year's treatise, also appears as a champion of tuberculin ; for diagnostic purposes he still considers it to be the finest and safest means for diagnosing tuberculosis in animals, in spite of the many opinions to the contrary.

We must point out the fact that the present scientific data with regard to the extermination of bovine tuberculosis clearly show this to be practically impossible without the aid of tuberculin. Naturally we must, in the first place, make a point of exterminating the cows affected with udder tuberculosis and general tuberculosis, since their milk is, more than all other, to be regarded as infectious. In the second place come the animals in which the clinical diagnosis can only be established by the help of tuberculin. These cows must first of all be separated from the healthy ones. The question whether they should be killed, and the proper time for doing so, will depend on the quicker or slower progress of the tuberculous disease. That is to say that, of course, not all the animals which react to tuberculin need be immediately slaughtered. Therefore we may say that, combined with the clinical and bacteriological examinations, the tuberculin test furnishes us with the safest means of obtaining milk free from tubercle bacilli as well as of rearing cattle free from tuberculosis.

GLANDERS.¹

By WM. HUNTING, F.R.C.V.S., London.

TO an assembly of veterinary practitioners it would be a waste of time to attempt an exhaustive essay on this disease. We are all well acquainted with its nature, its methods of spread, and with the heavy losses it has caused.

The object I have in view to-day is to demonstrate that the loss of human and equine life due to glanders will continue so long as the obsolete regulations of the Board of Agriculture are unaltered ; and to indicate how the disease may be economically and certainly stamped out by Orders based upon common sense and modern veterinary knowledge.

It has been said by the President of the Board of Agriculture that the information possessed by veterinarians is insufficient to warrant more stringent measures, based upon their advice, being enforced. Mr Hanbury's excuse for this statement is a paragraph in the Report of the Departmental Committee of 1899: "We feel that further research is needed on some of the most elementary facts of the disease before our prevention or treatment can be formulated with scientific exactness." It is perhaps unfortunate that an abstract opinion of this sort should have appeared in the report, but I venture to think that not more than one member of the Committee ever imagined that such a truism could have been used as an excuse for delay. The same words might be applied to every disease of animals that has been stamped out by State regulations, and to every disease of man that has been scheduled for control by the Local Government Board. If the Committee had believed that their pious opinion was a bar to

¹ Read at the Annual Meeting of the National Veterinary Association, London, 1902.

further legislation they would doubtless have refrained from recommending any change. They, on the contrary, recommended a number of alterations in the existing regulations—not one of which has been adopted by the authorities.

These considerations oblige me to give some details concerning glanders which otherwise I might have considered superfluous. This course will at any rate show that we are not altogether ignorant of some of the most elementary facts of the disease, and will form a basis for conclusions as to the best method of stamping out glanders.

History.

Glanders was recognised by Hippocrates about 450 B.C., and by Aristotle a century later. It was described by Apsyrtus in the fourth century of our era, and by Vegetius in the fifth. In 1618 the celebrated Italian anatomist Ruini described glanders, and declared it to be "a general disease and incurable." In 1682 Soleysel recognised it as a contagious disease. In 1734 Gaspard de Saumier advised that to stay the spread of glanders "the best practice was to kill quickly." In 1750 Bourgelat—the father of veterinary science—recognised the disease as contagious, but Lafosse wrote that the seat of glanders was in the nose, and that it was not contagious. For a century this question of contagion or non-contagion divided French veterinarians, and the two great schools of Paris and Lyons took opposite views, Lyons holding to contagion, Paris to non-contagion. Even so late as 1840 that great authority Professor Bouley taught that glanders might arise otherwise than from contagion. Viborg, of Copenhagen, in 1797 showed that there was a specific virus in the purulent discharges of horses suffering from glanders and farcy, that farcy and glanders were one disease, that the virus could be destroyed by heat and dessication, and that infection could be carried by harness or utensils, and more particularly by forage left in the mangers by diseased horses. It is sad to think of the losses which have since occurred by neglect of the work done by this Danish veterinarian. Among Continental veterinary surgeons the first to assert the exclusively contagious nature of glanders was Gerlach.

In Great Britain no veterinary writer of the eighteenth century has left any account of glanders worth notice. We only know that the disease prevailed widely and was not understood. White, who wrote in 1834, gave a very true description of the disease, and although he did not positively deny the possibility of the disease developing in some other way than as the result of contagion, he clearly held that contagion was the only cause. Blaine in 1841 asserted that "both glanders and farcy originate in contagion, and that the disease is one *sui generis*." Percival in 1858 published a series of volumes on *Hippo-pathology*. His article on "Glanders and Farcy" extends over 200 pages, and embodies all that was known at that time. He says: "I have no more doubt of glanders being a contagious disease than I have of syphilis or small-pox or itch being contagious." He quotes Professor Sewell as saying, in 1828, that "the lungs are the original seat of glanders, and the affection of the nose secondary." This opinion was not generally accepted till many years later, the majority of veterinarians looking upon glanders as a disease of the nasal membranes. The fact that glanders was a specific disease due only to

contagion was not generally accepted till after 1876. I remember in 1873 reading a paper before the Central V.M.S. in which I advanced that view, and failed to obtain the support of any other member. It is true that since 1840, when experiments made at Alfort determined beyond the shadow of a doubt that glanders was contagious, very few men have denied contagion as one cause; but many have held that it might be produced by other causes, as, for instance, by the introduction into the system of a healthy horse of purulent fluid, or by changes in the blood resulting from defective hygienic conditions.

After Pasteur had shown that every living organism arose only as the result of a similar previous organism, the theory of spontaneous generation of contagious diseases rapidly died out. In 1882 Löffler and Schütz discovered and isolated the specific organism of glanders, and so removed all doubt as to the cause. Much useful and indispensable work on the pathology of the disease was contributed by Rayer, Leblanc, Bouley, Leisering, and Chauveau, and more recently by Schütz, Nocard, and M'Fadyean.

Prevalence.

Glanders prevails over the whole world except, perhaps, in Australasia. Europe has been infected for many centuries. In China and India the disease prevails widely. In America glanders exists all over the United States and in some portions of Canada. South Africa is now reeking with the disease, which has been imported by horses from Great Britain, Canada, and the United States.

In Great Britain glanders is distributed all over the country, but chiefly confined to the large towns. Before the introduction of railways the coaching roads were hot-beds of glanders, and the disease was disseminated widely through posting establishments and carriers' stables. It is quite safe to say that fifty years ago we had twenty times more glanders than now. The decrease is due to the better knowledge of the disease possessed by veterinarians and horse-owners, assisted by the regulations in force for its control by local authorities.

At the commencement of the nineteenth century the British Army suffered frightfully from glanders, and Professor Coleman of the Royal Veterinary College was given a position as Chief Veterinary Adviser to the Cavalry. His efforts were successful in greatly reducing the prevalence of the disease, but it was not till 1891 that glanders was stamped out of the Army, when Dr George Fleming was Director of the A.V.D. For eight years the Army remained free, but the troubles in and connected with South Africa caused it again to become infected. The constant danger to the Army of permitting the prevalence of glanders in large towns has always appealed to me as a strong argument for stamping it out in civil stables, but, so far as I am aware, no word of warning or protest has ever been uttered by an Army man.

No statistics as to the prevalence of glanders are available before the year 1870, and the returns for that year made to the Privy Council under the Contagious Diseases (Animals) Act of 1869 are quite misleading; not a tenth of the cases existing were reported. In 1874 the returns became a little more exact by the inclusion of farcy. By about the year 1881 I think the returns began to fairly represent the

prevalence of the disease, and we may now accept the annual figures of the Board of Agriculture as practically correct.

The following table shows number of horses attacked in each year since 1881. The total loss is 35,651 horses, which, at £20 each, represents a value of £713,020. Most of this might have been saved by proper regulations.

TABLE I.—*Horses Attacked with Glanders in Great Britain.*

<i>Year.</i>	<i>Number Attacked.</i>	<i>Year.</i>	<i>Number Attacked.</i>
1881 . . .	2,110	1892 . . .	3,001
1882 . . .	1,389	1893 . . .	2,133
1883 . . .	1,244	1894 . . .	1,437
1884 . . .	1,123	1895 . . .	1,594
1885 . . .	946	1896 . . .	1,294
1886 . . .	1,114	1897 . . .	1,629
1887 . . .	1,482	1898 . . .	1,385
1888 . . .	1,581	1899 . . .	1,472
1889 . . .	2,246	1900 . . .	1,858
1890 . . .	1,808	1901 . . .	2,370
1891 . . .	2,435		
			<hr/> 35,651

It will be noticed that the prevalence of the disease in 1901 is rather greater than in 1881. The conclusion irresistibly suggested is that at the end of another, twenty years, unless different regulations are enforced, we shall continue to lose an average of 1700 horses per annum.

It is interesting to see how glanders, like other contagious diseases, fluctuates. The following chart shows exactly how the disease has fallen and risen during the last twenty years. The left hand column commences with the numbers 3200, and decreases by 200 on every line till 1000 is reached.

In 1881 there were 2110 cases reported. The lowest point was reached in 1885 with 946 cases, and the highest in 1892 with 3001 cases. From this it dropped in 1896 to 1294, and has risen again last year to 2370.

In 1880-81 a virulent epidemic of influenza prevailed among horses all over the kingdom. In 1891-92 influenza again prevailed. When we remember how an acute febrile disease has the effect of developing latent glanders, and thus converting non-infective into actively infective animals, there seems reason to connect the prevalence of influenza with the rise in the number of horses attacked in 1881 and 1892. The increase shown in 1900 and 1901 cannot be so explained, as no specific febrile disease has been unusually prevalent among horses during the last few years. The use of mallein has, during the last three or four years, added to the number of horses diagnosed as glandered; and the movements of horses due to the South African War has increased the facilities for the transmission of the disease. The fall of glanders is more easy to explain than its rise. Whenever a disease prevails more widely than usual it attracts attention, and the persons interested become more alert, measures of prevention and control are more energetically and carefully applied, with the result

that a decrease of the disease is soon evidenced. This decrease usually lasts for a year or two, then less care is taken and a rise

TABLE II.—*Chart Showing Fluctuations of Glanders in Great Britain.*

follows. The extreme rises are, however, due to positive conditions which increase the amount of contagion or facilitate its transmission.

In Table III., copied from the Annual Government Returns, I

TABLE III.—*Prevalence in Counties—Ten Years.*

<i>Counties.</i>	1892	1893	1894	1895	1896	1897	1898	1899	1900	1901
Bedford	—	7	—	1	1	5	6	1	2	4
Berks	1	—	8	2	1	—	1	2	1	4
Buckingham	2	4	10	2	1	2	12	5	5	8
Cambridge	3	5	4	16	—	—	—	12	13	14
Chester	10	10	5	21	11	6	5	6	2	2
Cornwall	—	—	—	—	—	—	—	—	—	—
Cumberland	—	—	—	4	13	—	—	—	—	1
Derby	—	—	—	2	—	2	2	22	3	1
Devon	1	—	—	—	—	1	—	—	—	1
Dorset	—	—	4	—	1	—	—	—	1	—
Durham	1	2	1	9	28	8	—	7	3	1
Essex	70	55	52	81	49	57	49	62	75	119
Gloucester	—	6	—	—	3	22	—	—	—	3
Hants	12	6	7	8	—	1	2	3	8	2
Hereford	—	—	—	—	—	—	—	—	—	3
Hertford	14	28	19	8	18	11	12	11	8	16
Huntingdon	4	2	13	1	—	—	—	—	—	—
Kent	26	28	19	18	21	24	8	12	39	51
Lancashire	14	24	10	44	52	34	24	38	14	21
Leicester	—	—	—	—	—	1	—	1	—	—
Lincoln	2	3	—	—	1	—	1	1	1	1
London	2526	1619	093	104	2845	966	800	896	1387	1828
Middlesex	121	121	82	109	52	73	94	77	96	93
Monmouth	—	—	—	6	—	—	—	—	—	—
Norfolk	4	3	3	3	—	15	14	10	39	6
Northampton	—	4	—	6	1	—	2	1	13	6
Northumberland	—	2	—	1	21	3	—	16	19	2
Notts	1	1	—	—	1	15	—	1	—	1
Oxford	7	1	1	—	—	—	—	2	1	—
Rutland	—	—	—	—	—	—	—	—	—	—
Salop	—	1	—	1	1	—	—	—	—	1
Somerset	1	—	—	—	—	—	—	2	6	1
Stafford	11	12	8	22	3	1	1	1	8	5
Suffolk	4	—	6	1	—	9	1	7	—	6
Surrey	35	45	16	23	26	20	9	20	22	17
Sussex	4	8	6	11	5	7	3	19	8	44
Warwick	14	42	16	32	19	21	21	15	15	15
Westmoreland	2	2	—	1	—	—	—	3	—	—
Wilts	2	1	—	1	—	—	2	—	—	7
Worcester	3	4	3	—	2	4	1	3	3	—
Yorks	21	12	14	11	20	16	3	13	—	3
Wales	6	4	2	1	—	8	1	—	—	2
Scotland	79	71	134	106	98	297	251	203	41	64
(<i>Lanark</i>)	77	60	129	98	96	279	245	199	41	41
										1344
										1265

have attempted to show the distribution of the disease in England, Scotland, and Wales. We may leave Ireland out of consideration, as glanders there only appears in sporadic outbreaks when an infected horse is taken into the country.

The table shows clearly that glanders is mostly prevalent in large towns where numbers of horses are crowded together and where facilities for contact are greatest. From the great centres infection is carried to surrounding districts and occasionally into distant places which suffer small outbreaks and then are free for years—until disease is again imported from permanent centres of infection.

Cumberland, Devon, Dorset, Huntingdon, Leicester, Lincoln, Monmouth, Notts, Oxford, Salop, Somerset, Westmoreland, and Wilts show only occasional and rare outbreaks, in which the animals attacked number perhaps one or two, and then the disease disappears for some years—until again introduced. Cornwall and Rutland seem to have escaped infection altogether, as did Hereford until last year. London is the great centre of disease, and the surrounding counties naturally suffer from the infection which is constantly introduced from outside their boundaries.

The following table shows at a glance the prevalence of glanders in and around London, and the relative proportions of the disease in the metropolitan district and the rest of England.

TABLE IV.—*Prevalence of Glanders in London and Surrounding Counties compared with Rest of England.*

<i>Year.</i>	<i>London.</i>	<i>Middlesex.</i>	<i>Essex.</i>	<i>Kent.</i>	<i>Surry.</i>	<i>Hertford.</i>	<i>Sussex.</i>	<i>Total.</i>	<i>Total in whole of England.</i>	<i>Per cent.</i>
1895	1042	109	81	18	23	8	11	1292	1487	86·8
1896	845	52	49	21	26	18	5	1016	1196	84·9
1897	966	73	57	24	20	11	7	1158	1324	87·4
1898	860	94	49	8	9	12	3	1035	1133	91·3
1899	896	77	62	12	20	11	19	1097	1269	95·5
1900	1387	96	75	39	22	8	8	1635	1814	95·6
1901	1828	93	119	51	17	3	44	2155	2304	93·5

In the seven years no less than 89 per cent. of all the glanders in England existed in London and the surrounding counties, a fact which suggests that some special measures might with benefit be applied to this constantly infected district.

If we look back at Table III. we find that other large towns, and counties which include manufacturing centres, must be looked upon as permanent centres of infection. During the last ten years Lancashire has averaged over thirty cases per annum. Warwick is nearly as bad, whilst Cheshire, Durham, Stafford, and Yorkshire have never been quite free.

Of the forty-one counties in the table which had returned cases of glanders during the last ten years, no less than twenty-five have had periods of freedom from the disease extending over at least one year, and in many of them over a series of years. In other words, twenty-five counties have succeeded in stamping out glanders, and thus demonstrating that there is no insuperable difficulty for any other

district doing the same. The difficulty is not so much in stamping out an outbreak as in preventing its invasion from infected areas.

In Scotland glanders is practically confined to Lanarkshire. The returns for Scotland for ten years show 1344 horses attacked, of that number no less than 1265 occurred in Lanark.

Seasons.

The prevalence of glanders varies not only year by year but month by month, and the following table shows the fluctuations in London for five years. From July to October inclusive we find the greatest amount of disease, and from February to April the least.

TABLE V.—*To show Prevalence of Glanders in London by Month or Season.*

	1897	1898	1899	1900	1901	Total.	Average per month for five years.
January	69	70	69	105	179	492	98'4
February	64	59	44	121	129	417	83'4
March	81	60	63	111	126	441	88'2
April	83	63	61	116	124	447	89'4
May	71	83	56	120	161	491	98'2
June	76	93	61	91	129	447	89'4
July	99	82	82	110	164	537	107'4
August	86	76	83	136	196	577	115'4
September	103	77	124	132	173	609	121'8
October	102	70	77	110	159	518	103'6
November	75	59	107	121	143	505	101
December	71	53	78	114	166	482	96'4

The explanation I offer of this fluctuation is that the hot months of the year are the most exhaustive, and that fatigue causes premature development of the disease which might otherwise have continued latent in its pulmonary form. I assume that all visible cases of glanders have previously suffered from nodules in the lungs for at least two or three, and possibly for twelve months. I know that a febrile condition, especially when accompanied by pulmonary congestion, frequently determines generalisations of the disease, and then the clinical symptoms appear. The summer and autumn seasons are those in which unfavourable conditions for the inhibition of glanders prevail.

Methods of Spread.

Experience has shown over and over again that if a visibly glandered horse be kept in a stable with other horses the disease will spread. No sane man wilfully tries this experiment—many make it unwittingly through not distinguishing between a comparatively harmless nasal discharge and one due to glanders. Given a glandered horse with an infective discharge the disease is spread to other

horses through nosebags, pails, mangers, or any other intermediate objects which may convey the active virus to the healthy horse. An open water trough in a stable or stable-yard at which glandered and healthy horses are allowed to drink is a fertile cause of the spread of disease.

There are two ways in which glanders does not spread in the stable, (*a*) by germs floating in the air of the stable, (*b*) by mysterious horses showing no signs of disease but constantly infecting others. If the floating germ were a method of spread it is impossible to explain the escape of horsekeepers passing twelve hours a day in infected stables. The floating germ theory was invented to account for the fact that in an infected stud cases are found in various parts of the stable. The fact is more easily accounted for by contagion through nosebags, mangers, and pails, and by the constant shifting of horses from one part of a stud to another.

The mysterious horse showing no signs of disease that works and eats month after month, whilst horses alongside are infected and destroyed by contagium from it, is an ancient, crusted legend. It dates from the days when glanders without a nasal ulcer was not acknowledged, and when an enlarged gland, a chronic cough, or an intermittent nasal discharge were not considered symptoms of glanders. It has fallen to my lot to investigate many outbreaks of glanders, and very seldom have I not been able to pick out four or five horses showing some clinical symptoms which had been overlooked by the owner and attendants. I venture to assert that a horse showing no sign of glanders is a non-infective animal even though his lungs be studded with nodules. Such a horse though non-infective is dangerous, because he may develop clinical symptoms very suddenly, and among them may be one accompanied by an escape of active bacilli.

The spread from stable to stable takes place through the introduction of infected horses, which have not developed visible signs of disease, but which subsequently become clinically diseased and infective. In large studs having many different stables glanders is spread by the constant changes of horses from stable to stable to suit the exigencies of business. When once infection is introduced to such a stud, the changes of horses spread the disease to every stable unless very careful measures are taken to prevent it. Newly purchased horses are another means by which glanders is brought into a stable. Purchasers who confine themselves to young horses from the country seldom introduce glanders, but men who buy aged town horses are never long before they have the misfortune to get an infected horse. Only a mallein test of all new purchases can protect buyers against the purchase of a latent case. Whilst it is possible by the aid of mallein to guard against the introduction into a stable of an undeveloped but infected glandered horse, it is impossible to guard against accidental infection from outside. A horse put up at livery may eat out of a manger which has been used by a glandered horse. A healthy horse may stand next to a diseased one in a shoeing forge, or in a railway horse-box. Drivers of horses may change nosebags and one of the horses may be glandered. Horses may be turned out to grass in the autumn and there meet with an infected animal. I once saw five visibly glandered horses in a field

with thirty others. Probably those animals belonged to ten different owners, and ten different stables would ultimately become infected.

TABLE VI.—*Return showing the Incidence of 1000 Cases of Glanders in the County of London during April to November 1901.*

<i>Occurrence.</i>	<i>Number.</i>	<i>Per cent.</i>
Cases occurring in studs where there has been continuous infection for five or more years	389	38·9
Cases occurring in studs where there has been occasional infection during three years	285	28·5
Cases occurring in studs where there has been no previous case	326	32·6
	1000	.

From this table I infer that 67 per cent. of glanders in London occurs as the result of continuous infection in studs which are never clear of the disease. The 32 per cent. of cases which are found in studs where no previous case has been detected are more difficult to explain. They are nearly all due to the purchase of horses suffering from the undeveloped disease, and if traceable would be found to have come from some of the permanently diseased studs or to have been infected at grass.

From town to town glanders is conveyed by infected horses which have been in contact with disease but which have not yet developed the disease so that it is apparent. Margate, Brighton, Worthing, Cambridge, are some of the towns into which glanders has been taken to my own personal knowledge by infected horses from London.

Out of the way places—villages and farms—have become mysteriously infected, and some such cases have been directly traced to travelling shows, circuses, and gipsies. Of course all sales, fairs, and markets may disseminate infected horses. The way to prevent them is—not to close the yards and markets, but to look after the infected animals.

One more method of spread I must refer to—the public water trough. It is rather curious that some men have picked out this most useful institution for special attack. I often wonder they have not proposed the closing of mangers and the prohibition of nose-bags. It is quite possible that some cases of glanders have arisen as the direct effect of drinking at a public trough, but they are very few and far between. I have an intimate knowledge of the stables of three contractors who have had during the last twenty years four outbreaks of glanders in their studs. Each outbreak was clearly and directly traceable to the purchase of a horse from an infected stud, and was stamped out at once without spreading. Save these outbreaks no glanders has troubled them, and yet their horses travel all over London and drink at any water trough they can reach. I feel con-

vinced that infection from water troughs is very rare, because in 90 per cent. of all outbreaks which I have personally investigated other methods of infection are traceable. Even if 5 per cent. of all outbreaks in London were traceable to the water troughs, the gravity of the harm would be no argument in favour of closing the troughs—especially in summer. The harm resulting to horses from being denied water all day would cause a mortality greater than is caused by all the glanders in the metropolis. The fact that an occasional case of glanders may be due to public water troughs is an argument for the extermination of the disease, but not for closing the troughs.

The spread of glanders from country to country by war and commerce is an important matter. We have imported the disease from Canada and from the United States. We have exported it to Holland and Belgium. Our wars have carried it from India to Egypt, from Liverpool to Cape Town. We imported glanders to South Africa from Canada and the U.S.A., and it is to be hoped that no horses from South Africa will be sent to other countries for some months unless they have been tested with mallein.

During the time of the Peninsular War our cavalry reeked with glanders. After the Franco-Prussian War both the opposing armies were saturated with glanders, and peace was followed by an enormous spread of the disease all over both countries.

During the American-Spanish War glanders prevailed among the American horses in Cuba. The sapient authorities shipped their infected horses back to the mainland and sold them in various towns, with the result of spreading disease widely. There is some excuse for mistakes and failures in war time, but provision against loss and inefficiency might be made when we are at peace. The way to guard our army against glanders is to stamp out the plague from our civil establishments. No one, save a few in authority, will ever know what this one disease has cost Britain and South Africa during the last two years. One may guess that the losses will not cease for many years to come unless the authorities there are more intelligent and more earnest than they are here.

Cause.

There is now no doubt about the cause of glanders—the disease arises only as the result of the *bacillus mallei* gaining access to the system of the horse. The bacillus is found in the discharges from farcy buds and nasal ulcers. It is found in infected glands, in the nodules of the lungs, and occasionally in nodules affecting other organs.

The bacillus is rapidly destroyed when exposed to direct sunlight. It may remain active in clean water for many days. It is destroyed by thorough dessication, and probably lives nowhere out of the animal body or a bacteriological laboratory for more than three months. It is easily destroyed by the more active disinfectants.

Modes of Infection.

There are three ways in which pathogenic organisms enter the body—(1) through the skin; (2) through the air passages; (3) and by the digestive tract. Direct experiment has demonstrated that

ingestion and inoculation are modes of infection. It is possible that inhalation may be another. Clinical observation suggests that ingestion is by far the most common channel through which the infective matter of glanders enters the system. As an academic problem the relative importance of the three modes of infection would supply a pretty subject for debate. As a factor in the practical question of prevention it is of little importance. No infection spreads except from a living glandered horse, and if we are permitted to find these living centres, to isolate them, or destroy them, we should speedily stop the spread of infection. This is one of "the elementary facts of the disease" which according to the Departmental Committee of 1899 required further research before "means of prevention and treatment could be formulated *with scientific exactness*." If we refrain from formulating measures against the spread of disease until our knowledge of each plague is scientifically exact we shall wait until disease has cleared the world of men and animals. If we had waited for "exactness" should we have stamped out sheep-pox, cattle-plague, pleuro-pneumonia, rabies, and foot-and-mouth disease?

Period of Incubation.

After the entrance of the virus of glanders into the system of a healthy horse, some time elapses before any noticeable changes in the animal are apparent. Direct inoculation through the skin or a mucous membrane, when it infects, causes lesions in from three to six days. Experiments in which virus has been mixed with food or added to drinking water have been followed by clinical signs of disease in from one to three months. Horses which have been submitted to the direct contagion of standing in a stable next to glandered horses have been known frequently to show no clinical signs of disease, and yet when killed a year or two later a *post-mortem* examination disclosed old lung lesions. Experiments by Schütz and Nocard show that lesions, resulting from ingestion of pure cultures of the bacillus mallei, are discernible in the lungs two or three days after infection, and clinical symptoms in six or eight days.

The term "period of incubation" is usually applied to the time which elapses between infection and the appearance of clinical signs of disease. Used in this sense there is no definite period of incubation in glanders; it may vary from three days to three years. If the term is used to describe the time which elapses between the day of infection and the day on which specific lesions are to be found on *post-mortem* examination we might perhaps put it at from ten to fifteen days.

Symptoms.

The development of glanders after infection is, as a rule, slow and variable, depending upon the age of the horse, its strength and vitality, its work and feeding, and upon its escaping febrile disease and local inflammations. Young horses develop the disease much more rapidly than old ones, and more often show it in an acute than a chronic form. Badly fed and overworked horses develop glanders very rapidly. Many horses continue at daily work unsuspected of any disease until an attack of influenza, or it may be an accident attended by injury to body or limbs, occurs. Then symptoms appear

which indicate glanders infection, and a *post-mortem* shows that the lungs have long been the seat of glanders lesions. In nearly every case the primary lesion is in the lungs, and there it may remain without causing any definite sign of its existence. In the majority of cases the first appearance of glanders is manifested by some of the signs which used to be distinguished as farcy. A swollen leg, an irregular painful swelling on the sides or on a limb, a small round tumour, well described as a "bud," are the most common first symptoms. With these there is a rise of temperature and constitutional disturbance out of proportion to the local lesion. In other cases the first symptom noticed is an enlarged sub-maxillary gland which may remain for weeks without being accompanied by any other lesion. In still other cases a horse is noticed a little amiss—off his food, feverish, and with a nasal discharge. The fever subsides, appetite recovers, but the nasal discharge remains from one nostril, and the sub-maxillary gland enlarges.

In many instances there are symptoms due to glanders which are overlooked—a hacking cough when driven at a fast pace, a fastidious appetite and continuous loss of weight, excessive staling in the stable (polyuria). Lameness is sometimes the first sign developed, and this may be accompanied by enlargements of synovial cavities, or it may exist without any apparent cause until a *post-mortem* examination discloses infected and enlarged lymphatic glands.

Ulceration of the nasal membrane is, of course, a most diagnostic symptom of glanders, but it may exist so high up in the nose as not to be visible. It may exist without any increased nasal discharge. An ulcer is not always diagnostic of glanders. But in every case I have seen, nasal ulceration not due to glanders presented a different appearance to the specific ulceration. The ulcer of glanders is deep, and the mucous membrane surrounding it swollen. Other nasal ulcerations are not deep, they soon granulate, and are often raised above the surrounding membrane. The glanders ulcer on the nasal membrane is preceded by a small white nodule, which soon alters by the loss of its covering membrane and rapidly extending ulcerous surface. Ulceration of the larynx is not uncommon, and is generally made evident by a sudden attack of roaring. Infiltration of the nasal membranes gives rise to snuffling, and may be so pronounced as to cause marked obstruction to breathing. The text-book division of glanders into chronic and acute is convenient but somewhat arbitrary. All fatal cases of farcy or glanders are acute. Some cases arise insidiously and progress slowly; these are chronic. Other cases arise suddenly, with high temperature and great constitutional disturbance; they are acute.

When a horse first shows clinical signs of glanders, when, in the common stable expression, he begins to "break up," there is always some febrile disturbance demonstrable with a clinical thermometer. The temperature rises to 103° or more, sometimes to 106° .

Before this stage is reached horses with lung lesions but no outward signs of disease, if kept at work suffer from intermittent rises of temperature. When mallein was not available, the more careful practitioners resorted to weekly examinations with a thermometer to enable them to pick out horses probably infected. Those animals that showed a rectal temperature fluctuating between 101.5° and 103°

were looked upon with suspicion, and noted for the horsekeeper to examine daily for any other sign. Continuance of this high temperature or exhibition of unusual fatigue, or loss of condition, cough, etc., were held sufficient reasons for isolation. Considerable success attended this method of controlling the spread of disease, but as it included horses suffering from other disorders it was inefficient and very tedious.

It is probable that some rise of temperature always attends the primary infection of horses, though it is overlooked. We know that in experimental infection a rise of temperature is evident on the third or fourth day after; with large doses of pure cultures a temperature reaction occurs in fifty hours.

TABLE VII.—*Symptoms shown in 1000 Cases of Glanders occurring in London between April and November 1901.*

<i>Symptoms.</i>	<i>Number of Horses.</i>	<i>Per-centage.</i>
Farcy, swellings, buds or ulcers, some verified by mallein test	414	41·4
Farcy and enlarged sub-maxillary glands	54	5·4
Nasal discharge, enlarged glands and farcy	82	8·2
Nasal discharge, ulcerated or infiltrated nasal membrane and enlarged glands	158	15·8
Nasal discharge, enlarged glands and mallein reaction	42	4·2
Nasal discharge, ulceration, enlarged glands and farcy	12	1·2
Nasal discharge and ulceration	27	2·7
" " emaciation	12	1·2
" " mallein reaction	17	1·7
Nasal ulceration and enlarged glands	5	·5
Enlarged glands	6	·6
" " and mallein reaction	40	4·0
Mallein reaction only	119	11·9
Diseased lungs and other diseases giving symptoms of a suspicious nature	12	1·2

The first two groups of this table numbering 468 horses, and the last four numbering 177 are free from nasal discharge, so that sixty-four per cent. of the glandered horses slaughtered in London would not probably infect a water trough or nosebag up to the day of their death. In 355 horses there was nasal discharge. Only 279 showed all the classical symptoms of glanders—nasal discharge, nasal ulceration, and enlargement of the sub-maxillary gland. Thirty years ago only these 27·9 per cent. would have been positively diagnosed as glandered.

Diagnosis.

A few years ago the diagnosis of some cases of glanders was most difficult. Other diseases occasionally were attended with one or

more of the symptoms we usually credit to glanders, and it was often necessary to wait days or weeks to be sure of the nature of the mischief. Since mallein was introduced we need have no doubts about diagnosis, as an injection will, in 98 per cent. of cases, determine the nature of the disease in twenty-four hours.

Every nasal discharge should be treated with caution until its nature is evident. An unilateral discharge, or a discharge which continues without odour, especially when the appetite remains good, should be considered suspicious. Farcy buds on the lips and extending up the cheeks may easily be confounded with horse-pox or what has sometimes been called stomatitis. Lymphangitis is frequently indistinguishable from farcy for a day or two, and lymphangitis epizootica is apparently not distinguishable. In all these cases mallein is a safe guide.

Other aids to diagnosis are the microscope, cultures on selected media, inoculation of small animals, and Widal's agglutinating test. Some of these are only of value in the hands of an expert bacteriologist, others require days for their development. Mallein is more trustworthy, simpler, and much quicker.

Post-mortem Lesions.

The only definite constant lesion is the nodule found in the lungs. Ulceration of the nasal membrane, of the larynx or trachea, may be found in developed cases which have shown clinical signs during life. Very rarely nodules are found in the liver or spleen, but such lesions are not diagnostic unless corroborated by examination in a laboratory.

The glanders pulmonary nodule is more easily discovered by the hand than the eye. If the hand be passed with gentle pressure over the pleural surface of a lung the nodules are easily felt, varying in size from a mustard seed to a pea, or even occasionally to the size of a hazel nut. The nodules are distributed very unevenly, and are frequently few in number. In some cases a careful search is necessary to find the two or three nodules that constitute the lung lesions. When a large number of nodules exist they vary in age. The youngest nodules are said to be yellowish gelatinous spots. I have never recognised them, but do not deny their existence. The earliest nodule I recognise is easily felt with the finger, and when cut into is seen to be circular in form, dark red with a grey or yellow spot in the centre and a lighter red zone around, but no well marked line of demarcation from the surrounding tissue. The older nodule has less red zone around it, and a larger white centre. A further stage and the white centre is softened, or the whole nodule may be caseous. When the caseous matter is gritty a further progress has been made, and the oldest of all is a hard calcareous mass which is quite separated from the surrounding tissue and can be enucleated.

These nodules when near the surface of the lung are usually accompanied by some thickening of the pleura, which is also opaque. To find the deep seated nodules it is necessary to cut the lungs into slices. The bronchial glands are seldom enlarged, but they, as well as the tracheal glands, are often found to have caseous centres when cut into.

Pulmonary glanders nodules are seldom if ever found in such numbers and so evenly distributed throughout the lungs as to be confounded with miliary tuberculosis. Nor can they be mistaken for the solitary large tumours which are also due to tuberculosis. In tuberculosis the bronchial glands are always enlarged when the lungs are the seat of marked lesions, in glanders this is not so.

In very few cases—only in those of long standing—do we find fibroid induration of large areas of lung due to glanders. In many cases there are large hepatised areas, but whether these are due to a specific glanders inflammation or to an inflammation which has arisen quite independently of glanders and possibly been the cause of the clinical development of the case I do not know. Glanders nodules have been found in the liver, spleen, and kidney—they are not common.

The blood does not contain glanders bacilli in the ordinary latent form of the disease, and probably only a few that rarely escape into the circulation in chronic cases. In acute cases *i.e.*, generalised glanders, there are probably increased numbers of bacilli in the blood. Coleman communicated glanders to an ass by transfusion of blood, and the animals in menageries have more than once suffered by eating the flesh of glandered horses. Schütz has also found the bacillus in the blood.

There is one very important point in reference to the *post-mortem* lesions of glanders. Under the Diseases of Animals Act a veterinary inspector's diagnosis is final "for the purposes of this Act," but he is not infallible. If, on *post-mortem* examination no lesions of glanders are detected the full value of the horse destroyed under the Act may be claimed by the owner. Suppose the veterinary inspector has corroborated his diagnosis by a mallein reaction and yet no lesions are found on *post-mortem* examination. Is the case one of glanders? There is argument in favour of the inspector, but I hold that some definite material change should be visible to support a diagnosis followed by compulsory slaughter. Therefore a *post-mortem* should disclose nodules in the lung before an opinion can be sustained that a horse is glandered. One exception I make to this, and that is where during life undoubted symptoms of farcy are detected.

Schütz and Nocard have both experimentally produced glanders by causing horses to swallow pure cultures of the glanders bacillus. In some of Nocard's cases clinical symptoms were evident on the eighth day after infection and a mallein reaction was given on the sixth day. This rapidity of development has not been previously shown by any ingestion experiments. Probably the explanation is that all the old experimenters used natural discharges from glandered horses as infecting media, whereas the pure cultures now used contain a thousand times more bacilli. The dose is a hundred times greater and the effects consequently more rapid and intense.

Infection produced by these virulent cultures is not only attended by an earlier appearance of clinical symptoms but by more marked internal lesions. Horses killed fifteen days after injection were found by Nocard to have their lungs full of miliary tubercles in all stages of development, and many of them entirely translucent. Horses

killed three months after infection had their lungs literally crammed with tubercles in all stages of evolution—calcareous, fibrous, and caseous.

Schütz made *post-mortem* examinations on the fourteenth, fifteenth, and twenty-fifth days after infection and found nodules in the lungs. He also found, what Nocard has not described—enlarged mesenteric glands, nodules in the spleen and intestines, enlarged abdominal lymphatic glands, and distended chyle vessels. I do not think any British veterinarian has seen these lesions, which probably only occur during the earlier stages of infection by ingestion, and possibly only when virulent cultures of the bacilli are employed as the infecting agent.

Schütz thinks that the glanders nodules in the lungs are secondary in their nature, and referable to the glanders lesions in the intestinal lymphatic glands. Apparently the lung changes are seen as soon as these in the intestinal glands, and if both exist both seem to have equal claim to be called primary. The lung lesions are permanent, the intestinal lesions (with the exception of a rare nodule in the liver or spleen) are temporary.

To state the time during which a horse has been infected with glanders is a rash proceeding. Even with the *post-mortem* lesions as guides we ought to be very cautious. Nocard found calcareous nodules in a case only three months after infection. I should have thought no nodules were even "gritty" till six months after infection, and I feel certain I have seen cases more than six months old in which not a single nodule had become calcareous.

TABLE VIII.¹—*Lesions shown in 1000 Post-mortem Examinations of Glandered Horses in London between April and November, 1901.*

<i>Lesions.</i>	<i>No. of Horses.</i>
Lungs fairly full of glanders nodules	295
„ Nasal membrane ulcerated	101
„ Nasal membrane infiltrated	95
„ Trachea ulcerated	19
„ Larynx and trachea ulcerated	16
„ Nasal membrane and larynx ulcerated	9
Lungs full of glanders nodules	121
„ Nasal membrane ulcerated	128
„ Nasal membrane, larynx and trachea ulctd.	124
„ Larynx and trachea ulcerated	26
„ Nasal membrane and trachea ulcerated	16
„ Larynx ulcerated	4
Lungs contained few glanders nodules	46

Of the 1000 cases 462 presented nodules in the lungs as the only internal lesions. The nasal membrane was ulcerated in 378 horses, the larynx in 179, and the trachea in 201.

¹ For the information in this and two preceding tables I am indebted to the courtesy of the Chief Officer of the Public Control Department of the London County Council.

In the 46 horses whose lungs contained only a few nodules, no lesion of membrane was found. Probably nearly all these were free from clinical symptoms and were slaughtered on a mallein reaction only.

Mallein.

The discovery of mallein in 1890 by Kelning, a Russian veterinary surgeon, has altered and simplified the whole process of preventing and suppressing glanders. It has also enlarged our knowledge of the pathology of the disease. To Professors Nocard and M'Fadyean we are chiefly indebted for establishing the claim of mallein to be a valuable aid in diagnosis.

Mallein is an extract of the *Bacillus mallei* made by growing the organism in a suitable medium, then removing the bacillus and diluting the extract to proper proportions. I believe it is not yet possible to standardise mallein, but the skill of the makers enables them to supply a finished material, a definite dose of which produces a specific action. The dose of mallein is 1 cc. or 18 drops. As the English hypodermic syringe is marked it is easier to use 20 drops, but I do not think it matters whether a few drops more or less are used. The injection is usually made on the side of the neck. The site is handy to get at, the skin is easily raised into a fold, and the swelling shows up on the firm structures underneath. The needle should be a fine one as it penetrates easily. It should not screw on to the syringe but fit over the nozzle. When the syringe is discharged, it and the needle are removed together. Of course if the most careful methods be adopted, washing and disinfecting the skin of the neck should precede injection. Not once in a thousand times is any ill effect observed after injection of mallein through an unwashed skin.

A small quantity of mallein injected under the skin of a healthy horse causes no definite effects. A similar quantity injected under the skin of a glandered horse is followed by two effects—a rise of temperature, and a swelling at the point of injection. These two phenomena together constitute the specific double reaction which glanders gives to mallein.

The reaction to mallein is a gradual process extending over 24 hours—sometimes longer. The following table shows a typical reaction :—

<i>Temperature.</i>		<i>Diameter of Local Swelling.</i>
Temperature at 8 p.m., time of injection	100	
„ 8 a.m., 12 hours after	104	3 inches
„ 12 p.m., 16 „	104.5	4 „
„ 4 p.m., 20 „	105	5 „
„ 8 p.m., 24 „	105	6 „

Although in many cases there is a rise of temperature to 105° and a local swelling measuring 6 inches across we do not always obtain a double reaction so pronounced. The swelling may not reach more

than 4 inches across, and the temperature may not rise higher than 104° and yet I should accept the reaction as diagnostic. I think it is necessary to have a double reaction—both rise of temperature and some swelling—before it is safe to declare a diagnosis of glanders. I have seen a rise of 3 or 4° in the temperature with hardly any swelling, and in other cases a very large swelling, 8 or 9 inches, with a temperature rise of less than 2° . Such irregular reactions may be useful guides when the history or surroundings afford some help, but it is safest to wait and test again.

A temperature rise to 103° within twenty-four hours accompanied by a swelling of 4 inches in diameter I should consider sufficiently diagnostic to warrant isolation, and if the swelling were to continue increasing after the twenty-four hours I should accept the reaction as evidence of glanders. The rise of temperature following injection of mallein into a glandered horse may reach its maximum within eight or nine hours, but more often not till about the fifteenth or sixteenth hour after. There is not much fall in the temperature till after twenty-four hours or even longer. In a horse whose temperature is 102° at the time of injection the rise to a maximum is, I think, usually more rapid than in cases when the temperature is 100° at the start. To inject mallein when the temperature is over 102° is inadvisable. The usual effect of a mallein injection with a temperature of 104° is to lower it one or two degrees within twelve hours.

The local reaction is valuable by itself in cases where the temperature is high and clinical symptoms exist at the time of injection. To wait for a normal temperature means the loss of days, whereas the appearance of a large swelling is sufficiently indicative to warrant a diagnosis of glanders. The absence of swelling must not, however, be accepted as evidence of freedom from infection, and we should wait for the return of a normal temperature to repeat the test.

The swelling which indicates glanders is one that rises and increases slowly for twenty-four hours, and which then continues to increase in size. It is usually well defined and painful when touched.

There are some doubtful reactions to mallein even when every care is taken, as for instance when we have a maximum temperature of 102.6° and a well defined 3-inch swelling. The only safe proceeding is to wait and repeat the test.

I have not found that one injection of mallein in a glandered horse has much effect in spoiling the reaction to a second test. I think an interval of a fortnight is quite sufficient, and I have often seen a good reaction follow a test repeated after an interval of only a week.

Mallein does not give a reaction in a horse infected with glanders a few hours previously. It is safe to say that fifteen days after infection mallein will act, and in a number of Nocard's cases a good reaction was obtained six days after infection. Schütz tried the mallein test on horses with a high febrile temperature and was dissatisfied because he obtained no rise. A fall of temperature followed his mallein injection, which was in itself very suggestive.

Some observers have recorded reactions to mallein in horses which

on *post-mortem* examination were found to be suffering from other disease, and not, apparently, from glanders. I have tested with mallein in cases of strangles, lymphangitis, nasal discharges, urticaria, purpura, tuberculosis, influenza, and septic-pneumonia without any save a negative effect.

An injection of mallein into a glandered horse has often a peculiar effect disclosed on *post-mortem* examination. It produces hæmorrhagic patches on and in the heart, red patches and streaks across the pleura, and a peculiar dropsical condition of the lungs which men who make many *post-mortems* of these cases recognise at once. These changes are only seen when the horse is slaughtered and examined within a day or two after reaction has taken place.

It is remarkable that the violence of a reaction to mallein is no indication of extensive or long-standing infection. So far as I know there is no guide to the condition of the disease within a horse by observance of the reaction. You may see a first-class double reaction and on *post-mortem* have difficulty in finding a few nodules in the lung. You may also have a poor reaction and find after death both lungs full of typical nodules of all sizes and ages.

I do not claim for mallein infallible diagnostic power. It requires brains and experience to interpret its indications sometimes. In 98 per cent. of cases it may be relied on, and these, be it remembered, in horses affording no sign of disease to the eye or hand of an expert.

Mallein in Repeated Doses.

In cases of doubtful reaction to injection of mallein it is necessary to repeat the test, and it has been noticed that in some cases each reaction becomes less marked until, finally, a proportion of the horses operated on cease to react. When the injection is made at short intervals the time of failure to react soon arrives, but if a long interval be allowed and the injection repeated a reaction may again be obtained. A second test for diagnostic purposes may safely be made a week or ten days after the first.

The effect of repeated injections of mallein is a matter of great importance, because if we are convinced that a horse which does not react to mallein is free from glanders we may safely and logically permit such an animal to continue at work in association with other horses. It is of course important to be sure that the cessation from reaction is a permanent condition due to the absence of bacillary infection, and not a temporary effect due to some passing immunity conferred by super-charging the system with mallein.

A large amount of experience bearing upon this question is now available, some public, some private. We know certainly that repeated doses of mallein do not prevent all horses developing clinical symptoms of glanders. We know that some horses, after giving a typical reaction to a diagnostic mallein test, live for many months without showing a sign of glanders. We know that many horses after giving a typical reaction three or four times gradually cease to react and continue at work in apparent health for years. We know that this last class when slaughtered and carefully examined give evidence, in the form of typical glanders nodules in the

lungs, of having been infected with glanders, but when these nodules are tested by culture and inoculation no active bacilli are detected.

We know further that when "ceased reactors" are closely confined for months with healthy horses, forced to use the same water trough and the same manger, no infection of the healthy horse results.

In the following table I have attempted to show the results attained or claimed in a few prominent studs where publicity has been given:—

TABLE IX.—*Published Results and Percentage of Horses Reacting in Five Studs.*

	Horses in Stud.	Reacted.		Slaughtered.	Ceased to React.	
			Per cent.			Per cent.
Reported by Nocard (Omnibus) . . .	10,231	2037	20'0	687	338	16'5
Gt. Western Railway . .	1,114	67	6'1	27	40	59'7
Glasgow Tramway . . .	4,439	278	6'2	3	275	99'7
London County Council . .	650	63	9'6	32	31	49'2
Departmental Committee Prof. M'Fadyean . .	75	23	30'6	7	16	69'9

The sub-columns showing percentage refer to the proportion of "reactors" to the number tested, and the proportion of "ceased reactors" to those that reacted.

The high proportion of reactors in the Paris omnibus horses is what one might expect in a stud long infected and which was losing heavily by slaughter of clinical cases previous to the test. The comparatively low rate of recoveries probably depends upon the fact that the repetitions of mallein were infrequent and only used for diagnostic purposes.

The Railway Company killed a number without waiting to submit them to a long course of mallein, and thus probably reduced the proportion of recoveries.

The Glasgow Tramway, unlike all the other studs, was not, and had not for years been, badly infected. One clinical case, and two cases recognised on *post-mortem* examination of the lungs, was the previous history of disease. On such a visible exhibition of disease it is impossible to understand how 6 per cent. of the stud could be infected. It is also opposed to all other experience to have 98 per cent. of recoveries. The explanation probably is that the standard of reaction was put too low, and that a very large proportion of those credited with reaction were really not infected at all.

The London County Council made their tests upon distinctly infected studs, but abruptly closed the experiment at the desire of the owners. Thus a number of horses were slaughtered which might have recovered and so improved the result.

The stud referred to by Professor M'Fadyean before the Departmental Committee showed a very high proportion of infected horses and also a high proportion of recoveries.

Probably the conditions and surroundings of horses that have reacted to mallein largely affect the result of continuing them at work

whilst periodically injecting with mallein. If the feeding is good and the work not too exacting we should expect a larger proportion of recoveries than if work was excessive, or food and stabling defective.

If we omit from the above table the Glasgow Tramway results as too doubtful, and take the average of the others, we find that nearly 50 per cent. of the reacting horses submitted to repeated mallein injections recovered. A recovery of 50 per cent. which may be acquired without much loss of work, with no great risk to other horses, and at no great expense to the owner or to the local authorities, is deserving of attention.

There is one experiment with mallein which has perhaps been carried further than any yet published. It formed part of the work carried out by the London County Council but has since been made more complete by further experiments undertaken by the Board of Agriculture.

In April, 1900, a stud of 164 horses, in which an outbreak of glanders had occurred, was tested with mallein. Of these, 21 reacted and were removed to one stable from which they continued to work throughout the experiment. Table X. shows the repetitions of mallein, the observations made, and the result. It has been published before but I quote it in full, with the appended notes.

It will be seen from this table that the reacting horses were submitted to monthly injections of mallein from May to November. In August two horses, "Bobs" and "Emblem," developed swollen legs (farcy) and were slaughtered. In November two horses, "Deal" and "Ernest," which from age and lameness were unable to work, were destroyed. Then a period of about three months was allowed to elapse without injections, so that any temporary effects of the mallein might have an opportunity of passing away. On 20th January 1901 the remaining seventeen horses were injected with 20 drops of mallein, the result of which is recorded in the final columns of the table. The horse "Chesterfield," who had reacted to every injection, again gave an indicative reaction. The two horses "Surefoot" and "Drastic" showed a rise of temperature to 103° , but no local swelling, and were considered doubtful. The three were killed, and the fourteen non-reactors were declared not infected.

The seven horses slaughtered were carefully examined by Professor M'Fadyean at the Royal Veterinary College. In the lungs of each were found glanders nodules similar to those seen in ordinary cases of glanders. Perhaps the chief points of interest may be shown best in tabular form:—

<i>Name.</i>	<i>Temp. at last Test.</i>	<i>Why Slaughtered.</i>	<i>Bacteriological Exam.</i>
2. Bobs	103.8	Clinical signs	Positive
3. Emblem	104.4	" "	"
11. Deal	100	Worn out	Negative
12. Ernest	101	" "	"
23. Chesterfield	104	Positive reaction	Positive
26. Surefoot	103	Doubtful reaction	Negative
24. Drastic	103	" "	"

TABLE X.—Results of Repeated Mallein Injections.

Name of Horse.	1st Test.			2nd Test.			3rd Test.			4th Test.			5th Test.			6th Test.			7th Test.		
	Date.	Temp.	Local swelling.	Date.	Temp.	Local swelling.	Date.	Temp.	Local swelling.	Date.	Temp.	Local swelling.	Date.	Temp.	Local swelling.	Date.	Temp.	Local swelling.	Date.	Temp.	Local swelling.
STUD A.	1900, April 21	105.4	3 by 3	1900, May 20	104.4	8 by 8	1900, June 6	104	5 by 6	1900, July 22	103.4	3 by 3	1900, Aug. 18	102	3 by 2	1900, Sept. 9	102.4	Nil.	1901, Jan. 20	103	Nil.
Surefoot . .	" "	105	5 " 3	" "	103.4	6 " 6	" "	103	5 " 5	" "	103	4 " 4	" "	101.6	2 " 2	" "	101.4	"	" "	103	"
Drastic . .	April 28	103.5	6 " 7	" "	103	5 " 5	" "	101.2	Nil.	" "	101.6	2 " 2	" "	102	3 " 2	" "	100	"	" "	101.6	"
Gilbert . .	" "	103.4	6 " 6	" "	101.6	5 " 4	" "	103.4	2 by 2	" "	102	Nil.	" "	101	Nil.	" "	101.4	"	κ	—	—
Ernest . .	" "	105.5	2 " 2	" "	102.2	2 " 2	" "	102.8	Nil.	" "	101.6	3 by 3	" "	101.6	"	" "	101.4	"	" "	100	"
Shamrock .	" "	104.6	5 " 4	" "	101.8	4 " 3	" "	104	5 by 7	" "	102	Nil.	" "	101.2	2 by 2	" "	101.5	2 by 2	" "	100.4	"
Ben . .	" "	105.8	5 " 5	" "	102	6 " 8	" "	104	2 " 2	" "	102	"	" "	101.4	Nil.	" "	101	"	" "	101	"
Riley . .	" "	104	7 " 7	" "	102	6 " 6	" "	103.2	8 " 10	" "	103.8	6 by 6	κ	—	—	" "	—	"	" "	—	—
Bobs . .	May 5	104	6 " 6	" "	103	6 " 6	" "	103.8	4 " 4	" "	103	3 " 3	" "	101.4	4 by 4	" "	101.2	Nil.	" "	101.6	Nil.
Smuggler .	" "	102	7 " 9	June 6	102	2 " 2	July 22	101	Nil.	Aug. 18	101.4	Nil.	Sept. 9	102	Nil.	Oct. 20	101	"	" "	—	—
Brownlow .	May 19	104.8	7 " 8	" "	104.4	5 " 4	" "	104.4	6 by 6	κ	—	—	—	—	—	—	—	"	" "	—	—
Emblem . .	" "	103.2	4 " 9	" "	105	5 " 8	" "	105	4 " 4	" "	102.6	4 by 2	" "	102	Nil.	" "	101	Nil.	" "	102.6	2 by 2
Abel . .	" "	106	5 " 6	" "	104.4	5 " 5	" "	103.8	6 " 6	" "	103.4	3 " 2	" "	105	5 by 5	" "	104.4	4 by 4	" "	104	3 by 3
Chesterfield	" "	105	5 " 5	" "	103.8	Nil.	" "	102	Nil.	" "	101.6	3 " 3	" "	102	Nil.	κ	103	3 " 3	" "	—	—
Deal . .	May 26	105	5 " 5	" "	104	6 by 6	" "	101.6	3 by 3	" "	101	3 " 3	" "	101	"	" "	102	Nil.	" "	100.8	"
Doctor . .	" "	103	4 " 6	" "	103	5 " 6	" "	102	3 " 3	" "	101.2	3 " 4	" "	101.4	"	" "	103	2 by 3	" "	100.4	"
Eaton . .	" "	105	7 " 8	" "	105	5 " 5	" "	103.4	4 " 4	" "	102.6	4 " 4	" "	101.4	2 by 3	" "	102.8	3 " 3	" "	101.2	2 by 2
Cob . .	June 17	104	6 " 6	July 22	103	2 " 2	Aug. 18	101.2	2 " 2	Sept. 9	102	Nil.	Oct. 20	103	3 " 4	Nov. 11	101	2 " 3	" "	102	Nil.
Dublin . .	" "	102.2	4 " 5	" "	102.4	3 " 3	" "	102.4	3 " 3	" "	101	3 by 3	" "	101.2	3 " 3	" "	100	2 " 3	" "	100	3 by 3
Buck . .	" "	102.4	5 " 5	" "	104	6 " 6	" "	102	5 " 5	" "	101	4 " 4	" "	102	2 " 2	" "	100.6	2 " 3	" "	101	3 " 3
Captain . .	" "	102	6 " 5	" "	103.8	3 " 4	" "	102.4	3 " 3	" "	101.4	3 " 3	" "	102.4	2 " 3	" "	100	Nil.	" "	101	3 " 3
Napier . .	" "																				

The temperature reaction is the maximum of three thermometric observations made between the 9th and 24th hours after the injection of mallein.

The bacteriological examination of the lesions found in each horse was made by Professor M'Fadyean, who has published full notes of his research in *The Journal of Comparative Pathology and Therapeutics*. I have placed numbers in front of the names of the seven horses in the table on p. 236. These are the case numbers under which the details of *post-mortem* and bacteriological experiments are related in *The Journal*. Anyone having the time to spare will be interested in referring to them.

The fourteen horses that ceased to react to mallein are alive and well. Four of them—perhaps more—will some day be slaughtered and submitted to most careful examination, the result being published.

Meantime, although we cannot say that repeated injections of mallein are a cure for glanders, I think there is strong evidence that mallein has some curative action—that it assists the natural action by which colonies of glanders bacilli are surrounded and sometimes completely isolated in the lungs.

To the information given on this page from *The Veterinary Record* of 19th October 1901, I am now—July, 1902—able to add something further. The 14 horses which had ceased to react continued at work and were permitted to mix and cohabit with the whole stud. No case of glanders has appeared in the stables, and the history of the fourteen is as follows:—

Gilbert :	Killed, worn out.	Ben :	Remains at work.
Abel :	" "	Smuggler :	" "
Captain :	Died, intestinal twist.	Brownlow :	" "
Shamrock :	Sold to Board of Agric.	Cob :	" "
Riley :	" " "	Dublin :	" "
Doctor :	" " "	Napier :	" "
Eaton :	" " "		
Buck :	" " "		

The diagnostic injection in these horses was made in April, 1900. In July, 1902, six remain at work with other horses and no case of disease has appeared. Five after working for a year were sold as a favour to the Board of Agriculture, were used in an important experiment and then slaughtered. Each horse was carefully examined, all lesions submitted to culture and inoculation tests, but not one disclosed active bacilli. The three that were killed or died were all aged horses, but I have no definite reports as to the *post-mortem* appearances. They showed no sign of disease during life.

The history of these animals is sufficient to demonstrate that horses may react to mallein, that after repeated injections they may cease to react, and that cessation is proof of non-infection and non-infectivity.

Control or Stamping Out?

Legislation with regard to glanders commenced with a simple attempt to protect horse-owners from the indiscriminate movement in public places of visibly glandered horses. Very slowly and gradually it has progressed until now it aims at preventing the spread of disease, and even, in a weak and futile way, giving some little assistance towards suppression.

In 1853 an Act of Parliament prohibited bringing glandered horses into markets, and turning them out on unenclosed lands.

In 1869 the Contagious Disease (Animals) Act scheduled glanders as a contagious disease, and provided for the compulsory burial of carcases of horses affected with glanders. Under this Act the Privy Council were given power to issue Orders which should have all the effect of the Act.

In 1873 by an Order of Council farcy was included amongst the diseases scheduled under the Act, but simply for the prevention of movement of diseased horses.

In 1878 an amended Contagious Diseases (Animals) Act was passed, and by an Order under it glanders and farcy were made "diseases," and horses, mules and asses "animals" under the provisions of the Act. Thus compulsory notification of glanders and farcy was introduced, local authorities were empowered to make regulations as to movement, and owners were required to slaughter horses suffering from glanders, and to isolate those suffering from farcy.

In 1892 an Order was issued by the Board of Agriculture making glanders and farcy one disease, and giving local authorities permissive power to slaughter on payment of compensation. The compensation was fixed at half the value of the horse with a limit of £20. The result of this Order was that the largest local authority declined to slaughter, holding that the cost would be too great. It is rather curious that a Superintending Inspector of the Board of Agriculture who gave evidence before the Departmental Committee of 1899 is reported thus—"Did this Order in any degree lessen the number of outbreaks?—Yes, in a marked degree, as the outbreaks fell from 1657 in 1892, to 1381 in 1893." As a matter of fact this Order had no effect whatever upon the disease. Hardly an authority in England enforced it, and London refused to put it in force and fell back upon their power to compel isolation. The best proof of its inutility is that it did not survive quite two years, and was then superseded by the Order of 1894 which is now in force. The Order of 1894 is worse than that of 1892, as it introduced a novel and mischievous system which permitted local authorities to slaughter and pay compensation on different scales—one authority might slaughter and pay carcase value, a neighbouring one might slaughter and pay one-fourth the value or any less amount.

The Order now in force compels :—

Notification of the disease by the owner or person in charge.

Inspection by Local Authorities Officers.

Prohibition of movement or exposure of diseased animals.

Disinfection of place in which disease has existed.

It permits Local Authorities to make regulations for :—

Slaughter and payment of compensation.

Disposal of carcases, manure, forage, litter, etc.

Movement of diseased or suspected animals.

Movement of animals into or out of any stable or other place in which glanders exists or which have been in contact with any diseased or suspected horse.

Of course the permissive powers are of no avail unless a Local Authority uses them, and some do not. The regulation as to the

movement of horses which have been in contact with diseased horses would be most valuable if it were not rendered nugatory by a proviso as follows:—"It shall operate so long only as a deceased animal remains on the premises and until the regulations as to cleansing and disinfection have been carried out." In other words the law is, that as soon as a glandered horse has been slaughtered and removed the inspector has no more power in that stable, though he may feel certain that a fourth of the horses therein are infected animals.

The veterinary inspectors of the Local Authorities by the Order of 1894 are men who go and look at such diseased horses as the owners wish killed and removed; sometimes assisting in the diagnosis of the case, but as a rule playing the part merely of registrar of death. The control of glanders in private stables is really dependent upon the care of the private veterinary practitioner and the intelligence of the horse-owner. The result is that the returns of glanders for last year are a little higher than they were twenty years ago.

It is unnecessary to argue that glanders should be controlled and that the State should assist. The existing law recognises all this but fails to attain its object. The result of this failure is that our Army is in constant danger of infection and consequent loss of some efficiency, that five human beings every year lose their lives from a loathsome and preventible disease, that 1700 horses perish annually with loss to many owners and ruin to some.

The existing regulations, which I have shown to be a failure and which permit such great losses, are expensive to administer. London alone spends £6000 a year in carrying out these useless regulations, and the rest of the country is mulcted in a corresponding amount. All the facts I have been able to collect indicate that these charges will continue, and that 1700 horses and five human beings will perish every year unless more rational measures are adopted for suppressing the disease. For a very slightly increased expenditure, accompanied by more logical and sensible regulations, we could in a very few years save all the loss of human and equine life, all the cost of compensation, and nearly all the cost of the organisation now wasted upon an attempt to control the disease.

Between attempts to control and attempts to stamp out there is a wide difference. The history of legislation against diseases of animals in this kingdom affords us full information as to the results attending both methods of procedure. In 1865 we tried to control cattle plague and failed. We then adopted measures for its extinction, and with immediate success.

For many years we played about with pleuro-pneumonia and foot-and-mouth disease—adopting half-hearted measures of control, without success. Then a direct effort was made for stamping out, and success came at once. For some years the Board of Agriculture has been attempting the control of swine-fever and glanders. The result has been an expenditure of £130,000 a year by the Central Authority and a very large sum by the Local Authorities. With what success? Absolutely none. The taxpayer and the ratepayer have not yet recognised the scandal and folly of the thing, the press ignores it, and the politician naturally makes no move.

That glanders might be certainly and rapidly stamped out is an inference from well-known facts, accepted by every authority on the

matter. The British Army stamped it out after some trouble and expense. Many large companies have stamped it out—some more than once. In every county in England outbreaks of glanders have occurred, but very few remain long infected. Twenty-five counties have stamped it out. What has been done in one stable or in one place can be done in others. Private effort can clear the private stud, but the State only can make regulations that will protect from re-infection due to centres where neglect and wilful deception keep the disease alive.

Really only a slight advance on the existing Orders would be sufficient to stamp out the disease. Of course they should be made uniform for the whole country, and compulsory instead of permissive. The legislation for glanders should, in short, be made similar to that which has successfully exterminated sheep-pox, cattle-plague, foot-and-mouth disease, pleuro-pneumonia, and rabies. The principle adopted with these diseases was to slaughter the diseased and either slaughter or isolate all the probably infected, *i.e.*, those in contact with the disease. No stud of horses is ever wholly infected with glanders. We find perhaps one or two visibly diseased at each outbreak, and we know that often a number more are infected but have not yet fully developed the disease. Under the existing regulations an inspector may visit a stable, on notification by the owner, see a case of virulent glanders, have it destroyed and removed, have the place cleansed and disinfected. This may be repeated weekly, and yet he has no power to detain any horse in that stud. The owner may sell his infected stock either publicly or privately and the law finds no fault with him and affords no protection to the innocent buyer of these undeveloped cases of disease. The Board of Agriculture has been advised of all this by its officers. It has been recommended by a Departmental Committee to make alterations in the Order. Deputations of horse-owners and Local Authorities have prayed for more earnest action. The two largest Local Authorities in the Kingdom have asked for greater powers to deal with the disease—and been refused. The plain fact is the Board does not know and will not learn.

One excuse for doing nothing is found in the statement that "Horses are not like cattle; horses cannot be prevented from working without great loss or even ruin to the owner, whereas cattle may be isolated and their movement prohibited without loss or injury." This statement is not true. Prohibition of movement of cattle, sheep, or swine is frequently attended by very great loss and injury. The carriage of food and water to stock which must not be moved is a heavy expense, the loss of markets just when animals are ready for sale is a very serious loss. The individual stock-owner is, not unjustly, made to suffer for the general good, and the horse-owner surely cannot expect better treatment. But the absurdity of the argument is best seen when it is known that the only detention of horses asked for is one of twenty-four hours to enable the authorities to detect which are healthy and which are diseased. Now no sensible man would say that twenty-four hours detention of suspected horses was a serious injustice, and no one could honestly protest against the detention of diseased horses. Before mallein was discovered there was a strong argument against the detention of in-contact horses,

because such detention might have lasted for weeks before the state of the horse could have been determined. Since mallein has been tried and approved, the detention of horses need never be for more than twenty-four hours, and the occasion of such detention can be made a matter to suit the convenience of the owner.

Those who insist upon the unfairness of detaining a £20 horse from an old glandered stud should reconcile their objection with their approval of the compulsory slaughter of a £100 bull which has the misfortune to be in a place where one case of pleuro-pneumonia has appeared.

The detention of horses forming part of a diseased stud is no new thing, no hardship due to legislation—it is a voluntary act in scores of stables every week of the year. It is a voluntary act of the horse-owner possessing intelligence and guided by sound professional advice. It is objected to by the more ignorant owners whose studs are permanent centres of infection—and it is objected to by the Board of Agriculture.

Conclusions.

That notification by owner should be permitted either to a veterinary inspector of the district or to a police constable.—This is one of the recommendations of the Departmental Committee of 1899. The object is to save time by allowing the notification to go direct to the officer who must see the horse. Another object is to save owners who voluntarily report the annoyance of visits from the police, and in many cases from the serious injury of having a constable posted outside the premises until the diseased horse is removed. In towns this causes a gaping crowd to assemble all day, and a collection of children to see the knacker's cart, and, if possible, the slaughter.

One of the members of the Committee objected to this alteration of the Order on the grounds that it would "cause inconvenience and confusion." He made his objection apparently in ignorance of the fact that the universal practice in London is to report direct to the veterinary inspector, and that it has been adopted and carried on solely because convenience was best suited by it.

That horses that the veterinary inspector may consider to have been exposed to contagion should be dealt with in the same manner as suspected horses.—This is one of the recommendations of the Departmental Committee of 1899 and is the most necessary alteration of the Glanders Order. Until the infected horses of a large stud are subject to regulations it is little less than folly to expect any favourable result from killing diseased horses and permitting the sale of others. To protect the owner from injustice he should have the power to demand that any horses detained as "suspected" shall be tested with mallein at the expense of the Local Authority.

That horses that react to the mallein test should be considered as suspected horses and should only be worked from an isolated stable or part of a stable.—The Departmental Committee made a recommendation "that horses that react to the mallein test should be considered as possible sources of infection." For this reason I suggest they should be brought together in one stable. It is quite true these are "possible sources of infection" but the positive stage of infection is only reached when clinical signs of disease are shown. So long as clinical signs of

disease are not apparent there is no reason why "reacting horses" should not work.

That the slaughter of all animals showing clinical symptoms of glanders should be made compulsory.—Slaughter is now permissive to Local Authorities with the result that on one side of a road glandered horses are killed and compensation paid, whilst on the other an owner is forced not to move the horse or to kill it at his own risk. This is a flagrantly unjust proceeding where difficulty of diagnosis exists or where the authority's inspector is a timid or a doubtful man. The administration of an Order should be carried out in every district with as much uniformity as possible. Where methods vary there is an invitation to the smaller and less reputable owners to move diseased horses into the district which is most liberal or least offensive. This alteration of the Order is also a recommendation of the Departmental Committee of 1899.

That compensation for horses slaughtered should be one-fourth value for all diseased animals—no animal to be valued at over £25.—The Departmental Committee recommended a maximum payment of one-fourth value but offered no protest against the minimum of £2 now permitted by the Order.

Whatever the amount of compensation allowed it is obvious that it should not be different in every county; it should be uniform. Upon what facts or statements the Committee based their opinion I am unable to guess, but they say "Considering that an animal that shows clinical symptoms of glanders is practically only worth the amount that could be obtained for the carcass, the Committee are of opinion that the compensation as above mentioned (the minimum of £2) is sufficient in those cases in which the animal shows definite clinical symptoms prior to slaughter."

This statement of the Committee is based upon the erroneous view that once clinical symptoms are apparent the horse is valueless. It is a gross error, and I have no hesitation in saying that quite one-fourth of the cases of farcy that occur may so far recover as to present no sign of the disease and then can be sold. Small owners know this and have a great temptation to neglect the notification of a £20 horse for which they will be paid only carcass value. A more liberal offer of £5 (the one-fourth) would induce notification, and so stay the spread of disease by sale of "patched up" clinical cases.

That compensation for horses slaughtered solely on account of reaction to a mallein test should be half the value—no horse to be valued at over £40.—The Departmental Committee recommend compensation "on a higher scale" and suggest a liberal one by fixing the maximum payment at £50. If Local Authorities found themselves liable to pay £50 for a "reactor," I fear the use of mallein would not be encouraged. Rich men know the dangers of infection and are easy to deal with even with no compensation. The majority are not rich, a minority are poor and artful. It is for these we want a liberal and a uniform treatment.

Healthy horses (non-glandered) slaughtered by a Local Authority either in error or by negligence should be paid for in full.—There is no hardship in this, as anyone with patience, ordinary intelligence, and mallein need not kill a horse till they are certain of its condition.

The value of any horse killed on a mallein reaction should be agreed

between the owner and the veterinary inspector before slaughter. Section 14 of the Order should be made intelligible. It provides for ascertainment of value of a horse by proceedings which commence six days after the animal has been buried or boiled. It seems to me a little difficult to arrive at the value then.

There are two recommendations made by the Departmental Committee which are of less value: (1) That it should be obligatory on all veterinary surgeons to notify cases of glanders of which they become aware. (2) That occupiers or owners of knacker's yards should notify any case found in their yards.

This last suggestion was in force in London for some years, but no cases were notified save those reported to the authorities. The knacker and his men said "How are we to know glanders from other diseases?"

The Departmental Committee also recommended "That the Board of Agriculture should exercise a more extended supervision of the working of the Glanders Order." This is a very useful suggestion if by it is meant that the Board should act decisively when appealed to by an owner or a Local Authority who disagree over a case. I have known more than one instance of appeal, but the Board simply evaded and postponed action as though afraid to act. One possible explanation of the Committee's recommendation, viz., that the Board should take an active part in the looking into all large or new outbreaks, I consider useful, provided it is not entrusted to lay inspectors. It would be absurd in the extreme to send a layman down to assist in preventing the spread of glanders in a town, and still more absurd to entrust him with an inquiry into the origin of outbreaks. That is work for a veterinary expert.

EDITORIAL ARTICLES.

DIPHTHERIA OF THE COW.

THE question whether bovine animals ever contract diphtheria in natural circumstances, that is to say, apart from cases of experimental or intentional infection, is obviously one of much practical importance. On various occasions during recent years medical officers of health and others have brought forward evidence to prove that the question must have an affirmative answer, but as a rule careful analysis of the facts cited in these cases has shown that, at the most, they only warranted the conclusion that diphtheria had been spread among human beings by the agency of milk. It is obvious that the occurrence of an outbreak of diphtheria involving a number of families who have nothing in common except the source of their milk supply falls a long way short of the evidence necessary to prove that diphtheria is a disease naturally contracted by animals of the