon a butter-fat basis instead of the content of total-solids. Essentially the cheese factory requires non-fatty solids for the manufacture of the product, and excess of butter-fat may even be an embarrassment. It is highly illogical, therefore, that the cheese manufacturer should pay a premium for a constituent that he does not require, whilst the premium-earning milk may actually be deficient in the proteins necessary for cheese-making.

It is to be hoped that in the future full attention will be paid to non-fatty-solids in the selective breeding of pedigree dairy stock, but it must be appreciated that improvement of milk productivity and milk quality by orthodox selective breeding is likely to be a disappointingly slow procedure. Calculated over a ten-year spell between 1934 and 1944, the records of the U.S.A. Dairy Herd Improvement Association showed an average gross increase of 319lbs. of milk and 14lbs. of butter-fat. These figures show just how slow improvement can be.

Under these circumstances it is inevitable for thoughts to turn to those indeterminate hybrid cows which are to be found in many South African dairy herds, and upon which the farmer relies to bolster up the quality of his bulk milk. The parentage of these hybrids is usually obscure, but some are obvious crosses between Afrikander and recognised dairy breeds, whilst the Friesland x Jersey cross is also encountered. There is, however, little or no hybridisation of this type carried out as a definite policy and none of the crossing is carefully controlled.

In this connection attention can be drawn to an experiment that is being run at the Beltsville Research Centre under the control of the Bureau of Dairy Industry, U.S.A. Department of Agriculture. The writer is unaware of any full report of this experiment, but a very brief progress report is given by O. E. Reed, Chief of the Bureau (Reed, 1946). It is not practicable to quote the data *in extenso,* but the following comprise some of the salient points:—

Only proved pedigree sires and dams are being used and 'proving' consisted of mating each animal with selected mates of its own breed, and noting the quality of the offspring in respect of milk productivity and milk quality. Using such proved parents, the following crosses were tried, the first named indicating the sire:—

Jersey	x	Guernsey
Jersey	x	Holstein
Holstein	x	Jersey
Holstein	x	Guernsey
Holstein	x	Red Dane
Red Dane	x	Jersey
Red Dane	x	Guernsey

In the first set of experiments the group of 32 crossbred heifers averaged in the first lactation 12,842lbs. of milk and 592 lbs. butter-fat, the butter-fat test average being 4.64 per cent. The crossbreds surpassed their pure-bred dams by 139lbs. butter-fat. Moreover the crossbreds showed much less variation between maximum and minimum output than the pure-bred dams, whether output was judged as pounds of milk or butter-fat. For instance, one Holstein x Guernsey heifer, during the second month of lactation, gave 50lbs. of butter-fat. This figure gradually rose to 60lbs. and remained between 50lbs. and 60lbs. for 12 months. Of all the first batch of hybrids, the lowest annual gallonage was given by a Jersey x Holstein heifer, this being 9,784lbs., whilst the highest given by a Red Dane x Holstein cross was 16,949lbs. In regard to butter-fat, the lowest return of 449lbs. came from a Red Dane x Holstein heifer, whilst the highest of 683lbs. came from a Holstein x Guernsey cross.

Taking the Red Dane bull as an example, when mated with Red Dane Cows the progeny of this sire surpassed their dams by 1,139lbs. milk and 45lbs. butter-fat. When mated with other breeds, the heifers from this sire surpassed their dams by 5,374lbs. of milk and 193lbs. of butter-fat.

The underlying importance of this American experiment is that it confirms the indications regarding the value of cross-breeding that have oniy been vaguely realised by South African dairy farmers. It is, however, unfortunate that the report in question does not touch upon the question of total solids. It is urged that this American Experiment should be repeated in South Africa, with the inclusion of the Afrikander dam of milk type. There is every reason to hope that the crossing of proved dairy sires with Afrikander dams would produce a cross having reasonable productivity of, say 9,000lbs., coupled with high butter-fat and with non-fatty-solids at least no lower than that of the pure dairy breeds. In addition the incorporation of Afrikander blood would enhance the stamina, resistance to disease, and ability to thrive under lean conditions, factors conspicuous by their absence amongst pure-bred dairy stock.

SUMMARY.

1. Records are given of the chemical quality of bulk milk samples collected five days a week during a three-year survey.

Butter-fat fell below 3.5% in 14 out of 35 months.
Non-fatty-solids fell below 8.5% in 23 out of 29 months.
Total solids fell below 12.0% in 24 out of 29 months.

2. This unsatisfactory position is attributed to:—

(i) Diseased udders,
(ii) Inadequate or unbalanced feeding,
(iii) Unsuccessful breeding methods.

TABLE 1.

Chemical Quality of Milk Produced for the Fresh Milk Trade.

MONTH.	*PERCENTAGE OF BUTTER-FAT IN YEARS AS SHOWN.			*PERCENTAGE OF NON-FATTY SOLIDS IN YEARS AS SHOWN.			*PERCENTAGE OF TOTAL SOLIDS IN YEARS AS SHOWN.		
	1943.	1944.	1945.	1943.	1944.	1945.	1943.	1944.	1945.
January . . .	—	3·4	3·5	—	8·17	8.43	—	11·57	11·93
February . .	3·5	3·5	3·5	—	8·12	8·41	—	11·62	11·91
March	3·6	3·6	3·4	—	8·07	8·51	—	11·67	11·91
April	3·6	3·7	3·7	—	8·26	8·53	—	11·96	12·23
May	3·7	3·6	3·5	—	8·32	8·53	—	11·92	12·03
June	3·5	3·6	3·5	—	8·36	8·68	—	11·96	12·18
July	3·5	3·4	3·5	8·37	8·32	8·62	11·87	11·72	12·12
August	3·3	3·4	3·5	8·34	8·44	8·54	11·64	11·84	12·04
September . .	3·3	3·4	3·4	8·37	8·47	8·43	11·67	11·87	11·83
October	3·3	3·4	3·4	8·33	8·43	8·49	11·63	11·83	11·89
November . .	3·3	3·5	3·4	8·29	8·45	8·48	11·62	11·95	11·88
December . .	3·4	3·5	—	8·28	8·46	—	11·68	11·96	—
Average samples tested monthly	60	60	45	36	60	46	—	—	—

* Percentage recorded is average of all tests done during the month.

TABLE 2.

Proportion of each year when Milk was Chemically Substandard.

1943	Butter-Fat Substandard	5	out of	11	months
	Non-Fatty-Solids Substandard	6	out of	6	months
	Total Solids Substandard	6	out of	6	months
1944	Butter-Fat Substandard	6	out of	12	months
	Non-Fatty-Solids Substandard	12	out of	12	months
	Total Solids Substandard	12	out of	12	months
1945	Butter-Fat Substandard	4	out of	11	months
	Non-Fatty-Solids Substandard	5	out of	11	months
	Total Solids Substandard	7	out of	11	months

3. The probable value of crossbred heifers (by dairy bulls out of Afrikander dams) for improving milk quality is suggested, and in support of this contention, an American experiment in the hybridisation of dairy cattle is quoted.

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ACKNOWLEDGMENT.

Thanks are due to the Director, Abattoir and Livestock Market Department, for permission to publish this article.

THE PORT VETERINARY OFFICER.

M. BERGH,
Durban.

In reading this short paper here to-day I must say that I was inspired by the Senior Veterinary Officer for Natal, Major Daly, who quite rightly held that very few Government Veterinary Officers or other colleagues know what the duties of a Port Veterinary Officer are unless they have had first-hand experience, and it might be of interest to them to know just what takes place at a port of entry. In addition it is felt that our regulations need revision, and for that reason I hope that I will to-day stimulate a discussion of a critical nature and, if necessary, a resolution will be passed bringing to the notice of the Department the necessity for the drafting of amending regulations.

The important factor about the "Port Veterinary Officer" is that he is, first of all, a Government Veterinary Officer, and in addition to this he stands on guard to prevent an untimely addition to our list of "aangiftepligtige" diseases of stock..

Let met once assure you that my duties do not consist of meeting at the quayside dainty little Fidos tripping at the end of leashes operated by some of nature's masterpieces. When a passenger liner with livestock on board docks, the port veterinary officer goes on board and seeks out the butcher, who is responsible for all livestock on board a passenger liner. From him all information is sought and if the owners are not on board the necessary certificates, etc., must be handed over by him. Should the owners be on board, then they have the papers. As a rule it takes a very short space of time to get everything on board in order, to issue a landing permit, and if necessary arrange for quarantining of the animals in question ; the whole procedure comes to a very speedy and prosaic ending. There are of course occasions, and during the late war these occasions were quite frequent, when people were evacuated to South Africa at a moment's notice and no papers were available. Then of course matters do not progress quite so easily.

On board cargo ships the animals are usually of the utility type, such as horses, cattle, sheep, and greyhounds. Since cargo vessels do not carry butchers as a rule, the official to seek out here is the First Officer or as he is called, the First Mate. He is here responsible for the animals and their papers. During the war, again, the papers which were often forwarded on vessels other than the one the animals travelled on were not available owing to the interest taken in such affairs by the enemy. Cabled certificates were then called for as a rule. On board freighters the matter in hand was dealt with even more expeditiously than on passenger liners.

The reason for this is that owners of stock when they are present are sometimes only there as a nuisance and nothing more. The owners of dogs are, of course, the worst offenders and on occasion can even make quite exhibitions of themselves. One finds, of course, that the people who give the least trouble and only try to be helpful in a practical way are Governors of territories, Royalty, diplomats and much travelled business men. The attitude these people take is that of in effect saying: "There's the animal, do the necessary and give me the bill." This attitude is one that frequently breaks down and lesser mortals especially the female species frequently bursts into tears on being told that six month's quarantine is necessary for her "Pookie." Such harsh treatment will kill little darling is the theme of the lament. We have not yet lost a "Pookie" I may say.

The Port Veterinary Officer also is in charge of the Veterinary Quarantine station. There is always a superintendent on the premises with the rank of Assistant Stock Inspector or even Stock Inspector. In Durban the permit clerk is also the Superintendent. Owing to the area in which the quarantine stations are situated not being exactly "select" the Superintendent generally gets free quarters.

The Department only supplies free accommodation, cleaning and water, as the owners are responsible for attendance, feeding and even veterinary care. With large animals there is as a rule no trouble, the owners or their agents always providing the attendance and the necessary feed. The trouble is with dogs. When there are many dogs it becomes impracticable to allow every owner free access to the quarantine station for the purpose of feeding his or her dog, so the Superintendent as a rule arranges with them to feed their dogs for a flat rate. Should many dogs be in quarantine he may break square or make something, but with only a few dogs he loses on the transaction.

It happens of course that sometimes people arrive who think that they have something of a canine prodigy and then of course the fun starts. Recently a couple arrived from India with three dogs. They were not satisfied with kennels or even a loose box, but requested the Superintendent to have them in his flat. This he foolishly allowed until he found his privacy completely vanished as these people arrived to visit their dogs at all hours of the day and night. The Superintendent arranged to feed the dogs, but they insisted on a diet of bacon, veal, gorgonzola cheese, chicken liver, sirloin, roast leg of mutton, tinned sausages, tinned roast, bully beef, fried eggs, chicken essence, liver, chicken, turkey, bovril, milk, malted milk, brandy and ham. The Superintendent of course informed them that under the present conditions his catering potentialities fell far short of this menu. They then had to feed the dogs themselves, and the result was that the lady stayed on in Durban and bought a spring bed, with an inner spring mattress and bed sheets for the dogs. She made four taxi trips to the quarantine station daily, two trips in at 8 a.m. and 2.15 p.m. and two trips out at 12.45

p.m. and 6 p.m. On one occasion a French poodle stayed with us with a European maid, and this dog had its own toilet roll.

I could, of course, go on quoting may of the peculiar incidents the port veterinarian comes up against which the ordinary government department does not consider part and parcel of its usual routine. For the present the examples mentioned should suffice.

The importation of animal and plant products presents no difficulties at all. No examination on board is necessary and the agents come to the office with the necessary papers and the permit is issued in the office provided all the papers are in order. In the case of meat-, blood-, bone- or fishmeal the Customs extract a sample for bacteriological examination at Onderstepoort.

The animals most frequently imported are cattle, horses, dogs and sheep. Wild animals and birds for zoos are frequently met with and it goes without saying that reptiles and other unfriendly animals only receive the most perfunctory examination on board.

Animal products imported consist mostly of bags of bone, meat, blood and fishmeal, horse and cowtail hair. A very important importation is hog casings, by which name the trade calls sausage skins. These hog casings arrive in Tierces. Tierce as you know is French for "a third" so I looked up in the dictionary for a definition of this particular tierce and this is what I found: "A cask larger than a barrel and smaller than a hogshead, hence a liquid measure formerly legal at forty-two wine gallons or one-third of a pipe." In trying to get this properly I spent an informative half-hour with Webster and got thoroughly confused between wine gallons, ale or beer gallons, Portuguese pipas, cubic inches, pounds and litres, so gave it up.

The Customs and Harbour Police are the first line of defence and to them we look for assistance as they are the people who must stop all livestock, animal products and plants without the proper permit from entering the Union. Here it is quite clear that the serious nature of such diseases as surra and psittacosis is not always adequately explained to them as I have considerable reason to think that some members — and I think the recruits especially — have been "talked" into letting some of these prohibited items into the Union. Some liaison work via our head offices seems to me to be indicated. Seamen, white or lascar, know all the answers in the smuggling game so the Customs and Police must be alert.

It will be asked whether attempts are made from time to time to smuggle livestock into the Union. This happens quite frequently and the animals that are the most often the subjects are parrots. Because these birds are subject to a very nasty and chronic disease it seems that they are in great demand and the profit on the illegal introduction of these birds seems to be worth a great many dangers. The lascar crews of ships seem to be the ones mainly implicated in this nefarious traffic and the result of detection is always a heavy fine, plus the lethal

chamber for the birds. Attempts have been made to smuggle dogs and the bribes that are as a rule offered for their illegal introduction as well as for shortening the quarantine period would astound most people.

Export duties do not form a great part of the Port Veterinary Officer's activities. Occasionally animals are exported from his own area, when he must undertake the tuberculin test, or again animals occasionally arrive some time before the sailing of the ship and then he must undertake the tuberculin test. For this purpose certain premises are usually inspected and approved as suitable for detaining cattle while awaiting shipment.

Exporters of animal products such as wool, hides, skins, etc., make a sworn declaration on Form U.A.D. 563 and bring them to be signed by me. This I do with my tongue in my cheek as I am accepting the word of the exporter who as a rule has not the faintest idea what he is declaring or where the products originate from. That the importing countries are very fussy about these things is proved by the fact that I was badly on the carpet once for not actually signing a set of forms that were destined for Canada but using my rubber stamp which is a facsimile of my signature. Incidentally I am told that this latter stamp is even legal on a cheque in this country.

The introduction of animals into the Union is partly governed by Act 11 of 1911 which deals with agricultural pests and by proclamation No. 115 of 1937 the following classes of animals are not permitted to enter the Union except by permit and under the conditions which the Department may prescribe: Mollusca, Nematoda, Crustacea, Myriapoda, Insecta, Arachnida, Amphibia, Reptilia, Aves and Mammalia. Needless to say I have not been able to comply with the terms of this proclamation as I am sure that some of the more minute members of some of the types have eluded my vigilance.

Under Section 2 of Act No. 14 of 1911, the Governor-General by Proclamation No. 166 of 1940, defines livestock for the purpose of the Act as all Primates other than man, all rodents, all ungulates, all insectivora, all carnivora, elephants, all equine animals and all birds including poultry. The same Act as amended by Act No. 37 of 1937, Sections 3 to 8 gives direction as to the importation of livestock and Section 4 empowers the Principal Veterinary Officer, to give written permission for the introduction of livestock. This section also empowers the Governor-General to proclaim in the Gazette the names of countries whence livestock or a particular type of livestock may not be introduced.

That this power has been freely made use of is quite clear if I may mention here a formidable array of proclamations and government notices which govern the introduction of livestock into the Union.

There are nine proclamations which are the following, No. 132 of 1923, No. 68 of 1924, No. 63 of 1925, No. 178 of 1927, No. 149 of

1928, No. 233 of 1935, No. 238 of 1925, No. 23 of 1933, No. 258 of 1937, and No. 22 of 1941. The Government Notices are sixteen in number and are the following: No. 638 of 1915, No. 40 of 1926, No. 1562 of 1927, No. 631 of 1928, No. 833 of 1930, No. 1456 of 1930, No. 35 of 1931, No. 598 of 1933, No. 105 of 1934, No. 526 of 1934, No. 1607 of 1935, No. 1742 of 1940, No. 1034 of 1941, No. 1755 of 1940, No. 872 of 1944 and No. 197 of 1945.

All the regulations contained in the above-mentioned proclamations and government notices on analysis mean that no livestock is allowed to enter the Union from any state, country or territory situated in Greater Asia, or South America, and all states, territories and countries North of Southern Rhodesia and from any port North of the Port of Durban.

Dogs and cats are also prohibited entry into the Union from all European countries except the United Kingdom. When they originate from the United Kingdom, Australia, and New Zealand no quarantine is required.

Sheep and pigs are allowed in from the United Kingdom with fourteen days quarantine. From the rest of Europe twenty-one days quarantine is imposed and no quarantine when they originate from Australia and New Zealand.

Cattle. From the United States of America, Canada, United Kingdom and New Zealand they are allowed entry provided they are quarantined here for thirty days as well as being subjected to the tuberculin test. Cattle must be quarantined in England for two weeks prior to being shipped to South Africa. Cattle are not allowed to enter the Union from Australia owing to contagious pleuropneumonia but from Tasmania entry is allowed under similar conditions as for those from other countries. This ruling may be varied from time to time according to the extent of foot and mouth disease in the countries of origin. Veterinary certificates must state that animals are free from contagious abortion.

Horses. Equine species are allowed into the Union without quarantine from the United Kingdom, but with thirty days quarantine with a mallein test from the United States of America, Canada, and New Zealand.

Poultry. Poultry is generally introduced from all countries without quarantine provided that such poultry is accompanied by a certificate specifically stating that the birds are free from and originate from areas free from such diseases as: enzootic- and epizootic cholera, tuberculosis, fowl plague, Newcastle disease and salmonellosis. Turkeys must be guaranteed in the same way to be free from trichomoniasis, blackhead and hexamitiasis. Fowls must also be declared free from epidemic tremor, blackhead and infectious laryngotracheitis. The tests for the usual salmonellas in the different species too have shown negative results.

Parrots are quite unwelcome and are prohibited entry.

Aviary Birds. These are allowed in provided they are certified to come from aviaries free from psittacosis. Pigeons and pheasants must come from countries free from equine encephalomyelitis.

Mice and Rabbits. These are allowed entry only by special permission.

Wild animals. As already stated these are allowed in at the discretion of the Principal Veterinary Officer to an approved zoo, after they have been kept under observation in an approved zoo in the country of origin, even India.

General. In addition to the above-mentioned restrictions it must be stated that all permits are issued subject to the general provision that:

1. All animals must be accompanied by a certificate signed by a State Veterinarian or a Veterinarian authorised to do so by the country of origin, to the effect that the animals were healthy at the time of shipment and that they showed no symptoms of contagious or infectious diseases and originated from areas free of such disease.

2. All animals must enter either at Cape Town or Durban, these being the only ports of entry in terms of the Act.

3. No intermediate ports may be made between South Africa and the country of origin.

4. No unexplained mortality had taken place en route.

5. Before a landing permit can be issued the Port Veterinary Officer must examine the animals on board and find them healthy.

It may be pointed out here that the Principal Veterinary Officer has overriding authority to issue a permit to introduce stock from any country, even countries prohibited from exporting animals to the Union. This is done only in special cases and under special conditions imposed by him. Similarly the Principal Veterinary Officer also has the right to stop importations from any country whence animals usually enter the Union. Should an entry permit be refused in this way and the owner considers that he is being discriminated against for any reason, he has no redress except that allowed him by Section 19 of Act 14 of 1911, by which he may appeal to the Minister of Agriculture whose decision is final. From this decision there is no further appeal and no litigation may result.

The introduction of animal products is governed partly by the Fertiliser, Farm Foods, Seeds and Pest Remedies Act. Under this Act (Act 21 of 1917) regulations are also framed as well as under the Stock Diseases Act (Act 14 of 1911). The regulations are contained in Government Notices No. 372 of 1932, No. 74 of 1937, No. 1956 of

1937, No. 1821 of 1938, No. 371 of 1939, No. 1907 of 1940, No. 1910 of 1940, No. 2425 of 1942, No. 2426 of 1942, No. 128 of 1943 and 1071 of 1943.

These notices all contain regulations which boil down to the fact that Asia is out of bounds for the importation of animal products. From all other countries animal products may be introduced provided they are accompanied by a veterinary certificate of sterilisation and that the products originate from healthy animals or animals which originated from disease-free areas. The sterilisation certificates must say among other matters that no Bacillus anthracis or organisms of the gas gangrene type are present. Hog casings must be in salt for at least six weeks prior to shipment. *Hides and Skins* must be accompanied by a certificate that they are free of anthrax, foot and mouth disease, rinderpest and contagious bovine pleuropneumonia.

The Director of Veterinary Services can give an overriding permission for the introduction of any animal product even if prohibited as mentioned above, and he states the conditions under which his permit is granted.

Vegetable products are also provided for in so far as their introduction is affected, in Government Notices 74/37, 1956/37 and 284/39 and they were framed under Sections 16 and 23 of Act 14 of 1911. They provide that straw must be accompanied by a certificate of sterilisation if used as packing. Hay, fodder, straw (other than packing) maize stalks and kaffir corn stalks must be accompanied by a veterinary certificate stating that the countries of origin are free of sheep pox, contagious bovine pleuro-pneumonia, foot and mouth disease, etc., etc., or must have been stored under Government control for four months free from contact with the above-mentioned diseases. All plants and plant products are prohibited from countries where foot and mouth disease exists.

Here again the Director of Veterinary Services can give overriding permission on his own initiative.

Before discussing the regulations I wish to make it clear that I had hoped to have at my disposal for purposes of comparison the regulations covering the importation of livestock into the United Kingdom, Canada, Australia, New Zealand and the United States of America. Dr. Diesel very kindly undertook to procure these but unfortunately the response was nil (A case for UNO). No comparisons are thus possible.

In any remarks made in this discussion I would like you to bear in mind that all animals introduced into the Union can only enter provided they are accompanied by an official certificate stating they are healthy and originate from healthy areas.

The first point may be only of value from the administration point of view but I do think that animals and especially small animals should be not only housed and watered by the Department but also fed by

the Department and a flat rate charged the owner. The reason I say the Department should take this responsibility is that as matters stand now owners cannot be prevented from feeding their animals and especially their dogs with the result that at all hours — reasonable and otherwise — they are in the Quarantine Station, turn the place into a social centre and make a general nuisance of themselves. By undertaking the responsibility the Department can lay down definite visiting days and hours. Any fancy feeding can be curbed and the "cuisine" can be Departmental. There is something to be said for the same being done for large animals.

A clear definition should be given as to when any piece of integument is still a hide or skin or can be classed as leather, fur or any piece of fancy tanned goods. It frequently happens, especially with hunters, that they arrive with trophies that have been prepared by taxidermists. In such a case the telephones buzz between Durban and Pietermaritzburg and on to Pretoria and vice versa and in the end the Port Veterinary Officer is asked for an opinion, which does not prevent a query on his monthly return because no record exists of all this telephoning.

The Port Veterinary Officer should here be in a position to have standards for his guidance so that he may have the necessary powers to act within those standards, without having to refer.

A more specific definition of the different parts of the world should be given. Does 'America' include the West Indies and where does Polynesia come into our regulations ? However no great volume of traffic exists in these directions, so this is more of academic interest.

The next point is one that depends on so many factors at the moment and has been the subject of so many arguments at the Port of Durban and doubtless at Cape Town so that I think it worth while to discuss it in extenso.

Broadly speaking, it may be asked why are animals and especially pets prevented from entering the Union from certain parts of the world when adequate precautions for their control can be taken all along the line.

I have yet to learn what the function of a Veterinary Quarantine Station is if it is not as a precaution against the introduction of stock diseases. Let us take dogs. Why are dogs prevented from entering the Union from Asia for example. Great Britain allows dogs from India to enter provided six months quarantine is spent by the dogs in certain approved kennels; note, not official veterinary Quarantine Stations. Why this strictness in South Africa, when a country like Great Britain is free from Rabies in spite of her seeming laxity in comparison with us ? What is the use of a veterinary certificate or a Quarantine Station if not to assist owners of livestock, who after all

are our clients and who eventually pay the piper ? Are we as a profession for ever going to admit defeat and never reach the stage which our sister profession arrived at years ago, where they do not hamper international movements but take good care that no disease arrives with visitors or immigrants ?

I feel that even in the case of birds, including parrots, the quarantine period should be established to eliminate such diseases as psittacosis, laryngotracheitis and equine encephalomyelitis. Let me at once mention an anomaly. Animals of all descriptions from all parts of the world are allowed into the Union from Zoological gardens that are disease free and here parrots also are included. The animals and parrots again are allowed to Zoological gardens where they come in daily contact with hundreds of people apart from the other inmates of the Zoo. What is the matter with the Zoo Department of the Veterinary Quarantine Station, Durban ?

There seems to be no policy, and as a result strange things happen. For example by what criteria is the Director of Veterinary Services guided in granting or refusing permits say for equines from areas other than the United States of America, United Kingdom, Canada, Australia, or New Zealand ? Why cannot the Port Veterinary Officer be notified at the Port to enable him to act within the terms of a defined policy? Why cannot the Port Veterinary Officer be kept posted of the occurrence of stock diseases in other parts of the world so that he can talk authoratively to any stock owner instead of prevaricating and inventing answers? I always quote the following case: A soldier comes into my office during the war, and says he has a canary on board a troopship. This canary he bought at Aden for a relative who breeds them in Durban. He wants permission to land it but he has no papers. I telephone the Senior Veterinary Officer. Dr. Diesel telephones Pretoria. The Deputy-Director of Veterinary Services is not available ; by the time his refusal is available the ship has gone. Puzzle: Find the canary.

In reading this short paper I hope that my colleagues will have been stimulated to discuss the present regulations freely and critically, because I feel that at the moment we are inclined to say " No " merely because by saying " Yes " the puzzle may become harder ; which indicates in turn that we are primarily public servants and only secondarily scientists, instead of being scientists who give their services to the State fearlessly and without scruple or diffidence.

TRICHOMONAS FOETUS INFECTION AND OTHER CAUSES OF INFECTIOUS ABORTION IN CATTLE.

E. M. ROBINSON,
Onderstepoort.

So much has been written about brucella infection as a cause of contagious abortion in cattle that other causes are liable to be regarded as of little importance or may be overlooked. The purpose of the present article is to review some of the more recent articles on Trichomonas foetus infection and some of the other organisms capable of causing abortion in cattle. At one time or another many different organisms have been incriminated as occasional causes of contagious abortion but apart from Trichomas foetus and Vibrio foetus infections none plays any big role in its causation.

Trichomonas Infection.

This condition has been recorded for the first time in South Africa by the author (1937) and since has been diagnosed several times in different parts of the country. It is very probable that it is much more widespread than is realized at present and may be responsible for some of the pathological conditions occurring in the genital tracts of cattle and leading to sterility in some cases. A good many articles on Trichomonas infection have appeared in recent years, dealing with different aspects of the subject. It is not proposed in this article to give a detailed description of the parasite with its culture methods, etc., but to deal with the symptoms, pathological changes, diagnosis and treatment only. A good deal of valuable information is contained in two articles by Kerr (1943) and it is from them that many of the points to be referred to have been taken.

In an editorial in the Veterinary Record (1942) it is stated that the distribution of the disease in a herd may vary considerably over a few years. In the absence of treatment it recedes slowly and may spread to other centres. A definite diagnosis should always be made as early as possible because it may be difficult once treatment has started. If energetic treatment or prophylactic methods are introduced the course of the disease may be greatly influenced. As one would suspect, it is often associated with brucella infection.

The symptoms vary considerably in different outbreaks and secondary infection may modify them. One should suspect trichomanas infection when (a) abortions occur at an early stage of pregnancy (b) cows keep on returning to the bull (c) endometritis or pyometra occurs with anoestrus and (d) if there are cases of cervicitis or vaginitis.

Cases of abortion may easily be missed and may not be suspected. Cows may appear to hold to service for two to five months, when a clear discharge from the vagina may appear. Per rectum the uterus

may appear normal for the stage of pregnancy but the corpus luteum may not be palpable. The discharge is often scanty and only marked at oestrum. It is clear, colourless and mucoid. White streaks and flecks of pus in it are rather characteristic, but secondary infection alters the discharge a lot. The occurrence of vaginitis is not constant and when it occurs it is often caused by secondary organisms or a virus such as vesicular exanthema.

In a recently infected bull there may be a mucopurulent discharge from the prepuce and the mucous membrane of the penis may show reddish nodules but the secretion may be very scanty and some bulls show no obvious symptoms. When there is an acute or severe local inflammation it is often associated with trichomonas infection but is due to some other cause. It is no proof of absence of infection if some cows served by a bull calve normally. Some cows carry infection up to the time of calving and are consequently very dangerous. As one would expect, trichomonas infection is often associated with brucella infection and this should always be kept in mind.

In an annotation in the Veterinary Record (1943) a number of observations on the disease are made. In a herd containing 125 females of breeding age, seven examinations were made of the animals over a period of eighteen months. In addition to the cows, 20 bulls were examined. Of the infected cows, 10% had live calves and of the uninfected 70%. Of the 20 bulls, 6 were found to be infected. The disease appeared to be self-limiting.

Out of 560 uteri of cows examined at a packing plant, 8 showed trichomonas infection. Of these 5 were non-pregnant cows with pyometra. One was in an apparently healthy cow, but the amniotic fluid showed a heavy infection.

In 1577 uteri, 997 of which were pregnant and 580 not, Tr foetus was found in 2 pregnant ones and in 13 pyometra cases in non-pregnant cows. In 211 bulls no infection was found. It was not possible to find trichomonads in the ureters, vasa deferentia or testes of bulls and no proof was found that Tr. foetus occurred in the blood or urinary tract. Infection was found to persist in bulls for at least 20 months in some cases and might become permanent. Transmission of infection may occur at every service. An important point is that semen taken for artificial insemination may be infected. There does not appear to be any relationship between Tr. foetus and the vaginal trichomonad of women.

Kerr (1943) in an article on trichomoniasis in the cow gives a very clear picture of most aspects of the disease. He states that there is a considerable variation in the clinical picture. The symptoms may even vary with the locality. Pyometra is frequent in some areas, but is rare in Northern Ireland, where he made most of his observations. Sterility is common in that area. A big complicating factor in trichomonas infection is secondary bacterial ones caused by corynebacteria, streptococci, etc.

Symptoms in Natural Cases

There are several features which may influence the infection. These are (a) the virulence and the number of the organisms present, (b) secondary bacterial infection, (c) the farming system employed and (d) the constitution and environment of the animal.

The symptoms may be divided into five types :—

(1) low-grade metritis, (2) catarrhal endometritis, (3) vaginitis and cervicitis, (4) pyometra and (5) anoëstrus.

About 80% of cases would fall under (1) or (2) but vaginitis may occur in any group.

(3) *Vaginitis.* Inflammation of the vagina and vulva occur a few hours after service according to some observers, but Kerr only saw it from the third day, reaching its severest degree about the ninth. Haemorrhagic papules occur round the clitoris and pencil lines occur along the lateral walls of the vagina. After the ninth day the mucous membrane becomes diffused and dark with proliferation of the lymphatics. Some observers have described a characteristic granulation of the anterior vaginal wall. Flecks of whitish pus occur round the clitoris. With secondary infection these flecks become creamy or golden yellow. This pus may disappear but reappear when oestrum occurs. In mild cases the symptoms may disappear in a few days. Cervicitis is seen but is usually an extension of the vaginitis and probably a secondary infection but an examination should be made for trichomonads.

(1) *Low Grade Metritis.* Oestrum returns at the third or sixth week with little or no discharge. Though the animal is mated at each period conception fails to occur. Flecks of pus around the clitoris are seen about the second day after the return to the bull. After about six months, conception may occur indicating that the condition is self limited. Recovery often occurs without treatment. Diagnosis is often difficult as the vaginitis is transient as is the occurence of trichomonads. Palpation of the uterus is of no value.

(2) *Catarrhal Endometritis.* Conception occurs with death of the foetus between the sixth and twelfth weeks. There is a slight whitish mucoid discharge which may be delayed until the third or fourth month, increasing in quantity and at its height about 14 days after it appears, gradually subsiding. It usually disappears after six weeks and then the only symptom is persistent sterility. The foetus may be aborted intact in its membranes. It is whitish and macerated. It may be so completely macerated that it passes unrecognized. Trichomonads are most frequent before the discharge reaches its peak, and repeated examinations should be made. The cow may come in season again when the discharge diminishes even if no treatment is instituted. A few animals may conceive and have normal calves, but most remain sterile. The uterus is thickened and both horns are affected. The cervix is flaccid and open. Any condition between a low-grade and catarrhal endo metritis may occur. It is with animals in this group that diagnosis by serological tests is of most value.

(4) *Pyometra.* If conception occurs and the foetus dies but is not expelled, pyometra, may occur. The foetus dies and becomes macerated in the uterus. It is normally found bleached and hard and about three inches in length, but it may not be traced. Hypertrophy of the uterus occurs corresponding to the size it would be at the expectant stage of pregnancy. The contents of the uterus are fluid but rather thick and creamy. There is no smell. The colour is whitish but may be yellow or of a slightly brown tinge. The cervix is closed. The fluid is rich in cells and trichomonads of a very active type. Palpation of the uterus reveals a doughy feel but it may be tense. The uterine wall is normally thickened and no foetus can be felt.

(5) *Anoestrus.* Up to 10% of animals in a herd affected with trichomoniasis show anoestrus but the percentage is usually lower.

A complicating factor is poor condition in the winter months when sterility due to trichomoniasis is most evident. The farmer does not notice any symptoms apart from the fact that the cow is not in calf and does not come in season. No uterine changes can be felt and the corpus luteum often persists.

Diagnosis in the Cow

The history is very important and may be characteristic. In examination of a suspected case some of the discharge is mixed with three parts of normal saline and incubated for one hour at 37° C. The trichomonads live for 24 hours outside the body in secretions. Active parasites are easily recognized in wet preparations, but dead ones are useless for diagnosis. The parasites are often sporadic in their occurrence so frequent examinations should be made. The search for the parasites should be made before treatment commences. Fœcal contamination should be avoided as there is a trichomonad Tr. ruminantium present sometimes in foecal material from cattle. Material for examination should be taken from deep in the vagina, for preference near the cervix which is a predilection site for the parasites. A speculum may be used to facilitate the taking of material and the discharge should be aspirated with a pipette with a bulb. The pipette should have a smooth end. If there is very little discharge, pipette some Locke's fluid into the vagina, re-aspirate and repeat several times. Usually cotton wool swabs wetted with normal saline are sufficient, but they must be kept moist if not examined soon. If there is nothing in the vagina, try the uterus itself. Where abortion has occurred, try the foetal membranes and foetal stomach.

The material should be examined as fresh as possible, even on the spot, and if it is negative another specimen can be taken. Dried and stained preparations are not satisfactory, but can be made if there are plenty of trichomonads. A fresh unstained preparation should always be examined first. A drop of 2% Eosin solution will show the parasites up. The organisms are very motile in fresh, warm preparations and show a gentle waving movement of the flagella for some hours, even in the cold. Examination of stale material is very unsatisfactory and

diagnosis is rendered difficult. In thick mucus the movement is slowed up. Movements of the flagella are best seen in sluggish parasites. There is some variation in size, but they are larger than leucocytes and smaller than epithelial cells. They are pear, leaf or egg-shaped. They may appear flat, but usually are oval or spherical.

The parasites have three flagella in front and one along the body with an undulating membrane. They are sometimes difficult to find but usually are frequent. In preparations from bulls they are usually infrequent, but in cows they can usually be found when present. In difficult cases an examination should be made during the oestral period, preferably the first or second day.

Diagnosis of all cases is not necessary. What matters is that the disease is diagnosed in the herd. If this is found to be difficult, particular attention should be paid to the heifers. Those recently served should be examined two to six weeks after service.

Treatment.

Curative treatment is very successful, but must be instituted early or the breeding capacity may be interfered with and the animal becomes sterile in spite of treatment. All sorts of treatments have been used but that with iodine has been the most successful. Weekly irrigation should be undertaken until oestrus recurs spontaneously and the discharge disappears. Two to three irrigations are necessary as a rule using lactic acid in addition to iodine and a further irrigation is given two weeks after oestrus but without lactic acid. For irrigation 2 drams of Liquor iodi aqueous are added to 12 ounces of water and 30 minims of lactic acid are then added. The animal may be served when two normal periods of oestrum have occurred after the first.

Intrauterine irrigation may have to be done. The catheter must not go into the uterine horns as it may cause perforation. Only 3 to 5 ounces of fluid are used as larger amounts may rupture the uterus. The cervix and uterine canal must be irrigated.

In pyometra, expulsion of the corpus luteum is recommended, with massage of the uterus, followed by uterine irrigation.

Prognosis.

In cases of low-grade metritis it is very good and 90% respond to treatment. In catarrhal endometritis the prognosis depends on whether the salpinx is involved. If the os uteri is flaccid and open the prognosis is not good. Anœstrus often responds to irrigation combined with hormone treatment. Two irrigations may be necessary in a chronic case.

Artificial insemination may be a way of avoiding the spread of infection.

In a further article, Kerr (1942, 1943) describes the disease as it affects the bull according to his experience in Northern Ireland. Like many other observers he regards the bull as the pivot of infection. Bulls may not show the infection but may be lazy at service, and one can only trace the parasites by allowing service of susceptible

heifers. In Northern Ireland, in certain areas, most of the bulls are exposed to infection and many become infected. One method of combating the disease is to cease service of cows for six months, disposing of the bull and fattening off badly-affected cows. One may have little in the way of symptoms in bulls when the cows in the herds are badly affected and vice versa.

Kerr produced a case in a bull by infection in the prepuce. On the 16th day the bull found difficulty in serving a cow. Extrusion of the penis was difficult so there was no desire to complete coitus, which took half an hour. This is known as lazy service. The penis showed a grey catarrhal condition and areas of petechial hæmorrhage. Fourteen days later service took an hour to complete, and at a later date service was impossible, though the desire to mate was not lost. There was pain in passing urine, particularly the first drops, and there was dribbling of urine. Ten weeks after infection it showed islands of granular tissue at the base of the penis and at the involution of the preputial sheath. There was a halo-like zone of inflammation round these islands of tissue. Only a few inactive trichomonads were found in the mucus. Five months after infection, orchitis developed. The testes were twice the normal size and were red, swollen and painful. The animal was killed, and at post-mortem an abscess was found in the epididymis with trichomonads in the pus. There was a secondary bacterial infection. The anterior portion of the urethra showed inflammation as did the ischial arch. Trichomonads were found throughout.

Two bulls from infected herds were examined. One showed a grey catarrhal condition of the mucous membrane of the penis with islands of granulating inflammatory tissue. Trichomonads were found for 18 inches along the genital tract. The other bull showed no symptoms, but produced symptoms in a cow which he served.

Direct examination of a bull is difficult, and epidural anæsthesia usually has to be given. If the penis is drawn out, pin-point hæmorrhages and raised granular groups of lymphoid tissue are very suspicious, and washings should be examined for trichomonads. Lazy service is not always due to trichomoniasis, but may lead one to suspect it.

Diagnosis in the bull.

Diagnosis is not easy as a rule. One can make a diagnosis indirectly by allowing the bull to serve a susceptible cow, and the clinical history of the cows in a herd may be useful. As has been mentioned, epidural anæsthesia is usually necessary before the examination can be done. Some bulls are intractable, especially if they have to be treated a few times. A canula with a brush inside it has been recommended for taking specimens. The genital organs may be examined for lesions under epidural anæsthesia.

Treatment.

Although in the cow restraint and treatment are simple, in the bull, owing to his strength and temperament, without anæsthesia restraint

is very difficult, especially if several treatments are necessary. The genitalia of bulls are complex. If the urogenital tract is affected, treatment must not injure the mucous membrane. It is usually possible to do one or two irrigations. Dyes are often used for irrigation, but must not be irritant. Irrigation may push the infection further up the genital tract. The folds and crypts of the preputial sac make it difficult to flush the mucous membrane efficiently, especially when the penis is at rest. Dye-containing ointments may be of more use. An ointment containing ·5% trypaflavine for treating the prepuce and penis has been recommended, with 1 to 3 ounces of ·1% trypaflavine into the urethra as well; ·5% acriflavine or 5% lactic acid has been recommended under the same conditions. Tests in vitro show dyes in high dilutions to be ineffective in killing trichomonads, and lactic acid is ineffective in a lower concentration than 2%. Iodine in a ·001% solution and M & B 693 in a ·01% are effective. A bull may be cast and under epidural anæsthesia the mucous membrane is exposed by gentle traction on the penis and the mucous membrane smoothed out before the application of the ointment.

In dealing with other causes of infectious abortion in cattle one must mention vibrio fœtus as another one which is often met with. Since Snyman (1937) first described the disease in South Africa, a number of outbreaks, but more often individual cases of abortion in cattle, have been diagnosed. Neither trichomoniasis or vibrionic abortion appears to have caused much trouble under South African conditions up to the present, but there is no doubt that they would be diagnosed more often if a more intensive investigation were made into cases of abortion not due to brucellosis.

Plastridge and Williams (1943) mention the lack of literature on the subject of vibrionic abortion, and put it down to the low incidence of the disease and the difficulty of diagnosing it. They met with 13 cases in nine brucella-free herds. Abortion occurred at 2 to 7 months, 10 of the cases occurring after the seventh month. They consider vibrionic abortion to be primarily a placentitis, and infection of the fœtal tissue as secondary, so cultures from the fœtal stomach may fail, though the agglutination titre of the cow's serum is positive. A reaction at 1 – 50 or less is considered negative, 1 – 100 suspicious and 1 – 200 or over positive. About 75% of positive reactors become negative within three months. Results of agglutination tests indicate that a relatively high incidence of positive reactors occurs, which may or may not be accompanied by a high abortion rate, depending on the stage of gestation and whether or not there had been previous infection.

Johnson and Graham (1945) mention the association of various organisms with abortion in cattle. Amongst these are C. pyogenes, hæmolytic streptococci, the avian type in tuberculosis, B. prodigiosus, Bicoli, various molds and fungi, and Listerella.

They record their finding of three different bacterial species in aborted fœtuses. In the first, a fœtus at the fifth month, S. choleræ suis var Kunzendorf was isolated from the stomach contents and fœtal organs. Cultures of the organism produced abortion when inoculated intravenously into a pregnant heifer. In the second case, Erysipelothrix rhusiopathiæ was isolated from the stomach contents and heart's blood of an aborted fœtus at the fifth month. Five abortions had occurred in the herd in two years. Heifers infected with the organisms carried their calves to full time, but one had a dead one from which the organism was isolated. In the third case, C. pyogenes was isolated from a five-months'-old fœtus. A culture inoculated into a pregnant heifer produced abortion, and the heifer died later from internal abscessation.

Bishop, Schatz and Canham (1943) described the isolation of S. enteritidis var Dublin from the stomach of a fœtus from a cow which had aborted at $7\frac{1}{2}$ months. There was some evidence that the organism was associated with cases of abortion and temporary sterility in a herd of cattle in Natal.

Canham (1937) described a few cases in a pedigree Ayrshire herd where abortion was due to tuberculosis infection.

Bert (1943) described frequent cases of S. enteritidis var Dublin infection with abortion in a herd of cattle. The organism was found in a fœtus from a herd where it was difficult to get the cows in calf. Brucellosis was absent. Five cows in the herd were found to be excreting S. enteritidis var Dublin, and three of them had recently aborted.

Summary.

In this article the author has drawn attention to the importance of certain organisms as the cause of abortion in cattle where brucellosis has been excluded. In particular a plea is made for the more intensive investigation of conditions in cattle associated with sterility, such as vaginitis, endometritis, pyometra, etc., in order to see whether there is any association with trichomonas infection in particular.

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AN EPIZOOTIC AMONG CROCODILES.

A. D. THOMAS,
Onderstepoort.

A heavy mortality of crocodiles in Lake Rukwa was reported by the Senior Veterinary Officer, Southern Highlands Province, Tanganyika Territory, in October, 1943. Although a few formol fixed specimen were submitted at the time, it was hoped to obtain more exact information and material suitable for bacteriological study at a later date. Unfortunately this has not proved feasible, and lest the observation be lost entirely it is recorded in its present incomplete form.

Lake Rukwa occupies a deep, flat-bottomed trough situated between the southern end of Lake Tanganyika and the northern end of Lake Nyassa. It is fed by several small rivers and streams, but as it has no outlet to the sea, its waters, unlike those of the latter big lakes, are brackish, and fluctuate in level according to seasonal rains. This lake is stocked with an abundance of excellent edible fish. In fact there is a flourishing fishing industry here which supplies fresh fish to the Lupa goldfields near by, and dried fish to the Native trade. Natives come on foot from distances of 100 miles or more to obtain this dried fish, which is greatly relished as an article of food. This rich aquatic life is mentioned because it probably accounts for the large numbers of crocodiles which infest this lake and also for the very numerous birds to be found on and around it.

The information received about the dying crocodiles was very meagre indeed. It was reported that crocodiles in hundreds came out of the lake on to the beaches and among the sedges and reeds. Some were apathetic and disinclined to move and re-enter the water, others were more obviously sick and "gasping" or showing other respiratory distress, while many died causing an unbearable stench. The specimens received were from a single case shot while sick, and the illustrations give a fair idea of the lesions seen.

The most extensive type of lesion was a chronic indurative swelling or growth characterised by multiple nodules with light brown, crumbly, cheesy centres. Such lesions apparently were found in the skin

(fig. 3) or on the abdominal and thoracic organs. These nodules were embedded in more or less massive dense connective tissue which formed protruding swellings on the ventral skin, and adhesive growths on and between internal organs.

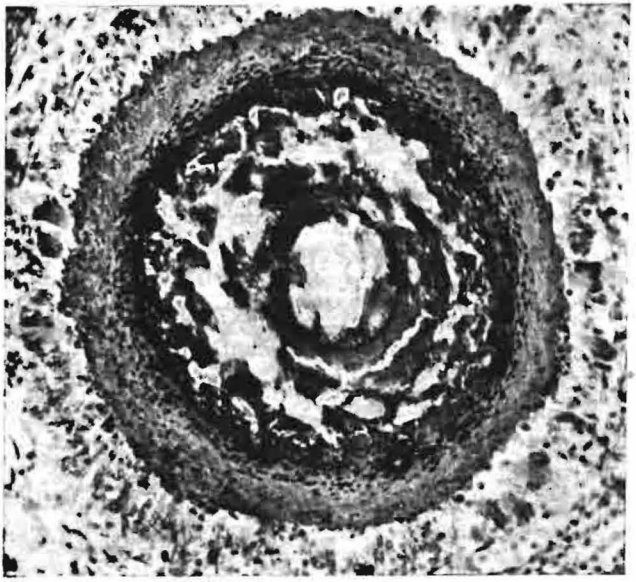

Fig. 1. — *Typical caseous nodule surrounded by palisade and giant cells.*

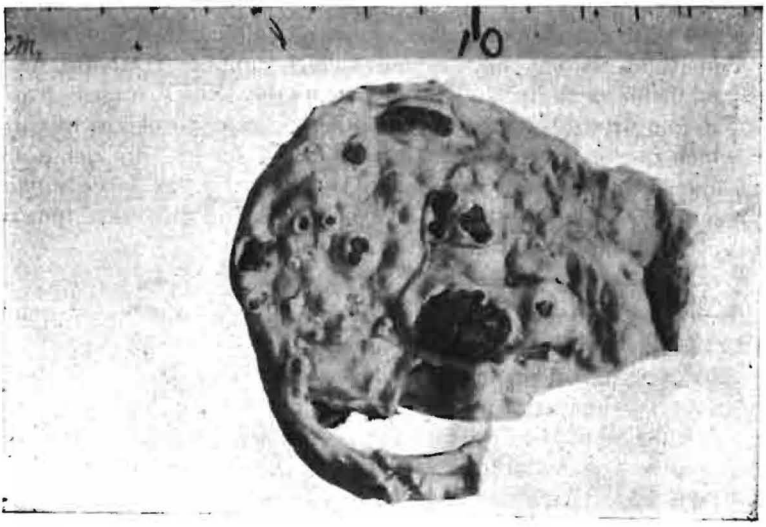

Fig. 2. — *Indurated mass from the abdominal cavity showing caseous centres.*

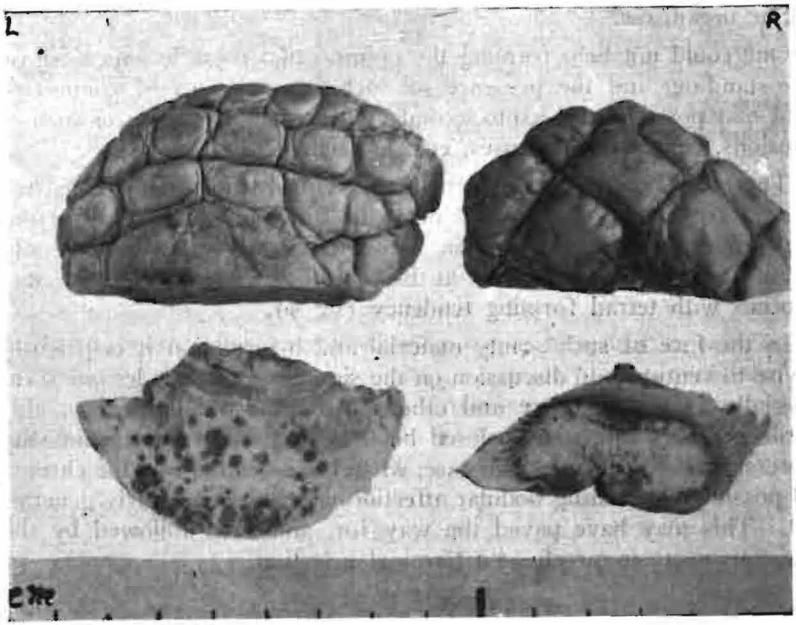

Fig. 3. — *Portions of abdominal scales and skin. Left: showing superficial and sectional appearance of indurative nodular lesion. Right: a necrotic centre.*

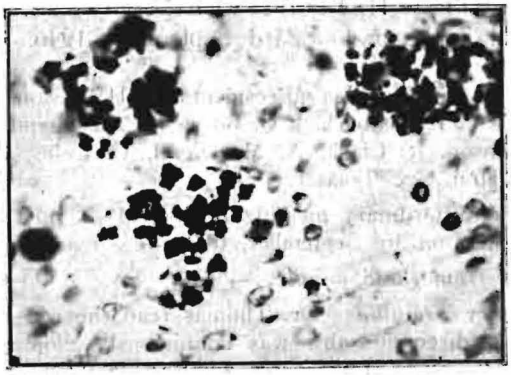

Fig. 4. — *Tetrad organisms seen in-tissue necrosis.*

The microscopic appearance of a typical nodule shows concentric layers of caseous matter consisting of closely packed dead cells and debris, the exact nature of which could no longer be identified. Surrounding this was a radially arranged layer of histiocytes and giant cells (so-called palisade cells) and outside that again, masses of connective tissue. The use of the special stains such as Ziehl-Nielsen

for acid fast bacilli and Gentian Violet for fungi failed to demonstrate specific organisms.

One could not help forming the opinion that these lesions were of long standing, and the presence of such large masses of connective tissue may possibly have led to secondary functional disturbances such as adhesions, contraction stenoses, etc., as well.

The other type of lesion seen in the ventral skin (fig 3) consisted of areas of active and recent necrosis, apparently spreading from breaks in the folds of soft skin between ventral scales. The only obvious micro-organism present at the edges of these necrotic areas was a coccus with tetrad forming tendency (fig 4).

In the face of such scanty material and information it is patently unwise to venture into discussion on the significance of the lesions seen, especially as the histology and other anatomical peculiarities of this reptile are very much of a closed book. Nevertheless, one gains the impression that the primary disease, whatever its cause, was the chronic and possibly debilitating nodular affection which seemed fairly generalised. This may have paved the way for, and been followed by the cutaneous necrosis merely as a terminal infection.

S.A.V.M.A.

COUNCIL MEETING HELD AT VELRA HOUSE, PRETORIA, at 7.30 p.m. on 23rd September, 1946.

Present: C. J. van Heerden (President). J. H. Mason, D. G. Steyn, A. D. Thomas, A. C. Kirkpatrick, J. G. Boswell, P. S. Snyman, R. Alexander, E. M. Robinson, R. Clark, A. M. Diesel, D. Coles, and S. W. J. van Rensburg (Hon. Sec.-Treas.).

(1) *Minutes* of Ordinary meeting held on 19th July, 1946, and of Special meeting held on 3rd September, 1946, were read and confirmed.

(2) *Arising from these minutes*:—

(a) *Veterinary Training*: Dr. Thomas read the report of the sub-committee. After discussion this was unanimously adopted and it was decided that the members of the sub-committee should discuss the report with (a) Faculty, (b) the Veterinary Board, (c) representatives of the Native Affairs Department, and (d) the Department of Agriculture.

(b) *Tuberculin Testing*: The report of the sub-committee was submitted, discussed and adopted. It was decided that this be sent to the Director of Veterinary Services and that the reply of the Director be considered by the Committee before it is submitted to Council.

(c) *National Health Council,* and *Health Foundation:* Drs. R. Alexander and J. Quin respectively were appointed as the Association's representatives on these two bodies.

(d) *Memorandum to Prospective Students*: The Secretary explained the difficulties experienced by the sub-committee in drawing up a suitable memorandum and sending it out within the prescribed period. After discussion it was decided that no further action be taken in this matter.

(3) *New Members*: Acceptance of the following is to be recommended to the General Meeting: J. F. Brownlie, J. D. Daly, J. Louw and N. R. Reid.

(4) *Resignation*: The Secretary was instructed to ask Dr. E. K. Mager to reconsider his decision to resign.

(5) *Votive Cards*: It was unanimously decided to print 1,000 votive cards and to send these to members with a request that such a card be sent whenever it is necessary to condole with friends or relatives in a bereavement and that instead of a wreath a donation be sent to the Benevolent Fund.

(6) *Council Vacancy*: Dr. M. C. Lambrechts was elected to fill the vacancy created by the election of Dr. A. M. Diesel to the Vice-presidency.

(7) *Standing Committees*: The following were elected:—

EDITORIAL: E. M. Robinson (Editor), P. J. du Toit, R. Clark, C. Jackson and B. S. Parkin.

FINANCE: R. Alexander (Convener), B. S. Parkin and A. D. Thomas.

LIBRARY: D. Coles (Convener), E. M. Robinson and W. D. Malherbe.

GENERAL PURPOSES: P. J. J. Fourie (Convener), R. Alexander, A. M. Diesel, P. S. Snyman and A. C. Kirkpatrick.

BOOK FUND: W. D. Malherbe (Convener), A. D. Thomas and D. Haig.

(8) *Finance*: Dr. Alexander presented the auditor's report for 1945-46. This was approved.

(9) *Secretarial Assistance*: It was decided that the Finance and Editorial Committees consider the avisability of cutting down expenditure on the Journal, for instance by discontinuing the supply of reprints free of charge, in order to increase the present salary of the typist. The latter will then be required to do all the bookkeeping in addition to the typing.

(10) *General*: (a) *Time of Meeting*: Decided that Council meetings in future commence about 2 p.m.

(b) *S.P.C.A., Johannesburg*: Letter re appointment of veterinary surgeon read. Decided that Drs. Mason and Kirkpatrick discuss the matter with the Secretary of the S.P.C.A.

(c) *Advertising*: An alleged case of advertising by a veterinarian was referred to the Board.

The meeting adjourned at 11.30 p.m.

S. W. J. van Rensburg,
HON. SEC. AND TREAS., S.A.V.M.A.

SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION
41st GENERAL MEETING,
held at Onderstepoort on 24th and 25th September, 1946.

Present: C. J. van Heerden (President), J. H. Mason, G. D. Sutton, B. J. Brummer, A. D. Thomas, V. Cooper, D. Coles, J. R. Scheuber, A. S. Canham, P. S. Snyman, J. S. Watt, J. L. Dickson, N. T. van der Linde, A. Matthew, G. McIntyre, W. J. Rijksen, R. Clark, J. R. Frean, F. B. Wright, G. J. de Wet, J. Zwarenstein, B. M. McIntosh, L. T. Edwards, J. Quinlan, O. T. de Villiers, J. Thorburn, L. L. Daly, A. M. Diesel, N. C. F. Steenkamp, E. B. Kluge, D. E. Truter, J. S. van Heerden, P. P. Hugo, D. J. Louw, Jac. Louw, T. C. W. Wessels, J. P. van der Merwe, P. G. Joubert, I. P. Marais, W. G. van Aswegen, W. O. Neitz, J. D. Daly, F. J. D. Hempstead, W. C. Viljoen, M. H. V. Brown, L. Stonier, C. H. Flight, G. Bishop, J. H. R. Bisschop, M. W. Henning, R du Toit, N. C. Starke, A. C. Kirkpatrick, J. H. Viljoen, M. de Lange, W. D. Malherbe, J. I. Quin, E. M. Robinson, L. W. Rossiter, B. S. Parkin, J. M. de Wet, P. H. Brown, P. J. du Toit, R. E. Hartig, E. C. S. Dawe, H. G. Franz, B. C. Jansen, H. H. Curson, H. P. A. de Boom, N. Barrie, W. J. Wheeler, H. H. Sigwart, J. G. Boswell, J. Nicol, R. A. Alexander I. Mowat, J. G. Townsend, J. W. A. Brookes, Campbell Dickson, H. P. Steyn, R. Painter, G. de Kock, J. H. Schoeman, A. J. Louw, T. N. Osborn, H. N. Botha, W. G. Barnard, G. C. van Drimmelen, C. F. B. Hofmeyr, M. C. Lambrechts, P. R. Mansvelt, S. W. de Villiers, J. D. Smit, J. G. Williams, A. A. Albertyn and S. W. J. van Rensburg (Hon. Sec.-Treas.).

Apologies for Absence: S. T. Amos, J. G. Keppel, N. F. Viljoen.

Obituary: A motion of condolence with relations of the following members who had died during the year was passed, viz., J. Chalmers, W. A. Elder, M. M. Neser, W. Orr and C. M. Sharpe.

Minutes of General Meeting held on 23rd and 24th October, 1945, weer confirmed.

New Members: The following were accepted: H. N. Botha, J. G. Brandsen, J. F. Brownlie, W. H. B. Buhr, J. D. Daly, G. J. J. du Preez, A. S. Erasmus, A. Grist, J. Louw, T. B. Nel, B. T. Paine, N. R. Reid, J. A. Schutte and J. P. van der Merwe.

Resignations: J. I. Taylor accepted.

Election of Council: The following were declared elected for 1946-47:

President: J. H. Mason.

Vice-President: A. M. Diesel.

Hon. Secretary-Treasurer: S. W. J. van Rensburg.

Members: R. Alexander, J. G. Boswell, P. J. du Toit, A. C. Kirkpatrick, M. C. Lambrechts, P. S. Snyman, D. G. Steyn and A. D. Thomas.

Opening: The opening address was delivered by the Secretary for Agriculture, Dr. C. H. Neveling.

Presidential Address: After delivering his address, the retiring President, Colonel C. J. van Heerden, vacated the chair, which was then taken over by Dr. J. H. Mason.

Standing Committees: The reports for the various committees for the current year were presented and approved.

Exhibits: The President drew the attention of members to the exhibits displayed by the following firms, viz., Agricura Laboratoria, Bayer Pharma, Cooper and Nephews, Maybaker, Roche Products, Scherag, S.A. General Electric Company and Westdene Products.

Papers: After the tea interval, the following papers were submitted and discussed:—

11 a.m.: The present campaign against tsetse flies in Zululand: R. M. du Toit and E. B. Kluge.

12 noon: Supplementation of winter feed for sheep: R. Clark and J. I. Quin.

2 p.m.: Immunization of sheep against blue-tongue, with special reference to the use of embryonated egg culture virus: R. Alexander.

3 p.m.: (a) Toxicity of species of Eucalyptus, (b) Myoglobinuria, Kimberley Horse Disease or "Bewerasie": D. G. Steyn.

Wednesday, 25th September:—

9 a.m.: Film: Ruminant digestion; Demonstration: The application of the Stader Splint (by courtesy of the South African General Electric Company).

10 a.m.: Parasympathetic control of body function and its role in veterinary practice: J. I. Quin and R. Clark.

11.15 a.m.: Some problems in relation to lamsiekte: E. M. Robinson.

12 noon: Some Union Act provisions of interest to veterinarians: A. M. Diesel.

2 p.m.: Calfhood mortality in the Northern Transvaal: N. T. van der Linde.

Resolutions, 3 p.m.: The following were passed unanimously:—

(1) This meeting of the S.A.V.M.A. views with concern the findings of the Centlivres Commission of Inquiry as published in its Fourth Report relative to the salary scales of veterinary officers, and instructs its Council to take any action it thinks fit to remedy the position in the interests of the whole veterinary profession.

(2) This meeting suggests to Council the appointment of a Publicity Committee whose duty it shall be to instruct the public in general, as well as various organised bodies interested in the animal industry, regarding the true position of animal health problems in South Africa, and the requirements for an adequate veterinary service commensurate with those problems.

(3) This meeting wishes to convey its thanks to the following bodies for the efforts they have made to get an improvement in the scale of salary of veterinary officers, viz., S.A.A.U., Cape Eastern Agricultural Union, O.F.S. Agricultural Union, Transvaal Agricultural Union, Shorthorn Society of South Africa, Winter Rainfall Area Association, S.A. Woolgrowers' Association.

The meeting closed at 4.30 p.m. with a vote of thanks to the President.

S. W. J. van Rensburg,

HON. SEC.-TREAS., S.A.V.M.A.

MOVEMENTS OF OFFICERS.

Dr. J. P. van der Merwe from Umtata to Vryburg, 2/12/46.
Dr. F. W. Langbridge from Vryheid to Ladysmith, 13/11/46.
Dr. J. J. Zwarenstein from Port Shepstone to Eshowe, 25/11/46.
Dr. G. C. van Drimmelen from Bloemfontein to Onderstepoort, 17/12/46.
Dr. N. T. van der Linde from Grahamstown to Bloemfontein, 2/12/46.
Dr. M. Bergh from Durban to Johannesburg, 30/11/46.

NOTES.

Dr. C. C. Wessels, who was in private practice at Krugersdorp, has been appointed City Veterinary Medical Officer to the Durban Corporation.

Dr. U. van Backström, who was in the service of the Bechuanaland Protectorate, has started in private practice in Krugersdorp.

OBITUARY.

THEODOR LUDWIG ZIEHN.

With the passing away of Dr. Ziehn on November 9th, 1946, at the age of 77 years, the Association has lost another of its stalwart pioneers. He was a man imbued with a high regard for the profession of his choice. He faced the many difficulties and hardships of pioneering in this field — and achieved success.

Ziehn was born and educated in Germany and attended the Royal Veterinary School at Hanover. Unfortunately owing to straitened circumstances he was unable to complete the final qualifying examinations. At the age of 29 he came out to South Africa and had been here only a short time when he met Theiler, then Veterinarian to the Transvaal Government. As the latter was due to take six months' leave in Europe, he was glad to entrust his charge, the artillery remounts, to the care of Ziehn.

After the Boer War, Ziehn started private practice in Pretoria on his own account and has continued to build it up ever since.

In 1905 he married Miss Helena Thoms, who has stood by his side and assisted him very ably in his work.

Of a retiring disposition, Ziehn did not take a very active part in veterinary politics, but he proved himself a very worthy member of the Association. For many years, in conjunction with the S.P.C.A., he conducted a free clinic for the animals of the poorer non-Europeans of Pretoria.

To Mrs. Ziehn and her two sons we extend our deep sympathy in their loss.

ERRATUM.

Vol. 17 No. 3. September, 1946.

On page 172, " Diabetes mellitus in a Dog " the word " rumination " should read " urination."

AUTHOR INDEX.

SUBJECT INDEX.

TWO MODERN STOCK REMEDIES NOW AVAILABLE

World-wide research by Imperial Chemical (Pharmaceuticals) Ltd., led to the production of a new range of Stock Remedies. Two of these are now available, and are of particular interest to the Veterinary Profession.

SULPHAMEZATHINE BRAND OF SULPHADIMETHYLPYRIMIDINE. — This preparation has been used generally in the same directions as sulphanilamide and sulphapyridine, and has proved oustandingly successful against **pneumonia, meningitis, hæmolytic streptococcal infections and** *B. coli* **infections of the urinary tract.** Its chief advantages are: (1) it is less toxic than the other sulphonamides and higher blood concentrations can, therefore, be maintained; (2) the acetylated derivative is more freely soluble and, therefore, the risk of deposition of crystals in the kidneys and urinary tract is much less than with other sulphonamides; (3) it is well tolerated by animals and does not cause nausea or vomiting.

"Sulphamezathine" is indicated in the prevention and treatment of **cæcal coccidiosis in chickens.** In the course of well-established epidemics of the disease considerable protection is afforded and losses are greatly reduced. It is also effective against coccidiosis of adult fowls, pigeons and rabbits.

Veterinary surgeons may obtain sulphamezathine at 3/11 per container holding 25 x 0.5 gramme tablets.

"AVLON" BRAND OF NEUTRAL PROFLAVINE SULPHATE. —Laboratory tests of neutral proflavine sulphate against *Staphyloccus aureus, Streptococcus pyogenes* and *Pseudomonas pyocyanea* have shown it to be more effective than either acriflavine or proflavine sulphate. Similar tests against *Bact. coli* indicated that the activity of the three products is about equal.

Neutral proflavine sulphate is the flavine of choice as **a general purpose antiseptic and is indicated for all types of wounds and burns at a strength of 1/1,000. In contaminated wounds** the direct application of neutral proflavine sulphate powder or neutral proflavine sulphate with sulphanilamide is a rational treatment which produces excellent results uncomplicated by the risk of local reactions.

For infusion of the udder in **bovine mastitis** neutral proflavine sulphate is preferred to any of the other flavines as it is somewhat **more active against the ordinary udder pathogens and its administration is not followed by any undesirable local effects.**

This preparation is sold in containers holding 25 x 0.875 grain tablets at 1/9 to Veterinary Surgeons, and containers holding 100 similar tablets at 5/3.

In addition to the above, we shall shortly be able to supply the following I.C. (P). products: **'Cetavlon'**, an antiseptic detergent; **'Diaquone'**, a purgative; **Phemitone B.P.**, an antiseptic; **Pholedrine**, circulatory stimulant and restorative; **Procaine Hydrochloride B.P.**, an anæsthetic; **Sulphonamide E.O.S.**, an anti-bacterial; **Tetrachlorethylene B.P.C.**, an anthelmintic; **Trypan Blue**, a piroplasmocide. **Phenothiazine** is already available.

Cooper & Nephews, S.Af. (Pty) Ltd.

Distributors of I.C. (P.) and other Stock Remedies,

Dips and Insecticides based on "Gammexane," etc.

P.O. Box 2963, JOHANNESBURG.