Proceedings
FORTY-SEVENTH
ANNUAL MEETING
of the
UNITED STATES
LIVE STOCK SANITARY
ASSOCIATION

HOTEL LA SALLE
Chicago, Illinois
December 2, 3, 4, 1943
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Records of the early meetings of the Interstate Association of Live Stock Sanitary Boards are very meager. The first meeting of the organization was held in Fort Worth, Texas, September 28-29, 1897, primarily to inspect a vat for dipping cattle and sheep that had been constructed in that city.

The name of the organization was changed at the 13th annual meeting held in Chicago, Ill., in 1909, to the United States Live Stock Sanitary Association. All meetings since 1909 have been held in Chicago.

<table>
<thead>
<tr>
<th>Meetings</th>
<th>Date</th>
<th>Place</th>
<th>President</th>
<th>Secretary</th>
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<tr>
<td>1</td>
<td>Sept. 28-29, 1897</td>
<td>Fort Worth, Tex.</td>
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<td>2</td>
<td>1898</td>
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<td>3</td>
<td>1899</td>
<td>Chicago, Ill.</td>
<td>E. P. Niles</td>
<td>F. T. Eisenman</td>
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<td>4</td>
<td>1900</td>
<td>Louisville, Ky.</td>
<td>W. H. Dunn</td>
<td>Hon. W. P. Smith</td>
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<td>5</td>
<td>Oct. 8-9, 1901</td>
<td>Buffalo, N. Y.</td>
<td>W. E. Bolton</td>
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<td>7</td>
<td>Sept. 22, 1903</td>
<td>Denver, Colo.</td>
<td>Hon. W. P. Smith</td>
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<td>8</td>
<td>Aug. 23-25, 1904</td>
<td>St. Louis, Mo.</td>
<td>Chas. G. Lamb</td>
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<td>9</td>
<td>1905</td>
<td>Guthrie, Okla.</td>
<td>W. H. Dalrymple</td>
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<td>11</td>
<td>Sept. 16-17, 1907</td>
<td>Richmond, Va.</td>
<td>John F. De Vine</td>
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<td>14</td>
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<td>Chicago, Ill.</td>
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<td>33</td>
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<td>C. A. Cary</td>
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<td>35</td>
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<td>A. E. Wight</td>
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* Information not available.
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<td>Walter Wisnicky</td>
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<td>R. W. Smith</td>
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<td>H. D. Port</td>
<td>Mark Welsh</td>
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<td>E. A. Crossman</td>
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<td>1941</td>
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<td>I. S. McAdory</td>
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</table>
ADDRESS OF THE PRESIDENT

BY W. H. HENDRICKS, D.V.M., B.Sc.

Salt Lake City, Utah

It is my pleasure to greet you here and to invite you to participate in the proceedings of this meeting. Some thought was given to the advisability of holding a meeting during these critical times, but after discussing the situation pro and con, it was decided that this organization should be kept alive and functioning, that in the interest of the livestock industry we should keep abreast of the times in our efforts to protect and preserve the livestock of the nation. It was felt that the men who are charged with these responsibilities through affiliation with this association should meet and endeavor to lend their organized efforts to the solution of the many perplexing problems that confront us.

It has been my pleasure to work with the new secretary, the vice-president, and the various committees in the preparation of this timely and instructive program, which we hope you will enjoy.

I think I should announce to you at this time that your most able and efficient secretary-treasurer, Dr. Mark Welsh, who was re-elected a year ago at our meeting held here, found it necessary to resign his position on September 15, 1943. Dr. Welsh, as you know, did a very fine piece of work during the time that he served the association and he is to be highly commended for the efficient way in which he has performed his duties. It has been my opportunity to work with him and I have appreciated that very much. It is to be hoped that he can continue to give his active support to the association. Dr. Welsh had already made some preparations for this meeting and he has given us much valuable assistance since he resigned.

It was necessary for your president to select a new secretary at a time when the business of the organization was pressing and at a time when arrangements had to be made for this meeting. All of the work of preparing a program had to be completed and it was necessary to select a capable man who could, on short notice, take over the work of the secretary and carry on. Dr. R. A. Hendershott was appointed and he very graciously agreed to assume the duties and responsibilities of the secretary-treasurer. I feel that the association was fortunate in securing the services of Dr. Hendershott. He responded when we needed him and he has done a swell job. I am sure you will agree with me, as this fine program is evidence of his work, and I want to thank him because he has helped me over a rough spot.

When I reflect over the history of this organization, and call to mind the many splendid, high type, and accomplished men who have, at various times, served as president and officers of this association, together with the many achievements to their credit, it makes me feel very humble, indeed, to be honored with this responsibility. It is my hope that, with your help, we can uphold and carry on the traditions, aims, and ideals of the organization for the protection and preservation of the livestock industry.

The annual meetings of the United States Live Stock Sanitary Association have
always been interesting and instructive to the members who participated and, also,
of immeasurable service to the livestock industry. This meeting, we hope, will be
no exception, although it is held under stress, anxiety and somewhat trying cir-
cumstances brought about by the great conflict in which we are engaged.

At this, the 47th annual meeting, many of the members who have been active in
the past are now serving in the armed forces. More veterinarians and livestock
sanitarians are today serving their country than at any other time in our history.
We have met at a time when our nation is engaged in a life and death struggle for its
very existence. At no other period in our history have so many homes been
represented in a military endeavor. Perhaps at no other time has the necessity for
food production and conservation been so great. With the responsibility of feeding
the armed forces, the civilian population, and the starving and war-torn countries
of the world resting on this country, the membership of this organization certainly
has a task that it cannot take lightly in the protection of the livestock of the nation.
We must formulate the policies and point the way for the prevention and control of
livestock diseases, scourges, and losses, whether or not these losses occur from
specific infections or as a result of faulty nutrition.

Livestock and livestock products constitute a large and important part of our
national food supply. The industry must be protected and a greater percentage of
the livestock population preserved from disease mortality. The opportunities for
study, research, and practical experience in disease control, which we have enjoyed
in the past, now make it our responsibility to protect and conserve the livestock of
the nation. We must be on the alert to prevent the introduction of livestock dis-
eases from foreign countries during and following the war, and we must assist in
protecting and preserving the public health at all times. Much is expected of us
and it is with the concerted and united efforts of all of us that we hope to live up
to that expectation.

Over a period of 40 years or more, there have been many changes in the individual
memberships of this association. We have seen many experienced men who have
accomplished a great deal leave the ranks for one reason or another, and new and
energetic men take their places and prepare to assume and carry on the great
responsibilities of the various phases of livestock sanitary and disease control work.
The ultimate aims of the United States Livestock Sanitary Association remain the
same, but new personalities lend their efforts from time to time to help carry us on
to an accomplishment of that aim. The benefits that have and will continue to
come to the livestock industry as a result of the policy-making programs inaugurated
through the association are difficult to estimate in dollars and cents, but our efforts
have always been toward the preservation and improvement of the livestock
industry.

One of the experienced and accomplished men who has been so prominently
identified with the United States Livestock Sanitary Association, and who has
helped to guide its destinies, is Dr. John R. Mohler, recently retired Chief of the
United States Bureau of Animal Industry, a man who has many wonderful achieve-
ments to his credit and a leader in livestock sanitary work. We will miss his per-
sonal contacts and his wise counsel in our future work. We extend our appreciation
and our best wishes to Dr. Mohler.
The responsibilities of the United States Bureau of Animal Industry have been assumed by a new chief, Dr. A. W. Miller, a man who has been trained and experienced in the activities of the Bureau of Animal Industry and an active member, booster, and friend of this association. We know that we will continue to have the fine leadership and cooperation of the Bureau of Animal Industry through Dr. Miller. We congratulate him and welcome him as one of the men who is assuming new responsibilities to help carry on our work.

In past addresses a progress report has been given with respect to tuberculosis, contagious pleuro-pneumonia, cattle scabies, sheep scabies, foot and mouth disease, anthrax, and Texas fever. I shall not, therefore, refer to them. In fact it is not my intention to speak at length of the various livestock diseases, but only to call to your mind a few that I feel should receive attention.

Brucellosis is undoubtedly the outstanding problem before us today. Even though much has been done, we are far from a uniform and successful control program. Calfothd vaccination has received considerable attention and has given very promising results. In some sections of the United States, it is the program of choice while in other sections it is less enthusiastically supported. There are still many opinions to be correlated and unified.

The cattle industry is looking to this association for guidance toward a uniform and practical control program. At the present time, we need to conserve our food supply and perhaps it will be necessary and probably advisable to change, somewhat, our methods of control, for the time being at least, and endeavor to keep many more of our valuable breeding stock in the reproduction line. Without a doubt we shall be called upon to furnish foundation and breeding stock for the ravaged and wartorn countries of the world. Our responsibilities, therefore, in this respect, are truly great. I hope we are equal to the occasion.

There is another point with respect to this disease that I would like to call to your attention. Cattlemen, livestock shippers, and all who have anything to do with interstate movement of livestock are complaining, and rightly so, with respect to the numerous and varied state sanitary requirements on Brucellosis. Certainly this association should endeavor to bring about more uniformity in this respect.

Equine encephalomyelitis is still causing a great deal of concern in some sections. Horses and mules are finding their place in our American way of life again and if for no other reason than through necessity, will be more in demand. While the development and use of an effective vaccine gives us an assurance in a safety program of preventive control for this disease, yet, it is our duty to see that this program is properly and adequately applied.

In some parts of the United States Rabies is a menacing problem. Opinions vary regarding control methods and results are anything but constant; therefore, any new facts that can be given on the control of this disease should be made available. It is a difficult problem for regulatory officials to cope with and this association should continue to give a great deal of thought to its suppression and control.

At this particular time our attention should be directed, as far as possible, toward the war effort. This means the increased production and conservation of our food supply. Our particular responsibilities in this respect will be with livestock and livestock products—namely, meat, milk, poultry, eggs, etc. It can and should be
our duty to lend support to any disease control program that will increase and preserve these products.

There are some diseases of dairy cattle that should receive particular attention, and an honest effort made to prevent and control them if we are to increase dairy food products. I refer to sterility which may be caused both by nutritional or functional disturbances and by infections. Entirely too many valuable breeding animals are lost to us because of these conditions.

Mastitis is probably responsible for more cows being removed from producing herds than any other one factor. Certainly our efforts should be redoubled in devising ways and means of preventing and controlling this serious disease of dairy cattle.

Diseases of new-born calves are very serious in many cases. Too large a percentage of calves succumb to gastro-intestinal infections, particularly so-called calf scours. These diseases should receive more attention in our livestock sanitary control work.

We are happy to state that a very interesting and instructive symposium on dairy cattle diseases will be presented for your pleasure on this program and we invite you all to participate with us.

Anaplasmosis is an insidious and serious disease of cattle that is causing more losses than we are perhaps willing to admit. Some sections of the United States have a real problem with it and we have reason to believe that affected cattle are being shipped from one section to another. We should inaugurate measures to control and prevent the dissemination and spread of this disease.

Parasitic diseases are responsible for enormous losses in every class of livestock, and in some instances these diseases are directly transmissible to humans. Therefore, renewed efforts should be put forth to control them.

As of August 1, 1943, it was estimated that there were in the United States 11 million more hogs than during the previous year. This is a comforting situation if we are able to feed them to marketable age and weight. Many, however, succumb to enteric diseases in addition to numerous losses from other diseases which persist even though there are biologic preventive and control measures available. The enteric diseases cause enormous losses of our food supply derived from the swine population and we are critically in need of new information for the prevention and control of these diseases. Certainly we have a responsibility to the livestock industry in this respect.

A very vital and large portion of our food supply is derived from poultry. The demand for turkeys, chickens and eggs has been and is great. The armed forces have been taking great quantities of these products. We have been asked to increase the supply. In view of the shortages of both high protein and grain feeds, it would seem that our attention should be centered on a program of poultry disease prevention and control. It is estimated that in many sections adult poultry mortality will vary between 25 per cent and 35 per cent. Certainly our efforts should be redoubled in an endeavor to save at least a portion of this loss. I would like to call your attention to the very fine program sponsored by the United States Bureau of Animal Industry in cooperation with the leading poultry interests of the nation to conserve poultry and poultry products. The program is under the able direction of Dr. Cliff Carpenter. It is known as a "Poultry Conservation For Vic-
tory Program." Some very fine work has been done already and this association had a voice in its inception. I think we should get behind this program with our cooperation and our support.

Perhaps at no other time has so much need arisen for the science of meat and milk inspection. Today more trained men are engaged in this work than ever before. As the conflict continues and the food situation becomes more acute, the need for increased vigilance in meat and milk inspection work will become apparent. Therefore it is our responsibility, in the words of the by-laws, "to study livestock sanitary science and meat and milk hygiene and disseminate the information relating thereto." If the organization functions as was contemplated, we now have additional reasons for discharging our responsibilities with respect to meat and milk inspection work.

One of the responsibilities of this organization, as stated in its by-laws, is the unification, so far as possible, of the laws, regulations, policies and methods pertaining to milk and meat hygiene and the prevention, control and eradication of transmissible livestock diseases.

Certainly we are in need of more uniformity in our various laws and regulations governing interstate movement of livestock. A regular committee, and at times a special committee, has had this assignment each year. While a great deal has been accomplished along this line, there is much more desired uniformity to be attained. I should like to recommend that the association give more attention to this important responsibility.

I want to thank the various committee members for their splendid efforts in helping to prepare the program. I am indeed grateful to them as I am to all of those appearing on the program. It has been a pleasure to have been associated with you. I think we all owe a debt of gratitude to our new and able secretary, Dr. R. A. Hendershott, who has so kindly agreed to help us out and who has done such a fine job.

In conclusion, may I say, it has been a pleasure to have had the privilege of working with you and of serving in this capacity. I have faith in the United States Live Stock Sanitary organization and I believe the livestock industry will continue to be greatly benefited by its policies.
**REPORT OF THE SECRETARY-TREASURER**

**FINANCIAL STATEMENT**
December 1, 1942 through November 30, 1943

**Receipts**

| Item | Amount  
<table>
<thead>
<tr>
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<tbody>
<tr>
<td>Cash in Prince George's Bank &amp; Trust Company 11/30/42</td>
<td>$2,815.04</td>
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<tr>
<td>Interest U. S. Treasury Bonds</td>
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<tr>
<td>Official Memberships</td>
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<tr>
<td>Individual Memberships</td>
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<td>Registration Fees</td>
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<td>Reprints and Proceedings</td>
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<td>Displays</td>
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Total | $5,619.17 |

**Disbursements**

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<td>Clerical Assistance</td>
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<td>Fidelity Bond Premium</td>
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<td>Postage</td>
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<tr>
<td>State of Delaware tax</td>
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Total | $3,415.67 |

Cash on hand First Mechanics National Bank, Trenton, N. J. 11/30/43 | $2,203.50 |

**Assets**

| Item | Amount  
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<td>U. S. Treasury Bonds</td>
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Total | $7,219.00 |
REPORT OF THE SECRETARY-TREASURER

Liabilities
Balance 11/30/42 ........................................ $7,731.54
Net loss ......................................................... 512.54

Net Worth November 30, 1943 ......................... $7,219.00

R. A. HENDERSHOTT, D.V.M.
Secretary-Treasurer

REPORT OF THE AUDITING COMMITTEE

C. U. DUCKWORTH, Chairman, Sacramento, California; C. C. FRANKS,
Des Moines, Iowa; and FRANK L. CARR, Columbus, Ohio

Your special Auditing Committee has examined the books of the Secretary-
Treasurer and find them in good order and the Treasurer's report true and correct
as presented.
It was moved and passed that the report of the Auditing Committee be accepted.
MEMORIAL SERVICE
United States Live Stock Sanitary Association
December 1, 1943
By J. L. Axby, D.V.M.
Indianapolis, Indiana

The men, whose names I shall read, have passed away during the past year.
Frank Foster McNeely (K.C.V.C. '17) of Colfax, La., former B.A.I. inspector, died April 24, 1942.
W. L. Martin (K.C.V.C. '11) of St. Louis, Mo., former B.A.I. inspector, died July 12, 1942.
John C. Hargrave (McGill '95) of Medicine Hat, Alberta, Canada, former inspector for the Health of Animals Branch, Dominion Dept. of Agriculture, died October 15, 1942.
Benjamin F. Swingley (Ont. '82) of Freeport, Ill., former state veterinarian of Illinois for 23 years, died October 16, 1942.
Ira V. Carpenter (Ind. '13) of South Bend, Ind., former B.A.I. inspector, died December 6, 1942.
D. S. Tamblyn (McGill '01) of Kingston, Ont., former district veterinary inspector for the Health of Animals Branch, Dominion Dept. of Agriculture and formerly chief of the Canadian Army Veterinary Corps, died January 2, 1943.
Howard H. Cohenour (McK. '01) of Salt Lake City, Utah, former inspector in charge of tuberculosis eradication, B.A.I. died January 15, 1943.
H. K. Berry (O.V.C. '02) of Glen Rock, N. J., inspector for the New Jersey B.A.I. for 25 years, died January 24, 1943.
Frank A. Ingram (Ont. '92) of West Hartford, Conn., former B.A.I. inspector and deputy Commissioner of Domestic Animals for Connecticut, died January 28, 1943.
Edison Bodwell Ungar (Ont. '19) of Moncton, New Brunswick, former inspector in charge of the Health of Animals Branch, Dominion Dept. of Agriculture, died February 2, 1943.
George A. Johnson (I.S.C. '86) of Orlando, Fla., former B.A.I. inspector, died February 3, 1943.
David Crozier Tennent (Ont. '06) of Toronto, Canada, former assistant chief inspector, Health of Animals Branch, Dominion Dept. of Agriculture, died February 4, 1943.
MEMORIAL SERVICE

Chas. J. O'Brien (McK. '08) of Chicago, Ill., former B.A.I. inspector, died May 1, 1943.

John H. McLeod (McG. '04) of Charles City, Iowa, former assistant state veterinarian of Iowa, died May 8, 1943.

Earl Louis Burnett (Corn. '23) of Ithaca, N. Y., former associate professor of poultry diseases and prominent investigator of these diseases at Cornell University, died May 14, 1943.


Maynard Rosenberger (I.S.C. '03) of Tarzana, Calif., former B.A.I. inspector and investigator in diseases of cattle, died June 11, 1943.

William Ross Cooper (I.S.C. '92) of Kansas City, Kansas, former B.A.I. inspector for 35 years, died September 24, 1943.

Geo. T. Casper (McK. '05) of Klamath Falls, Ore., former inspector of the B.A.I for 20 years, died October 7, 1943.

M. Jacob (U.P. 1899) of Knoxville, Tenn., Treasurer of A.V.M.A. since 1918, and Dean of Agriculture, Knoxville, Tenn., died March 22, 1943.

F. A. Imler (O.S.U. '06) of Kansas City, Mo., inspector in charge of serum and virus control for the B.A.I. in Kansas City, Mo., died September 17, 1943.

F. A. Laird (C.V.C. '02) of San Antonio, Tex., ex-state veterinarian of Illinois, died August 4, 1943.

STANDING SILENT PRAYER BY ALL

These men were all engaged in some phase of livestock health and sanitation. Some attained fame and wide recognition in their respective paths of duty and service. Each one left some memory filled with acts of love and kindness and in deference to what they did and what they were, we humbly honor and memorialize them today.

They were typical Americans in the use they made of their opportunity. There will be few such men at best, and we will not look into the mystery of how and why they come; but in knowing them, we will have our own faith supported in the old, old story of an all wise Providence and Jesus and his love, bravely proclaiming,

"I know not where His islands lift
Their fronded palms in air
I only know I cannot drift
Beyond His love and care."

We shall not let the good that was in them be interred with their bones, but rather emulate the good they did. Thus, their lives go on.

We shall carry out their precepts of service, righteousness and goodness, hoping for a comparable courage to stand as—

"They stood upon the world's broad threshold; wide
The din of battle and of slaughter rose;
They saw God stand upon the weaker side,"
That sank in seeming loss before its foes.
Many there were who made great haste and sold
Unto the cunning enemy their swords,
They scorned their gifts of fame and power, and gold,
And, underneath their flowery words,
Heard the cold serpent hiss; therefore, they went and
Humbly joined them to the weaker part, well content,
So they could be nearer to God's heart
And feel its solemn pulses sending blood
Through all the widespread veins of endless good.

Thus they came, lived and died. Now they await that last clear, clarion call
to arise and go to that place, the man from Gallilee has gone to prepare for all who
believe in Him.

Until that time may their souls rest in peace.

Mr. President, I move this service be entered upon the permanent records of
the Association and that a copy be sent to each family.
WHY A LIVESTOCK CONSERVATION PROGRAM?

BY FRED H. LEINBACH, B.S.A., M.S., PH.D.

Chicago, Illinois

It was with pleasure that I accepted the kind invitation of your Secretary, Dr. Hendershott, to appear on this program to discuss the subject of livestock conservation. It is a subject we are all mightily interested in and one which to many of you has been close to your thinking for a long, long time.

Livestock conservation implies the guarding, protecting and marshalling of our livestock resources in the light of available feed, manpower and labor to obtain the maximum production of meat, milk, eggs and other animal products. It calls for maximum efficiency of production, a matter which warrants far greater consideration than it has received sometimes in the past.

It is not my purpose to cite many figures in this discussion. However, it might be well to keep in mind that a conservative estimate of the annual loss sustained by the livestock industry from the more important diseases and parasites is nearly one half billion dollars—that the loss from the failure to generally adopt improved production practices which have been developed is probably greater than the combined loss sustained from disease and parasites.

Serious as is this monetary loss—and it represents a loss to livestock producers of an estimated nine dollars out of every one hundred received from the sale of livestock and livestock products—it also can greatly affect our whole war effort. The fact is we need every pound of meat, every drop of milk, every dozen of eggs and every bit of every other animal product we can possibly produce.

Feed supplies are less bountiful this year than they were during the past year. Already liquidation of commercial breeding herds and flocks is occurring in some sections of the country. The problem then is one of producing as much or more than we did last season with reduced feed supplies. It is well to ask “Can this be done?” The answer to that question is “yes” and in support of that answer a few illustrations of savings which can and must be effected are in order.

The average pig when born represents a feed investment of 140 pounds according to U. S. Department of Agriculture figures. A saving of one additional pig per litter in 1943 would have allowed the same total production with only 86 per cent as many brood sows kept for farrowing and would have allowed a saving of more than fifty one million bushels of feed, (corn equivalent), if all death losses had occurred at farrowing time. There are just too many instances of the better hog producers who are raising 8 or more pigs per litter year in and year out to convince me that our national average of a little over six pigs raised per litter isn’t lower than we ought to be satisfied with or that it isn’t possible to increase this by at least one pig per litter.

Illinois farm surveys of a few years back show that hog men using approved sanitation methods raised from 1.6 to 2.7 more pigs per litter than others who produced their pigs under the permanent hog-lot system, yet the fact that a goodly proportion
of our pigs are still being raised under the permanent hog-lot system attests to our either being unable to convince old hog producers that swine sanitation pays or to our not being able to get that story to tens of thousands of newer hog producers or both. In any event it calls for a more intensive, more thorough and more convincing educational program than we have had.

Again let us consider our losses from hog cholera. The incidence of this disease is now at an all-time low. Yet, before all the pigs farrowed in 1943 are marketed, it will have exacted a toll of more than two million pigs from that crop, have wasted more than twenty million bushels of needed feed supplies and prevented some two hundred million pounds of needed meat supplies from reaching market. Is it not possible to still greatly reduce the extent of this loss?

I am informed that surveys conducted in some of the intensive dairy sections indicate that milk production is being reduced as much as 25 per cent as a result of mastitis. The decreased output not only greatly affects profits, it represents an enormous feed wastage and a greatly reduced supply of a product greatly needed for the war effort. We are aware of the vast difficulties which accompany the effort to reduce this loss but I dare say no one in this audience is willing to admit that it can't be greatly reduced.

A great deal has been done to reduce the loss occurring from Bang's Disease in dairy and beef cattle herds in this country. Yet a great deal can and needs to be done to reduce this still further.

One needs only to observe the high percentage of "pee-wee" or cull lambs that reach some of our markets to realize that needless production losses are not peculiar to any one group of livestock producers but rather that they are more or less common to all. Many additional illustrations could be cited but those already enumerated should prove sufficient to illustrate the extent of these types of losses we are at present sustaining.

Many persons engaged in the fields of Veterinary Medicine and Husbandry are fearful that during the present winter season many market and breeding animals may be inadequately fed both on a quantitative and on a qualitative basis. It is absolutely essential that pertinent information relating to the nutrition of farm animals be assembled and gotten to livestock producers in simple, understandable language so that efficiency of production will not be lowered and to prevent permanent damage to breeding stocks. This problem, unfortunately is not peculiar to this season—it is one which is merely apt to be accentuated because of reduced and changed feed supplies.

In discussing this problem of livestock conservation brief mention should be made of the gains which improved breeding can bring about in increasing the efficiency of our animal units. That we should go far in exploring the possibilities in this direction, there can be not the slightest doubt. We have come a long way in increasing efficiency by the use of better sires, but we are still far from reaching our ultimate goal. The vast difference in feed utilization of offspring of different sires where fed and handled under similar environmental conditions drives this point home in no uncertain terms.

As an animal husbandman, I am concerned with the future of the livestock industry in the post-war period. It seems to me that we must consider the possi-
bility of outside competition for our markets and reduced prices for our products. Unless we can do something about reducing the extent of the wastes in production and otherwise increasing the efficiency of production we may find ourselves confronted with a "Peck of trouble." But I should add that I, for one, am optimistic that producers with all our help can and will successfully meet this challenge.

The National Livestock Conservation Program is a cooperatively conducted campaign designed to acquaint producers with facts relating to the extent of present losses, wastes and inefficiencies in production and marketing which are now being tolerated, and to help them in reducing these losses thus helping to insure a maximum supply of meat, milk and other animal products needed for Victory and a sound livestock industry, in the post-war period. The Program is sponsored through the National Committee on Boys' and Girls' Club Work in cooperation with the U. S. Department of Agriculture, State Agricultural Colleges, 4-H Clubs, Future Farmers of America, the livestock, dairy and poultry industries, and other interests and agencies closely allied to agriculture.

Was there ever a more opportune time for all groups interested in the welfare of the livestock industry to unite in a cooperative and constructive effort to help eliminate these wastes in production and to develop a more efficient, a more substantial and a more profitable livestock industry for the future?
THE CURRENT FEED SITUATION AND WHAT LIES AHEAD

BY DR. R. M. BETHEKE

Ohio Agricultural Experiment Station

Much as I would like to bring a bright and encouraging picture on the current feed situation to you gentlemen, I regret very much to tell you that the feed situation is anything but bright in terms of the production goals and our livestock population.

We are trying to do the largest food production job in the history of this country. In fact, I am not too sure whether it is a job that is bordering next to the impossible when we consider feed supplies, manpower shortage, transportation and distribution facilities and difficulties, and maybe in some respect governmental policies.

It is not my purpose to discuss governmental policies, the manpower shortage, transportation or distribution facilities, but rather to give you a brief picture, in my humble way, of what the feed situation looks like in terms of available supplies, to feed our present livestock and poultry population.

You gentlemen know as well as I that the country as a whole has been short of protein feeds ever since last fall or a year ago. Last January the Feed Industry Council, of which Mr. Boling is Vice-Chairman, estimated that we were about 20 per cent short in high protein concentrates. Based upon the experience of men in the industry, and from what they tell me, I am informed that the estimated 20 per cent which we were short last year, if anything, was low. I am told by men in the industry that we were probably closer to 30 per cent short in terms of the demands.

Bear that figure in mind. If we were 20 per cent short last year, and according to government estimates on January 1 of this year we have 10 per cent more live stock on our farms and in our feed lots in the United States, and the government telling us that we will not have any more feed supplies, that we will have about the same amount of high protein concentrates available this feeding year as we had last year, but with 10 per cent more animals to feed, and if we were conservatively 20 per cent short last year, you can readily see what the picture is like this year—anything but bright.

What about the grain picture? Again, the overall grain picture gives little cause for comfort, in spite of the fact that Mother Nature and our good American farmers have blessed us with a good grain crop this year. Our grain crop this year is not comparable to our bumper crop of last year.

As far as I can determine, according to government figures our corn crop this year is between 3 and 4 per cent less than last year. Wheat production is off 15 per cent; oats are down nearly 16 per cent; barley 22.5 per cent, and the sorghum grains not quite 6 per cent.

What about roughage? Hay production, according to government figures, is 8,000,000 tons less than last year, or approximately 6 per cent. Another thing we should not lose sight of, and which we must take into consideration, is the rate of feeding.
The rate of feeding during the past year has been so heavy that our carry-over of grains into this feeding year has now been reduced almost to the bare minimum. In other words, we are facing a situation where we are almost scraping the bottom of the bin, and we will have to depend more and more upon what we actually produce in a year, and not two, three or four years ago.

The October feed situation report of the Bureau of Agricultural Economics has this to say in connection with the disappearance of feed grains: "Disappearance of all feed grains, including wheat and rye feed on farms, was about 35 per cent greater during the period July to September this year than last. With the disappearance of feed grains running at a very high level through September this year, it seems likely that a larger than usual proportion of the total supply will be consumed during the first half of this feeding or marketing year. Should this occur, the pinch in feed supplies probably will become more acute during the spring and early summer of 1944."

All of the foregoing, I think, can be summarized in the brief statement which is currently appearing in government reports, to the effect that we have between 12 and 15 per cent less feed per animal on farms in this feeding year than last year. In other words, to state it in another way, according to government figures we now have about seven animal mouths to feed, and only enough feed to take care of six of them.

Since our reserve stocks of feeds have been largely used up, it is evident that every day we permit seven animals to eat out of our national feed trough we shorten the feed period that our feed supplies will last, and we will soon reach the time when we can only feed six, five or four.

With hay production down 6 per cent, oats off 16 per cent, barley 22 per cent, grain sorghum 6 per cent and corn between 3 and 4 per cent, it is obvious, to any thinking individual that we cannot hope to make up our feed grain deficiency through the unlimited use of wheat, for this year's crop is about 15 per cent smaller, and our carry-over greatly reduced.

If we were to depend upon wheat we might possibly invite a bread shortage. Furthermore, fall moisture conditions in a large section of the winter wheat belt have been anything but encouraging. I am informed that for the past few days the milling industry has been together and discussed the wheat problem. They have informed the government in very emphatic terms that the government will have to safeguard some of the wheat we have on hand if we are to prevent a possible bread shortage. I think you will agree with me that human food is going to come before animal feed.

I would like to give you a few figures which in my estimation tell a very convincing story. One of our difficulties in connection with the feed situation is that we have been thinking too much in terms of certain geographical areas. I am thinking of the feed situation on a national basis.

The problem in one geographical area, naturally, is different than in some other geographical area, and in discussing the feed situation with your friends it is well to impress upon them the national situation as well as the local situation, and endeavor to get them to think in terms of the national situation.

We hear a great deal about the bread basket of America, or the corn belt, or the corn belt versus, you might say, the rest of the country. According to figures, in 1941–1942 there were produced in the six cornbelt states 2,100 pounds of feed per
animal unit, that is, grains and concentrate feeds. We consumed in that year, in the corn belt, 1,747 pounds per animal unit. It is estimated that during that particular year 8 pounds of wheat were fed per animal unit.

In 1942 the production in the corn belt was 2,123, up a little bit from the year previous. Our consumption, instead of being 1,747 pounds, was 1,882 pounds. Our wheat consumption stepped up from 8 pounds to 65 pounds in the corn belt.

In 1943, or this feeding season, we are told that the production per animal unit in the corn belt is on the order of 1,896 pounds. If we continue to feed as we did last year, when we consumed in the corn belt 1,882 pounds, you can readily see that there is just about enough feed to take care of the animals in the corn belt. That leaves nothing for the rest of the country.

What about the other 42 states? In 1941–1942 the production in the other 42 states was on the order of 1,205 pounds per animal unit; the consumption was 1,441. The wheat consumption was 41 pounds. In 1942–1943, the past feeding season, the production was 1,242, and the consumption was 1,605. The wheat consumption jumped from 41 pounds to 194 pounds.

The production this year in the non-corn belt states was on the order of 1,114 pounds. If the non-corn belt area expects to feed as they did last year, they would require in the neighborhood of 1,600 pounds of grains in addition to 165 pounds of wheat.

Where are we going to get it from if they can't get it from the corn belt? Well, you say, how about imports? In this connection we naturally think of our northern neighbor, Canada, who might help out. We are just entering the season when navigation on the Great Lakes is closed, and the boats are few and far between. The boxcar situation, as you gentlemen know, is very tight. Thus we can expect little help from Canada at present.

Furthermore, the Canadian grain crops were much smaller this year than last, and Canada also has an increased live stock population; so I doubt very much whether Canada is particularly anxious to release any of their grains to this country.

Accordingly, we will have to depend more and more on our own feed supplies in order to feed our live stock.

On October 7 the War Food Administration made the following recommendations on the utilization of feed in order to attain the food goals for 1944; in fact, their recommendations are the food goals for 1944. I would just like to briefly review them for you:

One was that there should be sufficient feed to maintain milk and egg production on the 1942–1943 level.

Second, sufficient feed, and according to the federal government the feed should be so distributed, particularly protein supplements, to maintain 95 per cent of the number of range cattle that we had in 1942–1943.

Third, enough feed to feed our cattle to weights 5 per cent below average and to good grade.

What about hogs? For hogs, sufficient feed to finish out to normal market weights the 1943 spring and fall pig crops, and to raise a 1944 spring crop the same as the smaller 1942 size. This would mean farrowing about 20 per cent less pigs in the spring of 1944 than in 1943, and cutting down on the average market weights by about 25 pounds below those of 1943.

Fifth, sufficient feed to raise 80 per cent of the broilers raised in 1943.
Sixth, enough feed to raise 95 per cent of the number of turkeys, ducks and chickens, other than broilers, raised in 1943.

Seventh, sufficient feed to maintain the present sheep and lamb population.

These recommendations sound encouraging, but frankly I am not enough of an optimist to think it can be done with our current visible feed supply.

Government officials tell us that our feeds were used inefficiently last year. They cite the figure that it required three-quarters of a ton of feed to produce a unit of live stock product in 1942–1943, whereas in 1941–1942, or for that matter the average for the five years preceding 1942–1943, was only two-thirds of a ton. In other words, last year, according to their figures, we used three-quarters of a ton of feed to produce a unit of live stock product, and the average of the five years previous was only two-thirds of a ton. They claim that if we increase our feed efficiency during the current year to the 1941–1942 level, we can meet our 1944 production goals with available feed supplies.

I think it is only proper to ask, what has happened to the feed in 1942–1943 as compared to the previous five years. In my opinion there are several valid reasons for these differences in the utilization of feed on the part of the American feeder. Producers of hogs, as you know, were asked to carry them to greater weights, which they did. This naturally means less efficient conversion of feed into animal products. I doubt whether the government took into consideration the feed consumed by the thousands of backyard poultry flocks. Undoubtedly heavier feeding on the part of the producers to get more milk, meat and eggs was a factor. To what extent lack of help or manpower on the farm or in the feed lot was a factor, is not known, but it is reasonable to assume that it was a contributing factor.

Undoubtedly the short supply of protein supplements last year exerted their influence. Probably there are other valid reasons. The point I wish to make and leave with you gentlemen is this: I hate to see the American farmer and feeder accused of being wasteful, especially during a war emergency. In my estimation the American farmer and feeder is just as loyal, if not more loyal, than any other group in America, and I think we should take off our hats to the American farmers and feeders for the magnificent production job they have done in spite of the many difficulties with which they have been confronted.

What is indicated for the future? There is only one thing I know of. If we don’t have enough food to feed the livestock we have on hand, we will have to get rid of some of them.

If liquidation is necessary—and I think it is—I believe we should impress upon everyone that the liquidation should proceed in an orderly fashion, and not in a chaotic manner. In order to proceed in an orderly fashion, the government or somebody will have to give consideration to the manpower problem that is facing the packing industry. Some consideration will have to be given to geographical areas. Maybe some consideration will have to be given to different species of animals. The thing to do in certain sections of the country is to reduce our pork or poultry population percentage-wise more than our dairy population.

Whatever the solution might be, it is the duty of every one who is directly or indirectly concerned with the food production program, to do his just share. We must not only conserve feed but we must make every pound of feed count more than ever before, if we are going to do the job right.
CONFUSION AT THE TERMINAL MARKETS

BY JUSTIN C. CASH

*President, Kansas City Stock Yards Company*

I am honored in being asked to speak at your Association's Forty-Seventh Annual Meeting. I trust it will be valuable to both of us.

Confusion seems to be the word which indicates the state of mind of many people in the country, especially now, when rules and regulations come as variable as the weather and the uncertainty of the situation adds to the confusion. Confusion at the terminal markets is not as great in regard to livestock sanitary rules and regulations as about other things. I did not know the degree of confusion from market to market, and in order to get the point that caused confusion in the minds of market operators, I wrote to 40 markets and asked them 11 questions which would give me some idea of how they judged the livestock sanitary rules and regulations of their state and the states with which they customarily come in contact. Replies were received from 26 markets in 23 states. The answers to these questions may not be legally correct. They may be the answers of men who are not fully conversant with what the law is and how it operates, especially in regard to their competitors, yet it is the opinion of those who are in the livestock business and they express their opinions from a practical standpoint.

There seems to be a lot of variation in state regulations regarding the importation of livestock, except the movement of hogs. This is the most consistent and uniform of all regulations. All other rules seem to be changed in accordance with conditions in the particular state, or just left unchanged because of custom and habit.

It might be well to go over the questions and see the variation of replies in order that you may follow the confusion at the terminal markets in knowing whether a given kind or class of livestock can be admitted to a certain state. The first question:

1. **Q.**—Does your state have a veterinarian at your yard for inspection and enforcement of the livestock sanitary regulations?

   **A.**—Seventeen markets replied that they do have a state veterinarian at their yards. Seven answered "no" and two had lay inspectors at their yards.

2. **Q.**—Does your state have or enforce any livestock health regulations at the auction or community sales within its boundaries?

   Is there thorough inspection at these yards?

   **A.**—Nineteen of the markets indicated that there were regulations governing the auction markets within their state boundaries. Six of them said they had no regulations governing the auctions and one gave no answer. On the inspection at the auctions there were a lot of variations in replies. Eight said "yes, there was inspection at the auction markets"; six said there was no inspection at the auctions, and eight indicated that there was supposed to be inspection and that the state sanitary official tried to carry on some inspection work but it was only partly accomplished. Four of the markets did not reply to this question.
3. Q.—Can all sheep enter your state from your stock yards without being dipped?
   A.—Eighteen of the markets indicated that dipping was not necessary for sheep to move from their terminal market to their state. Five required them to dip under any conditions, and two required them to dip in all except inclement weather and gave certain months when the temperature was too low for dipping.

4. Q.—If you have to dip all sheep before "going to the country", what does the state require of sheep sold at auction or community sales, or of those coming through railroad feed yards to country points, in your state?
   A.—Thirteen of the terminal market operators indicated that it was not necessary for sheep to be dipped when leaving an auction market to go to the farms in their state. Eight indicated that it was necessary to dip the sheep before leaving the auction, and five failed to answer this question. Of course you would not expect a terminal market operator to know too much about the requirements of his competitors.

5. Q.—What does the state require before a cow or bull can be taken from your stock yards into the country?
   A.—It seems that regardless of whether the nation is accredited or not, T.B. tests are still in vogue. Fourteen of the markets indicated that both tuberculin and Bangs tests were necessary to move a cow or a bull to the country from the yards. Four of them indicated that the T.B. test was the only one necessary. Three of them required Bangs test only. Three required no test whatsoever and one failed to answer. The variation seems to be primarily in the class of livestock industry within the state. Most of the range territory was more liberal than the dairy sections.

6. Q.—Are stocker and feeder steers admitted to your State without tuberculosis or Bangs test? What restrictions, if any, are there on calf heifers for fattening?
   A.—Twenty markets do not have to test steers going to the country. One of them gave no answer. Five markets are still required to test steers going into their states. I believe it was Pennsylvania which checked the number of T.B. reactors in steers and found it very low—a small fraction of 1 per cent. It would seem that, from a practical angle, the testing of steers is unnecessary. On the admission of the stuff to the country from the yards, eight markets required both T.B. and Bangs test, two required Bangs only, three required the animals to be quarantined on the farm, seven required no test whatsoever, and four of the market operators failed to answer this question. There seems to be a lot of variation in the handling of cattle at the terminal markets for the states in which they are located, and others.

7. Q.—What state restrictions are there on the movement of hogs from your yards to the country?
   A.—The lack of uniformity in cattle is reversed when we consider the hogs. Two of the eastern markets have no regulations whatsoever by which feeder pigs may be removed from their market to the country. Two markets are allowed to move feeder pigs on permits, and twenty-two require the animals to be treated for hog cholera and either dipped or sprayed.

8. Q.—What particular veterinary rule or restriction of your state has given you the most trouble, and why?
A.—You might be surprised to know that the majority of the men indicated that they had no trouble nor any complaint. Those who did complain seem to have a pet peeve on something with which they run into trouble, usually some local condition which they thought unjust, unfair, or discriminatory to their market compared to other terminal markets or the small markets throughout the state.

9. Q.—What regulations in other states give you the most trouble and how would you remedy the situation?

A.—Those markets which complained most about the rules and regulations outside of their state were those which either handle feeder cattle or feeder lambs, or have quite a trade in dairy cattle. These, of course, are most of the Midwestern or mountain-state markets where there is a surplus of animals either to go to the Corn Belt or to the East. One Midwest market which handles a lot of feeder cattle and lambs makes this comment on requirements within their state and the movement to other states:

"The requirement that all cattle moving from here to country points within the state be shipped in cleaned and disinfected cars has recently been lifted, owing to the shortage of labor and difficulty in cleaning cars in cold weather. This regulation delayed movement of stock here awaiting proper equipment. Feeder sheep must move in cleaned and disinfected cars to country destinations within the state.

"The regulations applying in Wisconsin on feeding cattle are more rigid than those of other states. Permits must be obtained by the feeder designated by a number before shipping into the state. Otherwise they must be Bangs and tuberculin tested. Females must be tested irrespective of test charts showing test within 30 days.

"The State of Michigan has a regulation applying on feeding sheep which discriminates drastically against the public market. Movement through public markets are practically cut out because shipments must carry health certificates from the state of origin. As we get considerable far western shipments of lambs, out of which fats are taken, and in some cases lambs originating within the state are added, it practically eliminates our handling of feeder sheep in the state of Michigan. Our suggestion for improvement is that the state of Michigan be asked to accept a certificate from a B.A.I. inspector who would know if the lambs are from a scabie-free area."

10. Q.—What would you like to see done to make state livestock sanitary regulations more uniform?

A.—Most everyone indicated that they would like uniformity between the states in their rules and regulations governing the movement of livestock. A lot of the men felt that the regulations governing the auction markets and concentration yards in the country should be the same as those governing the terminal livestock markets. Some indicated that they thought the federal government should take a supervisory hand in the matter and see that all regulations were practical and uniform and by this means remove a lot of the variation in regulations.

11. Q.—Does your state chief livestock sanitary officer ever visit the yards and does he have a speaking acquaintance with the larger posted stock yards operations, cleanliness and freedom from contagious diseases?

A.—This is sort of a check on yourself to see how good a job you do in getting
CONFUSION AT TERMINAL MARKETS

around to the various spots, at least where the people expect you to be. Eighteen of the men said that you visited them regularly, that you had a good understanding of the situation and practical operations. Three indicated that the state veterinarian rarely visited their yards, three indicated that it was seldom, if ever, that the state veterinarian called at their yards, and one did not answer. Sometimes compliments come to those who do a good job but rarely are compliments given in business. This one state official has done a good job, selling himself at least. As stated by the yard manager:

“He is thoroughly familiar with every part of the yards and with general procedure, with the things that are done to prevent infection and he is also familiar with the auction yards. He admits quite freely that the chance for infection is very much greater at the auction yards, and he believes the handling of the majority of the livestock through the central markets from 1900 to 1930 had a great deal to do with the elimination of contagious livestock diseases.”

My main reason in asking this question of the various operators was to find out if state officials actually knew what was going on at the terminal yards. On occasions when I had the opportunity and need to discuss cleanliness of terminal markets with state livestock sanitary officials, I have usually found them inclined to think of terminal yards as a source of contagion and infection. It usually takes a federal B.A.I. inspector in charge of that market to fully impress the state official that all terminal posted yards are kept free from all contagion and infection at all times.

If the state sanitary official visits the yards often he knows our ways and method of operation, he is fully conversant with the fact that the animals are inspected on their trip through the yards and that if any diseased animals are found, these animals are quarantined immediately, are sold only for immediate slaughter unless they can be treated in some way to eliminate the disease, and all the pens, alleys, and viaducts that these animals have contacted in their trip, are locked up with a government lock and not released until all of the territory is thoroughly cleaned and disinfected. Instead of being infected yards, we are 100 per cent free of contagion and infection unless some specific case is found and it is promptly handled.

In talking with several federal veterinarians at various markets, I have concluded that they are practical because they come in daily contact with the customary work that goes on at a market. They jokingly indicate that most of the rules and regulations of states are fair and reasonable and that they have less trouble with the rules and regulations than they do in trying to educate a new state livestock sanitary official who, like a new broom, is sweeping clean and is more technical than practical. After a year or two of education the state livestock sanitary official usually understands the practical interpretation of the rules and things run smoothly until there is a change.

There are, however, some things which the terminal market operators feel should be removed and here are a few of the suggestions.

From a Mid-Western market comes this suggestion:

“The dipping regulations of feeder lambs to Iowa and Minnesota have given us the most trouble. These states will usually admit feeder lambs handled at railroad
feed yards where no inspection is maintained and where no disinfecting is done, while forcing dipping if handled at our market."

From one a little farther north comes this suggestion on sheep:

"On feeder sheep, Illinois and Iowa give permits on yards-sold animals, but we have trouble on through shipments to Iowa as there is no one here to sign agreements, except in a few cases of large shippers who have designated certain commission men to sign for them."

And he goes on to give the remedy:

"To remedy the situation and have more uniform regulations, we believe the government veterinarians located on the markets could and would police these movements of feeder sheep into the various states when issuing health certificates covering the shipment. If a clean bill of health was furnished on shipments from clean areas, that should be sufficient for their entry into any state. At outside points the shipper should wire for permit, and if from a clean area, the shipment should be allowed to enter, or be turned down if from an infected area, unless dipped."

This seems to give the idea that a few of the states are a little strict on their regulations; in fact this man believes they are too strict, or if not too strict, are partial to some of their competitors.

Suggestions on cattle are primarily those of testing all steers, which seems to be a rather impractical requirement in a tuberculosis-free Nation, and the testing of females under six months of age or other stock, which might be used for breeding, when going strictly for feeding purposes.

A south central market makes this observation:

"All states to which we ship require the Bangs test. Some states however require the tuberculin test, retarding the movement of cattle, males and females, for 48 hours longer than the Bangs test requirement, and since tuberculosis is not prevalent in the Mid south, the tuberculin test is both expensive and unnecessary in cattle coming from these yards. One Southern state to which we occasionally ship requires dipping of all cattle before animals may cross the state borders, an antediluvian requirement probably dating back to the stone ages."

A Southwestern market seems to be more fortunate than most of us, and has had some dairy cattle business in the recent past. He makes this remark:

"The non-enforcement of the Bangs test is our complaint. The board makes little effort to enforce it except at the public markets. Consequently, receipts of dairy cattle at this market, except for immediate slaughter, is negligible."

No one makes any remarks concerning the movement of hogs, and everyone seems to feel that he is treated fairly and practically. In order to bring about some of the things which they would like done, here are some other suggestions.

This reply came from a Pacific Northwest market:

"In order to enable livestock to move freely in interstate commerce within the United States, uniform state livestock sanitary regulations, not only as applied to the central public markets, but also to places where auction or community sales are conducted, should be adopted, and enforced. A serious outbreak of animal disease, originating at one of the points where no inspection or sanitary regulations are enforced, could have far-reaching effects as far as the central public livestock markets within a state are concerned."
A small Corn Belt operator has this to say:

"We would like to see state veterinarians at all public markets and they could police all auction markets in a radius of 50 miles. The auction market is where all our grief originates. The seller takes his sick and off-stock to auction or community sales and scatters disease all over our state. We would like this to stop. We watch our market as best we can but we are not veterinarians."

You will notice in both of these that the men are giving additional work either to the state or to the federal government thinking that the inspector at the terminal market might extend himself over a little more territory. They are not blaming the individual in the state in the enforcement of the rules and regulations at an auction market, but all of them seem to feel that it is lack of funds, primarily, or the fact that the inspector for the state is a local man paid for, in many instances by the auction market operator. As they have said many times, "the man who controls the money, controls the man."

A Northwestern market says this:

"The state has certain laws that are supposed to cover health regulations at the auction or community sales, but as far as we have been able to ascertain these laws have not been enforced. The lack of enforcement appears to be due to an insufficient number of enforcement officers to cover the many sales rings throughout the state, and the lack of interest by the producers and the patrons of these auctions and community sales. We have had several cases called to our attention where livestock has been bought at these sales rings, and later found by the buyer to be diseased. We would like to see the same sanitary and inspection rules and regulations enforced at the auction and community sales, as are enforced at our public central market."

You will notice in this paragraph that the operator has a different angle than expressed before. He is blaming the producer for not taking sufficient interest in his own business and in his neighbor's business to market clean and healthy animals and not try to peddle sick animals in the neighborhood.

Another man in the northern Corn Belt has somewhat the same idea:

"Personally we think that the farmer who avoids the small charges required for the testing of feeder animals when he is running them with a dairy herd is extremely short-sighted, and our relations with the State Department of Livestock Sanitation have been much more amicable since the people on our market have developed a better understanding of this fact."

They know that the rules and regulations are made for the benefit of the livestock producer as a whole, and feel that the individual should be sufficiently interested to have some understanding and acceptance of the rules set forth.

From all of these remarks, I believe you will learn that the Terminal Market operators do not consider the state veterinarian as any "big bad wolf." They understand some of the trials and tribulations of a state sanitary official and only wish to give constructive criticism to the point where practical betterment might be made.

I believe the first principle on which a state livestock sanitary official should base his rules is that a Terminal Public Market is free from contagion and infection and that the inspection given there is valuable to the health of the animals entering the state of destination. The operators feel that if a state livestock sanitary official
'discriminates against a livestock terminal market in favor of country loading points or railroad feed yards, he is misinformed or uninformed concerning the Terminal Market. They feel, in most cases, that the laws of the state are ample to control diseases but think in general, that the inspection at country points is poor, due primarily to lack of funds to fully enforce the laws on the books.

There are a few instances where it is felt the regulations should be changed in regard to feeder sheep moving into some of the Corn Belt states. There should be some uniformity in handling beef type feeder females to the feed lots in states where the dairy industry is important. If testing of cattle is necessary at the market, tests should be run simultaneously, delaying cattle as short a time as possible.

In all of the answers received, not one criticized the state livestock sanitary official personally, or referred to him as incompetent or unsatisfactory. If any mention was made of the man in charge, it was most complimentary. From this, I believe that it is not a case of differences between the market operators and livestock sanitary officials. The problem was expressed by one operator, who blamed himself for not cooperating further with the state officials and obtaining sufficient money to enforce the rules and regulations for the protection of the animals' health. Last but not least, the big request is for uniformity and similarity on rules and regulations governing the importation of animals into the various states. It is particularly stressed that adjoining states should be similar, but it is generally felt that more uniformity could be easily brought about, and that with this uniformity, confusion to a large degree, would be relieved at the terminal markets.
WHAT THE LIVESTOCK AUCTION MARKETS HAVE DONE FOR NEBRASKA

BY COLONEL JAMES WEBB

President, Nebraska Livestock Auction Market Association

History tells us that the auction method of marketing livestock, both in this country and abroad, is the oldest method of bartering between the buyer and the seller.

The first record of livestock auctions goes back to the year of 1836, in the State of Ohio, where livestock was bought and sold at public auction, thereafter spreading throughout Ohio and Kentucky and other eastern states.

The history of the development of livestock auction markets in Nebraska dates back to the year 1912 with the opening of one livestock auction market in Grand Island, Nebraska. In the years following, up to date, they have spread all over the entire state with 130 auction markets operating in the State of Nebraska at the present time. Some thirty-six of these markets are now posted markets, operating under the Packers and Stockyards Act, the balance operating under state regulation. Nebraska has more than twice the number of posted markets in the State than any other State in the Union.

Nebraska auction operators have an auction association with practically all of the larger markets belonging to this association in the state. This association is governed by a Board of Directors, Secretary and Treasurer, Vice-President and President, as well as a Counsel and Rate Expert. The volume of business done by all of the auction markets within the State amounts to many, many millions of dollars annually.

Two sale barns located in the City of Grand Island handle an annual turnover in livestock of about $15,000,000 per year.

ARE SALE BARNs AN ASSET TO THEIR TOWNS AND COMMUNITY?

Sale barns are essential to the progress and development of their respective communities. Business men in the towns and cities where sale barns are located, as well as civic organizations, Chambers of Commerce, farmers and feeders, and ranchmen realize the importance of a good livestock auction market located in their community.

A livestock auction market brings people from far and near to attend these livestock auction sales, where people can congregate and either buy or sell, as they wish. Many of these livestock men bring their womenfolk along, the ladies spending their time in town purchasing needed supplies from the city merchants. Likewise, under the present war conditions, the sale barns have proven that they are essential to the war program because they are rendering a specialized service to agriculture, furnishing concentration and marketing service to farmers and feeders.
necessary to the war effort in the production of meat. Livestock markets have been declared essential to the war effort by the War Labor Board. The livestock auction markets have done much to develop and encourage the small farmers and feeders to purchase a few head of hogs or a few head of cattle on the auction markets and take them home, feed them out and sell them back on the markets again. This in itself, all over the State, has done much to make Nebraska one of the foremost livestock producing states in the United States, and this will continue to increase in the future as it has in the past.

It would be almost impossible for any one person to try to measure the value of the livestock auction markets by what they have done to not only improve the breeding and feeding of livestock, but every city and hamlet receives its proper share of the good felt by each community wherein a good livestock auction market is located.

REGULATIONS, PAST, PRESENT AND FUTURE

We all realize in the early development of livestock auction markets, regulations, both sanitary and otherwise, were unheard of, but as the auction markets have developed, so have other essential improvements such as handling, yard facilities, weighing and state brand regulations, health and sanitary regulation, that have meant much to encourage the patronizing of these markets.

Nebraska operators of livestock auction markets feel that we have a health regulation that protects both buyers and sellers alike, and that our State Director, Dr. J. R. Snyder, is meeting with whole hearted cooperation from every livestock operator who is a member of our association.

Here and now I would like to propose that all livestock auction markets be either under state or under federal supervision. The present operation of our markets, part being under federal and part under state regulation creates the rankest kind of discrimination. For numerous reasons I favor all of the markets being under state regulation. Those of us who are acquainted with the workings of the original Packers and Stockyards Act of 1921 understand that this was never created to cover auction markets but was intended for terminal markets. This would be a very important move and would do a lot to further develop livestock auction markets. It is only reasonable to realize that the difficulties that might arise at any time could be much easier handled by state direction rather than federal. I hope this proposal will be given due consideration and acted upon accordingly.

FUTURE OF THE AUCTION MARKETS

It is reasonable to assume that the future of the auction markets depends much upon the auction market operators themselves in complying with all proper rules and regulations together with the proper equipment to handle livestock and the necessary sanitary conditions to avoid the spreading of any contagious disease of any kind, in hogs, sheep, cattle or horses.

It has been called to my attention that some auction markets are operating without the proper facilities to offer this protection to their buyers and sellers. I believe this condition should be corrected where at all possible. I also oppose any
one securing a license from our state officers to operate an auction market, in the future, unless he fully complies with all requirements such as bond, and this bond should be at least $5,000, and has the proper equipment such as scales, suitable sale ring and pens for the handling and the proper care of all livestock.

There is no question but what we are faced with more critical handicaps now than ever before with OPA price regulations and rumors of price ceilings discouraging many livestock feeders and producers. It is more important now than ever before that we handle our business more efficiently.
REPORT OF SPECIAL COMMITTEE ON OPERATION
OF LIVESTOCK AUCTION MARKETS

J. M. SUTTON, C. E. FIDLER, AND J. R. SNYDER,
Committee on Community Auction Sales

During the last twenty years a large number of local livestock auction markets
have been established in practically all States in the Union; especially throughout
the Southern States.

Judging entirely by local enthusiasm and support given these markets they
render the livestock industry a real, valuable, service. These markets, because of
their convenience, are of specific interest to the small producer. Of the livestock
offered at these weekly auction sales 90 per cent to 95 per cent are produced within
a radius of less than fifty miles of the market at which they are sold.

In sections where less than twenty years ago livestock production was limited to
a few hogs for home consumption, where the production of beef cattle was
almost unheard of, because of inadequate marketing facilities, today many of the
better livestock auction markets offer more than a thousand head at their weekly
sales.

Unquestionably these markets have been one of the most vital factors in stimu-
lating interest in livestock production in Georgia. More than 95 per cent of Georgia
grown livestock is marketed through local auction sales. Practically 100 per cent
of Georgia farm grown livestock is now driven in trucks directly from the farm to
the auction mart whereas a few years ago livestock peddlers roamed the country,
bought a hog here and a cow there holding them until a full carload had been
accumulated then shipping them to some established slaughter house center and
accepted whatever was offered them. In view of the risk and cost unavoidably
connected with this system the livestock dealer did not pay the farmer much.

Under the old system the farmer sold at a great depreciation in price to the local
dealer or he shipped from 100 to 700 miles to some established market center: he
paid the freight, feed bills if any, shrinkage in weight and loss in transportation if
any, then the commission firm deducted their commission and what little was left
gone to the producer. If by chance the market price did not suit the producer
he was practically helpless. In the meantime his neighbor in the market for beef
or pork products had to pay all the above expenses plus freight, refrigeration and
sales commissions from the slaughter house center back to his home.

Now most well established auction markets sell much of their offering to local
butchers who, in turn, supply the local markets. Consumers pay less, farmers get
more because an enormous overhead expense and middle-man profit have been
eliminated.

But like Colin's ram this husky, sprawling youngster has grown up without
restraint and has acquired, in spots, some undesirable habits.

The major weakness of this marketing system is the general disregard of live-
stock sanitary precautions.
Most leading auction market owners or operators recognize this deficiency and would welcome suitable State regulations promulgated and enforced by State Livestock Sanitary Boards.

In drafting and enforcing such laws and, or, regulations all red tape should be studiously avoided; the major service required should be to avoid, as far as humanly practical, the spread of infectious or contagious diseases. With this in mind only qualified Veterinarians should be employed as inspectors for such service.

Each truck load should be inspected before and after unloading.

Before loading animals after the sale for movement to the farm the trucks should be cleaned or cleaned and disinfected as conditions may require.

Owners knowingly sending animals out of sick herds to the sale should be prosecuted.

To eliminate, as far as practical, unscrupulous persons from operating such markets, thus avoiding unnecessary litigations and law suits, each auction market should operate under license issued by the State Livestock Sanitary Board, and should be required to furnish the State a substantial bond to guarantee faithful performance in compliance with the law. If the owner or operator fails to cooperate with the State or willfully disregards the regulations governing the operations of livestock auction marts the State can revoke the license until the recalcitrant operator gets in line with the State requirements.

If any State does not have the funds necessary to render this service the law should authorize the Livestock Sanitary Board to charge a nominal fee per head for inspection service, say two to five cents per head, this money to be paid to the Livestock Sanitary Board for the employment of qualified Veterinarians.

If a worthwhile service is rendered no farmer or stockman would complain about this fee, and if they did, the operator of the sales establishment could well afford to pay it out of his commission. His customers would appreciate the service and a satisfied customer is recognized as a valuable business asset.

Your committee sincerely appreciates the generous response received from the various State Officials in charge of Livestock Sanitation giving your committee full information in regard to the subject under consideration. Only a few States have specific laws enacted for the express purpose of regulating the operation of livestock auction markets or sales rings. Some States control the situation under regulation issued by their Livestock Sanitary Boards.

Believing it would be impractical to write, in detail, a law that would meet various local environments your committee offers in brief the basic principals which such a law should cover. These basic principals are concisely stated in the Caption of a law enacted in 1937 by the General Assembly of the State of Wyoming which, with minor amendments is hereby offered and recommended by this committee as worthy of your consideration and adoption.

LIVESTOCK AUCTION MARKETS OR SALES RINGS

An act defining and providing for the establishment and regulation of Livestock Auction Markets or Sales Rings; requiring any person operating such Sales Rings to obtain a license and fixing the fee therefore: requiring a bond of any person
operating any such sales Rings and fixing the amount and terms thereof; empowering the _________ Livestock Sanitary Board to issue rules and regulations to be observed in operating Sales Rings; providing for the investigation of Livestock handled or sold in such Sales Rings and the issuance of a certificate of such inspection; to require operators of such Sales Rings to establish and maintain adequate quarantine pens for possible emergencies; prescribing penalties for the violation of the Act, and the rules and regulations made pursuant thereto: providing for the disposition of monies received by the said Board through the operation of this Act, and repealing all Acts or parts of Acts in conflict therewith.
COOPERATIVE BOVINE BRUCELLOSIS WORK IN THE UNITED STATES

BY A. E. WIGHT, D.V.M.

In Charge Tuberculosis Eradication Division, Bureau of Animal Industry, Agricultural Research Administration, United States Department of Agriculture

Those of us connected with the brucellosis work during the last year have, of course, observed a considerable decrease in the volume of work done in connection with this activity. This decrease is chiefly due to the shortage of veterinarians. Many of those who were formerly identified with this work are now in the armed forces. Travel conditions and other factors caused by the war also have seriously interfered with the progress of the work, especially that under the area plan. We must expect to encounter such obstacles as long as the war lasts, but every effort should be made to hold the ground already gained. We should also proceed with the immunization of young cattle against brucellosis where such a procedure is indicated.

STATE AND FEDERAL FUNDS

The legislatures of all but four States have convened since our last meeting, and they have provided approximately $4,450,000 for operating expenses and indemnity during a 12-month period. In most of these States, a similar amount has been made available for an additional period of 12 months. The United States Congress provided approximately $4,500,000 for this work, including operating expenses and indemnity, for the fiscal year ending June 30, 1944. Under present conditions, this will be more than enough to carry on the work as outlined at present. Provisions for indemnity have been made in all of the States except California, Colorado, Indiana, Massachusetts, Nevada, Oklahoma, Texas, and Utah. In a few of the States some of the operating funds are provided by the counties.

METHODS FOLLOWED IN CONDUCTING THE WORK

The brucellosis work among cattle continues to be conducted under the individual herd plan, as well as on an area basis. The Federal Government does not have any rules or regulations requiring the testing of herds, but when it has been decided to take up cooperative work under the area plan in a given district, such as a county, it is compulsory under the State laws that all dairy and breeding cattle over 6 months of age be submitted to the test and the reactors be either removed for slaughter or placed under quarantine. About half the total number of cattle tested are in sections where the work is conducted under the area plan.

During the fiscal year ended June 30, 1943, 392,636 herds, containing 5,185,228 cattle, were tested, disclosing 197,329 reactors, or 3.8 per cent. However, 53,558 of these reactors were held under quarantine in infected herds where calfhood vaccination against brucellosis was being conducted. The owners do not receive any indemnity for reactors held under this plan, but dispose of them for slaughter when they become unprofitable.
On November 1 there were 40,986 herds, containing 841,485 cattle, classified as fully accredited. This is a considerable decrease in the number of such herds reported at this meeting last year, partly due to the fact that a review of the records disclosed that at a number of the stations the herds in which the accredited plan had been discontinued had not been removed from the total number reported. In some sections, area work had superseded work conducted under the accredited herd plan.

Reports received from several of the field stations show that the owners of large numbers of cattle in a number of States have indicated that they wish to have their herds placed under supervision for the control and eradication of brucellosis. Many of these herds are in localities where the work could be conducted under the area plan if there were sufficient help to conduct it.

INCIDENCE OF THE DISEASE

The approximate extent of brucellosis among dairy and breeding cattle 6 months of age or over in this country, continues to be about 5 per cent. This is slightly higher than the results shown on the official testing because the 5 per cent estimate includes areas in which a considerable degree of infection exists and in which but very little, if any, brucellosis work has been conducted. The results of the testing of cattle for this disease also include a number of retests, which makes the percentage lower than it would be if testing were confined to original tests only. During the year 1934, when the cooperative work was first taken up by the State and Federal Government, it was estimated that brucellosis among the dairy and breeding cattle of this country 6 months of age and over was about 10 per cent.

A map of the United States, which accompanies this paper, shows that there are 2,427 counties, out of the total number of 3,070, in which the incidence of brucellosis in cattle is estimated to be not more than 6 per cent. Of this number, 583 are in what is known as the modified accredited Bang's disease-free area. The counties in this status are shown in white on the map, and the approximate incidence of the disease in the other counties is shown by the type of shading.

INDEMNITY FOR REACTORS

In States where the owners may receive payment for reactors, the cattle are appraised and disposed of for slaughter under State and Federal regulations.

During the last fiscal year, the average appraisal for reactors was $128.03; average salvage, $64.87; average State indemnity, $18.63; average Federal indemnity, $16.77. Thus the average loss to the farmer was $27.76 per head. Ten per cent of the reactors slaughtered during the year were registered purebred cattle. On account of the great increase in beef values, the average salvage was the highest ever reported since the work was taken up.

 BREEDING STATUS OF POSITIVE COWS

Many of you will probably be interested in learning of the results of some observations made with reference to the breeding status of positive reactors to the test for brucellosis. The information given here has been collected by the inspectors in charge of the brucellosis work for this Bureau in 8 States located in various sections of the country. The veterinarians who handled the animals in the field obtained
the information direct from the owner and did not record any that was not believed to be authentic. There were two surveys made—one during December 1941 and the other during June 1943. The results were as follows: Of 6,432 reactors reported, 4,294, or 66.8 per cent, had calved normally during the previous 12 months; 1,445, or 22.5 per cent, had aborted during that period; and the remaining 693, or 10.7 per cent, were classified as barren or as poor producers.

**THE USE OF BRUCELLA VACCINE**

We are to receive important information on the Brucella vaccine this afternoon from Drs. Haring and Traum of the University of California, who have had wide experience in the use of this material. There will also be a discussion on this subject by Dr. R. R. Birch of Cornell University, and Dr. Melvin Rabstein of the University of Maryland.

The volume of calfhood vaccination conducted under official supervision has increased to a considerable extent since the last meeting of this Association. During October 1942, 17,028 calves were vaccinated under official supervision, whereas in October of this year, 31,277 were thus treated, or an increase of 83 per cent. Since January 1, 1941, about 473,000 calves have been vaccinated under the official cooperative plan. Under one of the official plans for calfhood vaccination, the adult reactors are removed for slaughter and the owners receive indemnity for them. This is known as Plan B in many of the States. The plan previously mentioned wherein the owners may retain the adult reactors under quarantine and dispose of them for slaughter when nonproductive, is usually called Plan C. The records of the Bureau indicate that at present about twice as many calves are vaccinated under Plan C as under Plan B. The total number of herds in which the calfhood vaccination is conducted under official supervision under both of these plans is about 50,500 now, as compared to about 23,200 a year ago.

The proper handling of Brucella abortus vaccine by veterinarians in the field in order that it may retain its proper potency, continues to be of much importance. All the Brucella vaccine prepared by commercial concerns is tested at the Animal Disease Station at Beltsville, Maryland, and is not released for use unless it passes the required test. This product is marketed in 1-dose bottles exclusively.

In March of this year, a final report of the results of calfhood vaccination against brucellosis under field conditions was released by Dr. John R. Mohler, then Chief of the Bureau of Animal Industry. As it has been widely distributed, it will not be included in this paper. Copies are available for anyone who has need of them. This report lists some points that should be observed in order to obtain best results, one being that premises contaminated as a result of an abortion should be properly cleaned and disinfected, as probably 95 per cent of the exposures in infected herds result from the placental tissue and discharges from aborting cattle or normally calving cows having uterine infection. Such intense exposure tends to break down the immunity in pregnant heifers or cows vaccinated as calves and makes the maintenance of maternity stalls, barns, and hospital pastures desirable.

**PUBLICITY HELPFUL TO THE PROGRAM**

Through the press and radio, much reliable information is reaching the public that is helpful in conducting the campaign. The State officials in a number of
States have prepared appropriate literature for distribution, giving the farmers an opportunity to read accurate information that will assist them in handling their herds in a manner whereby they may be kept free from this disease.

CONCLUSION

At the conclusion of my talk before this Association last year I suggested that provision be made for a forum on brucellosis this year, but I notice that the Committee in charge of the program decided to have a panel discussion on cattle diseases. No doubt some interesting points on the different phases of brucellosis will be brought out in this discussion.
THOUGHTS ON THE CONTROL OF BRUCELLOSIS IN CATTLE

By R. R. Birch, D.V.M.

Cornell University, Ithaca, New York

The basic knowledge of Brucellosis in cattle, though still incomplete, is a sufficient foundation on which to build effective measures for its control. By control I mean near-eradication with relatively inconsequential losses, such as we have with tuberculosis, and not a mere reduction and level annual distribution of heavy losses such as we have with hog cholera. With Brucellosis anything less than near-eradication means needless compromise; anything more is probably not attainable. With real control in view, then, we can measure our progress by the number of clean herds established, and not by the number of tests made, the number of calves vaccinated, nor by any other standard. The only herd that does not suffer in some degree from Brucellosis is the one in which it does not exist.

I have a purpose in naming here the chief measures that have been extensively in attempts to control Brucellosis in cattle. These are: drugs; proprietary remedies with unknown formulas; local treatment of the genital tract; bacterins; vaccines prepared from fully virulent Brucella strains; vaccines prepared from non-virulent strains; test and slaughter; test and gradual elimination with herd management to minimize spread within the herd, and—a late and important arrival—calf vaccination with Brucella Strain 19.

Of these measures, drugs have been abandoned because they were ineffective; no single proprietary remedy ever proved itself; local treatment of the genitals, effective only in salvaging individual cows as breeders, is known to be inconsequential as a measure for control; bacterins are in the discard because they failed to protect; vaccines representing fully virulent strains have been abandoned because they involved some danger and were not dependable, and vaccines prepared from non-virulent strains likewise did not immunize effectively.

These rejected measures, some with no scientific backing whatever, some supported by widely quoted but limited work of our most careful investigators, have all been proclaimed as the partial or sole solution of our Brucellosis problem, and all are discredited today because they broke down under wide application. I bring them back today—only momentarily I hope—as a needed reminder that with an erratic disease like Brucellosis, careful and extensive researches, supported by wide field use, with careful observation and interpretation of results, are the only dependable guide. The man who has done this, or done that, with the usual wonderful success, sometimes adds a useful bit to the larger picture, but his enthusiasms often delay or prevent the orderly expansion of sound methods.

After eliminating the acknowledged failures, we have remaining the following useful methods: Test and gradual elimination with herd management to minimize the spread; test and slaughter; vaccination with Strain 19; and, I want to emphasize a fourth, a combination of these measures. I place the emphasis on the combination because our tendency has been to rush from one method to another, attempting to
apply each method to all herds in all circumstances, then to commend or condemn, retain or abandon, according to our individual experiences. These experiences have depended in turn, not chiefly on the potentialities of the method itself, for each is useful in its place, but on the circumstances under which it has been used. We have not yet attained the flexibility which enables us to suit the method to the conditions in the individual herd, though in the setting up of the plan involving test and hold with vaccination of the calves as indicated, a step has been made, more important, perhaps, than is yet fully realized.

We may profitably examine the useful measures named, not for the purpose of pointing out failures well known to us all, but in the hope of bringing out the reasons for these failures, defining the limitations of each method, and salvaging its sound elements for future use.

Test and hold, with gradual elimination including the handling of reactors so as to minimize the transmission of Brucellosis in the herd had proved its worth long before we had any official programs. The owners of purebred herds were the first to adopt this plan, and it is fortunate indeed that we have had their assurance and support in formulating later official plans. While the usefulness of this plan is limited, in the main, to carefully managed, raised herds, its effect has been to encourage the establishment of just such herds, and to discourage the practice of picking up a cow here and one there, a ruinous practice in which the danger is by no means confined to Brucellosis. This plan required care, it involved inconvenience, and it carried with it its disappointments, but it was, and is, generally successful in raised herds, and it sustained a truth we will do well to relearn—we can raise a herd no higher than we can raise its owner. The plan was the entering wedge looking toward real control, and it still is yielding rich returns in careful hands.

Test and slaughter, applied to Brucellosis, came through the gate when it was opened to a cattle reduction plan, under the momentum supplied by its success with tuberculosis and the weight of public opinion which fairly shouted that if cattle were to be killed at all they obviously should be the reactors. The points missed were that we may kill reacting cattle without establishing herds free from Brucellosis and that the inter-herd transfers set in motion by the application of this policy in badly infected, large herds, were ruinous in their effect.

I do not believe there were many men schooled in the handling of Brucellosis who ever believed that test and slaughter as a blanket measure could be made to control the disease but an irresistible logic was against this minute and helpless minority, and in some quarters at least, the plan was taken up with reluctance and misgivings.

Time and experience have shown that in small, lightly infected, raised herds, test and slaughter has its place, but that in heavily infected, large herds, replaced by purchasing, it is both prohibitive in cost and futile—even destructive in its effects. And the future will prove if we will only limit its use and give it the opportunity, that test and slaughter will be useful in removing the few straggling reactors that remain in herds that are nearing the culmination of a plan based on test and hold with gradual elimination, with or without calf vaccination. It has no place where large numbers of good cattle are reactors, or where replacements must be purchased.

Because it has been injudiciously used, and not because it is inherently futile or even too expensive when wisely limited, test and slaughter has left at least one
misconception in its wake from which we shall be slow to recover. Many disappointed breeders regard all blood testing as part of a plan involving test and slaughter, and brand all testing as a failure.

Calf vaccination, in its place, can be made a most effective weapon against Brucellosis in that it prevents many abortions and, by lengthening the gestation period in cows that otherwise would abort, it increases the flow of milk. It is the only weapon we have so far that promises to yield much in herds sustained in effective production by buying and selling. Again, unfortunately, we are in the process of seizing an effective but limited weapon and casting aside all others. We are rapidly approaching a point, not by official decree, but by actual practice, when our efforts to control Brucellosis are bound up in the standard advice "you had better vaccinate" or by way of variation "Why didn't you vaccinate?" We have made it that simple. And the proposal is in the lobby but has not yet, so far as I know, reached the floor of this association, to give all officially vaccinated animals, without test, clean bills of health for inter-state transfer. Once again we are expecting a single measure to do everything. On, what do we base the assurance that it will? What do we know of it, and what remains to be found out?

I shall endeavor to answer these questions drawing freely on all known-reliable sources, including our own researches, which for the most part either confirm or establish the known facts which I shall enumerate. These researches, conducted by Dr. H. L. Gilman, Dr. W. S. Stone and the writer, are based on calf vaccination with adequate controls in a herd of about 60 animals kept for about seven years. Vaccinates and controls have been kept under constant, or at least, frequent exposure. In addition, we have drawn on extensive data based on testing in the field.

Let us first enumerate the established facts as they apply to the pathogenicity of Strain 19 and to vaccination with vaccine prepared from it:

1. Vaccinated calves kept subsequently under natural exposure acquire valuable immunity that prevents many abortions.
2. There is good evidence that, if the exposure is sustained, protection effective in preventing extensive abortions tends to last through several gestation periods, but there are enough exceptions to this rule to cause considerable loss.
3. Vaccination causes most calves to react from three to six months, and a few to react indefinitely.
4. A vaccination reaction cannot be distinguished from an exposure reaction.
5. Vaccinated calves maintained subsequently under natural exposure frequently contract Brucellosis in a degree that causes them later to spread the organisms in the milk and in the uterine discharges at calving time, and to react indefinitely.
6. Strain 19 has produced typical Brucellosis in cows, and it has been isolated from the testicle of a bull suffering with severe orchitis. This bull was vaccinated as a calf.
7. Strain 19, as originally reported, is much lower in virulence than are the ordinary field strains.

We still lack answers to these questions:
1. How dependable will be the protection in calves vaccinated at the usual age, but not actually exposed to virulent field strains until several years later?
2. What are the dangers and benefits of adult vaccination, single or successive?
3. How long following the vaccination of mature cows does it require to build up dependable immunity?
4. In clean herds, will Strain 19 used in vaccinating the calves endanger the adults appreciably?
5. Will Strain 19, known now to possess low virulence and high immunizing qualities, continue to maintain these qualities?

These knowns and unknowns clearly indicate that we are right in using vaccination systematically on calves in badly infected herds, wrong in using it as an only measure or in trusting it too far. It is clear that it prevents many abortions, but that it is not an effective preventive of a milder type of Brucellosis accompanied by actual and frequent spread of the causative organism. These results have been brought out in controlled experiments in which demonstrated exposure accompanied by regular transmission has been present. It will take much longer for them to appear in field herds in which exposure depends on chance, often is long delayed, and the actual policy, frequently unstated, is gradually to eliminate reactors.

While we still are considering the uses and limitations of vaccination, I want to ask each member of my audience to answer for himself this question: Assuming that Strain 19 were to lose suddenly all of its immunizing power, how long would it be before the deficiency would be discovered in the field, and corrected? This is no mere academic question, for while we have every confidence that our present facilities assure within reasonable limits distribution of smooth cultures of Strain 19, we must reckon with the possibility that even these smooth cultures may not retain their present valuable immunizing qualities. A species tends strongly to hold to its governing characteristics—single strains within a species are more subject to change. We do not test the immunizing qualities of each batch of vaccine. Thus, the deficiencies, should they occur, must be discovered and corrected in the field several years later, or not at all. And in the meantime thousands, perhaps millions, of cattle could have been vaccinated with the defective vaccine.

Returning to the original question, my own estimate is that it would require at least six years for a complete failure in the immunizing power of Strain 19 should it occur, to be detected and corrected in the field. It would be only two years, of course, before the vaccinated heifers came into production, but actual transmitting exposure would be so slow to overtake the majority of the herds that the number of those who had “vaccinated and had fine results” would be sufficient to delay for a long time recognition of a complete failure of the vaccine at its source. A building up of the virulence of the Strain also is a possibility, and would be especially objectionable where adult vaccination is practiced, but this is less likely to occur.

Please understand that this is in no sense an argument against using a valuable vaccine, according to established indications, as long as it will maintain its present qualities. It is intended as a caution against using it under all circumstances as a substitute for all other measures. The momentum behind the tendency to substitute vaccination for all other measures is tremendous. Starting from a solid nucleus consisting of a vaccine with proved value and proved limitations, it has fanned out in all directions into the unknown and uncontrolled. Again, all-or-nothing is on the way, supported as always by oft-repeated half-truths and catch...
phrases, rich in superficial and questionable logic but ill-sustained by fact. It is worth our while to examine a few of these half-truths and see where they are leading us.

Here is one familiar to us all. A man with a clean susceptible herd is "sitting on a keg of dynamite." The disasters that overtake some of our clean, susceptible herds are well known. Some of these disasters are unexplainable and unavoidable, some grow out of the phrase itself which insidiously breaks down morale by leading breeders to believe they are powerless to protect their herds, and that a break in a clean herd is proof that blood testing is a failure. Somewhere in my files I have a letter reading in effect—"My herd has been clean for 16 years and now, out of a clear sky, I find two blood reactors in it, so I’m through with blood-testing." Even if we grant that a real disaster was in the making, is not this man better off for having succeeded in protecting his herd for 16 years? Mostly the breaks occur in the larger, less well-isolated herds into which new animals are being introduced. But let us balance the entire picture by stating that thousands of breeders have been "sitting on kegs of dynamite" for years and have profited immensely by the experience. The reward for maintaining clean herds usually is in proportion to the effort put forth, and those who make the attempt are better off than those who do not.

Again: "There is no harm in vaccinating and it may do some good." This is another familiar one. It was applied originally to calves but it has crept up to include older heifers, then open cows, then bulls and pregnant cows, reactors or not. Well, it stands to produce a few abortions in mature cows, how many we do not know, and it certainly will create in them persistent blood reactions that will render impossible for a term of years any progress based on blood testing and elimination. The chief harm, however, springs from creating in the minds of cattle breeders the impression that nothing more need be done, and in delaying the time when they will begin to apply all proved available weapons. The physician who engages to cure our diabetes, or to reduce our weight, at the same time permitting us to eat as we wish may cause us to exclaim "Here at last is a practical man!" He may not harm us, and may even help us with his remedy, but he delays the time when the truth may be known and the case taken in hand. The switch again instead of the main track. When a choice is offered, there is always the understandable tendency to choose the convenient and promising against the inconvenient and substantial.

One more: "My client wants the calves and milk—he doesn’t care whether his cattle react or not." Here is a point of view seemingly so logical that it sells itself. Some are surprised that anyone should question its soundness or fail to accept it. I’m sure most of us understand it, and sympathize with it too, in selected individual herds, but we hope we see farther. An expedient that legitimately may alleviate some individual breeder’s problem, if widely used may also be the basis of a ruinous public policy. The problem is greater than the sum of its parts.

Let me illustrate. Many in this room have had the experience of attending autopsies of tuberculin reactors and have heard men murmur: "What harm did that little nodule do?" That cow gave us calves and milk and we keep cows for no other purpose." There again were the logical and the seemingly obvious, and the majority too, pitted against the beginnings of a sound far-reaching public policy,
and we are fortunate that we followed the "theorists" and "idealists" of yesterday instead of the self-styled "practical" men who couldn't see so far down the road.

I am citing some of these half-truths to reveal something of the harm they do individually, but chiefly to illustrate a broader principle. Let me state it here. In the control of Brucellosis, the chief difficulties now are not those that rest on deficiencies of our knowledge of the disease itself. They are the obstacles that we ourselves interpose.

We expect, and may legitimately expect, vaccination to aid in the control of Brucellosis, but by our action or inaction we confess ourselves powerless to control the vaccine itself when we have it in bottles, and the origin is known. It goes everywhere, is sold everywhere, and is used everywhere, living or not, refrigerated or not, by those who know nothing of its preparation, use, or keeping qualities. It is in many hands merely a bottle of medicine to be injected under the skin of each and every bovine. There are no records, only the usual crop of those who have "vaccinated and had fine results."

I suggest for next year that the committee on legislation be requested to make a thorough study and to prepare a statement that will let us know exactly what obstacles exist, and where they are. Is any state really limiting the sale of vaccine to channels that will insure its effective use? If so, how is this being done? Is any state making the attempt and failing? If so, what obstacles are being encountered? Where do the powers of the federal government actually begin and end? Are the powers now granted being used to the utmost? How may these powers be expanded?

Again, Boards of Health have injected themselves, not always understandingly, into the picture. They will remain there and their influence will be felt, especially in relation to adult vaccination. They have a legitimate interest in this phase of the problem, but in other directions unnecessary and sometimes galling restrictions have been interposed. Thus they create man-made obstacles, troublesome, but not insurmountable.

Vaccination itself, sufficiently governed and tied in with other methods of control, can be made to help us toward our goal, which is more and more clean herds, less and less infected ones; but it is time we reestablish the goal and cease to tell our breeders it's impossible to protect their herds. It is time we encourage them to try, and aid them in their efforts. There will be individual failures with the goal before us, but only confusion and failure where it disappears.

I want to raise one other question after telling you in advance that I can't answer it. Can our plans to obtain clean herds with or without calf vaccination survive the impact of contemporary plans involving vaccination alone? That is, can the two plans live and develop effectively, side by side, each in its own field? I ask this because there are strong and legitimate arguments for a plan involving official calf-vaccination in otherwise unsurpervised herds with tagging of the calves so as to provide, in the future, a reservoir of semi-immune breeding stock for replacements.

Among the herds that would fall naturally into this class would be many in the range states, and, temporarily, those whose owners had slipped in their efforts to maintain clean herds, but also the careless, the indifferent, the plunger, and the owners of poor cattle. It would not be difficult to qualify in this class—nor to remain in it. Would free choice between vaccination alone as an inviting and popular
expedient, and a plan including vaccination as indicated but looking toward clean herds, result in a wide breaking down of the latter objective? I hope I'm wrong, but I incline to the belief that this tendency would be serious, leaving us in the end with half a loaf instead of a loaf.

Before closing, let me describe what I regard as a sound approach to the problem of Brucellosis in the individual herd. First a test of the entire herd, except calves; second, the setting up with informed and local professional advice, of a plan suited to the herd and to its environment, this plan in include protection so far as possible of the mature clean animals, vaccination of the calves as indicated, and the final testing out in the course of time of straggling reactors, so that the disease will not again creep back into the semi-immune herd. It seems to me that official measures, in their essentials, should be made to do no less, and can do little more, than to reinforce this orderly process by giving aid to those who are starting out, recognition to those who succeed, and mounting safety as the years go be to those who purchase cattle from approved herds.

I was about to say that given reasonable release from artificial obstacles the control of Brucellosis in cattle is easy. I am saying that of the men who founded this association, the few who looked south from the Mason and Dixon Line across the areas that eventually were to be freed from the Texas fever tick were facing a bleak prospect involving difficulties far greater than those confronting us in the control of Brucellosis today. If we cannot control Brucellosis with the combined and judicious use of the proved weapons and trained personnel now at our disposal, it is only because we have become a lesser breed of men.
THE EFFECT OF BRUCELLA ABORTUS STRAIN 19 ON CATTLE OF VARIOUS AGES AND ITS BEARING ON ADULT CATTLE VACCINATION

BY C. M. HARING, D.V.M., AND JACOB TRAUM, D.V.M.

University of California, Berkeley

This paper includes observations on the incidence of Brucellosis as influenced by the use of Strain 19 on cattle of various ages under controlled experimental conditions as well as in commercial dairy herds. Limited printing space does not permit reference to numerous publications on this subject by others.

Data regarding the relative immunity produced by Strain 19 when injected into cattle of various ages appear to be absent from the literature, and this seems to be an appropriate place to present, for the first time, a comparison of the immunizing effect of Strain 19 on cattle in four age groups, i.e., young calves, four to five months, inclusive; older calves, six to eight months, inclusive; adolescent heifers, nine to sixteen months; and sexually mature heifers and cows, sixteen months to seven years.

In infection trials conducted by the writers, 41 vaccinated and 46 control animals were exposed by instilling a heavy dose of virulent *Brucella abortus* organisms into the conjunctival sacs. The percentage of infection resulting was 53.6 in the vaccinated animals and 91.3 in the controls. These data have been arranged in the following list to show the percentage incidence of infection in the four-age groups which may be considered significant in their relation to vaccination practice.

<table>
<thead>
<tr>
<th>AGE AT VACCINATION</th>
<th>NUMBER VACCINATED</th>
<th>PERCENTAGE THAT DEVELOPED BRUCELLOSIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. 4 to 5 months, inclusive</td>
<td>6</td>
<td>66.6</td>
</tr>
<tr>
<td>II. 6 to 8 months, inclusive</td>
<td>9</td>
<td>66.6</td>
</tr>
<tr>
<td>III. 9 to 16 months</td>
<td>12</td>
<td>58.3</td>
</tr>
<tr>
<td>IV. Over 16 months</td>
<td>14</td>
<td>35.7</td>
</tr>
<tr>
<td>46 head not vaccinated</td>
<td>0</td>
<td>91.3</td>
</tr>
</tbody>
</table>

The resistance produced by the vaccination was apparently greater in the animals which were adolescent or mature at the time of vaccination than in calves vaccinated when younger than nine months. It is a question if vaccination at less than four months of age conveys any appreciable resistance to infection. Our observations indicate that association of calves up to the age of four one-half months with infected cows and the ingestion of milk known to contain virulent *Brucella abortus* do not prevent the development of typical Brucellosis accompanied by abortion when the animals eventually become pregnant and are in contact with infected cows.

The relation of age at the time of vaccination to the persistence of agglutinins induced by Strain 19 is a matter of practical interest. Chart I shows graphically what happened in 752 animals, ranging from four months to aged cows at the time of vaccination. By "negative test" in the chart, we mean less than complete agglu-
EFFECT OF BRUCELLA ABORTUS ON CATTLE

Titration at 1:25 dilution. These results occurred in infected herds. If the animals had been in herds free from Brucella infection, perhaps the percentage of those whose titers became negative would have been greater.

It is noteworthy that the agglutinins in heifers vaccinated when over 16 months of age showed a greater tendency to persist than in the older cows.

CHART I. Relation of age at vaccination to titer recession to negative (752 animals in 7 infected herds).

In states where adult cattle vaccination is extensively used, will the dairy and livestock industries have to live continually with the disease and be eternally burdened with the expense of vaccine and the veterinary service that goes with it? To answer "yes" or "no" with respect to entire states would be merely to express an opinion. With regard to certain individual herds, however, the answer is "no." For example, in previous papers, the writers have described the disappearance of infection from the herd at San Quentin Prison, in which adult heifer vaccination as well as calfhood vaccination was used. The following point regarding this herd is announced for the first time. The disease incidence had dropped from 44 per cent to zero and all vaccination was stopped in the fifth year in order to test further the absence of Brucellosis. It is now the tenth year of our work with this herd. A group of 35 nonvaccinated young cows are in production and none has shown a positive reaction. All mature cattle in this herd are now negative to repeated blood tests.

Our interpretation of results in this herd has been criticized because the cattle are owned by the state, and it is said that such good results would be impossible under commercial conditions. In answer to this we can submit data from our work in Del Norte County, where dairying is conducted under very primitive conditions. The following description is a fair example of the results in that county:

Herd "U", with 34 adult cows, suffered severely from abortions during the years 1935 to 1939, warning of which was given by an increase of reactors to the blood test
from zero per cent in 1933 to 27 per cent in 1934 reaching a peak of 57.3 per cent in 1935. During this time, the owner reported 18 abortions in a herd of 34 adult cows, or 52.9 per cent. In 1939, calves and all adult animals, except those pregnant at the time, were vaccinated with Strain 19. Subsequent to that time, only calfhood vaccination has been practiced. The breeding and calving records kept by the owner since vaccination of the herd show a 100 per cent normal, vigorous calf crop each year except in the 1939-1940 season, when there was one premature birth of a weak calf, which the owner destroyed.

The reactors in this herd were 55.6 per cent in 1940, 35 per cent in 1941, 22 per cent in 1942, and none in 1943. This astonishing record occurred with a number of cows still in the herd which had aborted from Brucellosis in 1938 and 1939. Poor production was the only reason any cows were removed from the herd. At present the herd contains 39 adult cows, 19 of which are completely negative, and 20 cows showing partial or complete reactions at 1:25 or 1:50, but none complete at the 1:100 dilution with United States Bureau of Animal Industry antigen.

This is only one of 20 herds in Del Norte County where good results have followed calfhood and adult cow vaccination.

Recovery from Brucellosis was an important factor in the disappearance of the disease from this herd. Dr. B. A. Beach and his associates in Wisconsin have reported that, under certain circumstances, a high percentage of reacting animals will revert to negative. Our observations in Del Norte County indicate that such results may be expected under conditions of commercial dairying. In this connection, it should be explained that adult vaccination need be practiced only during one breeding season, since it can later be limited to calfhood vaccination unless a program of revaccination is instituted.

The effects of calfhood and adult vaccination on the incidence of Brucella infection following its introduction into a herd are shown by studies conducted by Drs. B. N. Carle and O. W. Schalm at the University of California. The herd studied consisted of 177 adult cows at the time infection occurred, and had been free of reactors for over 10 years. Calfhood vaccination was started in this herd in 1939. In 1941, an aged cow showed a positive reaction and, by the first of the next year, so many cattle had become infected, that attempts to control the disease by blood test and removal of reactors were abandoned.

It was decided to vaccinate all of the adult cows which were still negative to the blood test and either open or not more than 4 months pregnant. This resulted in a herd with three groups of cattle, i. e., cows vaccinated as calves, cows vaccinated as adults, and a group of nonvaccinated adult animals. The percentage of abortions in the three groups was 13.9 in the animals vaccinated as calves, 16.9 in those vaccinated as adults, and 40.9 in the nonvaccinated cows.

At the time of each calving or abortion during 1942, cultures and guinea pig inoculations were made with uterine discharges and milk in as many cases as it was possible to get the material.

In animals vaccinated as calves between four and nine months of age, Brucella abortus was isolated in 10.26 per cent of 39 attempts; in those vaccinated as adults in 2.6 per cent of 73 attempts, and in nonvaccinated cows in 41.9 per cent of 81 attempts.

From these results, it will be seen that, in 1942, after the infection had become
established, there was very little difference between the calf crop of the group vaccinated as adults and that vaccinated as calves. The calf crops in both of these groups were definitely better than that in which the animals were left unvaccinated. As would be expected, the number of reactors was greatest in the group vaccinated as adults. The ease with which Brucella organisms were isolated from the three groups would indicate that more actual infection was present in the vaccinated heifers than in the vaccinated aged cows. In view of the fact that the milk and uterine discharges of all animals in each group were not examined bacteriologically, it may be that this difference is merely coincidental. It is presumed, however, that the results of the Brucella cultures give a fair index of the presence of infection in the different groups.

THE VACCINATION OF REACTING COWS

In 1940, Dr. Maynard Rosenberger, who cooperated with the University of California (see Proceedings of the 1940 meeting, p. 77), reported no appreciable good or apparent harm from the use of vaccine on reacting cows. His statement was based on a study of 1228 cows that had been vaccinated while showing a positive reaction. Although we have no serious criticism of Doctor Rosenberger's conclusion that such practice is harmless, it has been observed that animals which are vaccinated after showing a reaction usually continue to be reactors for several years, while unvaccinated reactors not infrequently recover and become negative. Therefore, vaccination of reactors is a disadvantage if the ultimate aim is a Brucellosis-free herd, free from reactors caused by vaccine. Furthermore, our data on large numbers of animals indicate that no appreciable difference in the resistance to infection was found between vaccinated and nonvaccinated, reacting cows.

What harm results from the use of Strain 19 on adult cattle in Brucellosis-free herds? The injection of Strain 19 into pregnant cows after the fourth month of gestation has, in our experience, caused some abortion in rare instances, but associated, non-vaccinated cattle have not shown infection with Strain 19. Furthermore, if care is used to avoid the injection of cows after they have reached the fourth month of pregnancy, the abortions resulting directly from the vaccine will be negligible compared to losses which usually occur in unvaccinated cattle in infected herds.

To illustrate what happens when adult cows in Brucella-free herds are vaccinated, we can report that a company had three of its dairies free from Brucellosis by the use of frequent blood tests and the removal of reactors. Occasionally an animal would show a suspicious titer and would be transferred to a fourth dairy, ten miles away, which was known to contain infected cattle. In 1935, the manager asked the University to experiment with calfhood vaccination, and he was so encouraged with the results, he asked for experiments in adult cattle vaccination. The first trials were made in Dairy Number 7, which consisted of 189 cows, none of which, on test, showed even a trace of agglutination at 1:50. No bulls, heifers or calves were kept on this farm. The calves were removed at birth to a calf farm and the cows were injected with semen brought from bulls on one of the other farms owned by the Company. Vaccine doses of approximately 100 billion organisms were given to 91 of the cows in this herd. Fourteen of these were pregnant at the time of injection. The herd also contained 98 cows on which no vaccine was used. None of
these nonvaccinated cows showed agglutinins at any dilution above 1:25 nor at retests three and six months after the experiment started. This is particularly noteworthy because during that time the skim milk from these and also from the 91 head of vaccinated cows was being fed back to the herd by mixing it with their feed. Frequent guinea pig injections of the milk made during a six-month period following vaccination were all negative. The calving and sterility incidence in this herd after vaccination was as follows:

In the group of 14 cows vaccinated while from 15 to 195 days in gestation, one aborted; that is, 95.4 per cent gave birth to normal calves.

In the group of 40 head vaccinated while open, none aborted, but there were two still-births; that is, 95 per cent calved normally.

Eight head vaccinated while open proved sterile and 29 were sold too soon after vaccination to permit a record of results of breeding.

In the 57 nonvaccinated cows that calved, one still-birth occurred; that is, 98.3 per cent were normal. Thirty-five of the nonvaccinated cows proved sterile and six were sold too soon to permit any breeding records being made.

Some loss of milk production was probably caused by the shock of Strain 19 on the lactating cows, but the fact that the nonvaccinated cows, as well as those vaccinated, showed decreased yields indicated that weather conditions were chiefly to blame for decreases in productivity. The writers have observed that lactation may decrease as much as 20 per cent for three or four days immediately following vaccination, and it may be two weeks before normal production returns. The manager, however, was so pleased with the results of adult vaccination that he decided to have all his cattle vaccinated. These numbered about 1700 head. By doing this, he expected to eliminate the cost of segregation and testing. Since, under such conditions, no controls were available, the University withdrew from further cooperation. For economic reasons, particularly where it is very difficult to get labor, such a move was probably justified from the standpoint of this owner. The fact that thousands of dairymen throughout California are now following such a policy has resulted in a situation, the eventual outcome of which cannot be predicted at this time.

Since adult cattle vaccination as well as calfhood vaccination has been found to be superior to blood test and segregation or slaughter in many California dairies, it does not seem to the writers of this paper that the same economic benefits would necessarily follow such practice in many or any of the other 47 states where agricultural, economic, legislative and political conditions are so different.

In closing, we wish to admit that Strain 19 is far from being an ideal vaccine. More facilities should be made available for the development and testing of improved products. Strain 19, however, is the only product at present that has been used on thousands of animals with any degree of satisfaction and, until a better vaccine is developed and tried, our livestock and dairy industries will continue to depend upon it for benefit.
HISTORY AND RESULT OF CALFHOOD VACCINATION IN THE DUNLOGGIN HERD

BY MELVIN M. RABSTEIN, V.M.D.

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In September of this year, one of the finest Holstein herds in the country, owned by Dunloggin Farms, was sold at public auction. About 250 animals brought an average price of $1873, which was considered a record for the breed. The dispersal of this herd ended a calfhood vaccination experiment which we began in 1939. Accurate and complete records of this experiment were kept during the entire period and seem of sufficient interest to warrant a report of what has been accomplished.

The excellent results obtained in the herd were in no small measure due to the fine cooperation and intelligent interest on the part of the manager, Mr. Paul Misner.

PRE-VACCINATION HISTORY OF THE HERD

The original Dunloggin herd was composed of a large number of purebred Holstein cattle purchased from various sources in 1928, but it was not until five years later that testing for Brucellosis began. From 1935 until 1937 the entire herd was blood tested at regular intervals in an effort to gain an accredited status. Since only six reactors were found during this two-year period and there were no signs of active infection, it was thought that the accreditation of the herd would be a relatively easy process. Finally, on March 7, 1938, the herd of 283 animals, having passed the required number of blood tests over a period of one year, was accredited.

About nine months following this test for accreditation, we were told by the manager that several of his animals had aborted and that he wished to have another herd test immediately since he feared an incipient outbreak. We were not unduly alarmed because, during the nine months since the herd had become accredited, we had made nine different tests involving 89 animals that had either been sold or entered in shows without finding any reactors. Nevertheless, it was decided to make a herd test immediately to determine whether any infection did exist; and the whole herd, consisting of 264 animals, was tested. The results revealed that there were 13 reactors and four suspects, and these animals were immediately sent to slaughter.

Two weeks later, 250 animals were tested and five reactors and three suspects were found. Three of these reactors were outstanding cows, and it was desired to retain them in the herd. Since a separate barn and pasture were available, these three animals were isolated. As a future policy, it was decided to abandon the test and slaughter program but to continue frequent testing and to remove all reactors to segregation quarters. Tests were made on alternate weeks in an effort to keep ahead of the infection which appeared to be gaining ground rapidly.

We were unable to make any headway even with such repeated testing over a
period of three months (table 1). During this time 35 reactors and suspects were removed from the herd to isolation. Eight months following the appearance of the first reactors in the herd, there were 43 positive animals in the segregated group, and it became evident that there would have to be some change in the program if we were to succeed in our efforts to overcome the infection. We therefore recommended that the herd be taken under our supervision and a calfhood vaccination experiment inaugurated.

<table>
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<th>DATE OF TEST</th>
<th>NUMBER TESTED</th>
<th>NUMBER NEGATIVE</th>
<th>NUMBER POSITIVE</th>
<th>NUMBER SUSPICIOUS</th>
<th>REMARKS</th>
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<tr>
<td>12-20-38</td>
<td>264</td>
<td>247</td>
<td>13</td>
<td>4</td>
<td>All reactors and suspects sent to immediate slaughter</td>
</tr>
<tr>
<td>1- 7-39</td>
<td>250</td>
<td>242</td>
<td>5</td>
<td>3</td>
<td>Reactors and suspects isolated</td>
</tr>
<tr>
<td>1-27-39</td>
<td>245</td>
<td>242</td>
<td>1</td>
<td>2</td>
<td>Reactors and suspects removed to isolation</td>
</tr>
<tr>
<td>2-10-39</td>
<td>241</td>
<td>234</td>
<td>4</td>
<td>3</td>
<td>Same</td>
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<tr>
<td>2-21-39</td>
<td>233</td>
<td>225</td>
<td>1</td>
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</tr>
<tr>
<td>3-22-39</td>
<td>229</td>
<td>217</td>
<td>6</td>
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</tr>
<tr>
<td>6-16-39</td>
<td>232</td>
<td>180</td>
<td>35</td>
<td>17</td>
<td>Last complete herd test prior to start of calfhood vaccination. Eight more reactors found two months later</td>
</tr>
</tbody>
</table>

VACCINATION DATA

There was available for immediate vaccination in the herd at this time a group of 11 heifer calves, four to eight months of age. In addition, there was a group of 25 heifers which ranged in age from nine months to 14 months. It was necessary to decide whether to vaccinate these over-age heifers and run the risk of having many of them retain their post-vaccination titers, or to take a chance and place these very susceptible heifers with the rest of the infected animals. The advantages to be gained from vaccinating these animals appeared to outweigh the disadvantages of the few animals which might fail to clear up. The first group of heifers vaccinated, therefore, numbered 36, of which 25 were over eight months of age.

Our procedure in this herd was to test all calves before vaccination and two weeks afterward. They were then tested at frequent intervals, usually a month or two apart. Out of 230 animals vaccinated over a period of four years, there were only two which showed any pre-vaccination titer, one of which was + P --, the other ++ + P. Considering that many of these animals came from positive dams, this was a surprisingly small number. The animal with the + P -- titer was rather interesting in that she produced no post-vaccination titer. We revaccinated her two months later, following which she showed a ++ + P titer which disappeared in two months. She later had two normal calves. The ++ + P animal was 14 months of age when vaccinated, and her post-vaccination titer disappeared within five months.
In general the post-vaccination titer in the animals vaccinated between four and eight months of age disappeared rapidly. Six months following vaccination, 69 per cent of the animals were negative, and one year afterward 92 per cent were negative. Of the animals which had failed to lose their post-vaccination titer within a year, only one was still showing a suspicious titer at the time of first calving. The animals which were over eight months of age when vaccinated lost their titers more slowly than the others and some of these took as much as 30 months to clear up, but they did eventually become negative. Only one failed completely to lose her positive titer. This heifer was carried on test for three years, during which time she had two normal calves. Following each calving we collected milk samples and uterine material which were examined by the guinea pig inoculation method for the presence of Brucella abortus. We were never able to recover Brucella from this animal. She was later given to the laboratory for experimental purposes, and we tested two more sets of quarter milk samples and innumerable uterine swabs with continued negative results.

A few of the vaccinated animals showed what we call a fluctuating titer. Such animals lose their post-vaccination titers and then at a later date again show either a positive or more often a suspicious titer. Sometimes the titer will fluctuate from negative to + or + + over long periods of time.' There were nine such animals in this herd, all of which showed normal breeding histories and calvings.

It was the practice to blood test each vaccinated animal within several days following calving and we found that there was a tendency in previously negative animals to show a suspicious titer at this time, but it usually disappeared promptly. The vaccinated heifers were also bred, regardless of whether or not they were still showing a post-vaccination titer. This did not appear to prolong the time required for these animals to return to a negative status.

There were 109 normal calvings recorded in the vaccinated animals, 32 of which had one calf, 28 had two calves, and seven had three. There were four cases of retained placentae, which was not a higher average than was found in the negative unvaccinated group. Two abortions were recorded in first-calf heifers, both of which had passed many negative tests both before and after aborting. We were unable to recover Brucella abortus from either the fetuses or from the quarter milk samples which we drew later.

Quarter milk samples were drawn from 48 of the vaccinated animals following their first calvings. These were tested for the presence of Brucella by direct cultural examination and guinea pig inoculations. We were unable to recover Brucella from any of these animals.

**HERD HISTORY FOLLOWING VACCINATION**

By the time vaccination was begun in this herd, the infection had evidently reached its peak and had begun to decline, because although we did pick up eight new reactors on subsequent herd tests, there were no new abortions due to Brucellosis in the unvaccinated animals. The entire herd was tested periodically, usually about two or three times a year. In December, 1942, having met the state regulations and passed the required number of negative tests over a period of one year, the Dunloggin herd was reaccredited. Thus, it took four years from the time the
first reactors appeared in the herd to regain an accredited status by the use of calfhood vaccination.

The disposition and blood test records of the 43 reacting animals which remained in the herd at the time vaccination was started, offer some interesting and unusual information. Twenty-four of these animals returned to a negative status, although the time required varied to a considerable extent. Some required three years to become negative, others cleaned up in four months. The average time was about one year. Three of these 24 animals aborted twice, while six of them aborted once. However, almost all calved normally two or three times during their lifetime in the herd. The breeding history on this group showed that their calving efficiency was as good as that of the other cows in the herd.

Quarter milk samples were drawn from 12 of these animals after they had become negative, and were tested for the presence of Brucella with completely negative results.

Six of the 19 cows which remained positive were sold out of the herd. The remaining 13 animals never lost their positive titers. Six of these 13 aborted once, but all went on to produce a total of 37 normal calves during their lifetime with the herd. So far as breeding records were concerned, this group compared favorably with the negative cows in the herd.

**Discussion**

The number of animals involved in this herd is perhaps insufficient to warrant the drawing of conclusions. Nevertheless, the results obtained check closely with the information that we have gathered from many different types of herds over a period of years.

It would appear that it is not necessary to test calves prior to vaccination. Although most of the reactors in the Dunloggin herd were retained, we found only 0.8 per cent of our calves of vaccination age showing either a positive or suspicious titer. These animals promptly cleaned up after vaccination.

The administration of a potent vaccine will produce, with very few exceptions, an average titer of 1:500 in about two weeks. We concede that the post-vaccination titer is not a true indication of the immunity produced, since the animals remain immune after the titer has disappeared. We believe, however, that the post-vaccination titer is a good criterion of the viability of the vaccine used and that the blood testing of calves two weeks following vaccination should be resorted to whenever there is any doubt as to the potency of the vaccine.

The problem of the fluctuating titer is a serious one in that it is an undesirable side effect which has a tendency to alarm the owner in whose herd such troublesome animals appear, especially if the owner has an accredited herd and has been educated to look upon any titer, regardless of its cause, as reason for alarm. Such an owner must be told at the start of calfhood vaccination to expect a certain number of suspicious vaccinated animals in the herd and not to worry about them.

The reason for such recurrent titers is unknown, but we feel reasonably certain that such animals are not dangerous to the remainder of the unvaccinated herd.

The return to a negative status over varying periods of time of over 50 per cent of the original reacting animals should probably be considered as the exception
rather than the rule. The interesting aspect here is that the strain of Brucella involved in the outbreak in this herd was not one of low virulence, since almost half of the reacting animals aborted.

The main source of spread in bovine Brucellosis is the aborted fetus and the infected uterine material which is shed following abortion. Any immunizing agent which will reduce this symptom of Brucellosis to a minimum will certainly go a long way toward reducing the incidence of the disease. All our work on calfhood vaccination has been carried on in herds where the infection rate has ranged from 25 per cent to 100 per cent, yet in no herd has the percentage of abortions in the vaccinated animals due to Brucellosis exceeded 1.5 per cent. This record has been amply corroborated by other workers in the field. Until information proving otherwise is discovered, it must be admitted that vaccination against Brucellosis with Strain 19 does confer a substantial immunity.

The herd history here presented serves to illustrate how great an aid calfhood vaccination can be toward the building up of an accredited herd.
BRUCELLOSIS IN SWINE

BY L. M. HUTCHINGS, B.S., D.V.M., M.S.

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Brucellosis in swine is one of the important problems in the swine industry. This disease is of considerable economic importance to the swine raiser, but there are no satisfactory figures to indicate the incidence nor the financial losses resulting from it. Surveys that have been conducted indicate that roughly 20 per cent of the hogs in some sections are infected, others indicate a much lower percentage of infection. It is not uncommon to encounter herds of swine where this disease prevents profitable operation. The evidence available indicates that brucellosis in swine is on the increase as might be expected under prevailing conditions where little effort is being made to check or control its spread.

In this paper citations to the literature are omitted as much as possible for the sake of brevity. The paper of Dr. S. H. McNutt (1) presented before this association in 1938 is suggested for further references to the literature.

The importance of brucellosis in swine extends beyond the swine industry since it involves other kinds of farm animals, especially cattle and also human health. 

Brucella suis has frequently been isolated from cattle indicating that infected swine may act as a reservoir of infection for cattle.

A major part of the problem concerns human health, particularly among farmers and butchers. Porcine strains of the Brucella organisms are more pathogenic for man than bovine strains. In swine the Brucella organisms commonly invade the blood stream and also become localized in various edible parts of the hog carcass. This is in contrast to the corresponding disease in cattle where Br. abortus is rarely found in any tissues other than the uterus and the mammary gland. There is ample evidence to show that Brucella organisms invade the "unbroken" skin of man and animals. Thus, there is a possible public health problem resulting from the distribution of fresh pork products from hogs infected with brucellosis. In the light of our present knowledge it appears that the satisfactory control of undulant fever in man is dependent upon prevention of exposure to infected animals and thereby is largely a veterinary problem.

Relating to the public health importance of this disease in swine. Dr. Carl F. Jordan, M.D. (2), referring to cases of undulant fever reports, "among 205 Brucella strains isolated in Iowa during the 15 year period 1927-1941: 147 (71.7%) were porcine (Brucella Suis)." He further states, "In the aggregate, cases of the disease resulting from contact probably exceed in number those traceable to contaminated dairy products. Undulant fever of milk-borne character is apt to be of sporadic

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occurrence whenever the bovine type of Brucella (Br. abortus) is the causative agent."

"Unlike the bovine strain of Brucella, when the porcine species (Br. suis) gains access to the udder of one or more dairy cows to contaminate a raw milk supply, an epidemic of undulant fever may be fully expected. This is apparently due to the fact that Brucella suis is more virulent and more invasive than Br. abortus."

"Two outbreaks of milk borne epidemics of brucellosis caused by the porcine type of Brucella have occurred in Iowa in past years.... All blood cultures from patients yielded Brucella of porcine type. Br. suis was isolated from the cream of several dairy cows which reacted to the test for Bang's disease. Of twenty-four sows that had been allowed to occupy the same lot with dairy cows, ten showed positive and three suspicious reactions in the agglutination test."

An example of the importance of brucellosis in swine to human health which occurred recently in Indiana seems worthy of note. Thirteen out of 150 to 160 butchers employed by one small, but thoroughly modern packing plant, were ill with undulant fever at one time (3). These butchers were handling nothing but hogs. Obviously, this situation created a number of problems some of which can not be solved with our present knowledge. For example, the other butchers asked the management for assurance that they too would not become ill with undulant fever. The medical profession does not know how to successfully immunize people against undulant fever. The veterinary profession does not have sufficient knowledge or machinery to eliminate Brucella-infected hogs from those which the butchers are required to handle. It would be impractical and unsatisfactory to attempt to prevent infection in such butchers by requiring them to wear rubber gloves and practice other appropriate precautionary measures. Many farmers and butchers are in contact with Brucella-infected swine. Dr. Thurman Rice (4), state health commissioner of the Indiana State Board of Health, states, "Any physician who has practiced long among farmers realizes that entirely too many of them are prematurely crippled up with arthritis said to be due to rheumatism and other obscure causes. There is no doubt whatever that many of these cases are in this condition because of undulant fever."

Brucellosis in swine is caused by Brucella suis, a species of Brucella which was first reported by Traum (5) in 1914. Apparently swine are not infected with Br. abortus or Br. melitensis under natural conditions. The scientific evidence to date strongly indicates that Br. abortus is not regularly transmitted from cattle to hogs but that Br. suis is transmitted not infrequently from hogs to cattle.

The mode of infection and age of hogs susceptible to brucellosis has not been studied adequately. Some experiments conducted at Purdue University indicate that oral, conjunctival, vaginal and rectal exposures are all effective in weanling pigs. These studies further indicate that a high percentage of weanling pigs are readily infected which is contrary to the results of some previous investigations. A total of 60 heterogenous weanlings pigs were exposed to massive doses of Br. suis by six different channels. Subsequently, Br. suis was isolated from the blood of 40 of these pigs and all but one of the 60 gave some evidence of an agglutination response at some time but not necessarily at the time of isolation of the organism. Br. suis was isolated from various tissues of eight of a total of 18 of these pigs destroyed for bacteriological examination.
Twenty-three gilts from the above mentioned group of pigs exposed at weaning age were subsequently bred and without exception completed a normal gestation period. Fetal membranes, colostrum, all dead pigs, and at least one live pig from each litter were examined for Br. suis with negative results.

In one naturally infected herd the pigs were isolated from the sow at weaning time and placed in two separate groups. One group had no known contact with any other swine. In this group all pigs were tested when about three months and were negative to the agglutination test. The second group were on a creek bottom pasture where it was possible for water from the infected sow's pasture to reach the creek. In this second group there were 37 boar pigs of which 24 reacted in various maximum dilutions and 49 gilts of which 24 reacted to the agglutination test in various dilutions. Orchitis was observed in several of the young boar pigs. This indicates that infection was contracted by these young pigs either from ingesting infected sow's milk, contact with the infected sows, or from the possible water contamination after weaning.

Infection of the young pig may partially explain why the symptom of abortion is prevalent in some naturally infected herds and apparently less prevalent in others. In our field experiments, herds containing as many as 80 per cent positive reacting swine have been encountered and yet the owners had no knowledge of any abortions having occurred. Possibly these herds had been infected for some time and the replacement pigs had contracted the disease early in life, developed some resistance against the symptom of abortion and did not abort when pregnancy became established. In contrast with this some apparently recently infected herds have shown the symptom of abortion in a high percentage of cases. The percentage of abortions is usually much higher in gilts than in sows.

Evidence is accumulating which suggests that there may be a relationship between the time of exposure and the stage of pregnancy which influences the outcome of the pregnancy in Brucella-infected swine. In other words, it appears likely that sows and gilts may not commonly abort when exposed to Br. suis while in the non-pregnant state or when exposed relatively late in the pregnancy. Results so far suggest that in sows and gilts exposed at the time of breeding or shortly thereafter, the pregnancy is more likely to terminate in an abortion.

Another reason for the lack of observed and recorded abortions in some herds is the phenomenon of very early abortions. In our controlled experiments, abortions have occurred as early as 23 days after exposure, with Br. suis isolated from the fetuses. Also Br. suis has been recovered in natural outbreaks of the disease from fetuses aborted at 27 days after service. Such early abortions are very difficult to observe in swine and probably occur much more frequently than the farmers or their veterinarians recognize. It is mainly luck when such abortions are detected. As an example, one herd in which 75 per cent of the sows and gilts reacted to the agglutination test, had experienced no abortions to the owner's knowledge, yet upon examination of the breeding records, it was noted that 37 of the first 100 sows and gilts bred had been in heat again in from 21 to 52 days following the first service. Such unsatisfactory breeding results could well be explained by early, unobserved abortions.

Symptoms of this disease in swine, other than abortions, vary considerably de-
BRUCELLOSIS IN SWINE

Depending on the site of localization of the causative organisms. Lameness, abscesses, spondylitis, sterility and orchitis are the more common conditions that may be associated with Brucella infection in swine.

Spread of the disease probably occurs chiefly at the time of abortion and through service by the infected boar. Probably the disease spreads in other ways. In our experiments, Br. suis has not been recovered frequently from normally farrowing sows at the time of parturition even though reacting to the agglutination test. Ample evidence shows that the infected boar is an important source of spread. In some cases, however, by the time a clinical orchitis has appeared the boar may cease to breed, consequently he is frequently not as active a spreader after the clinical manifestations have appeared as during the early stages of infection. Brucella suis has been isolated many times from boar semen.

The diagnosis of brucellosis in swine is commonly based on the standard agglutination procedures used for the diagnosis of the corresponding disease in cattle. Conflicting views are held in regard to the interpretation and accuracy of the agglutination test when used as a diagnostic procedure for swine brucellosis. Some investigators consider a positive reaction at 1:100 dilution of serum to be significant while an apparent majority feel that considerable significance should be attached to reactions in a 1:25 or higher dilution of the serum. From our experimental studies, it appears that the agglutination methods as now employed are adequate as a herd diagnostic procedure, but may not be sufficiently reliable as a basis of diagnosis of brucellosis in individual swine. This tentative deduction is based on results of both controlled and field experiments. In the controlled experiments there were many cases in which Br. suis was recovered from swine showing negative or essentially negative agglutination reactions. In our field observations the practice of attempting to assemble a herd of swine free from brucellosis by purchases based on negative agglutination tests of individual animals from infected herds has failed. Also control attempts in naturally infected herds have failed too frequently unless all the breeding stock associated with the reactors was considered as infected.

Often sows that react farrow normal litters and do not eliminate Br. suis from the uterus or in the colostrum. In these cases, at no time has it been possible for us to demonstrate specific agglutinins in the blood of the new-born pigs before nursing. However, as short a time as six hours after nursing some of these pigs showed a low titer agglutination response if the colostrum of the sow contained agglutinins. This low titer agglutination response in such pigs disappeared rather rapidly after weaning and in many cases disappeared before weaning. This seems to indicate that there is frequently a concentration of agglutinins in colostrum of such sows which subsequently disappears from the milk during the suckling period; hence, some such suckling pigs no longer carry an agglutination reaction. Thus extreme caution may be necessary in interpreting the agglutination test in young pigs, since it is likely that positive agglutination reactions in these pigs may be the result of absorption of agglutinins from the colostrum of the mothers and are not evidence of active infection. It should be pointed out again that Br. suis was not recovered from these sows and gilts at the time of parturition.

On the other hand Mingle (6) has evidence which shows that entirely different agglutination results were obtained in pigs born from mothers that were eliminating
Br. suis in the colostrum at the time of parturition. Isolations of Br. suis from the blood as well as agglutination titers have been obtained from such pigs for as long as 20 weeks after weaning. Thomsen (7) reports that Br. suis may live in the organs of the pig—including the seminal vesicles and the bladder—for more than one year after the organism has been ingested by suckling pigs. One of these pigs, a boar, was found by him to excrete Br. suis bacteria with the semen. Thus, knowledge of the infection status of the mother at the time of parturition may be essential for a correct interpretation of agglutination reactions in weanling pigs.

Intradermal injections of Brucella extracts prepared by Dr. Huddleson (8) are being used experimentally at Purdue University in an attempt to develop a more accurate and possibly easier diagnostic procedure than the agglutination test. Results to date indicate that some of these products may be worthy of study on a larger scale. The chief criticism of these materials seems to be that they may cause reactions in sensitized as well as in infected animals. Reactions to the agglutination test may not occur for some time after infection is established. Errors in the skin test would not vitiate the success of a plan of control based on test and elimination of infected individuals.

Control procedures other than those already established for cattle, which do not seem to be adequate for swine, have not been used extensively. Vaccination attempts using Strain 19 have been unsuccessful. Other vaccines are being tried at various experiment stations, but results have not been reported as yet.

One of the chief objections to control based on the agglutination test has been the tedious process of obtaining suitable blood samples from swine. This objection can be readily overcome by bleeding swine of all ages from the anterior vena cava as described by Carle and Dewhirst (9).

A plan of control should take into account the principle sources of spread and the probable age groups affected. The principle source of spread is apparently animal to animal contact rather than infected premises. The age groups affected appear to be animals of all ages. At present the only method that can be recommended is agglutination blood testing with prompt removal or segregation of reactors. In the main our attempts to control the disease have been unsuccessful when the reactors have been segregated and held in isolation. Some veterinarians have been unsuccessful by testing and removing all reactors for slaughter. It would seem that more success could be affected if the entire breeding herd were considered as infected regardless of the number of reactors in this group. In other words, the animals in the infected herds should be handled in groups rather than as individuals.

Control in an ordinary herd where pork production is the main objective may be handled most economically by selling the entire herd for slaughter, and starting over with animals from a healthy herd. In the purebred herd where the sale of breeding stock is the main objective, a different plan must of necessity be employed. Here the reactors may be either sold for slaughter or isolated completely and the remainder of the breeding herd tested at least once a month until three consecutive negative blood tests have been passed. This plan has limited chance for success since some individual animals may not show a positive agglutination test even when they are carrying the organisms in the blood, consequently they may act as spreaders to the negative stock. Any plan in which segregation is used is dangerous because the principle source of infection is not removed from the farm.
Whatever plan of control is used it seems likely that the pigs should be removed and isolated from the infected sows at weaning time and that these pigs should be tested at weaning and rather frequently up to and during the first pregnancy. Only negative pigs should be kept as replacement breeding stock.

When the disease has been eliminated from a herd, reinfection should be avoided. This is not an easy task. It should be remembered that the practice of purchasing replacement stock is a common source of reinfection. In this regard it is well to remember that experience in assembling a herd of swine based on negative individual tests from infected herds has failed. This apparently means that replacement stock should be purchased, wherever possible, from herds in which all animals in the herd are negative to the test. If the replacement animals are purchased from infected herds, it is necessary to isolate the new animals and retest them before finally adding them to the herd.

Any infected boar should be eliminated immediately. It appears that often the owner is reluctant to dispose of an infected boar because of his appraised value and this one factor alone may result in the failure of the control attempt.

Success of a control program seems to rest with the completeness of isolation, the promptness of removal, and the handling of the breeding stock as infected groups rather than as infected individuals.

Recommendations have been made very general because in our work with this disease in the field the results of practical control measures so far have not been encouraging with the exception of the previously mentioned plan of complete herd sales for slaughter and re-entry into the swine business with clean stock. It is timely to direct attention to the urgent need for further research on the various phases of this disease. The method of diagnosis based on the agglutination test as now employed is the only weapon available but is not without rather serious limitations. The symptom of abortion should not be relied on as a basis of diagnosis nor should the fact that no abortions have occurred lessen the zeal for controlling this disease in a herd for despite this apparent lack of abortions in some herds the fact still remains that other symptoms and the public health problem is very important. In our own experimental set-up, where during the first year no abortions occurred, one of two barn men contracted the infection and became ill with undulant fever. Our attempts to protect these men from infection included liberal use of antiseptics, rubber gloves, boots and complete changes of clothing as well as shower facilities. It would seem that the ordinary handler of an infected herd of swine would have more chances of self-exposure under ordinary farm conditions.

Unfortunately no plan can be recommended at present that will always control the disease with certainty. This does not mean that we should not continue applying our knowledge of control to the best of our ability with the tools at hand in an effort to keep the disease in check until such time as more definite control procedures can be established.

REFERENCES

3. Eaton, Lyman D., M.D. Director District Health Department No. 1 Indiana. Personal communication.


Progress in the control and eradication of brucellosis in cattle has not been as great in the year immediately past as in previous years. This is a result of several factors, including the lack of sufficient available veterinarians due to war-time conditions. On October 1, 1943, approximately 2,220,000 herds, containing about 16,800,000 cattle, were under supervision in the cooperative program to control and eradicate brucellosis in cattle. Among these, approximately 40,500 herds, containing about 846,000 cattle, located in 41 states, are fully accredited Bang's disease-free herds. The number of cattle tested has decreased from an average of 575,000 per month last year to approximately 432,000 per month during the year which ended July 30, 1943. Thus, the number of cattle tested under the Federal-State cooperative program is only about 75 per cent of the number tested last year. Approximately 50 per cent of tests conducted this year are in sections of the country where the area-plan of control is used. The area-plan of control is being continued, and during the past fiscal year 46 counties were added to the modified, accredited Bang's disease-free area. There were 14 counties added on July 1, 1943 making a total of 582 counties in 24 states in this classification. There are about 5,462,400 dairy and breeding cattle in these counties. Area work is being conducted in about 135 additional counties in 19 states. Thus, the farmers in 717 (23%) of the 3,071 counties in the United States have chosen the area-plan for the control of brucellosis in cattle. Funds for indemnity in the control of brucellosis are provided in Puerto Rico, and all of the States except California, Colorado, Indiana, Massachusetts, Nevada, Oklahoma, Texas and Utah.

**Calfhood Vaccination**

Calfhood vaccination, which was made a part of the brucellosis control program in December, 1940, has increased in volume during the year. About 25,000 calves are being vaccinated each month under official supervision as compared to about 8,000 calves vaccinated per month one year ago. Since January 1, 1941, about 442,000 calves have been so vaccinated. The use of Brucella vaccine continues to be an aid in the program, but it is not a substitute for other procedures which it is necessary to follow in order to effectively handle this disease.

A recent release from the United States Bureau of Animal Industry shows that, in some herds applying calfhood vaccination, non-vaccinated, positive reacting, mature cattle are being retained in the herds instead of being eliminated by slaughter. This practice seems to have an advantage in some infected herds, particularly during war time. The figures show that of a total of 197,329 reactors disclosed during the
year ending June 30, 1943, 53,558 or 27 per cent were retained by the owners in herds where calfhood vaccination was conducted. Some control officials have come to the conclusion that better results from calfhood vaccination are obtained in herds where naturally infected mature cattle are maintained in contact with the vaccinated calves. However, in some sections it has been found unsafe to allow pregnant vaccinated heifers to associate with cows that are aborting from Brucella infection because the resistance conferred by calfhood vaccination is not always sufficient against infection from massive discharges from such cows.

Calfhood vaccination in brucellosis-free herds may be desirable in areas where the evidence of infection is high. The use of Strain 19 in healthy herds has been under observation for at least ten years and no evidence of transmission of Strain 19 infection to the associated non-vaccinated animals has been noted.

VACCINATION OF MATURE CATTLE

In some states, vaccination of mature cattle with Strain 19 vaccine is being employed in certain types of infected herds under official supervision. Also, many veterinarians and laymen are using the vaccines in mature cattle in infected and non-infected herds not under official supervision. Such practice seems widespread and popular in some quarters. Attention is directed to the fact that sufficient data from controlled experiments are not available at this time to establish the merits and safety of such use of Strain 19 in mature cattle. Some studies are being made of the use of the vaccine in adult cattle under field conditions, but a sufficient amount of this work has not been done to prepare a complete report concerning it. The vaccinated adult cattle usually continue to give a positive reaction to the agglutination blood test for brucellosis for a considerable and indefinite period of time. This interferes with the movement of such cattle from one place to another because of regulations and it also interferes with the marketing of milk in localities where the regulations require that all milk be obtained from herds, negative to the agglutination blood test for this disease.

Adult cattle vaccination has become general in many states. There has not been sufficient research work done nor are there sufficient data available for your Committee to approve the vaccination of mature cattle with Brucella abortus vaccine, Strain 19. We are not unmindful of the fact that under certain isolated conditions and especially during war time when it is our duty to conserve and promote production of beef and milk, that vaccination of matured breeding animals may be indicated when such vaccination is done under official supervision and when all such cattle are maintained in quarantine and handled as reactor cattle.

The Committee recommends the following broad principles for the control of bovine brucellosis for use during the period of war;

1. That control and eradication efforts, based on the agglutination blood test be continued, with emphasis on the elimination of unprofitable, reacting animals. During the present emergency, the judicious retention of reacting animals may be advisable in certain infected herds, provided that they are high producers or represent valuable blood lines, and further provided that the retention of such reacting animals does not jeopardise the total productive capacity of such herds by infecting clean cattle.
2. That more emphasis be placed on appropriate herd management and sanitation practices which have repeatedly demonstrated their value for increasing production efficiency in infected herds by decreasing the spread of the disease within the herd.

3. That the area-plan of control be continued and expanded to areas where it is feasible.

4. That we approve and encourage calfhood vaccination under proper supervision as recommended as an aid to control and discourage its use as a substitute for good herd management and proper sanitary practices. The ultimate objective in all cases to be a clean, non-infected herd in which the mature animals are negative to the agglutination blood test for brucellosis.

**BRUCELLOSIS IN SWINE**

Knowledge of brucella infections in swine from controlled experiments and from experience with natural outbreaks of the disease is inadequate. No definite control procedures that are known to be satisfactory are available. Researches are in progress concerning this disease in swine.

The importance of brucellosis in swine with respect to efficient pork production, public health and as a potential source of infection for cattle is becoming increasingly apparent. The evidence so far indicates that no satisfactory method of vaccination to prevent this disease in swine is available. This Committee strongly urges that an expanded and intensified program of research with brucellosis in swine be carried on, both in laboratories and under farm conditions.

**MORE RESEARCH NEEDED**

There seems to be a growing tendency on the part of many veterinarians and cattle owners to consider the problem of brucella infections as satisfactorily solved with the use of Strain 19 vaccines. The Committee considers it timely to warn against the tendency towards this conclusion, because the problem of brucellosis control is far from being solved. Money spent now for research would probably yield more benefit than millions spent for blood tests and slaughter under present conditions. Extensive laboratory and field trials should be made with biologics that are known to produce some immunity without the handicap of also causing positive agglutination reactions. The possibility of enhancing genetic resistance by selection and breeding and the effect of combining various methods should be studied.

**RECOMMENDATIONS FOR REVISED REGULATIONS PROVIDING FOR MODIFIED ACCREDITED BOVINE BRUCELLOSIS-FREE AREAS**

The extent of the area shall be determined by State and Federal agencies cooperating. When testing is started, the area shall be placed under quarantine and the following rules shall be enforced:

Section 1. All cattle six months of age and over, except steers and cattle for immediate slaughter, when moved into a quarantine area, shall be handled according to the following rules:

Section 2. Cattle from officially accredited brucellosis-free herds, and cattle from
negative herds in modified accredited bovine brucellosis-free areas, when officially 
blood tested with negative results within one year of date of shipment, may enter 
other modified accredited areas without being retested for brucellosis. All such 
cattle shall be individually identified and shall be accompanied by approved cer-
tificates.

Section 3. Cattle from herds under Federal-State Supervision for the control of 
brucellosis in which all animals in the herd over six months of age, were negative 
to an official agglutination blood test for brucellosis within three months of entry 
and the individual animals were negative to such an official test within 30 days of 
the date of entry, may enter a modified brucellosis-free accredited area or an area 
in the process of such accreditation without further test.

Section 4. Heifers under 18 months of age officially vaccinated as calves when 
4 to 8 months of age, and coming direct from officially brucellosis-negative herds, 
may enter modified accredited areas without test when individually identified by 
mark, brand, tattoo or other acceptable individual identification and when certified 
by the State Sanitary official of the State of Origin that they have been officially 
vaccinated when calves with brucella abortus vaccine, Strain 19, subject to the 
provisions contained in sections 8 and 10.

Section 5. Breeding cattle over 18 months of age, and not more than 2½ years 
of age, officially vaccinated as calves when 4 to 8 months of age, shall be blood tested 
for bovine brucellosis before entering any modified accredited brucellosis-free area. 
Animals whose blood sera shows a reaction not higher than incomplete in a dilution 
of 1:100, may enter such areas provided that they shall be maintained in quarantine 
until they give two successive negative blood tests made not less than 60 days apart.

Section 6. All other male or female cattle over 6 months of age now, except steers 
and cattle intended for immediate slaughter, shall be required to pass a negative 
officially recognized agglutination blood test for brucellosis within 30 days prior to 
the date of entry and shall be maintained in quarantine separate from other cattle 
and retested in not less than 30 or more than 60 days after the date of entry. If 
found free, they shall then be released from quarantine.

Section 7. All cattle 6 months of age or over, except steers, in the area shall be 
tested for brucellosis, and all cattle reacting to the agglutination blood test for this 
disease in dilutions of 1 to 100 or higher shall be slaughtered within 15 days of the 
test or permanently identified and placed in quarantine, and all premises where 
such reacting cattle were located shall be cleaned and disinfected within 15 days of 
the date of the removal of the reacting animals. The milk and milk products de-

derived from reacting cattle shall not be used or sold unless properly pasteurized.

Section 8. If, as a result of a test of all the cattle required to be tested according 
to the provisions of Section 7 above, the number of reactors does not exceed 1 per 
cent, nor the herd infection exceed 5 per cent, the area may be declared a modified 
accredited brucellosis-free area for a period of three years by the State and Federal 
cooperating agencies in charge of the work, provided that all infected herds shall be 
placed in quarantine and the cattle in them retested for brucellosis at intervals of 
from 30 to 90 days until all of them passed two consecutive negative tests and pass 
a further negative test not less than six months from the date of the second negative 
test. And further provided that herds in which brucellosis vaccine is being em-
BRUCELLOSIS

ployed and in which any cattle 6 months of age or over are positive to the test for brucellosis, shall be maintained under strict quarantine, except calf vaccinated herds in range and semi-range area where it is not practical and for purposes of herd percentage shall be classed as infected herds.

Section 9. At the expiration of the three year period the area may be re-accredited for an additional 3-year period if all previously infected herds and such other herds as are designated by the cooperating Federal and State officials, are retested and the percentage of reactors among the cattle retested does not exceed 1 per cent of all the cattle so tested in the area.

Section 10. It will be permissible in modified accredited brucellosis-free areas to have not to exceed 1 per cent of the total number of herds in the area maintained as infected herds under strict quarantine with positive reacting cattle permanently and conspicuously identified, provided calfhood vaccination against this disease is conducted under official supervision in such infected herds. The quarantine herds and cattle contained in them shall be included in the total infected herds and reacting cattle when determining the percentage of herd and cattle infection as provided in section 8 of these Regulations.

As above stated, your Committee recognizes and approves the vaccination of calves 4 to 8 months of age with Brucella abortus vaccine Strain 19. In making this recommendation, your Committee desires very forcibly to reiterate that the use of Brucella abortus vaccine is an aid in the control of this disease and is not to be accepted as a panacea in the control of bovine brucellosis. We desire also to very forcibly call to the attention of the livestock industry that sanitation and proper herd management are absolutely indispensable in the control of bovine brucellosis. In approving calfhood vaccination we are not unmindful that in many instances the test and slaughter method will prove a more reliable and quicker method of eradicating bovine brucellosis in certain herds, and under certain conditions in area tested. Veterinary medicine deals with the economics of disease and as a profession we must recognize and adopt the most economical method for the control and eradication of disease in the various areas and under the various circumstances in which disease may make its appearance.
REPORT OF THE COMMITTEE ON PARASITIC DISEASES


IMPORTANT EXTERNAL PARASITES OF FOOD-PRODUCING ANIMALS

Outbreaks of external parasites during the past year have greatly affected meat and poultry production. The urgent demand on this source of the food supply to the armed forces and civilian population led the committee on parasitic diseases to choose for its study important external parasites of food-producing animals. The animals considered are cattle, sheep, swine and poultry.

In obtaining information the committee was fortunate in receiving contributions on phases of veterinary and medical entomology from leaders in the United States Bureau of Entomology and Plant Quarantine, and in the Zoological Division of the United States Bureau of Animal Industry. The names of individuals, except those of the committee, who furnished information used in this report are given below.

SCREWWORM (FLY COCHLIOMYIA AMERICANA C. & P.)

The larvae or maggots of the primary screwworm fly (Cochliomyia americana) are very damaging to livestock, especially to sheep and cattle. Any abrasion of the skin that occurs between April and November may become infested with screwworm fly larvae. These larvae which hatch from eggs laid at the edge of a wound, make their way into it. The oral hooks and body spines, together with the muscular equipment of the maggots, enable them to burrow into the wound and lacerate the tissues. Their more or less constant movement and the secretions elaborated by them set up severe irritation and cause great annoyance to the animal. Prolonged reinfestations in vital organs cause death of the host.

The numbers of larvae in a wound may be considerable. Laake, Cushing, and Parish (1936) found that a female fly could lay 300 eggs in from four to six minutes. After 11 to 12 hours the larvae hatch from the eggs and begin their feeding period which may last from 3½ to 4½ days. The mature maggot approaching the pupal stage soon drops from the wound to the ground where, in the cocoon, the pupal stage may be completed in seven or eight days. In optimum conditions the entire life

1 Contribution No. 237 from the Department of Zoology, Agricultural Experiment Station, Kansas State College of Agriculture and Applied Science, Manhattan, Kans.
cycle from egg to adult may be completed in less than two weeks, sometimes within 11 days.

The adult screwworm fly is bluish green in color and has three dark stripes on its back. The space between and below the eyes is a reddish or orange color. It is seldom observed on dead animals, whereas one or two species of blowflies closely resembling it are observed on carcasses of dead animals. Observing habits and color markings, it usually is possible for one to recognize the primary screwworm fly (*C. americana*).

Typical of screwworm infestations was the case of a southwestern Kentucky farmer, who on castrating and docking 60 lambs early in June, found on the second day thereafter a watery exudate dripping from many of the wounds. On the third day conditions were worse and a young veterinarian diagnosed the condition as screwworm fly infestation. Larvae were found in every one of the 60 lambs either in the tail or scrotum. The characteristic screwworm odor and details of worm arrangement in the wound left no doubt that the screwworm was the invader. There were no other infestations on the farm although some young animals were being born.

The occurrence of screwworm flies in 1943, according to the information received, was most severe in Florida, southern Georgia, Texas, southern and eastern New Mexico, and southern Kansas and Oklahoma where heavy outbreaks were reported. States with medium infestations of cattle, sheep and swine included the central Great Plains States from Indiana and Illinois south to the Gulf and from Iowa, Wisconsin, Minnesota and South Dakota, southwest to southern California.

Detailed inspections of livestock for screwworm fly infestations were made during the summer and fall by agents of the United States Department of Agriculture. They graded infestations as follows: 0.5 per cent or less were considered light; 0.6–2.0 per cent medium; and infestations of 2.5 per cent or more were classed as heavy. Heavy infestations of screwworm fly were found in southern counties of New Mexico during the middle of summer. The evidence for Eddy County indicated that infestations were running up to 5.5 per cent in all classes of animals, and Sierra County was experiencing from 3.5 to 5.0 per cent of all classes of livestock. In southern Texas, infestations in various classes of animals reached 8.5 per cent. In the earlier part of the season, southeastern Oklahoma had heavy losses. Data on portions of the livestock showed 2.5 per cent screwworm fly infection in 25,000 cattle; 1.5 per cent in 13,000 hogs and 10 per cent in 1,000 sheep. Over the wide belt from coast to coast field men place the loss to producers from screwworm infestation of food-producing animals in excess of $25,000,000.

**Control.** The control of the screwworm fly, *C. americana*, involves several factors. The adult flies do not migrate regularly from one section of the country to another, but they may drift as far as 1200 to 1500 miles each year (Cushing and Parman, 1942). Such flies have been known to spread as much as 35 miles in a week. Other means of dispersal from infested to non-infested areas include shipment of infested livestock, especially cattle and sheep, to feeding and finishing areas hundreds of miles away. Open wind-swept plains and high mountain regions serve as controlling factors while sparsely settled grazing land covered with a heavy low growth of brush and timber favor rapid increase of the fly population.
The most important single factor in the control of screwworm is climate. The flies cannot survive the year around in localities where the average temperature is 50°F. or lower for a continuous period of three months. At least one generation must be produced during a four month period or the fly will be exterminated in that area. Hard frosts kill many of the adults and for every 100 pupae which are subjected to winter conditions probably not more than three adult flies emerge. In regions where the climate is too mild to furnish a three-month freezing period, the screwworm fly offers a year round threat to the livestock industry.

Treatment. Several of the contributors recommended benzol as a treatment for wounds infested with the larval or maggot stage of the screwworm fly, *C. americana*. The results of researches in recent years by entomologists of the United States Department of Agriculture have shown that it is possible to combine benzol as a killing agent with a repellent, diphenylamine, and certain other ingredients to make an effective smear treatment. The formula known as Smear 62 is made by mixing the powdered chemical, diphenylamine with benzol, turkey-red oil, and lamp black in the following proportions:

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>Parts by weight</th>
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<tbody>
<tr>
<td>Diphenylamine (technical grade)</td>
<td>3.5</td>
</tr>
<tr>
<td>Benzol (90 per cent commercial)</td>
<td>3.5</td>
</tr>
<tr>
<td>Turkey-red oil (sulfonated castor oil, pH 10 or neutral)</td>
<td>1.0</td>
</tr>
<tr>
<td>Lampblack (Germantown)</td>
<td>2.0</td>
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This mixture which has the consistency of thin paint can be applied to the wound with a paint brush. The smear kills screwworm and other fly maggots infesting a wound and gives repellent protection for several days against reinfection. Two applications a week until the injured tissues have healed are best. With smaller wounds, one treatment may be sufficient to kill the larvae and give protection against reinfection. Such treatment together with avoidance of wounds due to cacti, needle grass, loose barbed wire, and other sharp pointed objects, and the arrangement for dehorning, branding, and castrating out of fly season will greatly aid in primary screwworm fly control.

BLOWFLIES

The larvae of certain blowflies when coming in contact with the broken skin of sheep penetrate into the tissues and cause a condition known as Calliphorine myiasis. Even unbroken skin is sometimes invaded. Undocked lambs and the skin under soiled wool following lambing become infested in the spring in Texas, New Mexico and Arizona, and during the summer in Oregon and Washington. There is very general use of Smear 62. In the west, however, benzol and pine tar oil are most generally indicated for treatment and repellent purposes.

BITING FLIES

The small Black fly, *Simulium* sp., was a serious pest in certain areas during the summer of 1943. The bite of this fly leaves a swollen sore for a week or more. When animals are bitten by considerable numbers of *Simulium* they are greatly affected and persistent attacks may result in the death of the host. The reports
show two areas of heavy attacks. These were in the northeastern United States in the Adirondacks area and in the deltas of the Mississippi River. Other areas included Arkansas and the southeastern United States. Since larvae develop largely in stony brooks, control measures are difficult.

The Stable fly, *Stomoxys calcitrans*, and the horn fly, *Syrphona irritans*, were relatively unimportant this year in much of the country as reported by the contributors. A few peaks of abundance developed in different regions but the generally reduced rainfalls served as a check on the larval and pupal stages over much of the area. These flies and others including the house fly, *Musca domestica*, have been partly controlled about dairies by the regular use of standard fly repellents which stupefy a high percentage of the flies on the cows and kill perhaps 30 per cent of them. Repellents with such bases as Pyrethrum and Thanite were found to be safe for application to cattle in recent studies at the Kansas Agricultural Experiment Station. The sprays did not decrease milk production nor increase it. By stunning and killing flies the sprays made handling of cattle easier and doubtless reduced considerably contamination of milk and other foods.

The large Biting flies, *Tabanus* sp., were reported as offering serious problems with livestock throughout much of the country. Following are some of the comments. “Serious pests from May to August” (New York). “Abundant, causing great annoyance to cattle” (Eastern Kansas). “Bad as usual” (Arkansas). “Common in the coastal areas. Bites open way for screwworm infestation” (Texas). “Many species—bad day and all night on mules, horses, cows and swine” (Louisiana).

Tabanids of the *Chrysops* genus were reported from California as serious biters, and vectors of anthrax; from Louisiana as seriously attacking livestock, causing much loss of blood; from Texas where they are severe biters and are known to transmit Anaplasmosis. Because of the swiftness of flight of the adults and of the development of the larval stages in sluggish streams and pools the control of the Tabanidae is difficult.

**CATTLE GRUBS**

In the emergency of furnishing meat and shoes to the armed forces and the civilian population, cattle grubs, or ox warbles (*Hypoderma* sp.) present one of the principal hindrances. The maturing larvae or warbles discolor the prime beef about them and make holes in the skin of the back, the source of the thickest and best leather for shoes.

The summer season of 1943 witnessed another period of heavy infestations of cattle with this parasite. Whether the grubs are on the increase or whether stockmen are becoming more aware of their presence is uncertain, but many contributors place *Hypoderma lineata* as a major parasite throughout most of the regions. Of 20 cows purchased in southern Wisconsin, all but one had five or more grubs. They are reported to be numerous in the Central Plains States. Oklahoma reports showed 1,500,000 cattle infested and a loss to the producers from meat trimming and dockage of hides amounting to $4,500,000. In the Texas report *Hypoderma lineata* is given as a major pest throughout the state with producers’ losses up to $5,000,000. Other regions reporting cattle grubs as very important include Kansas, Idaho and California, with the pest not quite so serious in the southeastern United States. One
authority on the subject reports that 30 per cent of all hides are infested with grubs (Hypoderma sp.) and that this parasite is among the most important in the great plains states.

The complete life cycle and other important phases of the biology of Hypoderma sp. (cattle grubs) were presented to this Association last year by Schwartz (1942) who also recorded a loss of 11,500,000 pounds of beef the previous year.

Treatment. Treatment of cattle for grubs (Hypoderma sp.) has been extensively investigated and the necessary procedures have been determined. Three effective methods are now available: dusting, or washing for smaller herds and spraying for large herds. In the latest communication from the United States Department of Agriculture (September, 1943. E-602) reports are given of recent tests showing that rotenone dusts mixed with tripoli earth or with pyrophyllite are more efficient than those made with talc or sulfur. This is because they penetrate the hair to the skin better and also because twice as many animals can be treated with the same amount of derris. The new formula calls for one part by weight of ground cube or derris (five per cent rotenone) and 2.25 parts by weight of tripoli earth or pyrophyllite with 200-mesh fineness (standard cube-ground, rose tripoli).

These ingredients are thoroughly mixed and applied to the back of the ox to the amount of two to three ounces per animal. The shaker can, with about 15 holes one-quarter inch in diameter in the lid, is held in one hand and the powder worked down in the hair with the other hand. With two men working on opposite sides of a chute about 100 cattle can be treated in an hour.

The formula for the liquid treatment calls for one gallon of water, 12 ounces of derris or cube (five per cent rotenone) and four ounces of soap. The soap is dissolved in warm water; then a paste is made by adding a little soapy water to the powder. Water is then added and the mixture thoroughly stirred. A few holes made in a quart fruit jar with an 8-penny nail makes a suitable container for applying the liquid with one hand while a stiff brush is held in the other hand for working the derris solution down thoroughly to the surface of the skin. Six ounces of wettable sulfur may be used in place of four ounces of soap. One gallon of wash is sufficient to treat from 10 to 20 animals depending upon the size.

A power spray for the control of grubs in larger herds of cattle has recently been prepared by the United States Department of Agriculture. The application pressure recommended is 400-500 pounds for a formula consisting of 100 gallons of water, 7.5 pounds of derris or cube and 10 pounds of wettable sulfur. By use of a fine spray, which is preferred, about two quarts of the solution are sprayed on the back of each animal. With this apparatus cattle can be treated almost as rapidly as they can be put through a chute.

The results of tests show that the cattle grub holes in the skin begin to heal shortly after the grub is killed by the treatment. The directions prescribe that cattle be treated three to four weeks before they are marketed. Under these conditions all but the largest holes heal.

Reports from contributors showed wide application of the hand methods of dusting and wash applications for the control of grubs in cattle, with the power spray coming into use to some extent. The greatest activity in the late summer and fall appeared to be in the South Central States. In Oklahoma, 200,000 cattle were
given the rotenone wash or dust treatment. Arkansas also had large scale treatment of cattle with Rotenone-sulfur dust. In an effort to check a $5,000,000 producers' loss due to cattle grubs in Texas, Rotenone-sulfur treatment was applied as a dust, a wash and a spray. Kansas and Nebraska in November were organizing state-wide drives for cattle grub treatment.

Each member of the Association should do all in his power to promote treatment of cattle for grubs.

**SHEEP NASAL FLY**

Although no outbreaks of sheep nasal fly (*Oestrus ovis*) were reported in the present study, all contributors mentioned this fly as being of rather common occurrence. The majority either gave no treatment or control or stated that usually no control measures are being applied. Two mentioned lysol and one recommended night grazing, and tall forage pastures where practicable. One recommended as a nasal spray a three per cent saponified solution of creosol. There are indications that this fly (sheep bot) is increasing in the central states. Both Kansas and Wisconsin recognize a gradual increase of this important parasite of sheep.

**SHEEP "TICK" FLY**

The sheep "tick" fly (*Melophagus ovinus*) with its powerful proboscis is a damaging parasite. Economic losses were indicated from Idaho and California in the western area with somewhat lessened effects in the southern United States. Some of the Central Plains States consider it a serious problem with from 20 to 30 per cent of the sheep infested. In the northeastern part of the country it is considered the commonest external parasite of sheep, often severe.

Progress has been made in the control of the sheep "tick" fly by the work of Cobbett and Smith (1943) who give evidence of effective killing of "ticks" by a comparatively strong derris dip. In tests involving the dipping of nearly 10,000 sheep, Cobbett and Smith concluded that one dipping in a solution using six ounces of derris or cube powder (containing five per cent rotenone) per 100 gallons of water was effective in killing all "ticks" and in giving protection against reinestation.

Another contribution to the control of the sheep "tick" was made by Schwardt and Matthyse (1943) who described a portable sheep dipping vat and gave complete descriptions for its operation in the field. Many dipping formulae were tested but only three are recommended. All include wettable sulfur at the rate of 100 pounds to 1,000 gallons of water. One dip includes in addition 10 pounds of cube (five per cent rotenone); another dip calls for 10 pounds of fixed nicotine (Black Leaf 155) while the third is without either cube or nicotine. The authors found these dips to be very effective in killing both the "ticks" and the biting louse (*Trichodectes ovis*). The materials for each sheep cost about one cent.

Among the recommendations of contributors for the control of this parasite were rotenone and sulfur dips and also coal tar creosote dips. Owing to the thickness of the wool, dipping appears to be the only effective treatment for this parasite which spends its entire life cycle upon the host.
Cattle lice are considered to be important parasites in all sections covered by this study. In the northeastern portion, they are listed as the most serious external parasite of cattle. In the north central section the biting lice (Trichodectes or Bovicola) give trouble in the winter time in damp barns when the cattle huddle together. In this section there is a tendency not to try to eradicate the lice before cold weather. All contributors in the central plains area stress the abundance of cattle lice under winter conditions. In the south central area even with a milder climate there has likewise been widespread louse infestations, with Oklahoma reporting 1,500,000 animals infested, and retarded growth and development amounting to cattle losses approaching $2,000,000. Sections of the southern United States, including Texas, Louisiana, Alabama and North Carolina, show vast infestations of cattle lice, while the western section lists lice of bovines as important.

Treatment. Some of the contributors feel that louse control on cattle is being neglected. Others report some activity, while a third group report active work on delousing the year around. Those in the northern areas are inclined to use powder treatments such as rotenone-sulfur dust, while in the warmer areas dipping is the principal method, using either arsenical or rotenone-sulfur dips. One contributor (Dove) points out that the short-nosed sucking louse (Haematopinus eurysternus), which is very important on cattle in feed lots, is not controlled by arsenical dips.

A new treatment which is effective in destroying both biting and sucking lice was developed recently by Telford, Longwell, and Munro (1943). The treatment which is applied as a powder consists of a mixture of one part phenothiazine, two parts of sodium fluosilicate, and five parts of flour. It was noted also that sulfur could be used in place of flour as the diluent, and that sodium fluoride could be substituted for the fluosilicate.

Lice of sheep and swine

Little information on sheep lice was submitted by the contributors. One reported that the foot louse, Linognathus is often a serious parasite on lambs with long hair on the lower portions of the legs. Derris dip was recommended for this condition.

The large hog louse, Haematopinus adventiclus was reported from all sections of the country, those in the Central Plains States reporting serious economic losses in some cases. Quite a variety of treatments are being used. At the Nebraska station, where crude petroleum in vats or wallows is preferred in warm weather, care is urged in eradicating all lice from the sows before farrowing, also the inspection of newly purchased stock. In cases of heavy infestations indoors the producers are urged to burn bedding and spray the quarters with crude oil. In some of the northern states the producers are requesting a powder rather than a liquid treatment. Dusting with rotenone and talc is being tried.

Chicken lice

Lice on chickens were quite generally reported. Although seven species are frequently found, only three of these, the head louse (Lipeurus sp.), the body louse...
PARASITIC DISEASES

(Eumenacanthus stramineus), and the shaft louse (M. pallidum), were reported as of major importance. Like other lice, the entire life cycle is spent upon the host. What appears to be the most generally used method of control is to paint the top surface of the roosts with nicotine sulfate (Black Leaf 40) one half hour before the flock goes to roost. Heat from the bodies of the chickens vaporizes the material which rises through the feathers and kills the lice. The nicotine sulfate is applied undiluted with oil can, brush, or other means. Only a narrow line of the solution is needed on each roost. To determine whether repeated treatment is needed the necks and heads of some of the birds should be examined a few days after the first treatment. When but a few birds are to be treated for lice some prefer the use of sodium fluoride by the pinch or dip method.

MITES

The common mange or scab mites, Sarcoptes, Psoroptes, Chorioptes and Demodex seem to be almost entirely lacking in cattle and sheep over the great southern United States section; also in the western section of the country. Scattered cases are reported from the central plains area. Only the northeastern section reported a limited outbreak in 1943. These were the sheep scab mites, Psoroptes communis. Prompt dipping in one to 20 commercial lime sulfur solutions at 105° F. brought the outbreak under control.

Sarcoptic and follicular mange in swine, while reported from nearly all areas was not designated as important except in the central and southern plains areas where several thousand cases of sarcoptic mange occurred. The control and treatment of sarcoptic mange in swine is much like that for the control of hog lice.

The mites of poultry, according to the reports from contributors to this study, are fairly well under control. On the west coast there was an outbreak of tropical fowl mite, Liponyssus sp., on adult turkeys. The male fowls were more affected than the females. As the flock was on range without houses, treatment was difficult, but good results were obtained by sanitation and the use of nicotine sulfate (Black Leaf 40) which was smeared on the feathers in small amounts.

TICKS

Ticks of food-producing animals appear to be under pretty fair control over much of the areas covered in this study. The southern cattle tick, Boophilus annulatus, or Margaropus annulatus, seems to be very largely eradicated. There were sporadic occurrences of the spinose ear tick (Ornithodorus megmini) in different regions, but nothing approaching an outbreak. Only the lone star tick, Amblyomma americanum, appeared to be numerous on livestock in any section. In the south central plains area these blood-sucking ticks were reported by several contributors as being very abundant.

Following are recommendations for the control of spinose ear ticks: cotton seed oil, pine tar oil, and benzol applied with a swab at intervals of two or three weeks. Arsenical dips are recommended for the control of the lone star tick, but because of the multiple hosts of this tick it is very difficult to eradicate.
SUMMARY

1. With the aid of field and laboratory workers and of recent literature, information was assembled on the occurrence and control of important external parasites of food-producing animals.

2. Larvae of the screwworm fly, *Cochliomyia americana*, were among the most important external parasites in 1943 because of their tissue destruction in skin wounds, especially from barbed wire, dehorning, castration, parturition and separation of the navel cord from the lamb or calf.

The widespread infestations of screwworm fly larvae in livestock in the Central Plains States south to a coast to coast strip from the Atlantic to the Pacific included several million serious cases, many of them fatal.

A new treatment has been developed which combines a killing agent, benzol, with a repellent one, diphenylamine, together with turkey red oil and lamp black, the mixture being known as Smear 62.

3. Of equal or greater importance to the problem of national meat and leather supply are the larvae of the warble fly, *Hypoderma lineata*, which develop as grubs beneath the skin of the back. The developing larvae, which bore holes through the skin for respiration and develop in cysts that discolor adjacent tissues, result in serious losses of leather and beef.

Rotenone-sulfur treatments of the backs of infested cattle are being applied locally and in state-wide drives.

4. Progress is reported in the control of the sheep "tick" fly, *Melophagus ovinus*, by the use of a portable field vat for dipping sheep in aqueous solutions of derris (five per cent rotenone), of derris and sulfur, or of fixed nicotine, Black Leaf 155.

5. Progress is reported also in the control of biting and sucking lice of cattle; the new treatment, applied as a powder, consists of phenothiazine, sodium fluosilicate and flour.

6. An outbreak of the sheep scab mite (*Psoroptes*) in the northeastern United States was brought under control by prompt dipping in commercial lime sulfur solutions at 105° F. Another outbreak of mites (Tropical fowl mite, *Liponyssus*), which occurred among range turkeys in California, was brought under control by smearing on the feathers small amounts of nicotine sulfate (Black Leaf 40).

7. The results of the study indicate that the infestations of external parasites of food-producing animals in 1943 reduced by millions of pounds the volume of beef, mutton, pork and poultry products available for the armed forces and the civilian population.

LITERATURE CITED


REPORT OF THE COMMITTEE ON MISCELLANEOUS TRANSMISSIBLE DISEASES

ADOLPH EICHHORN, Chairman, New York City; C. E. FIDLER, Springfield, Ill.; L. M. HURT, Los Angeles, Calif.; HADLEIGH MARCH, Bozeman, Mont.; JOHN W. GEORGE, Jefferson City, Mo.; SAM McCue, Albuquerque, N. Mex.

In presenting this report, the members of the Committee on Miscellaneous Transmissible Diseases regret that it was necessary for Dr. Eichhorn to give up the chairmanship of the committee. The acting chairman took over the responsibility at a rather late date, and has attempted to make a survey of the field to be covered by this committee. Much of the information on which this report is based was very kindly supplied by the Pathological Division of the U. S. Bureau of Animal Industry.

The subject of miscellaneous transmissible diseases covers a wide and rather indefinite field, including bacterial and viral diseases, and diseases caused by protozoan and metazoan parasites. For purposes of this report, the field is limited definitely by the existence of committees on three specific diseases—tuberculosis, brucellosis, and rabies. It is also limited in an indefinite manner by the work of the committees on swine diseases, poultry diseases, cattle diseases, and parasitic diseases. As the time available for preparing this report has been quite limited, it has not been possible to establish liaison with these other committees, and there may be some overlapping in the material covered. However, any references in this report to material in the fields of the other committees will be in the form of brief general comments.

Since the last meeting of the Association, another year of world war has passed, without any significant increase in the incidence of transmissible diseases in the United States, and without the introduction into this country of any new infections, as far as the committee is aware. This condition seems to constitute evidence that the veterinarians and livestock sanitary officials of the United States have been highly effective in the work of disease control, although subjected to the stress of increased work, shortage of help to properly care for livestock, abnormal livestock movements, deranged market and feed conditions, and unusual difficulties in controlling importations. However, it is our belief that there are some non-spectacular and insidious increases in some transmissible diseases, such as swine diseases for instance, against which we must be on the alert.

FOOT-AND-MOUTH-DISEASE

According to data compiled for us by the U. S. Bureau of Animal Industry, from the limited information available from foreign countries under present conditions, there is no significant change in the foot-and-mouth disease situation, unless changes have occurred in enemy countries, from which no reports are received. The continent of Africa is infected territory, except for the Union of South Africa, which has remained free of the disease. Southern and eastern Asiatic countries from which reports have been received show about the usual incidence. Reports from Sweden indicate freedom from foot-and-mouth disease since December, 1942. Switzerland
is reported to have experienced a slight incidence of the disease early in 1943, following a period of practical freedom in 1942. In the British Isles, where there were extensive outbreaks in 1942, there have been relatively few infected herds in 1943. Foot-and-mouth disease has persisted with about the usual incidence in South America, except in Colombia and Venezuela, which were reported free of the disease. All countries of North and Central America have remained free of foot-and-mouth disease, though enforcement of regulations covering potentially dangerous products from infected territory has become increasingly difficult under existing conditions. Wartime emergencies inject serious problems into the picture. Livestock producers and sanitarians must continue to take measures to prevent any international political agreements which would allow any relaxing of the necessary quarantine regulations controlling the importation of meat products.

**EQUINE ENCEPHALOMYELITIS**

Records compiled by the Bureau of Animal Industry indicate a slight increase in equine encephalomyelitis over 1942. The number of cases officially reported to the Bureau up to November 16 was 3,945 in 34 states, as compared with 3200 in the same period in 1942. No new foci of infection with eastern type virus have been discovered, though few determinations of virus type have been made. The Bureau again stresses the point that virus samplings have been entirely too few in number. This committee suggests that the livestock sanitary authorities attempt to stimulate the collection of material for virus typing, although it is realized that from the standpoint of the man in the field it is difficult to obtain suitable brain specimens.

The U. S. Public Health Service reports 604 cases of human infectious encephalitis for the first 9 months of 1943, but none of these was reported as being of the equine type. We do not have information as to the extent of determinations of the virus type.

**DOURINE**

The Bureau of Animal Industry reports that dourine of horses and mules is under effective control, in the small area in the southwest in which it still exists. No cases were discovered in new territory, and the number of animals reacting positively to the complement fixation test up to November 19 has been only about 1:10th of the total for 1942.

**ANAPLASMOSIS**

Anaplasmosis continues to gradually invade new territory as it moves northward, and in some instances has been the cause of rather serious loss to cattle owners. As recovered cattle remain carriers of the causative organism, and as experiments have indicated that ticks, horseflies, and mosquitoes may act as vectors, the problem of control is difficult. Veterinarians in areas where the disease is rare or has not been diagnosed should be on the alert for the appearance of this disease, the cardinal symptom of which is a marked anemia. Apparently the most practical control procedure in newly infected territory is to attempt to remove all infected cattle from the range involved as soon as possible after a diagnosis is made, and dispose of them for slaughter if they recover.
SWINE ERYSIPelas

Although the mention of swine erysipelas invades the field of the committee on swine diseases, this disease deserves mention as a major problem, from the standpoint of its rather rapid recent increase in some areas where it has been seldom recognized in the past. Differential diagnosis and the question of the possible simultaneous occurrence of swine erysipelas and hog cholera in the same herd are causing difficulties for field men in control work.

MASTITIS

Bovine mastitis has been discussed in some detail in the 1941 and 1942 reports of this committee, and becomes of increasing importance as the war program demands increasing supplies of milk products. The discussion of this disease was made a major item on the program of the Conference of Research Workers in Animal Diseases, and undoubtedly will be covered thoroughly by the special committee on cattle diseases.

LISTERIOSIS

It might be appropriate to call attention to the increasing reports of the occurrence of listeriosis in sheep and cattle.

GENERAL CONSIDERATIONS

This committee has no constructive recommendations to make at this time, but in considering the factors involved in the control of the spread of transmissible diseases in general, two problems, neither of which is new, are of increasing significance in the more recently developed agricultural areas of the country. One of these is the problem presented by the livestock auction sales, which are rapidly invading new territory in the West, and, as has been recognized for many years, constitute a serious complication in the control of infectious diseases.

Another problem which applies particularly to swine, but which also has an indirect bearing on human and cattle disease, is that of the elimination of the feeding of raw garbage, which is a potential source of infection in the case of several infectious diseases, including trichinosis, with its relation to human health. The feeding of hogs has recently increased to a large extent in areas outside the corn-belt, and, with that rapid increase, the question of the control of swine diseases acquires added importance.

Finally, the committee emphasizes the obligation of all veterinarians to exert special vigilance under present unsettled conditions in making careful diagnoses of all livestock conditions which may be transmissible, and of instituting all possible control measures.
REPORT OF THE REPRESENTATIVE TO THE INTER-ASSOCIATION COUNCIL OF ANIMAL DISEASE AND PRODUCTION

BY MARK WELSH, D.V.M.

Ridgewood, New Jersey

It has been recognized for some years past that the work of several organizations interested in livestock production frequently duplicate their efforts. An individual worker, studying a given problem, makes his attack on the basis of his specialized training and experience. Many of our major problems are subdivided and a given segment is investigated from one point of view only and the results, of necessity, are inconclusive. It is, of course, obvious that more valid results might be obtained if we initiated the simultaneous study of the problem as a whole by men having complementary training rather than having these individuals work independently.

It was with this general objective in view that a member was appointed to serve on the Inter-Association Council of Animal Disease and Production from each of the following organizations:

- American Society of Animal Production
- American Dairy Science Association
- American Poultry Science Association
- American Veterinary Medical Association
- United States Live Stock Sanitary Association

This is the first annual report to be made to this association by your representative on the Inter-Association Council.

Preliminary meetings were held to discuss the work of the Council, its scope and objectives, and in May of 1942 an official meeting of the representatives was held following their appointment by the member associations. Meetings of the Council were held in August of 1942 during the annual convention of the American Veterinary Medical Association; in December, during the meeting of the American Society of Animal Production and the United States Live Stock Sanitary Association; and a special meeting was held in New York during July, 1943. For the guidance of the Council in its activities, a set of rules and regulations were prepared and a copy placed on file with the secretary of each member association.

The first endeavors of the Council were toward the development and preparation of well co-ordinated programs which would assist the livestock owners in meeting the production goals requested of them. Several consultants, highly regarded in their own fields, were requested to prepare workable educational programs on the various classes of livestock. These consultants were selected with the aid of the officers of the respective member associations, and the programs have been developed for virtually all of the classes of livestock. The Council now has under consideration various ways and means of getting these published and distributed, and put into action. It is hoped that this can be completed in the very near future.

Within recent months, there has been organized what is known as the National Live Stock Conservation Program. The Council has extended to this group an offer to cooperate with it to the fullest extent. The indications are that an arrange-
ment will be made which will enable the Inter-Association Council to render valuable assistance to the important undertakings of this program.

In the original resolution, which resulted in the formation of the Inter-Association Council, it was proposed that the National Research Council be requested to consider the appointment of a committee on animal health. The National Research Council saw fit to act upon this suggestion, and a committee composed of veterinarians, animal nutritionists, and animal production men has been appointed. A program concerned with research problems is well under way.

At the invitation of Dr. E. C. Auchter, Director of the Agricultural Research Administration of the U.S. Department of Agriculture, a conference was held in April of this year in Washington, D.C. Representatives were invited from the American Veterinary Medical Association, the United States Live Stock Sanitary Association, the National Assembly of Chief Live Stock Sanitary Officials, and the Inter-Association Council on Animal Disease and Production. The purpose of this conference was to discuss increased service by different branches of the Department of Agriculture concerned with the livestock interests of the nation, particularly during the existing crisis.

Many of the problems preventing the improvement of animal welfare and productive efficiency can be solved only by a pooling of the knowledge possessed by those engaged in different fields of endeavor, such as nutrition, farm management, genetics, sanitation, and veterinary medicine. Consequently, the Council felt that one of the biggest services that could be rendered the livestock industry would be the preparation of a report on the status of livestock production and disease control, which would embody the viewpoints of the various ones working from different aspects. Specific recommendations will be made for improving productive efficiency and rendering more effective service to the livestock industry.

The Inter-Association Council has been hampered in its work through a total lack of appropriated funds. The various members defray their own expenses, but there are certain postage and other expenses, and it is for this reason that the following motion was adopted at a recent meeting:

"Therefore, it is hereby moved that the respective associations be requested to provide an annual contribution to the Council on the basis of $25.00 for the first 500 members and $0.01 for each active member in excess of 500, with the monies to be used for incidental expenses such as postage, mimeographing, clerical help, and the like; such funds to be made available to the Council through the respective representative by October 1, of each year, beginning with the current year."

The Council has now had more than a year to study the large and involved problems concerned with animal disease and production. It has had the privilege of meeting and conferring with the officials of various national organizations, federal and state officials, outstanding scientists, and livestock and poultry producers. It is very apparent that co-ordination of effort is vitally needed and the means must be provided whereby different groups, agencies, and individuals may jointly discuss their problems, formulate plans, and initiate action. It is the hope of the Inter-Association Council that it can, in part, be instrumental in providing some of the facilities needed.
TWELVE YEARS OF VESICULAR EXANTHEMA

By C. U. Duckworth, D.V.M., Administrator

AND

B. B. White, D.V.M., Swine Disease Specialist

Division of Animal Industry, California Department of Agriculture

Veterinarians connected with the Division of Animal Industry of the California Department of Agriculture have for the past twelve years been concerned with a serious disease problem in hogs which, though having an humble beginning, has throughout the years taken its place among the major swine disease problems in California. It appears at the present time, that vesicular exanthema is now well established in garbage-fed herds of swine, is endemic on many of our larger garbage-feeding premises, and in all probability will continue to occur as long as raw garbage is fed.

The disease had its beginning on April 23, 1932 (1), at which time it was reported that garbage-fed swine located on a ranch near Buena Park, Orange County, were affected with a condition suspected of being foot-and-mouth disease and which was subsequently so diagnosed. Strict quarantine measures were immediately adopted and the hogs slaughtered as is the usual procedure in occurrences of foot-and-mouth disease. Eight days later the disease appeared near Bellflower, Los Angeles County, a distance of some fifteen miles from the site of the original infection, again in garbage-fed swine. The third infection in 1932 was discovered in garbage-fed hogs in San Bernardino County some thirty miles distant. The report of these three occurrences states that there was a definite history of contact between all three breaks, however, it was not possible to determine the source of the original infection.

Infected swine herds, including a small herd of cattle and goats exposed on one of the ranches, were slaughtered. The total appraised value was placed in excess of $203,000.00 with the State's share of the indemnity being over $101,000.00. Quoting from the official records of this break the following interesting statement is noted:

"The disease in the present outbreak affected only swine. Usually foot-and-mouth disease also attacks cattle, sheep, and goats. The virus of this outbreak failed to affect these animals, some of which were exposed. All swine involved were garbage-fed."

Thus was recorded the first occurrence of a virus disease of swine which later was to be termed vesicular exanthema.

The second occurrence of vesicular exanthema was on March 20, 1933, at which time a garbage feeder in San Diego County noted peculiar symptoms in his hogs, notably lameness and sore noses. Again immediate steps were taken to establish a diagnosis, to employ strict quarantine measures, to slaughter infected and exposed animals on the ranch, and to bring under control another outbreak of foot-and-mouth disease. The disease developed in hogs on three additional ranches in the
vicinity of the original one, the last on April 3; in all, 5,533 hogs on four ranches were destroyed in that year. The following quotation from official records of this occurrence (2) is significant in view of later developments:

"As it appeared in hogs, the condition was a picture typical of foot-and-mouth disease, but due to the confusion presented by the other test animals, no diagnosis was made. Everything was handled as in an outbreak of true foot-and-mouth disease, with the exception of burdensome and costly quarantines. The men on the ground felt that as they were unable to diagnose foot-and-mouth disease they were not justified in instituting quarantines which would have cost the people of the State large sums of money due to embargoes that would be laid down against us to protect the rest of the State and country. Because this condition in swine presents symptoms typical of foot-and-mouth disease, great care must be exercised should similar conditions occur in the future, for a mistake in diagnosis might have serious consequences. In handling this situation it would have been easier for those in charge to diagnose foot-and-mouth disease and no one could have questioned the diagnosis. The hard way was taken, however, and the people of California were saved large sums of money."

The quarantines mentioned in the above quotation refer of course to area quarantines and are not to be confused with regulatory procedures inaugurated on individual ranches which were naturally of a most stringent nature.

The "confusion" mentioned in the report refers to the fact that infective virus-containing material from affected hogs would not produce lesions of foot-and-mouth disease when inoculated into cattle and guinea pigs but did infect horses. Some of this material was sent to the Bureau of Animal Industry in Washington, D. C. for further study from whence some virus was in turn forwarded to Professor Waldmann, Riems Research Institute, Germany, for identification. Later reports from Waldmann (3) indicated that this virus was unlike any of the three strains of foot-and-mouth disease virus encountered in Europe.

Thus is recorded the second occurrence of the disease in question.

The third occurrence was in 1934, this time in the north central portion of the State a distance of some 400 miles from the sites of the two previous breaks. During this year the disease appeared in Santa Clara, San Mateo, Alameda, Contra Costa, and San Joaquin counties in the central portion of the State and later in Los Angeles and San Bernardino counties in the South. A total of 31 garbage-feeding premises and 95,000 hogs were involved during that year. Again, as in the two previous years, the infection was readily transmissible to swine, exposed swine developing lesions typical of foot-and-mouth disease. Horses, as also in the case of the two previous breaks were mildly susceptible to the virus and cattle and guinea pigs entirely refractory.

The United States Bureau of Animal Industry now declined to participate in the payment of indemnities which rendered eradication measures by the slaughter method impossible since State indemnities under California law are contingent upon Federal participation. Telegraphic advice from the Chief of the Bureau of Animal Industry relative to disposition of affected animals was to "handle the condition in the same way that vesicular stomatitis is handled in the Middle West."
Up to this time veterinarians connected with the United States Bureau of Animal Industry in California had been participating in quarantine, slaughter, and eradication measures. When, however, this advise was received from the Chief of the Bureau, all Federal participation promptly stopped. Quarantines established by the State Department of Agriculture were however rigidly enforced, infected ranches being held under quarantine until all evidence of the disease had subsided. During the 1934 break guards were placed on premises under quarantine. It was necessary that garbage trucks leave the premises daily for garbage collections, however, trucks were sprayed with a lye solution upon departure and steps were taken to insure that truck drivers and ranch attendants did not contact other hog ranches or livestock premises.

In spite of such stringent quarantines the infection in 1934 appeared, as previously stated, on 31 premises.

Dr. J. Traum, University of California, reported (4) in detail at the Twelfth International Veterinary Congress in August, 1934, upon the clinical manifestations of the disease encountered during the 1932 and 1933 breaks. At this time Traum suggested the name “vesicular exanthema.”

In 1935, there were encountered mild recurrences of the infection on four of the premises that had been under quarantine the preceding year. On these four premises, 5,500 hogs were maintained.

Crawford (5) working with virus harvested during the 1933 and 1934 breaks, reported that he had definitely identified four strains of the virus, each of which appeared to be an immunologically distinct type. Crawford attempted to determine the infectiousness of the virus by certain experimental procedures involving pen exposures. He concluded that experimentally this disease was found to be communicable as a result of direct infection but really only slightly so as a result of indirect infection. He further concluded that studies of vesicular exanthema suggest a closer relationship to vesicular stomatitis than to foot-and-mouth disease but definitely indicate vesicular exanthema to be a separate though closely related disease.

This conclusion that vesicular exanthema infection is only slightly communicable under indirect or natural conditions has, though not born out by field experience, we believe, resulted in a tendency among many veterinarians who have not worked with vesicular exanthema to shuffle it into the discard as an unimportant or a benign affair that should not occupy too much of one’s time or attention. On the contrary, our experience has shown that when this virus gains access to susceptible hogs the disease may spread very rapidly through the herd and in many instances the incidence of infection in a given group is 100 per cent.

In 1936 vesicular exanthema was encountered on 14 garbage premises. These 14 premises maintained a total of 19,000 hogs. Following release of these premises from quarantine, hogs were moved direct to slaughter on permit which was issued after inspection of all hogs on the premises. Slaughter shipments were not allowed to move through public stock yards and sales pens. This procedure was continued for a period of some 3 months and discontinued only after it appeared that the infection had completely disappeared.

From June, 1936, until December, 1939, a period of three and one-half years, no
known occurrence of vesicular exanthema was recorded. During this period, periodic routine inspections were made on garbage ranches in the major garbage feeding districts. These periodic inspections by no means included all the garbage feeding premises in the State, however, we believe this work was of sufficient scope to have detected any major occurrence of vesicular exanthema had the infection been present.

In December of 1939 the disease made its sixth appearance. This time on such a large scale and involving such a wide area throughout the State that it reached truly epizootic proportions. From December, 1939, until June, 1940, the infection had appeared on 123 premises, in 25 counties, and had involved some 222,500 hogs or approximately one-fourth of all hogs in the state.

As has been the procedure at the beginning of each of the previous five breaks, horses, cattle, guinea pigs, and swine were inoculated with fresh vesicular material taken from affected hogs in order to establish a proper diagnosis. After the initial series of animal inoculations were completed, and a definite diagnosis of vesicular exanthema was established it was necessary to place calves on each additional premise where the infection occurred as we had no way of knowing for example that a new break near Fresno was vesicular exanthema and not foot-and-mouth disease simply because the one near San Francisco the previous week had been.

Since 1940 we have discontinued recording individual breaks by specific number and for the sake of our official records record the known occurrences of this infection by calendar years. We have been continually confronted however, with the infection on many of our major garbage feeding premises since that time.

Our records show that out of the total of 517 herd infections that have been encountered during the 12 year period, there have been only 35 outbreaks in other than garbage fed herds. These 35 consisted of 21 ranch infections among grain fed hogs and 14 in stock yards and packing house pens. In most of these 35 cases, we have been able to establish definite contacts between the individual herd in question and an infected garbage herd. In not a single instance has there been a recurrence on a non garbage feeding ranch premise. Therefore we feel justified in the belief that vesicular exanthema is primarily a garbage hog problem and is probably perpetuated through the medium of infected raw pork scraps in the garbage. In this connection it is of significance to note that we have in California 16 State institutions at which swine herds are maintained on raw garbage accruing at the particular institution. Most of the pork supplies at these institutions are produced and slaughtered on the premises. None of these herds have to date experienced an outbreak of vesicular exanthema.

As previously stated, the disease has appeared in public stock yards and slaughtering establishments on several occasions. In May of this year for example, it appeared in the South San Francisco Stock Yards and two adjacent packing house holding pens. In one instance 2,000 eastern hogs imported for direct kill contracted the infection. Foot lesions were so severe among these animals that they could not be driven up the chutes for slaughter. Before this infection was reported, however, numerous lots of hogs in the initial stages of the disease were slaughtered and condemnations resulted from the fact that carcasses on post mortem showed infiltrations with a gelatinous edematous exudate characteristic in many cases of the early stage of vesicular exanthema infection and while these condemnations were made on a
diagnosis of swine erysipelas we feel that they were primarily due to vesicular exanthema.

These establishments were operating under Federal Meat Inspection Service which is now, as you know, under the jurisdiction of the Food Distribution Administration.

After proper field diagnosis had been made and calf inoculations conducted in order to exclude foot-and-mouth disease, the affected swine were segregated until recovery and the entire yards were thoroughly cleaned and disinfected.

Financial losses to one packing firm alone amounted to over $20,000. This loss was partially due to the fact that when fat hogs break with vesicular exanthema, the shrink in weight is usually in excess of 50 pounds per head. Add this to the death loss accruing from secondary conditions such as pneumonia and hog cholera which frequently occur when slaughter hogs are held at stock yards or slaughterers' holding pens for 30 days or more, and one can readily visualize the economic importance of vesicular exanthema.

California slaughterers normally import approximately 55 per cent of their slaughter hogs from midwest and intermountain states. Our total pork production in California is only 30 per cent of our consumption including all classes of pork products.

We are informed that tariff restrictions on dressed pork are such that the dressed product cannot be shipped to California from corn belt points and compete with pork derived by live hog shipments. It was clearly obvious, therefore, that in order for western packers to continue to operate efficiently, the control of vesicular exanthema was necessary at least insofar as centralized markets and packing house centers were concerned.

Vesicular exanthema is a local problem in that the infection has not been identified outside our own State. Despite this fact Bureau of Animal Industry cooperation was requested for participation in a state-wide system of ranch inspections and shipping permits on all garbage hogs before movement to slaughter. Such a system had been tried on 2 or 3 previous occasions on a local basis without noticeable improvement in the general state-wide vesicular exanthema situation. We have in California approximately 340,000 garbage-fed hogs which are spread over an area of 750 miles in length, hence the mechanics of any attempt to regulate hog shipments entail considerable personnel and expense. Nevertheless we are now attempting to inspect garbage hogs within 24 hours of the time they leave ranches for slaughter and requiring that they move only on permit which is issued after inspection has disclosed the fact that no infection exists on the ranch of origin.

This system has been in effect only four months and it is too early to determine whether or not it is doing any good as a control measure. We intend to continue these restrictions in the event future experience proves them to be worthy of the effort.

Throughout the past 12 years we have repeatedly recommended that funds be made available for additional research on vesicular exanthema as it is felt that our knowledge of the disease is so meager that any control attempts are merely groping in the dark.

We have seen vesicular exanthema shrink fat hogs, we have seen it cause consider-
able loss through post mortem condemnations, we have seen it result in loss in weight and condition on ranches to the extent that 30 additional days of feeding time may be required before market weights are attained, we have seen it cause the loss by death of many baby pigs, we have seen it produce abortions in pregnant sows; therefore, we are of the firm opinion that this disease is one which warrants study and investigation in order to effect control or eradication.

REFERENCES
VESICULAR STOMATITIS IN SWINE

By H. W. Schoening, V.M.D.

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There are three vesicular diseases of animals which are of importance to the livestock industry of the United States. These are foot-and-mouth disease, vesicular exanthema, and vesicular stomatitis.

The danger of foot-and-mouth disease to the livestock industry and the damage that it causes when it becomes established are well recognized. The other two diseases are not only of importance as a cause of loss to the livestock industry, but also in their great similarity and the need for their differentiation from foot-and-mouth disease. All are caused by viruses, and the similarity of symptoms and lesions produced are quite striking—so much so that the only sure means of a definite diagnosis is by animal inoculations and a study of the disease so produced.

In making a differential diagnosis by animal inoculation, horses, cattle, swine, and guinea pigs are used. The virus of foot-and-mouth disease produces lesions in cattle, swine, and guinea pigs. The virus of vesicular exanthema produces lesions occasionally in horses, and these are of a slight nature; not at all in cattle; regularly in swine; and not at all in guinea pigs. The virus of vesicular stomatitis produces lesions in horses, cattle, swine, and guinea pigs. Inoculations are usually made by scarification of the dental pad or the tip of the tongue, into which the virus is gently massaged. With foot-and-mouth disease virus in the susceptible animals, a local lesion forms at the site of inoculation, followed by a rise in temperature and the subsequent appearance of vesicles in other parts of the mouth, on the feet, and, in cattle, sometimes on the udder. With vesicular exanthema, the disease appears at the site of local inoculation in the susceptible animal, followed usually by the appearance of secondary vesicles. In vesicular stomatitis the lesions appear at the site of inoculation but are very rarely followed by secondary vesicles. In other words, the disease remains localized, although there may be a tendency for it to spread from the site of inoculation to tissues adjacent to the site of inoculation but there is no generalization as evidenced by the formation of secondary vesicles except in very occasional cases.

Foot-and-mouth disease is produced regularly by intramuscular and intravenous inoculations into susceptible animals. The same is true for vesicular exanthema. With vesicular stomatitis, the injections of the virus into susceptible animals by intramuscular inoculation has so far failed to produce the disease, and that fact can be utilized in arriving at a differential diagnosis between vesicular stomatitis and foot-and-mouth disease.

Vesicular stomatitis in the past has not been reported as a natural disease in swine, although swine can be infected by artificial inoculations, either by scarification of the snout or by an intravenous inoculation. When the disease is produced in swine by a local inoculation, the disease usually appears at the site of inoculation.
only, although occasionally there may be evidence of generalization as shown by the involvement of one or more feet. By intravenous inoculations of swine, however, the disease makes its appearance in a very frank and definite form and lesions may be found on all four feet and on the snout.

In August 1943 a vesicular disease of swine appeared at a hog cholera serum plant in a group of animals which had been hyperimmunized 12 days before. The disease subsequently appeared in a group of immune hogs which had been on the premises a few days. The disease was definitely determined to be vesicular stomatitis by animal inoculations, since horses, cattle, swine, and guinea pigs readily developed definite lesions after local inoculations. Cattle and swine inoculated intramuscularly with the virus failed to develop the disease. From the available history it appeared that the infection at the plant was introduced into the hogs through intravenous inoculation of the hypering virus, the development of the disease in the unhypered hogs appeared to have been the result of close contact with the hyperimmunized animals. The disease was severe in its effects. The animals were lame, off feed, and many developed secondary infections with resultant loss of the claws. Others developed a septicemia and died or were destroyed.

The exact source of infection has not been determined and one possible explanation is that a pig used for the preparation of hypering virus was carrying the virus of vesicular stomatitis in its blood at the time it was bled out, thus contaminating the hypering virus. While it was possible for this to have happened, it would be the first instance of the demonstration of vesicular stomatitis as a natural disease in swine. Inquiry into the farms from which the virus pigs were purchased failed to reveal any evidence of vesicular stomatitis in any species of animals.
PIG MORTALITY

BY H. C. H. KERNKAMP, D.V.M.

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Within the past few years the matter of pig mortality appears to have attracted the attention and aroused the interest of a greater number of persons than at any previous time. The need for a greater amount of pork and pork products for human food and a favorable price for marketable hogs, contributed to this position.

Pig mortality, as the subject will be treated, pertains to the loss through death of young swine within a particular period of chronologic time. The period, for the most part, is the first two postnatal months. This is the nursing period and con-

| TABLE 1.—Pig mortality statistics, R. S. B. laboratory, 1938-1942 inclusive |
|---------------------------------|-----------------|-----------------|-----------------|-----------------|
| STATION | Farrowed in Spring | | Farrowed in Fall | |
| | No. born living | No. | % | No. born living | No. | % |
| A | 3,135 | 1,335 | 42.5 | 2,103 | 520 | 24.5 |
| B | 4,964 | 1,738 | 34.8 | 2,426 | 746 | 30.7 |
| C | 1,532 | 407 | 26.5 | 1,667 | 482 | 28.8 |
| D* | 742 | 203 | 27.3 | 603 | 115 | 19.0 |
| E | 6,118 | 1,325 | 21.6 | 1,443 | 240 | 16.5 |
| F | 1,992 | 631 | 31.6 | 298 | 135 | 45.0 |

* (1940-1942 inclusive).

forms to the length of time allowed by most technical and professional swine husbandmen engaged in the study and investigation of breeding and feeding problems fundamental to the swine industry. Practically all of the life and death records used in the statistical treatment of this report, are for the period specified above.

Vital statistics dealing with pig mortality are contained in reports from the Regional Swine Breeding Laboratory. The reliability of these data make them especially suitable for a study of death rates for a particular age group of swine—the nursing period—in a fairly large population. A summary of the data reported by each of the cooperating State Experiment Stations for the six years, 1938 to 1942 inclusive, are shown in Table 1. This series gives us a grand total of 18,483 pigs farrowed in the spring seasons of the years specified and 8,540 farrowed in the fall. Of those born in the spring, 5,639 (30.5%) died between the time of birth and the time of weaning on the 56th day. The results in the fall litters was 2,238 (26.2%) dead before the weaning date. It is only fair to state that the swine under investigation in the laboratory are submitted to inbreeding and crossbreeding practices of

1 Paper No. 2133, Scientific Journal Series, Minnesota Agricultural Experiment Station.
lesser or greater intensities, which in some way, may influence the life and death statistics.

Fairbanks (1), speaking before this association in 1942, stated that of pigs alive at birth, a death loss during the nursing period approximating 37 percent, was calculated from the birth and death data on the progeny of 7,415 sows kept by various experiment stations in this country and at the Department of Agriculture in Beltsville, Maryland. The mortality rate from birth to weaning of the progeny of 518 sows at the Minnesota Station showed a loss equal to 24.1 per cent.

It will be seen that the loss among newborn and suckling pigs in droves or herds on which complete birth and death records are kept is of an order approximating 25 to 30 per cent. Are these values in line with what is occurring under general farm conditions? We believe they are not far off. In this connection, the following should be of interest. Late in January of the present year, myself and two associates (W. L. B. and M. H. R.) were meeting with a group of businessmen from a city in Minnesota that is located in a territory where swine production is a principal agricultural enterprise. In the course of conversation, the matter of pig mortality was discussed and we made the statement that approximately one-third of pigs born are lost by death before reaching two months of age. These gentlemen registered surprise at this and expressed a doubt as to whether the losses among newborn and suckling pigs was of that magnitude on the farms of that particular territory. The sincerity of their interest is attested by the fact they agreed to lend assistance to any reasonable plan that would yield more precise information on this subject from farms of that area. With the assistance of Professors Winters and Comstock of the Division of Animal Husbandry of the University of Minnesota, a plan was worked out that would furnish a reliable sampling of birth and death rates on young swine for a given geographic area. The area in this case was two townships. It provided that every 25th farm of those registered with the AAA program would be selected as a farm for collecting the required data. Thirty-five farms were included in the survey. The size of the farms selected and the equipment for swine production available represented average conditions of farms in that territory. The progeny of 291 sows were surveyed. They farrowed 2,332 pigs, a loss equal to 26.8% occurred between the time of birth and the time when the first of the spring farrowed pigs were marketed for slaughter (which is a longer period of time than is covered in our other data). At this writing, a complete breakdown of the data was not available but, an inspection of it shows that the loss was greatest during the first few weeks of the pigs' lives. This is a very small sector of the swine-producing business of the country and a record for but a single season. It is hoped other such surveys can be made.

Turning for the moment to the realm of speculation. If we use conservatively values obtained in these studies of pig mortality and apply them to records in the Bureau of the Census on the swine population, a more concrete idea of an actual number for pigs lost early in life might be gained. Using records for 1935 (2) on number of sows and gilts to farrow (slightly over 6 million) and then assuming that each sow and gilt farrowed five living pigs, the total number farrowed would approximate 30 million. If 25 percent failed to reach 60 days of age, this would mean that the life span for seven and one half million pigs was terminated early.
An investigation of causes for the loss of pigs between time of birth and weaning was undertaken at the Minnesota Station a few years ago. In the first series studied, there was a total of 2,335 pigs born living. Of this number, 800 (34.2%) died within 90 days of birth. Thirty-four per cent of the deaths occurred in the first week of life, 64 per cent during the second to eighth weeks inclusive and 2 per cent from the ninth to twelfth weeks. In a second series, involving 199 (35.5%) of the total number farrowed, the distribution of the loss during the 56 day nursing period can be divided into four categories. Forty-six per cent of the deaths occurred the first week, 35 per cent the second week, 16 per cent the third week and only 3 per cent throughout the remainder of the period. Magnusson (3) writing about pig mortality in Sweden says, "The average mortality varies in different age groups. In general, it is greatest during the first week; there is another period of weakness at about three weeks, and another at the beginning of weaning at six weeks." Doyle (4) records an experiment, the object of which was a study of the death loss in newborn and suckling pigs. He reports that 44 per cent of the pigs farrowed by sows receiving a low protein intake during the gestation period, were either dead at the time of birth or died within the first week. This contrasts with only 11 per cent dead at birth or expired within the first week for pigs born to sows whose ration had been supplemented with protein (dried skim milk, tankage or meat scrap).

That the causes for the death of nursing pigs may be many is axiomatic. From the records of the diagnosis service in the Division of Veterinary Medicine at the University of Minnesota, more than 35 different diseases, disorders or conditions have been ascribed for the death of newborn and nursing pigs. Nutritional anemia, internal hemorrhage (due to trauma), pneumonia, gastro-enteritis (catarrhal and diphtheric types) and the so-called "baby pig disease" syndrome are some of the conditions observed most frequently. Septicemias, due to one or another of a variety of micro-organisms, such as, streptococci, staphylococci, Eberthela, Salmonella, Pasteurella and Pseudomonas types, are not uncommon. Hog cholera has figured as the cause of death in quite a number of the cases in our laboratory. Swine erysipelas as the cause of death in nursing pigs has not been found very often. Congenital anomalies, omphalitis, peritonitis, hypothyroidism, tonsillitis and malnutrition with a concomitant inanition and cachexia represent disorders considered as the primary cause of death in some cases. A considerable number of young pigs have been examined but recorded as undiagnosed.

Statistics on pig mortality, such as have been presented here, force us to the conclusion that if similar situations prevail under general swine husbandry practices, then veterinarians and animal husbandmen alike must accept a challenge to learn more about the factors responsible for the loss of pigs between the time of birth and time of weaning and then discover measures that will reduce them.

LITERATURE CITED

REPORT OF THE COMMITTEE ON TRANSMISSIBLE DISEASES OF SWINE

F. L. Carr, Chairman, Columbus, Ohio; Frank Breed, Lincoln, Neb.; L. P. Doyle, Lafayette, Ind.; L. Van Es, Lincoln, Neb.; E. S. Brashier, Jackson, Miss.; R. Fenstermacher, St. Paul, Minn.

Your committee has endeavored to make a careful survey of the infectious disease problem of the swine industry. We are mindful of the fact that this year the marketing of a great per cent of animals farrowed is in compliance with the government request for more pork.

In making this survey, which at its best is none too satisfactory, we have been able to gather some information which is of value. In some instances, certain diseases have increased slightly while in others there is a decided decline.

Twenty letters requesting information were sent to the state livestock authorities; fourteen have made replies from which this report is compiled.

Hog cholera shows a decided increase in eight of the 14 states and a decrease in four, two states reporting the incidence of this disease about the same as usual.

The answers on Hemorrhagic Dysentery were somewhat surprising in that only one state reported an increase, while three showed a decided decrease, and seven reported no cases so far this year. Former reports showed this condition on the increase for quite a number of years.

Infectious Enteritis. Much to our surprise only two states made mention of this condition, one stated that the disease is decidedly on the increase, while the second reported none within the state. It does not seem possible that this disease, which has caused such economic losses in the past twenty-five or more years, could so completely drop out of the picture in one 12-month period. It prompts us to wonder if the state authorities and practicing veterinarians are giving this disease due consideration and reporting these cases.

Swine Erysipelas has become more prevalent in certain territories, especially those areas where the disease was first reported one or two years ago. Thirteen reports were received. Six showed increases, two decreases, four were about the same; and one claimed to have none.

Swine Influenza seems to remain about the same. Twelve reports were received, two showing increases, while ten stated that the incidence of this disease remained about the same as in former years.

Brucellosis in Swine. All the authorities seemed to have a decided interest in this disease for all fourteen reports contained some comments relative to it and a few had some definite statements relative to the prevalence and control of this disease. Three states reported an increase, three a decrease, six claimed no change in the incidence of the disease, and two reported none or of no importance.

Swine Pox was mentioned in only one state. It must be that this disease is fairly well confined within this state or that the veterinarians and state authorities of the other swine-raising states are not giving this disease much attention.
We are pleased to report that the occurrence of Vesicular Exanthema has not as yet been recognized outside the bounds of the State of California. Quoting from the Los Angeles County Live Stock Department 1942–1943 report:

"Vesicular Exanthema occurred on nine ranches and a public stockyards during the last half of the year. One ranch had two outbreaks since January 1943. The intensity of the disease has not been particularly severe. A few abortions have been noted and minor losses have occurred among suckling pigs. It was interesting to note the presence of lesions on the snouts of four-day old pigs on one ranch. This is the first time we have observed lesions of the disease on pigs of that age."

This disease will be discussed more completely at a later period in our program. In reviewing the reports for many years past and also searching through available literature, we fail to find mention of the virus disease, "Vesicular Stomatitis," occurring in swine. This year we will break that record and report that this disease has occurred in the Middle west, was definitely diagnosed and controlled by the U. S. B. A. I. authorities. This disease will be discussed thoroughly later in this program.

Summarizing the information gathered for this report, a few individual comments are timely. One state veterinarian reports:

"It is my opinion that we have about a 10 per cent increase in hog cholera over previous years, due to increased production, shortage of veterinarians and, possibly, delayed vaccination, coupled with an unusual amount of farmer vaccination and vaccination by so-called "professionals" throughout the state."

Another comment:

"It has been my observation throughout the year that the increased production with reference to the war effort has been responsible for the increased amount of swine diseases. I believe that the main factor is that veterinary service has been so much in demand that practitioners are entirely too busy to take care of all cases in the early stages of such outbreaks. We have reasons to be very proud of our erysipelas control program in Nebraska, which goes to show that any disease properly handled and controlled by products such as the erysipelas vaccine will certainly produce results as far as disease control is concerned. It is our opinion that more swine would reach the killing floor, if more attention was given to proper disease control and herd management of less numbers. There is no doubt but what we have tried to produce too many swine without taking into consideration the necessity of improvement in herd management, sanitation, and housing facilities."

Still another comment:

"There has been an increase in the number of cases of hog cholera reported in 1943 because many owners failed to employ professional services promptly when they first noticed their hogs were sick. Then, unfortunately, we also had a number of cases reporting the treatment of hogs by laymen with unfavorable results."
These comments are self-explanatory and should be given careful and thoughtful consideration by the swine raiser, practicing veterinarian and state livestock disease control authorities.

Your committee has endeavored to present the available information relative to the swine disease problem as it has existed in certain of the swine-producing states. Certain disease conditions appear to be under better control than in a few years past.

Our old friend, hog cholera, apparently still remains the number one disease of swine despite all the educational efforts put forth by the state and federal authorities.
REPORT OF THE VETERINARY COMMITTEE ON BIOLOGICS


A committee on biologics was first appointed by this association in 1940, and submitted formal reports which appear in the Proceedings of the forty-fifth and forty-sixth annual meetings of the association. In addition to these two reports, we would like to call to your attention the paper by Dr. D. I. Skidmore, in the forty-fourth annual report, entitled "Wherein Is the Virus-Serum-Toxin Law Inadequate for the Proper Control of Veterinary Biologics?".

A review of the above-mentioned data indicates two divergent points of view. One is to the effect that any given state is legally entitled to prohibit the admission, sale, or distribution of such biologics as may be deemed undesirable. These restrictions may be issued in the form of regulations by the properly constituted state officials, or may be a legal statute of the state. The obvious objective in either case is to prohibit or control the use of the particular product or products within the state. The other point of view is that, under federal statutes, an organization is given a license to do an interstate business and, under its provisions, shipment can be made into any state provided the product remains in its original container.

It has become apparent, and brought to the attention of the Committee, that the distribution and sales policies of some of our largest manufacturers of biologics have been influenced and changed because of the failure of adequate legislation whereby all manufacturers of such products, insofar as distribution is concerned, be placed upon an equitable basis.

It is also the opinion of your committee that more stringent control and testing of biologic products should be encouraged to the end that those products offered for sale shall have been found to have met such minimum standards of efficiency as to justify their use.

It will be apparent from a review of the literature previously cited that these and other questions of a similar nature were presented and discussed with the United States Secretary of Agriculture. It would appear that a new interpretation of B.A.I. Order No. 276, or definite congressional action taken changing the basic law, would be needed before change in our present status could be expected. It seemed inopportune to your committee that any change now could be effected under present conditions. We would, however, recommend that the committee be continued and instructed to initiate such corrective action as is possible when the opportunity is presented.

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ARMY INSPECTION OF MEAT AND MILK

BY COLONEL JAMES E. NOONAN, V.C.

Sixth Service Command, Chicago, Illinois

Within a few days, history will record two years of America's participation in World War II, a conflict which has taxed almost to the limit the resources of our country—both in material and manpower. The demands on all professions have been extremely heavy, ours being no exception. At the outbreak of hostilities, the Veterinary Corps had a very small force of trained Officers and Enlisted Men. This force was, for the most part, assigned to duty at stations scattered throughout the United States and its overseas possessions. Not unlike other branches of the Military Service, the Veterinary Corps was in the early stages of development. Considerable expansion was necessary in a comparatively short time if this service was to discharge the many responsibilities placed upon it. For the present we will consider only one of our responsibilities: the inspection of meat, poultry, dairy, and sea-food products for the Armed Forces of the United States. The first object of this inspection is to protect the health of the men and women in the Armed Forces against diseases transmitted through consumption of spoiled, damaged or contaminated foodstuffs; second, to protect the financial interest of the government by determining that the quality of the products purchased fulfill contract requirements, and third, to insure the delivery of only sound, wholesome products to troops stationed in the United States and overseas.

Within the Continental United States, the Army and Navy limits the purchase of meat and meat food products to items which have been produced in plants operating under the supervision of the Meat Inspection Division of the Department of Agriculture. This inspection agency guarantees the delivery of a product which is free from disease and contamination so long as the product remains under its control. However, we find many of the products inspected by the Meat Inspection Division require additional processing before they meet Army requirements. This additional processing is supervised by Officers and Enlisted Men of the Veterinary Corps stationed in plants while the product is undergoing manufacture. Perhaps the best way to illustrate the necessity for close inspection and supervision is to select one of the most common meat products purchased for the armed forces. The product I have selected is frankfurter style sausage, a product which is very popular with the service men. The meat components of this product, namely, pork and beef trimmings, may be prepared in one or more establishments operating under Federal supervision, shipped to a plant where they are combined with spices, seasoned, ground, and packed into containers for domestic or overseas shipment.

The procedure, as outlined above for the manufacturer of the product, may on the surface appear to be so simple that nothing could possibly go wrong. We, at home, eat frankfurters quite frequently and enjoy them. If at any time we purchase an article that does not suit our particular taste, we make our future purchases at some other store that handles the products we desire. However, the Army is
confronted with an entirely different problem. We must deliver to the soldier in Iceland, Egypt, Alaska, or the South Pacific, a product which is sound, wholesome, palatable, and nourishing. If, while the product is undergoing preparation, we permit the use of an inferior quality ingredient or deviate to any appreciable degree from the specification, the soldier will in turn receive an inferior quality and possibly an unsound or spoiled product. In such event, the contractor would have spent thousands of dollars preparing the product and the Government would have spent additional thousands in transporting and handling the product to have it arrive at destination unfit for consumption. Consequently, in making our origin inspections we must constantly keep in mind that the product may not reach the soldier for six months or more. It may require frequent handling—truck, rail, and water transportation will be involved. It will also be subjected to frequent changes in temperature and humidity, all of which tend to affect the quality of the product.

We now assume that the product meets all the detail requirements of the contract. It contains the specified amount of beef and pork trimmings; spices and moisture have been added. It has been cooked, smoked, processed and packed under our supervision in a plant maintaining satisfactory sanitary standards. However, we still have a perishable product on our hands, and only about four days along on a six months journey. At the time of arrival at destination, the product must be free of contamination and in sound condition. This is assured by conducting inspections while the product is in storage and along the route of travel. If at any time the inspector finds the product going out of condition, he recommends it be diverted to other channels for Army use or reprocessed and converted into another type of edible product. This procedure alone saves the contractor and the Government millions of dollars by preventing complete loss of the product as a result of spoilage. In addition, essential food products are conserved.

We may now consider some of the other products purchased in large quantities by the Quartermaster; namely, poultry, dairy, and sea-food products. Very few establishments processing such products are under supervision of a recognized or competent inspection agency. Consequently, we here enter the field of plant sanitation, in which case it is necessary to make a sanitary survey of the establishment and either approve or disapprove it as a source of supply. In case the establishment is approved, a Service Command or Quartermaster Depot will be called upon to assign one or more Veterinary Inspectors to the plant for the purpose of conducting ante-mortem and post-mortem inspections. They may also be required to grade the products for quality or other specification requirements, and supervise the processing and packing of the products for either domestic or overseas shipment. This class of inspection has increased to considerable proportions since the outbreak of the war.

We enter one more field where inspections are conducted by personnel of the Veterinary Corps. This field concerns itself with the supply of fluid milk for troops in the United States. I believe there is no use in my telling you the importance of a safe, clean, wholesome milk supply for our men in the Armed Forces. Dr. Haskell, of the United States Public Health Service, will cover this subject at a later time. But, I do propose to bring to your attention some of the problems
which have confronted us when we attempted to obtain the desired quality of milk in quantities sufficient to meet the demands of our service.

Problem I—SUPPLY. Large Camps were set up in congested areas, thereby placing a great burden on the local dairies. In some places, this was not much of a problem since the pasteurizing plants were large enough to handle the increased volume, provided the raw supply could be obtained. However, in other sections, large camps were established in practically rural communities where pasteurizing vats were almost unheard of, if they were present, and methods employed were faulty in many instances.

Problem II—LOCAL SUPERVISION OF THE MILK SUPPLY PRIOR TO ARMY INSPECTION. Local supervision varied widely in the United States from absolutely no control to a very complete and adequate supervision in other communities. This also held true of assistance given communities in regard to milk sanitation by various state agencies.

Problem III—PERSONNEL. At the onset of the present war, we did not have sufficient personnel to cover the wide field of milk sanitation, but we did have a job to do. Now you may ask just what has been accomplished. Well, to answer this question and cover all the details involved would require more time than we have at our disposal. However, I can select some typical examples of our accomplishments:

Example A—Deals with a large camp located in two large communities. Normally, one would expect proper sanitary supervision of milk in such areas. However, our inspections revealed this was not the case. Through the constant efforts of the Station Veterinarian and with the assistance of the United States Public Health Service, two pasteurizing plants were selected. The Station Veterinarian and his assistants visited over 500 producing farms, conducted an educational campaign in the production of clean milk, and finally selected approximately 150 producers, who were apparently willing to cooperate in the production of clean milk. During the month of May one plant received milk from approximately 100 farms. A bacteria count of the raw product from these farms ranged from 90,000 to well up in the millions. By the following September, the bacteria count of the raw product from these same farms fell below 100,000 range; many samples showed a count of less than 50,000. During the same period, the plant operator solicited and obtained the services of a Veterinarian connected with an educational institution in a nearby community for the purpose of making a detailed survey of the pasteurizing plant. Upon completion of the survey, the owner was advised of his faulty methods and changes necessary for the production of a high quality product. For the most part, these changes have been made and this station is now receiving a satisfactory supply of pasteurized milk. At the same time, its milk production steadily increased. The other plant selected, which had been in operation for many years with little or no improvement, has undergone complete renovation and new equipment has been installed. The improvements noted here were chiefly in the methods employed in handling milk. Incidentally, the surrounding communities are benefited.

Example B—is unique in that only one plant in a small town could be considered as a source of supply for a military installation that jumped from a few hundred to
many thousands within a few months. The local health authorities were without means to enforce a satisfactory dairy farm and plant inspection. In this case, a Veterinary Officer was assigned to the military installation and he, in cooperation with the owner, started a clean-up campaign both on the farms and in the pasteurizing plant. Some new equipment was added; old and useless equipment was discarded. This plant for the last year and a half has constantly produced pasteurized milk with a bacteria count of less than 30,000 per cc. At least four counts are made each month and it is not unusual to find counts ranging from 2,000 to 8,000 per cc. In addition, the laboratory reports invariably show a negative coliform content, plus a negative phosphotase test. No state or municipal supervision is exercised over the producing farms or pasteurizing plants.

SUMMARY

1. We have delivered sound, wholesome, nutritious meat, dairy, poultry, and sea-food products to every man and woman in the Armed Forces throughout the entire world.

2. No serious outbreak of disease has been reported which could be attributed directly to the consumption of meat, dairy, poultry, or sea-food products after they have undergone processing under our supervision.

3. The Army Veterinary Corps is, in many instances, the only health agency exercising control over the production of food products for both the Armed Forces and the surrounding community. I admit some individuals have apparently made mistakes; their judgment may appear to have been none too sound. At the same time we must admit that they were confronted with a situation which was new to them, but one which by custom was well established in the industry. Here, I say, in the majority of cases, the individual erred on the side of safety.

4. In many instances sufficient emphasis has not been placed on the ordinary everyday common-sense rules of cleanliness in the production of food products, especially those products derived from the dairy cow. It may be desirable, although not essential, to have the most modern equipment obtainable for the production of a quality product but it is a serious mistake to place this excellent equipment in the hands of people who are indifferent to the rules of sanitation.

In conclusion, the United States Livestock Sanitary Association is to be congratulated on its achievements. You have assisted in the eradication of many diseases which claimed as their toll millions of animals annually. Your association has been a bulwark against the introduction of animal diseases from foreign countries which would threaten our very existence as a nation. Your efforts have borne fruit and your accomplishments speak for themselves.
MILK PROBLEMS IN PUBLIC HEALTH

BY WILLIAM H. HASKELL, D.V.M.

Senior Milk Specialist, Federal Security Agency, U. S. Public Health Service

November 22, 1943

Milk sanitation is the science of sanitary conditions, of or pertaining to the preservation of health, as affecting milk. In a discussion of relatively short duration, it is not possible to adequately explore all the problems presented in providing our country with a milk supply of a satisfactory potable quality.

Obviously not all areas in this country are at present enjoying the blessings of a milk supply of unquestionable safety. The reasons for this condition are not difficult to understand, if one cares to explore the situation with an open mind.

The 1937 report of the Committee on Methods of Improving Milk Supplies in Small Communities of the International Association of Milk Sanitarians presented the following facts:

(1) That local milk control is attempted by only 1 in every 5 municipalities in the 1000 to 10,000 population group. There are approximately 5,500 such municipalities, and only 852 of these were reported as having local milk ordinances. This figure includes milk ordinances of all kinds, good and bad, complete and fragmentary.

(2) That on the average the State milk control staffs are not adequate to cope with the problem of milk control in small communities in the State. They would, in fact, have to be increased 4 or 5 fold before State control could possibly be adequate.

(3) That an average of only 38.6 per cent of the milk sold in communities in these population groups is pasteurized.

It has further developed that 66 per cent of all milk-borne outbreaks of disease reported to the Public Health Service during the past decade occurred in communities of less than 10,000 population.

The inevitable conclusion from these facts is that we cannot possibly hope to reduce materially the annual toll of milk-borne outbreaks in the United States until we have solved the very important problem of how to introduce adequate milk control in urban areas of less than 10,000 population.

It may be worth while, therefore to inquire more closely into the reasons why milk sanitation is so conspicuously lacking in this lower population bracket. Among the reasons may be cited the following:

(a) The fact that the smaller communities are much less likely to have full-time city or town health departments than are the larger communities and therefore less likely to be provided with an adequate milk control staff. A number of the smaller communities which have no health departments do nevertheless employ milk inspectors, but these constitute so small a percentage of the total number of small communities that this fact need not be seriously considered in a general analysis of the problem. The important fact is that milk control has tended to
come into being very little faster than the establishment of full-time local health departments.

(b) The fact that county or district health departments have only recently begun to recognize that milk control should form an essential part of their general public health program. Such “area” health departments have been increasing in number rapidly during the past decade as a result of the growing conviction that since many towns are too small to afford a full-time health department and since the area of most states is too large to permit an efficient centralized state control of all of the minutiae of public health (even if it were possible to secure the enormously augmented state appropriations which would be required) there must therefore be established some intermediate public health organization which can be held responsible for the details of public health work within its jurisdiction.

However, although supporting data are not available, general observation indicates that except in some areas in the South and other limited parts of the country where the enactment of the Public Health Service Milk Ordinance has followed closely the establishment of county health departments such local “area” health departments have not, in general, tended to include milk control as a part of their program.

(c) In many sections county or district health departments are not empowered to enact milk ordinances effective throughout the county or district and must therefore attempt to secure the adoption of a milk ordinance in each individual town in the area. This naturally retards progress. Furthermore even the expense of publication of a milk ordinance seems prohibitive to many of these small communities, and deters them from adopting one.

(d) There is a shortage of adequately trained inspectors, and in order to undertake milk control it is frequently necessary for local public health authorities to employ relatively untrained men with the expectation of securing training for them at a later date.

(e) In some states retardation of active local public health control has been the result of a divided state authority in matters of milk control. The Committee considered that the division of authority between the state health department and some other state department is unwise and that the public health control of milk supplies should be vested exclusively in one department.

The reason why so large a percentage of small communities do not enjoy milk control may therefore be summed up approximately as follows:

1. The individual communities themselves are too small to afford full-time health departments.

2. The county or district health departments, which are intended to provide public health protection for such local communities, have not as yet awakened to the need, or are not authorized to pass legislation and must therefore resort to the cumbersome and expensive procedure of having ordinances passed in each small community within the area, or have insufficient funds, or cannot easily secure adequately trained milk inspectors.

3. Many state health departments cannot undertake direct, detailed control from state headquarters and in many cases have not as yet undertaken a program to promote the establishment of adequate control on a county or district basis.
The remedy for this situation is not simple, but the Committee believed the following points should be stressed:

(1) All states which have not already done so should attempt to secure the passage of state legislation which will either

(a) empower the state milk sanitation authority to pass state regulations on milk sanitation which will be enforceable by county or district health departments, or

(b) empower county or district boards of health to pass milk sanitation regulations.

In either case the county or district boards of health should be authorized to enforce the regulations in all communities in the area except those which prefer to exercise their own direct control and which demonstrate within a reasonable period of time, to the satisfaction of the State, that they are able to exercise effective milk control.

(2) All state milk sanitation authorities, unless they are prepared to undertake direct and complete control in all small communities which do not undertake effective control for themselves, should set up an adequate milk sanitation staff empowered to carry out the following duties:

(a) Promote the approval and enforcement of the state milk regulations by the county or district health departments, or, in the absence of such regulations, promote the adoption of adequate, uniform milk regulations by the county or district health departments, or by the individual towns.

(b) Help secure and train local inspectors for the municipal, county, or district health departments.

(c) Make periodic investigations of the enforcement of the milk regulations in each municipal, county, or district milk shed.

(d) Assist the local departments in solving special technical problems for which certain expert technical assistance is needed.

(3) To supplement item (2b) all state milk sanitation authorities should promote the establishment of milk sanitation seminars or training courses at which local inspectors will be able to secure intensive training in the details of milk sanitation.

Thus far we have discussed the necessity of milk control in small communities and proposed certain remedies for the administration of the program. However, much of the success of the undertaking depends upon the selection of the type of program to be administered. Unless a comprehensive, enforceable, uniform program is used the possibilities of arriving at a satisfactory conclusion are greatly diminished.

The most successful type of milk control is designed primarily to be either recommended or made mandatory by State authorities but enforced by city, county, or district health authorities.

Complete public health control of milk supplies, including farm inspection, is carried out by State authorities in only two or three States, and with the possible exception of one of these, cannot be considered very satisfactory. In practically all States the burden of control falls primarily upon the local health departments.

In one or two instances, where it seemed appropriate, the passage of a State law
MILK PROBLEMS IN PUBLIC HEALTH

has been recommended, but even then it was provided that the enforcement of these State regulations be delegated to local health departments which so desire.

It is the conviction of the Public Health Service that no State law on milk supplies should be passed which would deprive local communities of their right to control their own supplies. This conviction is based upon the principle of conserving local self-government as far as practicable. State politics sometimes affect the enforcement of State laws by changes in personnel and appropriations, and if milk regulations are solely State enforced a political storm might easily disrupt the entire milk control structure. If, however, the control structure rests upon many individual local government foundations, no one political storm can wreck a large part of the structure. Any State laws which are passed should not include the detailed regulations for the production, processing, transportation and delivery of milk and milk products, but should delegate the authority for the preparation of such regulations to the State health department. This is because it would be almost impossible to secure as a State law an adequate detailed milk control regulation. There would be too much “sharpshooting” with the result that the final regulations would probably be a fragment instead of a complete and coordinated whole. There would certainly be very little chance under such a plan of securing uniform regulations as between States. Furthermore, it would be very difficult to keep such a State law up to date and abreast with new developments in milk control. On the other hand, if the detailed regulations are adopted by the State board of health or sponsored for adoption by local communities, it is much easier to keep them up to date and secure unification.

During the past twenty years much improvement has been secured in the sanitation of milk supplies in many sections of the country. The program to secure a satisfactory milk supply for all communities in our country is, however, far from completion. Certain retarding factors have had much to do with “slowing down” the rate of accomplishment. Among these retarding factors may be mentioned the following:

(a) Some communities, evidently from a desire to incorporate into ordinances individual hobbies, have included regulations either too severe to be properly enforced, or too lax to provide the necessary public health protection. In many instances these too strict requirements have no public health significance.

(b) Milk sanitation regulations, governing the public health control of milk supplies have often been used as a means of constructing “trade-barriers” designed to prevent legitimate interstate or intercity business.

(c) Lack of efficient enforcement of milk sanitation regulations, by individuals charged with administering the regulations has had much to do with the severe criticism often directed at the entire field of milk sanitation.

(d) A lack of agreement among milk sanitation authorities as to what should be included in milk sanitation programs has contributed largely to the confusion existing in many local areas as to what should be included in a milk sanitation program. In many instances local officials, as a result of this confusion, adopt no milk sanitation program. The most serious result of outspoken differences between milk sanitation authorities, and between higher branches of our govern-
mental agencies, is the creation of a consumer doubt as to the effectiveness of any regulations.

(e) Last, but not least, is the effect of the attitude of the milk sanitation officials of the larger cities in any given area. Smaller cities are prone to follow the examples set by larger communities. If inadequate milk sanitation programs are in effect in the larger centers, the areas surrounding these centers are usually operating under similarly inadequate programs. Unwise criticism of efforts directed at securing a potable milk supply should be viewed with caution.

Finally, any milk regulation is no more efficient than the effort expended in its enforcement. Interpretations of ordinance provisions are of value in proportion to the extent to which the interpretations are followed. Substitution of individual whims for official interpretations provides neither a uniform nor a legal-background of procedure.

Every person engaged in milk sanitation activities should realize the necessity for a "solid front" in providing the safe milk supply to which every citizen of this country is entitled.
REPORT OF THE COMMITTEE ON MEAT AND MILK HYGIENE

A. F. Schalk, Chairman, Columbus, Ohio; L. M. Hurt, Los Angeles, California; J. S. Koen, Storm Lake, Iowa; Warren P. S. Hall, Toledo, Ohio; T. B. Jones, Phoenix, Arizona; W. K. Lewis, Columbia, South Carolina

Your committee submits the following reports for your consideration.

MEAT

We submit the following requirements of the War Meat Board governing the preparation of meat for civilian use.

1. A safe water supply to be certified by the local and State Health Departments.
2. Adequate means of disposal of offal and refuse. Offal shall not be fed to swine. Septic tanks shall be provided when connections cannot be made with established sewage systems.
3. Plants must be so constructed and equipped that they can be easily kept in sanitary condition. Concrete or brick flooring shall be provided and at least four feet of wall surface above floors shall be constructed of material impervious to dirt and grease.
4. Adequate protection against flies, roaches, and rodents shall be provided. All outer openings shall be screened and equipped to exclude insects and rodents.
5. Cooling facilities shall be provided at the plants or within reasonable distance to the plants that is not more than thirty minutes drive from plants.

These regulations constitute the minimum requirements for beginning operation in meat plants. Meat inspection is a progressive service and your committee recommends that all agencies employing same shall add to and increase the efficiency of these requirements until all such service shall approximate the Federal system of meat inspection.

MILK

The adoption of the United States Public Health Service Milk Code and Ordinance is urged as the best approach to a safe milk supply and your committee recommends that this body accept, support, and advocate the code.
PROGRESS AND VALUE OF FEDERAL POULTRY INSPECTION IN WARTIME

By H. A. Weckler, D.V.M.

Assistant National Supervisor, Poultry Inspection Section, Office of Distribution

The Federal poultry inspection service was inaugurated in 1929 and has continued to grow ever since that date. Previous to the year 1928, little consideration had been given to the development of this kind of inspection. Shortly before that time the New York Health Department passed regulations under its sanitary code, prohibiting the sale of eviscerated or canned poultry in that city unless the poultry had been previously inspected and identified by an authorized agency acceptable to the Department. Naturally, these regulations particularly affected poultry canners who were selling their products in this area. A few years earlier, Congress had authorized the United States Department of Agriculture to inspect perishable farm products for quality and condition, and under this authority dressed poultry was inspected and certified for condition and wholesomeness. This service is now being conducted by the Dairy and Poultry Branch of the Food Distribution Administration.

Since the inception of our service several other cities have passed similar regulations prohibiting the sale of uninspected poultry products. Therefore, many canned poultry packers are utilizing the Federal inspection service. Their products are identified by the official legend which reads as follows: "Inspected and Certified by U. S. Department of Agriculture," with plant number indicated—a legend in which the consumer has learned to have confidence.

In the early days of the Federal inspection service, practically all the poultry inspected was that packed in poultry canning establishments. In the last few years, due to the desire of the leaders in the poultry industry and the growing demand of the American housewife, eviscerated poultry has been making rapid strides in out-molding old-fashioned methods of preparing poultry for sale.

Do you buy meat and meat products that are Federally inspected? That is a fair question to ask of anyone, and I doubt if anyone in this gathering would answer in the negative. It is just as fair to ask "Are you as careful in your selection of poultry and poultry products as you are in buying other meats?"

The keynote of American industry is progress and development. Federal inspection of poultry, as a guarantee of wholesomeness and quality, is progress that is of particular value in the present emergency.

I have been connected with this work since 1929, and have every reason to believe the rapid strides that have been made in inspection will continue providing of course, the public is reliably informed about the advantages of the service. It is still a relatively new field of endeavor so far as consumers are concerned, and much needs to be done to obtain full consumer acceptance and confidence.

Briefly, our service is administered in the following manner: The War Food Administration, which is the Agency under which we now operate, has authorization
for the inspection and certification of dressed poultry for condition and wholesomeness. This inspection service is carried on under cooperative agreements with the various State Departments of Agriculture. The entire cost of this service is borne by the users of the service. These costs to the plants operating under inspection consist of the full amount of the inspector's salary and certain additional amounts to cover administrative expenses and supervision. All of our inspectors assigned to plants where post mortem inspection is conducted are graduates from a recognized Veterinary College. The duties of an inspector are the post mortem inspection of poultry and the supervision of the labelling, packaging, and processing of poultry products. All plants must conform to our requirements relative to construction of building, equipment, sanitation and uniform manner of conducting this inspection. Provision is made for the disposal of all poultry, either in the raw or cooked form, that is unfit for food. I will not discuss operating details of the actual method of inspection, but if there are any questions at the conclusion of this talk, and time permits, I shall be glad to furnish you with this information.

During the present war emergency, everyone is doing his utmost to assist the war effort in every way possible. The production and conservation of food are among our most important tasks. Equally important is the maintenance of public health. Federal inspection of poultry and other commodities contributes to this job. Figures from our regional office in Chicago show that a substantial quantity of poultry was condemned as unfit for food during the first 6 months of this year. More than 46,000,000 pounds were inspected and 1.1 per cent of that quantity was found unfit for food. It was, of course, kept out of food channels.

It cannot be expected, of course, that disease in poultry can be eliminated entirely, but if there is any opportunity or means of reducing this percentage, every effort should be made to do so. Tuberculosis, for example, comprises the cause for approximately 60 per cent of our rejections. The remaining forty per cent of condemnations results from a number of different conditions and diseases, but I feel that the most important problem at this time is to control "avian tuberculosis," in which considerable amount of work is being done by various State and Federal agencies.

I might add at this time that consideration is being given by this service to the establishment of a pathological laboratory for the purpose of making laboratory diagnoses and to make studies of poultry diseases found in various parts of the U. S. to determine the causes and possible measures of control or elimination. I know I speak for the inspection service in stating that we are more than willing to cooperate with any representative agency to accomplish progress in eliminating diseases in poultry.

In conclusion, this Association, as well as the poultry inspection service and the veterinary professions as a whole should be and are interested in an educational program of poultry disease control, and also in a program to inform the consuming public of the advantages of inspected poultry.
THE USE OF THE AGGLUTINATION TEST IN DETECTING SALMONELLA TYPHIMURIUM CARRIERS IN TURKEY FLOCKS

By W. R. Hinshaw, D.V.M., Ph.D., and E. McNeil, Ph.D.

University of California, Davis

INTRODUCTION

Salmonellosis is one of the important causes of mortality in turkey poults. Thirty species from turkeys were recorded by Hinshaw (1) in a review of the disease. Since this compilation was made, several species not previously isolated from turkeys have been reported. In our own studies on this problem, 60% of the outbreaks recorded have been caused by the ubiquitous species, Salmonella typhimurium. It has been determined by several investigators that this species as well as many of the others can be transmitted through the egg in a manner similar to that of pullorum disease. The agglutination test, so successfully used for detection of pullorum disease carriers, has usually been considered of little value for detection of other Salmonella carriers. Most of the reported work has, however, been done without attention to the preparation of antigens to obtain the proper distributions of O and H types and the various components of these types according to the Kauffmann-White-Schema.¹

The studies reported herein were made in an attempt to utilize the existing knowledge of the antigenic structure of the Salmonellas in detecting carriers of S. typhimurium in turkey flocks. Typhimurium infection was selected as an example for study of the efficiency of the agglutination test as an aid in eradication of Salmonellosis from turkey flocks for two reasons. The first of these was because about 70% of all outbreaks in turkeys in California were due at that time to this one species. The second was that it was possible to make the studies simultaneously with a project on pullorum disease (Hinshaw, McNeil, and Taylor (4)), thereby utilizing the same blood samples for both projects. At the time the studies were started, this species was the only one isolated from turkeys in that area.

METHODS

When the project was first inaugurated during the 1940–41 season, only an H type of antigen was used. It was hoped then that the pullorum test which was set up simultaneously would serve to detect the O type because of the common O component XII. This plan was used during that season only. During the first, and part of the second season a dilution of 1–10 was used as a finding dilution for the H tests. Thereafter 1–25 was used as an initial finding dilution. The methods of preparation given below are the ones finally adopted for use in this project.

¹ The Kauffmann-White-Schema is based on the fact that the Salmonellae possess two distinct types of antigens. One of these is derived from the flagella and is known as the "H" type (from the German "Hauch" whip). The second is derived from the cell body and is known as the somatic or "O" type ("O" is from the German "Ohne" = without). See Kauffmann (2) and Edwards and Bruner (3).
DETECTING S. TYPHIMURIUM CARRIERS

The H antigens have been made essentially the same except for improvements in details of technic learned by experience. Cultures have been selected on the basis of smoothness, motility, antigenic component present, and efficiency in production of antigen. Each year before starting production of the season's supply of antigen these factors have been redetermined. One strain of S. typhimurium has always been selected because of its high content of the Phase 1 component "i". A second strain has been selected on the basis of its high content of phase 2 (1, 2, 3, components). A serum prepared by the injection of rabbits with the H antigen made from a strain of Salmonella kentucky has been used to test the "i" content of the antigens of Salmonella newport has served to test the "1, 2, 3," components.

Various media have been tested for the production of H antigens. These include meat extract nutrient broth, peptone water, beef infusion broth, turkey meat infusion broth and beef heart infusion broth. The best antigens have consistently been made with beef heart infusion broth. Factors taken into consideration in selecting a proper medium have included amount of antigen produced, type of flocculation, specificity of results obtained, and keeping quality.

After selection of the cultures they are inoculated into semi-solid agar and after at least two passages at 24 hour intervals are seeded into tubes of beef heart infusion broth. In 18 to 24 hours, five cc. of the broth culture is added to 1,000 cc. of beef heart infusion broth. It has been found that large-base flasks filled to a depth of two inches, give the best yield, in the 24 hour incubation period. At the end of 24 hours incubation at 37°C., a sample of antigen is removed aseptically for purity tests, and 0.6% of C.P. formalin is added as a preservative. Each flask of antigen is tested separately for purity, units per cubic centimeter and antigenic behavior. All flasks found to be satisfactory are pooled, retested for antigenic component content, and stored in the refrigerator until needed for use. For routine tests, the antigen is diluted with physiological saline to correspond with a direct reading of 6.0 with a Gates type of nephelometer. The diluted antigen is brought to a 0.2% content of formalin and is adjusted to a pH of 7.6 to 7.8 with sodium hydroxide. One cubic centimeter of antigen is used with 0.04 cc. of serum (1-25 dilution) and the tests are incubated for 4 hours at 50°C. in a dry air incubator fitted with a humidifier to keep it at near the saturation point to prevent evaporation.

The O antigens are prepared with the use of two nonmotile strains of S. typhimurium. Whenever a new lot of antigen is to be prepared, the cultures are tested for purity, plated on agar, and individual smooth colonies are selected by spot agglutination tests with specific serums, to make sure that all the O components (I, IV, V, XII) are present. Several colonies are selected and these are each used separately to inoculate Kolle or similar flasks of meat extract agar. Cultures are incubated for 48 hours, tested for purity and smoothness, and then the growth is washed off with a minimum amount of sterile physiological saline. To the saline suspension is added enough 95 ethyl alcohol to make a final suspension in 50% alcohol. It is stored in the refrigerator in this preservative until ready for use. Each lot is tested for antigenic properties before storage.¹

¹ Recent investigations indicate that 0.5% phenolized physiological saline can be used for washing off growth and for preservation of "O" antigens providing non-motile cultures are used in their preparation. In such cases no alcohol is added.
When ready to be used, it is diluted with sterile physiological saline to a turbidity equal to that of 6.0 with a Gates type nephelometer. No preservative is added and the pH is not adjusted since it usually ranges from 7.6 to 8.0. Only enough for one day’s use is diluted up at one time. One cubic centimeter of antigen is added to 0.04 cc. of serum and the tests are incubated at 50°C. for 20-24 hours in the same incubator mentioned above.

Various combination of antigens have been tried in an attempt to eliminate the number of tests necessary on one sample where pullorum disease and typhimurium tests are made simultaneously. After 3 seasons experience, it has been concluded that three tests are necessary for the maximum efficiency; i.e. 0 typhimurium, H typhimurium and pullorum.

RESULTS

The results of the three seasons’ testing are given below. They are tabulated according to the 4 groups concerned. Groups A and B are Cooperative Associations in the same community. Group A sells only hatching eggs, while Group B sells both poults and eggs. Group C is a turkey breeding farm from which group A bought its present foundation stock. Group D is a turkey breeding farm in another locality with which the University has had a project for 4 years. A summary of all tests is given in Table I.

Group A

1940–41.—The pullorum testing history of this organization was thoroughly known, and autopsy records from many groups of poults from the previous season were available. A total of 4245 birds (8 flocks) of the 21,129 birds (58 flocks) were tested in 1940–41 and 52 typhimurium reactors (1.2%) found on first test. Only 876 birds were tested a second time and 29 reactors were found (3.3%). No third tests were made. Of the 52 reactors, titers are available on 36. They were as follows: 1–10, 16; 1–20, 14; 1–40, 5; and 1–160, 1.

These infected flocks were not used as sources of eggs for local replacements. Of 153 groups of poults hatched locally during the spring of 1941, specimens were obtained for autopsy from 52. *S. typhimurium* was isolated from only one group. The principal media used for these and all other isolations referred to in this paper were tetrathionate broth for enrichment and desoxycholate agar for final isolations. After isolation and preliminary identification, all Salmonellas were antigenically typed by Dr. P. R. Edwards, University of Kentucky.

1941–42.—At the beginning of the season (first test) there were 23,131 birds in 48 flocks. Of the 48, only nine flocks (2,321 birds) were negative to the typhimurium test. Sixteen flocks (8745 birds) had less than 2% reactors but over 1.5%. The remaining 23 flocks (12,065 birds) had an average of 3.9% reactors. The average for all 23,131 birds on first test was 2.6%. The percentage of reactors in the males on first test was slightly higher than in the females—3.4% as contrasted with 2.5%. After the first test, however, the percentage was slightly lower in the males than in the females. In studying the incidence of pullorum disease in males and females (Hinshaw and McNeil (5), an appreciably greater percentage was found in females than males (4.23% and 1.77%).
On second test, there were 17,389 birds and 387 (2.2%) reactors. The incidence in males was 1.1% and in females 2.2%. On third test, there were 10,762 birds and 156 reactors (1.5%). The incidence among males and females was about the same. This was probably due to the fact that by then the males had been put with the females and had picked up infection from the breeder yards. On fourth test,

**Table I.**—Summary of results of testing four groups of turkey flocks for typhimurium infection

<table>
<thead>
<tr>
<th>TEST</th>
<th>GROUP A</th>
<th>GROUP B</th>
<th>GROUP C</th>
<th>GROUP D</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1940-41</td>
<td>1941-42</td>
<td>1942-43</td>
<td>1940-41</td>
</tr>
<tr>
<td>First:</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Birds</td>
<td>4,245</td>
<td>23,131</td>
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<td>Reactors</td>
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<td>604</td>
<td>18</td>
<td>37</td>
</tr>
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<td>1.2</td>
<td>2.6</td>
<td>0.8</td>
<td>0.96</td>
</tr>
<tr>
<td>Second:</td>
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</tr>
<tr>
<td>Birds</td>
<td>876</td>
<td>17,389</td>
<td>550</td>
<td>2,024</td>
</tr>
<tr>
<td>Reactors</td>
<td>29</td>
<td>387</td>
<td>7</td>
<td>30</td>
</tr>
<tr>
<td>% Reactors</td>
<td>3.3</td>
<td>2.2</td>
<td>1.3</td>
<td>1.5</td>
</tr>
<tr>
<td>Third:</td>
<td></td>
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</tr>
<tr>
<td>Birds</td>
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<td>10,762</td>
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<tr>
<td>% Reactors</td>
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<td>1.9</td>
<td>0.2</td>
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<tr>
<td>Fourth:</td>
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<td>2.6</td>
<td>0.2</td>
</tr>
<tr>
<td>Fifth:</td>
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</tr>
<tr>
<td>Sixth:</td>
<td></td>
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</tr>
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<td>Birds</td>
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<tr>
<td>% Reactors</td>
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</tr>
</tbody>
</table>

there were 4535 birds and 83 reactors (1.9%). On fifth test, there were 1595 birds and 13 reactors (0.8%). On sixth test, there was one pen of 189 birds with two reactors. It should be pointed out that nonreacting pens on ranches were usually not retested and also that only flocks with reactors were retested. If it was found that the same males had been in both infected and noninfected pens, both pens were then retested.

At the end of the season, there were 25 flocks that had had at least one negative
report. There were 15 flocks (11,472 birds) with an average infection of 1.99%, and there were 4 small flocks (657 birds) with an average infection of 3.2%. The retesting was continued throughout the height of the egg season. The total number of birds at the end of the testing season was 19,916 with an average percentage of infection of 0.7.

No eggs were kept for local replacements except from the 25 non-reacting flocks. With two exceptions, the eggs were either hatched at home or in a single hatchery which cooperated at all times with the Association. The University took the responsibility of advising which flocks were best for local replacements, and had complete records of eggs set and poultis hatched. Of 98 lots of poultis hatched, specimens were obtained for autopsy from 68, and no cases of typhimurium infection were diagnosed. These include specimens from every hatch except the two mentioned above. It should also be noted that there were no diagnoses reported in poultis hatched from eggs sold to non-members of the Association (800,000 eggs in all).

1942-43.—As has been stated, poultis from every hatch of the 1941-42 hatching season had been obtained for autopsy. Although no cases of typhimurium infection were found in this group, one case of *S. meleagris*, one of *S. kentucky*, one of *S. montevideo*, and two of *S. senftenberg* were diagnosed. The flocks in which these infections were found were tested for the species in question as well as for *S. typhimurium* and *S. pullorum*.

A total of 24,033 first tests were made on birds which were the immediate progeny of birds owned by this group during the previous season or hatched from eggs purchased from Group C. Eighteen reactors (0.88%) were found. These reactors were limited to one flock of 587 birds. On second test, there were 550 birds with 7 reactors (1.3%); on the third test 537 birds and 10 reactors (1.8%); and on the fourth test, 310 birds and 8 reactors (2.6%). A total of 233 chickens were tested on this ranch, and 6 reactors (2.5%) were found on first test. Although the adult turkeys and chickens were well segregated, the chicks were brooded near the turkeys. It is also known that typhimurium infection existed on the ranch from which the chicks came, and it is possible that the infection was introduced by purchase of the chicks.

**Group A1.**—This group of 784 birds comprises 3 flocks (500, 100, and 184 birds respectively), which were not the progeny of group A birds of the previous year. These flocks were tested at the request of three members of group A who wished to buy additional birds in 1942-43 and are not included in Table I. None of these flocks were kept by the Association.

The flock of 500 was the progeny of birds owned by group B, the previous season. These birds were tested under protest, since it was certain from the history of the preceding season that there would be reactors. There were 62 reactors (12.4%) in this flock.

The flock of 100 birds was purchased by one grower on the advice of his feed company, without the knowledge of the Association or the laboratory. There were 42 reactors (42.0%) so the flock also was sold immediately. It was discovered that these 100 birds had been in a yard adjoining 120 birds tested earlier.
DETECTING S. TYPHIMURIUM CARRIERS

in the season and found free. In another adjoining pen were some younger birds which had not yet been tested. After the results of the tests on the birds purchased from the outside, the other turkeys on the premises were retested. Five reactors were found in the group which had been free, and 10 in the younger first-test group. It was not surprising that the infection had spread, because the grower admitted that no attempt at isolation of the new birds had been made, and that they sometimes flew into the other yards.

The 184 birds in the third flock were purchased without our knowledge and again were tested under protest. There had been a mortality of 26%, with no poults submitted for diagnosis, and the hatchery from which they came had had losses from paratyphoid. There was a total of 26 reactors found (14.1%).

From these results it can be seen with what ease new infections can be introduced into an Association if strict supervision is not exercised at all times. A total of 1084 birds were a loss to the Association—784 purchased outside and 300 which had been in contact with infected ones.

Group B

1940–41.—Group B had undergone the greatest losses due to mortality in poults. In this group, 3836 birds (7 flocks) from a total of 8329 were tested in 1940–41 and 37 reactors (0.9%) were found on first test. On second test, there were 2024 birds and 30 reactors (1.4%).

The original tests (H type only) were set at a 1–10 dilution, and titer tests were made whenever there was sufficient serum. Birds were culled on the basis of a good reaction at 1–10 during this season, although it is probable that some of these may have been nonspecific agglutination. However, since the mortality had been severe, it was felt that even these low titer birds should be removed. Of the 37 reactors, on first test, H type titers are available on 27 of them. They were as follows: 1–10, 13; 1–20, 8; 1–40, 2; 1–80, 2; 1–320, 1; 1–640, 1. Six birds were obtained for autopsy, and S. typhimurium was recovered from two of them. One of the two had a titer of 1–320 and one 1–640.

1941–42.—There were 18,442 birds in 25 flocks on first test in 1941–42. In these, 1416 (7.6%) reactors were found. It should be noted that 774 of the reactors were in one lot of 1354 males which had just undergone a subacute attack of the disease and had not yet had opportunity to abort the organism. There were 15,925 females on first test with a percentage of infection of 3.9, which is probably closer to the true incidence of carriers. On second test, there were 11,549 birds and 366 reactors (3.2%). On third test, there were 3497 birds and 76 reactors, (2.2%) and on fourth test 1386 birds and 39 reactors (2.9%). This does not represent the true status of infection, because several growers were not willing to cooperate and refused to retest, so the University found it necessary to discontinue the project with them, and refused to accept further tests from them.

The results of the lack of retesting by this group were shown by the fact that it had to refund to its 1942 customers several thousand dollars because of losses from both pullorum disease and paratyphoid, while groups A, C, and D did not have to make any replacements for either one.
Group C

1940-41.—The owner of group C has operated an isolated breeding and hatching unit for many years, and has always had a pullorum-disease-free flock. During the summer of 1939 *S. typhimurium* was isolated from poults on this ranch and during the spring of 1940 there were two outbreaks of typhimurium infection in poults purchased from him.

There were 1797 birds on first test in 1940-41, and 19 reactors (1.1%) were found. Of these, titers are available on 17. Of the 17, five showed O type agglutination and 12 were H type. The O type reactors were very low in titer; four of them reacted at 1-10 and one at 1-20. Of the 12 H type reactors, five reacted at 1-10, three at 1-20, two at 1-40, two at 1-160.

On second test, there were 1750 birds and 30 reactors (1.8%). At this time there were seven yards, of which six were infected. In one pen of 89 birds, two reactors had been left in since the previous test. At the time of the second test, there were 15 reactors in this small yard. Of the 30 reactors, 13 were O type. Of the 13, 9 reacted at 1-10 and 2 at 1-40. Of the 17 H type reactors, 10 reacted at 1-10, 5 at 1-20 and 2 at 1-80.

On third test, there were 1545 birds and 21 reactors (1.4%). Of the 21 reactors, titers are available on 19. All 7 somatic reactors had a titer of only 1-10. Of the 12 flagellar types, 4 reacted at 1-10, 4 at 1-20, 3 at 1-40 and one at 1-80. On fourth test there were 904 birds and 7 reactors (0.8%). Six of the 7 were somatic type; two reacted at 1-10, three at 1-20 and one at 1-80. The one flagellar type reacted at 1-10.

1941-42.—On first test, there were 2076 birds and 45 reactors (2.2%). On second test, there were 1454 birds and 10 reactors (0.7%); on third test 933 birds and 9 reactors (0.9%); on fourth test 804 birds and two reactors (0.2%); and, on fifth test, 229 birds and no reactors. Progeny of these birds were secured for autopsy, and *S. typhimurium* was not isolated. The segregation and general sanitation were much improved over the previous year.

1942-43.—A total of 2088 turkeys were tested in 1942-43 and there were no reactors. Progeny from this flock secured for autopsy during the brooding season of 1943 did not yield *S. typhimurium*. It is impossible to know exactly how the infection got on this ranch in the first place, but the rodent and wild bird population is abundant in that region, and typhimurium infection was known to exist within a mile of it. A more strict rodent and bird control program has been in operation since that time.

Since the inauguration of the testing program on this ranch there have been no acute outbreaks of typhimurium infection traced to progeny of the tested birds sold to customers. On the ranch no losses in poults from the infection occurred after the intensive testing program was started, but the results of the tests in 1941-42 would indicate that some infection remained on the ranch which was transmitted to the flock that year. The results of the 1942-43 tests indicate that the infection was successfully eliminated from the ranch by the 1941-42 testing program.

Group D

1941-42.—The owner of group D experienced losses from typhimurium infection since the spring of 1939. In 1941 the disease was diagnosed in birds from four of
his five hatches. The mortality was high and continued from a few days after hatching until the birds were several weeks of age.

A total of 6345 tests were made for him during the 1941–42 season, and 146 reactors (2.3%) were removed. On first test, there were 1642 birds and 63 (3.8%) reactors. This was somewhat higher than either Group A or C, but was not surprising considering the history of mortality. On second test, there were 1540 birds of which 38 (2.5%) were reactors; on third test there were 1062 birds resulting in 12 (1.1%) reactors; and on fourth test, of 727 birds 22 (3.02%) reacted. All the remaining birds (1374) were retested in April, 1942 and 11 reactors (0.7%) were found.

During this season the general sanitation of the ranch was improved and an attempt was made to control the rodent and wild bird population. Poult's were examined from every hatch, and typhimurium infection was not found.

1942–43—During this season, 1621 birds were tested and no reactors found. Several lots of poult's were autopsied during the spring of 1943, and no paratyphoid found nor has it been diagnosed in poult's hatched from eggs sold.

As in case of group C, typhimurium infection has apparently been eliminated from this ranch. The intensive testing system of 1941–42, plus an improved sanitation program which included rodent control all were factors contributing to the success.

**INTERPRETATION OF RESULTS**

In conducting a testing program of this sort there are certain laboratory procedures which serve as criteria in deciding whether or not a flock is infected. The two most important of these are: (1) the determination of the titers of reactors with specific antigens and (2) autopsy of reactors. It is especially important to autopsy sufficient numbers of low-titer reactors in flocks having a history of freedom from the disease in order to establish a correct diagnosis. The following sections give the results on certain flocks which were chosen to illustrate the various types of reactions which occur, and how they may be interpreted.

**Titers**

In order to determine whether normal or nonspecific reactions to the H and O typhimurium tests exist in typhimurium noninfected flocks, and in order to determine if possible, minimum specific diagnostic titers, all reacting serums have been reset to find the end titers. By accumulation of such data, it has been possible to compare the results of the testing with results of autopsy of reactors and the diagnoses made on progeny of the breeders. In Tables II and III are given examples of the results of these studies.

A total of 10,912 tests were set on typhimurium infected flocks 1 to 6 inclusive and 24,943 on noninfected flocks 7 to 42. A few of the most significant differences are as follows: in infected flocks only 3.6% of the H reactions had a titer of 20, and only 28.6% had a titer of 40 or less. In contrast, 25.6% of the tests in the noninfected flocks had a titer of 20, and 60.0% reacted at 1–40 or less. Only 1.6% of the H tests in the noninfected flocks had a titer of 1–320 or greater, while 22.1% of the reactors in infected groups reached an H titer of 1–320 or higher.

In both infected and noninfected flocks there would seem to be more low titer O
<table>
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<tr>
<th>FLOCK</th>
<th>REACTIONS</th>
<th>&quot;H&quot; Typhimurium</th>
<th>&quot;O&quot; Typhimurium</th>
<th>Pullorum</th>
</tr>
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<tr>
<td></td>
<td>20</td>
<td>40</td>
<td>80</td>
<td>160</td>
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<td></td>
<td>Per cent</td>
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<td>2</td>
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</tr>
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<td></td>
<td>Per cent</td>
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<td>9.5</td>
<td>23.8</td>
</tr>
<tr>
<td>5</td>
<td>Number</td>
<td>3</td>
<td>11</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>Per cent</td>
<td>7.5</td>
<td>27.5</td>
<td>20.0</td>
</tr>
<tr>
<td>6</td>
<td>Number</td>
<td>0</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Per cent</td>
<td>0</td>
<td>38.5</td>
<td>0</td>
</tr>
<tr>
<td>*1–6 incl.</td>
<td>Number</td>
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<td>35</td>
<td>42</td>
</tr>
<tr>
<td></td>
<td>Per cent</td>
<td>3.6</td>
<td>25.0</td>
<td>30.0</td>
</tr>
</tbody>
</table>

B. Flocks free of both typhimurium infection and pullorum disease

| **7–42 incl. | Number | 32  | 43  | 31  | 17  | 2   | 125 | 17  | 8   | 2   | 1   | 0   | 28    | 5   | 5   | 1   | 0   | 11   |
|       | Per cent | 25.6| 34.4| 24.8| 13.6| 1.6 | 1.6 | 60.8| 28.6| 7.1 | 3.6 | 0   | 45.5 | 45.5| 9.1  | 0.0 |

* Total tests made in flocks 1–6 inclusive, 10,912.
** Total tests made in flocks 7–42 inclusive, 24,943.
reactions than low H reactions. In infected flocks 14.7% reacted at 1–20 with the O antigen and 53.9% at 1–40 or less, while in noninfected flocks 89.3% reacted at 1–40 or less. Of the reactions to pullorum antigen, 91% reacted at 1–40 or less in the noninfected flocks and 74.7% in the infected ones.

Of 157 reactors in the noninfected flocks 6 (3.8%) reacted only to pullorum antigen, 3 (1.9%) to both pullorum and typhimurium O, 24 (15.3%) to O alone, 124 (78.9%) to H alone and 1 (0.6%) to all three antigens. Of 335 reactors in the infected flocks, 8 (2.4%) reacted to pullorum alone, 52 (15.5%) to both pullorum and typhimurium O, 135 (40.3%) to O alone, 35 (10.4%) to O and H, 79 (23.6%) to H alone and 26 (7.8%) to all three antigens. Certain significant differences can be seen from these results, the most striking of which is the fact that in noninfected flocks only one bird reacted to both H and O antigens, while in the infected

**Table III.—Summary of types of reactions**

<table>
<thead>
<tr>
<th>FLOCKS</th>
<th>TYPES OF ANTIGENS AND REACTIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>H-type only</td>
</tr>
<tr>
<td>No. %</td>
<td>No. %</td>
</tr>
<tr>
<td>A. Flocks infected with <em>Salmonella typhimurium</em></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>5 22.9</td>
</tr>
<tr>
<td>2</td>
<td>42 35.9</td>
</tr>
<tr>
<td>3</td>
<td>1 1.9</td>
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<td>4</td>
<td>10 43.5</td>
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<td>5</td>
<td>17 20.0</td>
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<tr>
<td>6</td>
<td>4 11.1</td>
</tr>
<tr>
<td>Totals</td>
<td>79 23.6</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>B. Flocks not infected with <em>Salmonella typhimurium</em></th>
</tr>
</thead>
<tbody>
<tr>
<td>Totals (7–42)</td>
</tr>
<tr>
<td>124 78.9 24 15.3 0 0.0 6 3.8 3 1.9 1 0.6</td>
</tr>
<tr>
<td>157</td>
</tr>
</tbody>
</table>

flocks 10.3% gave such a reaction. Another important difference is the fact that in the infected flocks 23.6% reacted only to the H antigen, while in the noninfected flocks 78.9% reacted only to H.

One significant point of agreement in the infected flocks is that all of them showed reactions to both somatic and flagellar antigens. This was not true of the noninfected flocks. One would, therefore, hesitate to condemn a flock for typhimurium infection which had only H or only O type reactions. A common belief is that the pullorum test will detect most of the typhimurium reactors. Our results show that at a dilution of 1–25, the pullorum test might pick about 25% of them (Table III). However, 41.8% of the pullorum titers were only 1–25 and many of these were partial reactions.

From the data given in Table II it can be seen that there are many "nonspecific"
H reactions. In order to better interpret these reactions a new system of setting H titers was started in 1943. According to the Kauffmann-White Schema, the flagellar antigens consist of a specific and nonspecific phase, called phase 1 and 2 respectively. In most cases, phase 1 is denoted by small letters and phase 2 by Arabic numerals. In the case of typhimurium, phase 1 is denoted by the letter "i" and phase 2 by 1, 2, 3. Several other species, among them S. newport, have similar components of phase 2. If the serum reacting to S. typhimurium H antigen is set also with at least 2 other antigens one containing phase 1 component "i" and another the phase 2 components 1, 2, 3, it becomes easier to interpret results. S. kentucky (VIII, XX:i, z6... ) and S. newport (VI, VIII:eh, 1, 2, 3) are the two used routinely. In some cases Salmonella coli 1 (XXXI:1, 5) has been used to detect the phase 2 component 1. In one case, 9 reactors to the typhimurium H antigen gave higher titers to S. coli 1 than to either the other two.

By the use of this system of determining the phases represented in each sample of serum, it was learned that in flocks infected with S. typhimurium, the reactors were usually not in both phases. For example, in a group of 86 reactors from 5 infected flocks, 18 (20.7%) were in phase 1 ("i" only), and 43 (50.0%) were in phase 2 (1, 2, 3) only. A total of 35 (40.8%) of these had titers of 1-40 or less, while 51 (59.2%) had titers of 1-80 or greater. The highest titer recorded for the group was 1-2560. This was for a bird that was only in phase 2. Its 0 titer was 1-160. S. typhimurium was isolated from the intestine, crop, and liver of this bird.

A study of flocks proven to be free from typhimurium infection has revealed a small percentage of H type reactors that occur without any 0 types in the same flocks. These are probably caused by the presence of infections with coliform types that have similar H antigens to S. typhimurium or by the presence of other Salmonellas having at least one phase in common with S. typhimurium. A study of 121 such reactors from flocks representing 41,000 turkeys free of both pullorum disease and typhimurium infection, revealed that 91 (75.2%) were in phase 2 only. Twelve (9.9%) were in both phases and 18 (14.9%) were in phase 1 only. A total of 61 (50.4%) of the 121 had titers of 1-40 or less and 60 (49.6%) had titers of 80 or more. In this group the highest titer of any individual reactor was 1-320.

All of the 121 reactors referred to above were culled from the flocks, and retested, and the majority of them were either autopsied or cloacal scrapings taken for bacteriological studies. In no instance was S. typhimurium isolated from them or from other birds in the flocks from which they came. These reactions illustrate one of the problems of testing for typhimurium infection. Correct interpretation of results in such cases will depend on many factors. Most important is the history of the flock, and especially autopsy and hatching history. The possibility of other Salmonellas that have common antigenic components must be considered. A retest of the reactors after a week or two often is a valuable aid since in many instances these "nonspecific" reactors lose their agglutinins in a short time. Autopsy of a representative number if not all of them, and a careful bacteriological examination is recommended as the final means of arriving at a correct diagnosis.

In flocks known to be infected with S. typhimurium, one seldom finds the reaction limited to a single antigenic type. Both O and H types are found either alone in individuals or combined.
In typhimurium noninfected flocks, when reactions occur, the type is usually O alone or H alone and the percentage of reactors not high (less than one percent). As stated above, the majority of the H reactors in our experience with such flocks has been in phase 2 (1, 2, 3). This will depend somewhat on the types of para-colon, coliform bacteria or Salmonellas that exist in a community.

Consideration must be given to the fact that there are many organisms listed by Kauffmann (2) and by Edwards and Bruner (3) having all of the phase 2 components and many more that have one or more of the components of this phase. For instance, Edwards and Bruner list 58 Salmonellas that have at least the 1 component of phase 2; 16 that have 1 and 2, and 11 that are identical (1, 2, 3) to the phase 2 of S. typhimurium. Thus the presence of any one of these organisms in a flock might account for an H type of S. typhimurium reaction.

**Autopsy results**

Table IV gives 21 examples of reactors, from known pullorum disease-free flocks, on which we have complete titer records, and from which S. typhimurium was isolated from one or more tissues. These birds all appeared healthy when culled as reactors. It will be noted that 4 of the 21 reactors were culled by the pullorum test alone. It is now known that the O antigen, used at that time, was low in the XII factor, while the pullorum antigen was more sensitive than normal. It is a

### Table IV. Examples of agglutination titers of turkeys yielding only Salmonella

<table>
<thead>
<tr>
<th>TURKEY NUMBER</th>
<th>ANTIGENS AND TITERS</th>
<th>S. TYPHIMURIUM</th>
<th>S. PULLORUM</th>
<th>TISSUES YIELDING S. TYPHIMURIUM ON AUTOPSY</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>H-type</td>
<td>O-type</td>
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<tr>
<td>138</td>
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<td>160</td>
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<td>445</td>
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<td>247</td>
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<td>160</td>
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type of problem however with which one is confronted in such testing procedures, and illustrates the need for careful supervision of the manufacture of antigens. The high incidence of pullorum reactions given in the table is not a true picture of what can be expected in routine practice, because an effort was made when selecting birds for autopsy to obtain all those that reacted to the pullorum test. By this procedure a greater number were obtained than if random samples were taken. The results do show the importance of utilizing pullorum antigen as well as both an O and H type of typhimurium antigen to obtain maximum efficiency.

There appears to be no correlation between the titers obtained with any one, or any combination of types of antigens, and the organs infected. These facts make it difficult to arrive at a decision as to the optimum titer to select for culling reactors from a flock. It is because of these results, and additional studies, that 1–25 dilution has been adopted as a finding dilution for all three antigens.

The digestive organs of this group of birds yielded a much higher percentage of isolations than the reproductive organs. This is in contrast to pullorum disease where the incidence is higher for the reproductive organs (Hinshaw, McNeil and Taylor (4)). Of 18 livers cultured 12 (66.7%) yielded *S. typhimurium*; 12 of 18 spleens (66.7%); and 17 of 21 intestines (80.9%) also yielded it. Only 3 (16.7%) of the 18 reproductive organs yielded the organism. The other tissues referred to in the table were not routinely cultured. Recent studies indicate that the crop is an important organ to culture because of a high incidence of positive *Salmonella* isolations that have been made from this organ.

A group of 74 birds from flocks having a history of freedom from typhimurium infection and pullorum disease, but suspected of being carriers because of their reaction to either the typhimurium or pullorum antigens were autopsied. *Salmonella* species were not isolated from any of these birds. *Streptococcus* and *Escherichia* were found to be the predominant genera in these 74 birds. It has already been noted that coccus types were the predominant forms in the tissues of birds suspicious to the pullorum test (Hinshaw, McNeil and Taylor (4)).

**DISCUSSION**

These studies have shown the great necessity of using two types of antigens, one made to select birds having the O phase of agglutinins and the other made to select birds having the H phase. Cultures used for producing such antigens must be carefully selected for maximum antigenic component content, and for smoothness. *S. typhimurium* has 8 antigenic components (I, IV, V and XII in the somatic phase and “i”, 1, 2, 3 in the flagellar phase). The preparation of a complete antigen for this species is, therefore, equivalent to making 8 different antigens. It has often been necessary to use several cultures to get the proper mixture of components in the final product, because each culture has been largely in a single phase. This has proven more true of cultures used for producing H antigen, than for those used for producing O antigens. The preparation of antigens for conducting tests should be left to laboratories thoroughly familiar with the technic of making antigens to conform with the Kauffmann-White Schema. No person contemplating making tests should do so until he has thoroughly familiarized himself with this Schema. Without an appreciation of the antigenic structure of the *Salmonellas*, a laboratorian will find many pitfalls when making interpretations.
DETECTING S. TYPHIMURIUM CARRIERS

There are several organisms which may be responsible for nonspecific reactions. Kauffmann has found five lactose fermenting strains which he now designates as *Salmonella coli* 1, 2, 3, 4 and 5, respectively, because they have antigenic components found in salmonella species. Of these 5, two have the nonspecific component 1 in common with *S. typhimurium*, one has somatic IV, V and XII in common with it, and still another has IV and XII. These five strains were isolated from humans, but it is not improbable that certain similar strains may be found in birds. Since coliform types were found in the livers of some of the reactors in these studies it is possible that they may cause false reactions. It will be necessary to infect birds experimentally with various suspected strains isolated from "nonspecific" reactors and to test their sera against paratyphoid antigens before all these questions can be answered.

It is doubtful if typhimurium infection can be successfully eradicated from a community where it is prevalent, by a testing program alone, because of the ubiquitous distribution of the causative organism. Properly conducted the test can, however, be used to locate infected flocks, and even as an aid in eradicating the disease from a flock. When a ranch is found infected either by means of autopsy and bacteriological examination of diseased birds, or by means of the test, a careful survey of the ranch is necessary before deciding the proper procedure to follow to eliminate the disease. Every ranch will constitute a separate problem and must be handled as such.

It is evident from these studies that it is necessary to have a complete history of flocks which are being tested. As in the case of pullorum disease, only then can one attempt to interpret results and to make decisions. *Without such a history, and a willingness to use it, it is useless to test, because it leads to endless confusion.* The problem of spread of infection by animal and human carriers is much more important than is the case of pullorum disease. Humans, dogs, cats, rabbits, rats, mice, cows, horses, sheep, hogs, chickens, ducks, geese, pheasants, quail, chukars and pea-fowl are among the many carriers of this species. Two of the ranches which were free on first test in the 1942-43 season became contaminated during the breeding season. In one case quail were the most probable transmitting agent. By extensive autopsy of poult hatched locally these reinfections were detected, and eggs from these sources were no longer used locally nor for shipment. Such an autopsy program is absolutely necessary to protect both local growers and outside customers. It also aids in interpretation of testing results.

Whenever infection is found in turkeys or chickens on a ranch, all animals (including man) on the ranch must be considered as possible sources of reinfection even if the disease is eradicated from the fowls. It is useless to attempt eradication in the turkeys unless it is possible at the same time to eliminate other sources of the disease. Rodents, wild birds and reptiles should be considered as possible sources and the eradication program should include their extinction. Because of the fact that the majority of typhimurium carriers shed the organism in their feces a strict sanitation program following removal of the reactors is even more important than in the case of pullorum disease.

Eggs for replacement should come only from flocks free from the disease. These flocks should have been reared on ranches having a history of freedom from it. They should be hatched only by hatcheries accepting eggs of like status. In most
instances it will be advisable to sell infected flocks as market birds because of the problems connected with the frequent retesting necessary to keep the infection reduced to a minimum. In case infected breeding flocks are kept for production of hatching eggs, they should be tested at frequent intervals and all reactors promptly removed.

We do not intend to leave the impression that these studies show that a general testing program for eradication of typhimurium infection, such as is recommended for eradication of pullorum disease, is advisable at present. They do show certain possibilities as well as certain limitations of such procedures as adjuncts to other methods of eradication of Salmonellosis from a community. Probably the best summary of the merit of such a testing program is the fact that neither group A, C nor D of these studies has had to pay out any money for replacements since its inauguration, while group B has had to do so each season.

SUMMARY

1. This paper reports studies on the utilization of the existing knowledge of the antigenic structure of the Salmonellas in an attempt to eradicate *Salmonella typhimurium* from turkey flocks. Results on 151,626 agglutination tests made over a 3 year period for 4 groups of turkey growers are reported.

2. The studies show the necessity of using, as separate tests on each blood sample, two types of antigens at 1–25 as a finding dilution. These are (1) an H or flagellar type and (2) an O or somatic type. Successful preparation of such antigens entails a thorough knowledge of the Kauffmann-White Schema, and should be left to laboratorians familiar with it. The 1–25 dilution tube agglutination test for pullorum disease will detect about 25% of the typhimurium reactors because of the component XII, but it is definitely not a substitute test.

3. Types of reactions, determination of titers, and autopsy results are the 3 procedures which serve as criteria in deciding whether or not a flock is infected.

4. It has been found that the use of two supplementary specific antigens, one containing the component "i" (*Salmonella kentucky*) in common, and one containing the components 1, 2, 3 (*Salmonella newport*) in common, is a valuable aid in interpretation of the H type reactions.

5. A study of the bacteriological results on reactors autopsied revealed the following percentages of isolations of *Salmonella typhimurium* from various organs: intestines 80.9; liver 66.7; spleen 66.7; and reproductive organs 16.7.

6. Because of the ubiquitous distribution of *S. typhimurium* in animals other than turkeys, it is doubtful if it can be eliminated by testing alone. The results obtained show, however, that the tests described have possibilities as aids in eradicating Salmonellosis from a community. The limitations as well as the possibilities of their use are discussed.

7. The merit of such an eradication program as is described is shown by the fact that cooperating groups A, C, and D of these studies have not had to pay out any money for replacements since starting to test. In contrast group B which failed to conform has had to make readjustments each year.
ACKNOWLEDGMENTS

We wish to acknowledge the following agencies and people for their cooperation and assistance in carrying out this project: Ramona Turkey Growers Association for the use of its facilities, and especially Mr. A. E. Matlack, its manager, for his continued cooperation; the Zoological Society of San Diego for its laboratory facilities; Mr. T. J. Taylor, for his technical assistance; and, Dr. P. R. Edwards, University of Kentucky for aid in identification of Salmonella species isolated.

REFERENCES

A REVIEW OF THE LITERATURE ON NEWCASTLE DISEASE

BY F. R. BEAUDETTE, D.V.M.

New Jersey Agricultural Experiment Station, New Brunswick, New Jersey

INTRODUCTION

Usually the first description of a new disease escapes attention unless the author has found the causative agent. Such was the case when Kraneveld (1) described a disease in the Dutch East Indies in 1926. However, when what ultimately proved to be the same disease appeared in England in 1926, Doyle (2) found it to be caused by a filterable virus, and named the infection Newcastle disease. In the years that immediately followed, investigators in India, Dutch East Indies, Philippine Islands, Korea, and Ceylon, describes poultry diseases under a variety of names, but still these had many features in common. There were differences, notably the presence or absence of virus in the blood, but for the most part the diseases described were sufficiently alike to cause investigators to exchange viruses for purposes of cross-immunity studies. The studies made with virus exchanged between the Dutch East Indies, India, Philippine Islands and England confirmed the fact that, regardless of names and minor differences, the diseases were immunologically identical.

Somewhat later, the Japanese workers showed that their viruses were identical to the Philippine virus.

For a number of years, the absence of virus in the blood of birds affected with the Dutch East Indies virus was disconcerting, but then virus began to appear in the blood stream and organs of birds inoculated with the strain that had been carried continuously in the laboratory. And, curiously enough, about this time the same change was noted in spontaneous field cases. Therefore, the one important difference that had characterized the Dutch East Indies disease had now seemingly disappeared.

Although this disease has never appeared in the Western Hemisphere, the fact that it causes nearly 100 per cent mortality should be sufficient reason to attract our acquaintance. Moreover, by reaching England, the disease showed its ability to be transported thousands of miles so that there is no reason why it could not reach our shores as easily. Since the disease received but passing notice in a recently published book it is hoped that this review of the literature will serve a useful purpose.

SYNONYMS

The disease was first referred to in the literature by Kraneveld (1) in 1926, merely as one prevalent among poultry in the Dutch East Indies. In 1927, Doyle (2) proposed the name “Newcastle Disease” because the first outbreak identified by him occurred at this place on the river Tyne in England. An outbreak at Ranikhet in India prompted Edwards (3) to refer to it as “Ranikhet” disease in 1928. In the

1 Journal Series of the New Jersey Agricultural Experiment Station, Rutgers University, department of poultry husbandry.
same year Rodier (6) described it as the “Philippine Fowl Disease.” Picard (4, 5) reported the disease under the name of “Pseudo-Vogelpest or Pseudo-Fowlpest,” and in the Philippines Kee (8) published “Notes on an Outbreak of Poultry Epidemic.” Here it should be mentioned that the “Pseudo Fowl Pest of Egypt” described by Lagrange (9) in 1929, has since proved to be an atypical fowl plague. Konno, Ochi and Haschimoto (14) in 1929, described their “Neue Geflügelseuche in Korea” and compared it with the “Batavia disease.” In 1930, Gomes (17) described “An Avian Disease New to the Philippines,” and Farinas of the same place called the disease “Avian Pest.” The outbreaks in Australia were described as the “Pseudo-Poultry Plague” (25, 26, 35). In 1931, Kylasmaier (29) in India referred to the disease as the “Madras Fowl Pest.” The Japanese “Korea Hühnerseuche” or “Chosen Keieki” (35) proved to be the disease in question. The name “Avian Distemper” appears in Haddow’s (40) report in 1933 and he mentions that “Doyle’s Disease” has also been proposed as a name. In 1935, Haddow (51) used the name “Doyle’s Disease.” Again in India, Kuppuswamy (29) refers to the disease simply as “Fowl Pest” and the Japanese (58) report it as “Chosen Disease.” However, in 1933, Doyle (43) pointed out that because of the wide-spread distribution of the affection, “Newcastle Disease” was obviously unsuitable but should be retained to avoid confusion. He opposed the designation “Pseudo Fowl Plague.”

SPECIES SUSCEPTIBLE

Naturally:

1. **Chickens:** Practically all reports concern the spontaneous disease in this species so that citations serve no purpose.

2. **Pigeons:** The literature records only one instance of the isolation of the virus from a spontaneous case. In 1939, Iyer (65) found the virus in a pigeon that had been inoculated with pigeon pox virus for the production of vaccine. The bird developed a paralysis which suggested the examination. A filtrate of spleen suspension injected into a fowl caused its death on the tenth day. Its spleen suspension in turn, filtered and injected into a bird immune to Newcastle disease had no effect, but produced death on the eleventh day in a non-immune bird. A third passage was made from this bird and the inoculated bird died on the eighth day. From this bird 4 pigeons were inoculated of which all sickened but only 2 died on the tenth and eleventh days, respectively. Since the original pigeon had been inoculated with pigeon pox and was also found to be infected with Haemoproteus it would be interesting to know whether or not these diseases increased the susceptibility to Newcastle virus.

And yet, the single report notwithstanding, authors frequently mention pigeons as dying during outbreaks in fowls (1, 4, 5, 21, 22, 48, 57).

Doyle’s (2) attempts to infect young or old pigeons by natural means failed. Picard (4, 5) exposed 5 young pigeons (3–4 mos.) and 5 adults to 4 sick fowls and one young pigeon sickened on the sixth day and died on the ninth; the others remained well.

Kee (8) exposed 15 pigeons to sick chickens with negative results, and Crawford (23) reported the escape of pigeons exposed on infected farms.
3. **Turkeys:** This species is frequently mentioned as dying during natural outbreaks (1, 4, 5, 21, 22, 23, 57, 60, 61, 72). And yet, isolation of the virus from a spontaneous case is still to be reported.

4. **Ducks:** These too, are mentioned as dying during natural outbreaks (1, 4, 5, 21, 48, 57). Doyle's (2) attempts to infect ducks naturally failed, and ducks exposed to chickens on Kee's farm (8) remained well. Dobson (64) observed 20 ducks escape infection in association with infected chickens on range and was unable to infect ducks by natural exposure in the laboratory. Likewise, Crawford (23) observed no infection in ducks kept on the same premises with infected fowls. Rodier (6) claims to have seen natural cases and Picard (5) exposed 4 ducks to 3 chickens with the result that one sickened on the tenth day and died on the 14th, a second sickened on the 12th day and was killed on the 14th. Albiston and Gorrie (72) reported that while ducks exhibited a marked resistance during the first Victorian outbreak (mortality was less than 10 per cent) that none died during the second outbreak even though exposed.

5. **Geese:** Appear to enjoy about the same status as ducks as regards field reports, namely, that they occasionally contract the disease (4, 5, 6, 14, 57). Rodier (6) exposed a goose for 2 months with negative results and also exposed a goose with inoculated geese and it remained well. Geese were reported dying in the first Victorian outbreak (72) but not in the second. Picard (5) exposed 2 young geese to diseased chickens for more than 2 months and both remained healthy, but because of frequent field reports he believes they are susceptible.

6. **Guinea Fowls:** Field reports include this species as dying during outbreaks in chickens: (21, 22, 23, 60).

7. **Swans:** Reported by Picard (5) on field evidence.

8. **Parrots:** Reported by Picard (5, 22) on field evidence.

9. **Wild Birds:** As such, and on field evidence, these are reported as dying during outbreaks (5, 21, 48). Farinas (22) mentions mayas, a member of the martin family and a third not identified. Sahai (57) mentions sparrows, and Picard (48) mentions partridge. Death of crows is frequently reported as a feature of natural outbreaks in chickens (1, 5, 24, 57). And yet Crawford (23) reported that on 4 occasions sick or dead crows were found on or near infected premises but their blood, spleen and liver inoculated into fowls failed to produce the disease.

**Artificially:**

1. **Chickens:** The disease is easily reproduced in chickens as will be seen later.

2. **Pigeons:** In the beginning Doyle (2) pointed out that the susceptibility of pigeons to artificial inoculation constituted a valuable means of differentiating this disease from fowl plague. He used 8 pigeons ranging in age from 4 months to 4 years and, with one exception (heart blood and spleen), employed mouth exudate as virus. This was injected intravenously and intramuscularly in doses of 0.5 to 1 cc. with the result that 7 died. The exception was a 3 year old bird given 0.5 cc. of mouth exudate intravenously. Picard (4, 5) found a marked difference between young and old pigeons as regards susceptibility. He used 11 birds from 3 months to 1½ years old and employed saliva virus which was swabbed in the mouth and injected intravenously. Four failed to react of which 2 were swabbed (1½...
years and 3 months old), 1 injected intravenously (1 year old) and the only one injected subcutaneously (4 months old). Later he (48) observed that healthy pigeons did not contract the disease from sick ones if kept together in large cages, but in smaller cages which increased the opportunity of contact the disease was acquired.

Gomez (17) failed to infect a pigeon with 1 cc. of a Berkefeld filtrate of duodenal material given intramuscularly, but a second bird sickened on the 6th day and died on the 8th when given saliva in drinking water.

Cooper (24) reproduced the disease in pigeons with his own Ranikhet virus as well as with Doyle's Newcastle virus and Rodier's Avian Pest virus.

Purchase (28) regularly infected young or old pigeons with 1 cc. of spleen suspension which caused death in 7 to 9 days.

Kylasamaier (29) infected 2 pigeons with liver and spleen emulsion subcutaneously with death in 6 and 7 days.

Acevedo (39) produced illness in the 3rd day and death on the 6th with 1 cc. of citrated blood intramuscularly.

Dobson (47) infected pigeons with virus from the Hertfordshire outbreak in 1933.

Kuppuswamy (49) used 1 cc. of a 1–10 dilution of brain suspension from a dead bird subcutaneously and the bird sickened on the 6th day and died on the 9th. A similar inoculation with material from a live bird produced illness on the 6th day but death not until the 18th day.

Hudson (61) injected 2 pigeons intramuscularly with blood and they sickened on the 6th and 7th days, and died on the 18th and 20th days, respectively.

Reproduction of the disease in chickens and pigeons with virus from a spontaneous case in a pigeon by Iyer (65) has already been cited above.

Crawford (23) injected 4 pigeons intramuscularly with blood with the result that 2 died after 9 days, one sickened after 6 days and recovered, and the fourth showed no ill effects. Of 3 inoculated intravenously with heart blood, 2 died in 6 and 7 days, and the third was not affected.

Many authors mention that drooping of the wings and paralysis of the legs are prominent symptoms in pigeons and that dispnoea as seen in chickens is conspicuously absent. Picard (5) claims never to have seen mouth discharges and dispnoea in infected pigeons. And Kuppuswamy (49) noted the prominence of nervous symptoms and the longer course of the disease.

Manninger (33) reported that Newcastle disease was merely a form of fowl plague, but Doyle (50) pointed out that Manninger had failed to make use of pigeon inoculation as a differentiating feature. So, in 1936, Manninger (55) reported that in December 1930 and January 1931, he injected 13 pigeons serially and that of these 12 contracted the disease after showing convulsions and paralysis and died in 4 to 13 days; the 13th pigeon lived. Then after Doyle's second paper in 1935 Manninger used Doyle's strain which had been carried in chickens to inoculate 12 pigeons with 10,000 to 100,000 lethal dose for fowls and none contracted the disease. He therefore concluded that the pigeon test was too variable and not to be relied upon.

3. Turkeys: Picard (48) states that they are more resistant than ducks and pigeons and claims to have produced symptoms by intramuscular injections.

4. Ducks: Doyle (2) inoculated 2 ducks intravenously with 1 cc. of mouth exudate intravenously and one sickened on the 5th day and died on the 10th:
Picard (5) injected 5 ducks intravenously with 1 to 4 cc. of a 1–10 dilution of saliva or 1 cc. of non-diluted saliva. Curiously enough a duck which had failed to contract the disease after 3 week’s exposure to chickens became ill on the 8th day and died on the 11th with typical symptoms and post mortem findings. It had received 1 cc. of non-diluted saliva. A similarly dosed duck remained healthy as did one that received 4 cc. of a 1–10 dilution. The remaining 2 birds that got 1 and 3 cc. respectively, of a 1–10 dilution became sick on the 10th and 7th days and were well after 8 and 4 days respectively. Picard (5) also depicts a duck infected by simple contact.

Gomes (17) injected 4 cc. of a Berkefeld filtrate intramuscularly and produced death in 5 days.

Purchase (28) was unable to infect either of two ducks injected intravenously and intramuscularly with 1 cc. of spleen suspension.

Dobson (65) injected 3 groups of 10 ducks each intraperitoneally, intravenously and intramuscularly with an emulsion of liver spleen and mouth exudate and was unable to produce infection. In some cases he gave up to 5 cc. intraperitoneally.

5. Geese: Rodier (6) injected 1 cc. of saliva intravenously with negative results but this bird had been previously exposed for 2 months. A second goose got 2 cc. intravenously and became sick on the 8th day. It was killed on the 15th day and was said to be typical. A third goose received 3 cc. of a 1–10 dilution and remained well. Picard (48) found geese more resistant than ducks and pigeons but claims to have produced infection by intramuscular infections.

6. Guinea Fowl: Farinas (22) shows a figure of an infected guinea but further information is lacking. However, Hudson (61) instilled a few drops of a blood and spleen emulsion into the throat of a domestic white guinea of the Madagascan race as well as in a control fowl. The fowl died on the 7th day and on the 6th day the guinea showed difficult movement and paralysis. These symptoms became more marked and the bird died on the 13th day. No catarrhal symptoms or temperature were noted. The brain of the guinea was emulsified and instilled in the throat of a chicken which died of a hyperacute attack.

7 & 8. Swans and Parrots: No reports of inoculations.

9. Wild Birds: Farinas (22) injected mayas and a member of the martin family and produced death in 2 days but without typical symptoms. In mayas death occurred after 5 days when given contaminated water.

**Mammals**

Horse: Picard (5) injected 3 cc. of mouth mucus subcutaneously and 10 cc. of a 1–10 dilution of the same intravenously with negative results.

Donkey: Cooper (24) observed a distinct reaction after the intravenous injection of filtered mouth washings in an attempt to produce an immune serum.

Cow: Two cows gave negative results when inoculated by Picard (5) as described above for horses. Kylasamaier (29) was unable to infect calves with a liver suspension.

Sheep: Farinas (22) found these to be negative.

Pigs: Doyle (2) injected a 4 months-old pig subcutaneously with 4 cc. of mouth exudate with negative results. Also reported non-susceptible by Farinas (22).
Cats: Reported non-susceptible by Farinas (22).

Rabbits: One cc. of known infectious mouth exudate subcutaneously and intravenously failed to infect rabbits according to Doyle (2). Picard (5) failed to infect rabbits with large doses subcutaneously and intraperitoneally; and Acevedo (39) had negative results from a subcutaneous injection of 2 cc. of spleen emulsion. Dobson (64) injected 2 rabbits intravenously with 0.5 and 1 cc., respectively, of a liver and spleen emulsion without effect. Species reported non-susceptible by Farinas (22) and Konno et al. (14).

Guinea Pigs: Doyle (2) reported negative results in 2 guinea pigs injected subcutaneously with mouth exudate. Picard (5) failed to infect them with large doses given subcutaneously and intra-abdominally, or by feeding and duodenum. Acevedo (39) failed with 2 cc. of spleen emulsion subcutaneously, and Farinas (22) and Konno et al. (14) reported negative results.

Rats: Non-susceptible according to Farinas (22), and Gomez (17) was unable to produce infection with 1 cc. of a Berkefeld filtrate intraperitoneally.

Mice: Konno et al. (14) reported these as non-susceptible. Kraneveld and Nasoetion (63) were unable to infect mice with the largest tolerable dose given intracerebrally with any of 25 strains of Dutch East Indies virus. At the same time they succeeded in infecting mice by the same route with Holland and Switzerland strains of fowl plague. These findings were confirmed in a later report (69). The dose used was 0.5 cc. of a 1–10 brain suspension of which 1 cc. of a 1–100,000 dilution was regularly fatal for chickens. The virus remained strictly localized at the points of inoculation with one exception in 14 trials. Nine days was the longest time virus persisted. Even by serial passage at intervals of 1–2 days it was impossible to adapt the virus.

However, Burnet (73) instilled a high titre virus intratracheally and 24 hours later gave a second dose to spread the material from the primary lesion and observed that some mice died 3–4 days after the second dose and showed complete or various degrees of lung solidification—gross lesions indistinguishable from those caused by influenza virus. Virus was detected 2–5 days after the second inoculation but in smaller amounts than in influenza. He too failed to establish the virus by serial passage.

Ferrets: According to Burnet (72) intratracheal inoculation caused no symptoms or thermic reaction although 1 of 2 animals developed mild neutralizing antibodies.

GEOGRAPHIC DISTRIBUTION

Dutch East Indies:

The first known outbreak that ultimately came to be known as Newcastle disease occurred in Batavia, Java and was described by Kraneveld (1).

On the basis of reports from veterinary officers Picard (5) compiled a list of infected places throughout the Dutch East Indies as of Sept. 1927.

Sumatra: The towns of: Balige, Benkoelen, Fort de Kock, Keboemen, Koetaradjak, Padang, Pagaralam, Sibolga, Tandjoengkarang, and Teloekbetoeng.

Java: The towns of: Bandoeng, Bangkalan, Batavia, Bondowoso (Besuki),
Buitenzorg, Cheribon, Djember, Jagjakarta, Kediri, Koedoes, Magelang, Malang, Modjokerto, Pasoeroean, Pekalongan, Semarang, Soekaboemi, Soerabaia, Soerakarta, Tegal and Welteverden.

Madura: The town of Parnekasan.

Bali: The town of Singaradja.

Lombok: The town of Mataram.

Borneo: The towns of: Kandangan and Tangerang.

Celebes: The towns of: Garontalo, Makassar, Manado and Watampone.

No reports of outbreaks came from the Island of Soembawa, Flores, Soemba (Sandlewood) or Dutch Timor.

England:

The first outbreak known occurred at Newcastle on Tyne in Northumberland County, in March 1926 and was reported by Doyle (2) in 1927. Other outbreaks positively identified at this time occurred in Somerset and Staffordshire counties. Later he (50) reported that 11 counties were involved. No further outbreaks occurred until August 1933 in Hertfordshire (47). This one was eradicated and the disease had not reappeared up to 1939 (64).

Philippine Islands:

The first outbreak was reported in Manila by Rodier (6) in 1928. In the same year Kee (8) described an outbreak on the Manila South Road midway between Paranaque and Las Piñas in the Province of Rizal. Gomez (17) reported that from Sept. 1927 to Feb., 1928 the disease raged in Manila and neighboring towns in the Provinces of Bulacan, Rizal and Laguna and that the disease reappeared about the same time in 1928 and 1929. Kretzer (21) mentioned the towns of Malabon north and Los Baños south of Manila.

According to Farinas (22) the disease began in Manila in Sept. 1927 and was found in the districts of Sampalac, Malate, Ermita, Paco, Pandacan and Quiapo. Other affected villages in Rizal Province were San Francisco del Monte, San Juan del Monte and Malabon. In Laguna Province the disease occurred at San Pedro Tunasan, Calamba, Biñan and Los Baños. In 1928–29 and 30 cases came from Maypajo, Guadalupe, Pasay, Fort McKinley, Taguig, Pasig and Malabon in Rizal and from the districts of Santa Mesa, Santa Cruz, Quiapo, Malate, Sampaloc and Ermita San Lazaro, Singalong and Pandacan in Manila.

India and Federal Malay States:

The first known outbreak occurred in July, 1927 at Ranikhet and later spread throughout the Kumaon Hills according to Edwards (3). Cooper (11) reported an outbreak in the latter part of 1928 in the Garhwal district and the United Provinces, and others in January and April, 1929 at Lucknow, Turkhud (27) mentioned an outbreak at Kodaikanal in 1929–30. In 1931, Kylasamaier (29) reported widespread outbreaks in the city of Madras in 1929–30 as well as in the districts. According to Kuppuswamy (49) the disease appeared at Tassek, Province Wellesley in the Federated Malay States in October, 1933. Shah (52) stated that the disease occurs extensively in the Northern Provinces. In 1936, Naik (56) stated that he diagnosed
the disease first in the Bombay Presidency in 1932. At Christmas in 1935 an outbreak occurred at Ramabai Mukti and at Kedgaon in the Poona district on Dec. 20. of the same year. In January, 1936 he reported outbreaks at Bori and Dhond. Reporting for Bihar and Orissa provinces Sahai (57) stated that the disease was perhaps the most common there and likely also in other provinces. Finally, in 1937, Shirlaw (59) reported that the disease shows no sign of abating in severity or incidence and that it remains the most important disease of the Indian poultry industry.

**Ceylon:**

In 1930, Crawford (23) reported that outbreaks appeared in Colombo and surrounding districts in 1927-29 and 29.

**Japan and Korea:**

Ochi and Hashimoto (12) and Konno et al. (14) reported that the disease appeared in middle Korea in 1926, and thereafter spread to Northern Korea. They even say the disease has existed there since 1924. Nakamura et al. (42) found the Sato strain of virus isolated in Japan identical with the Korean virus.

**Australia:**

The first outbreak occurred in November 1930, at Dalyston near Wonthaggi and at Cheltenham according to Johnstone (26). The disease was diagnosed by Albiston on Dec. 11. Except for Dalyston all affected farms were within 20 miles of Melbourne, Victoria. In Johnstone's (37) report of the second outbreak he also mentions the town of Kyneton as involved in 1930 during which 72 farms having 22284 head were involved. The second outbreak was diagnosed Oct. 6, 1932 and the last occurred March 6, 1933. Albiston and Gorrie (72) stated the 1930 outbreak began at Anverloch a sea-side village and spread to suburbs of Melbourne.

**Kenya:**

According to Hudson (60) the Annual Report of the Director of Agriculture of Kenya for 1935, makes reference to a disease of birds from Mombasa in November, and that the disease had caused extensive losses for 12 months on Mombasa Island. The second outbreak occurred in the Eastleigh district of Nairobi. In a second report (61) it is claimed that the disease has existed for years in epizootic form along the coast of Kenya and Tanganyika, at least from Mombasa to Lindi and that periodically it flares up into an extensive outbreak.

The disease was diagnosed in Palestine by Komarov (67).³

**SEASONAL VARIATION**

Too few reports are available, but Konno et al. (14) state that the disease appears in the colder seasons (fall and winter) and subsides in the summer. On the contrary, Sahai (57) reporting for Bihar and Orissa provinces in India states that the disease usually appears about June or July after the rains and continues to Decem-

³ The review's copy of this paper and translation were lost in the mail so that its contents cannot be included.
ber or January. Occasionally it continues at all times, but tends to subside in extreme hot weather in enzootic areas.

TOPOGRAPHY OF LAND

According to Farinas (22) this is without effect in the Philippines.

BREED

Picard (5, 48) saw no difference in susceptibility among various breeds (Java), nor did Crawford (23) (Ceylon) or Sahai (57) (India). On the other hand Kee (8) decided that Leghorns were the most susceptible and then B. P. Rocks, R. I. Reds and the Cantonese. And Albiston and Gorrie (72) found mortality higher in Leghorns than in the heavier breeds like the Australorps and R. I. Reds (Australia).

SEX

Rodier (6) alone mentions sex and states that both are susceptible.

AGE

Kraneveld (1), Rodier (6), Crawford (23) and Picard (48) merely state that all ages are susceptible. Picard (5) observed no difference in birds purchased on the market for experimental purposes, except that birds 3 months old were not so typical as adults in that dispnoea and nervous symptoms were seldom seen. Generally speaking, natural outbreaks concern adults but this may be more a case of opportunity for infection than age. Thus Hudson (61) saw one outbreak in which practically all adults died but a number of chicks survived. In the 1933, Hertfordshire outbreak the farm was made up of 6000 birds 6–8 months old, 4000 from a few days to 6 weeks old, and 30 old hens. Young chickens of all ages died but the old hens were never affected according to Dobson (64). Then, all birds were killed and the premises cleaned (?), but seven weeks later 1500 day-old chicks were purchased and within a week the disease appeared in them. In laboratory contact experiments with this virus several old birds escaped infection which readily attacked birds 6 months old. But, after several passages the virus infected young and old alike by contact.

PERIOD OF INCUBATION AND COURSE

According to Doyle (2) the period of incubation varies from 4 to 11 days with an average of 5 whether infection is produced by injection or contact. However, filtrates or stored virus may prolong the period to 20 or 25 days. Five days as an average is also given by Picard (5) with 3 as the shortest and 14 the longest. Gomez (17) gives 4–6 days with an average of 5, but adds that in contact infection it may be prolonged to 8. According to Farinas (22) the period depends on the dose, some birds dying 2 days after a large dose while moderate doses provoke symptoms only after 3–4 days and with alternated virus it may be prolonged to 14 days. He gives 4–5 days as an average. Cooper (24) used filtered mouth washings injected subcutaneously and found the period to vary from 2–14 days, but the majority developed disease within 5 days. The average in 96 birds came to 3.55 days. Like-
wise the average duration of illness was 1.55 days. The following table records his data on 11 birds that recovered:

<table>
<thead>
<tr>
<th>Days</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day of first symptom</td>
<td>✘</td>
<td>✘</td>
<td>✘</td>
<td>✘</td>
<td>1</td>
<td>1</td>
<td>✘</td>
<td>✘</td>
<td>✘</td>
<td>✘</td>
</tr>
<tr>
<td>Duration of symptoms in days</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>3</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

With the Korean virus Nakamura et al. (35) found the period to be 5 days usually and death on the 7th day, but it may be as long as 17 days and the course an additional 13 days. In serial passage using brain virus given subdurally the minimal lethal dose decreased and the period of incubation and course were shortened. Thus in passages 1–5 the period was 4.7 days and the course 2 days. After 35 passages most of the inoculated birds were dead within 4 days so that the incubation period amounted to 1–2 days. Introduced subcutaneously, brain virus of the 1–15 passages showed an incubation period of 4 days, and death on the 7th or later, but after 32 passages or more, symptoms appeared after 2 days.

Shah (52) and Sahai (57) are in agreement that the shortest incubation period is 2 days and the longest 14 with an average of 3–5.

Konno et al. (14) found the period to vary, according to the mode of inoculation, between 2–8 days with 70 per cent falling within 4–5 days.

In Hudson’s (61) report death occurred on the average on the 9th day in 8 contact cases, on the 8th day in 3 injected intravenously and on 5.6 days in 11 birds injected intramuscularly and swabbed in the larynx.

Manninger (33) claimed to have reduced the incubation period and course of the disease by rapid serial passage of Doyle’s virus to the point where it approximated that of fowl plague. But on repeating his experiments Doyle was unable to confirm this. In fact he (43) states that the frequent passage of virus through fowls over a period of 8 years has failed to bring about any reduction in the period of incubation.

**SYMPTOMS**

In general, there is agreement among the various authors regarding the symptoms of this disease. Differences may be accounted for by observations on spontaneous cases by some authors and on artificially produced cases by others. Likewise, the symptoms exhibited probably depend on a number of factors such as the dose of virus, the mode of inoculation (in experimental cases) and on the resistance of the host. Some have described peracute, acute, subacute and chronic cases, but in an outbreak of almost any disease involving a large number of individuals examples of each type may be seen for it is natural that the most susceptible or those getting the largest dose should die first and from a peracute attack while the most resistant birds or those getting a small dose either escape entirely, or suffer a chronic attack. Between these extremes the acute and subacute cases are to be found.

In any event the onset of the disease is usually sudden (6, 29). Death is said to take place occasionally without symptoms (2). The bird is described as showing
dullness (1, 14, 23, 24, 29, 60), droopiness (17, 22, 23), indisposition (6), weakness (5, 17), rough feathers (5, 17, 22), droopy tail (5, 37), coughing (2, 29, 39), sleepiness (2, 5, 14, 37), half-closed or closed eyes (1, 2, 17, 60) or slowness (5, 22). Most authors agree that there is an early rise in temperature (2, 8, 17, 22, 29, 50, 57, 60, 61, 62). Doyle (50) found this to fluctuate between 0.8 and 3.2 F. with an average of 1.9. A few (24, 50, 62) report what the disease is non-febrile. One of these (24) however, employed filtered virus which may account for his findings. In fact, he reported a preliminary drop in temperature. The temperature curve, after the preliminary rise, may be irregular (17, 21, 22), but there is general agreement that it becomes subnormal before death (2, 5, 17, 22, 29, 50, 61, 62). Loss of appetite is an early sign (2, 5, 8, 17, 21, 22, 24, 29, 39, 49, 60). A few mention excessive thirst (5, 6, 8, 22) and one (39) the lack of it.

Diarrhea is another early symptom (5, 6, 8, 17, 21, 22, 23, 24, 37, 49, 57, 62), but one (60) reports it not common. The droppings are described as watery, profuse, yellowish white, algae green, greenish-white or greenish-yellow (14, 23, 29, 39, 49, 61) and several (5, 17, 37, 62) refer to their disagreeable odor. One (62) reports only soft droppings. Bloody diarrhea is mentioned (6), especially in fat birds (22) or only sometimes in early stages (62). The respirations are increased according to some (2, 23, 37), or there is dispnoea (5, 14, 23, 29, 48), but the majority refer to the characteristic prolonged inspiratory act with outstretched head and neck and half opened beak (1, 2, 5, 6, 8, 14, 17, 21, 22, 37, 49, 57, 60, 62). Doyle (2) observed this in 70 per cent of the cases and Sahai in 50 per cent. Gurgling (49), coughing (60) or sneezing and crooking (5) sounds may be produced. Another prominent symptom appears to be distention to the crop with gas, or a sour, foul, ill-smelling brownish watery fluid or undigest food (2, 6, 17, 22, 23, 29, 37). Invariably, in chickens at least, there is a mucus discharge from the nostrils (2, 6, 8, 14, 17, 21, 24, 60) and salivation (22, 62) or the collection of mucus in the mouth (2, 5, 6, 8, 14, 17, 21, 23, 24, 37). Sometimes the mucus hangs in threads from the beak (2, 5, 22, 29, 37, 49). Still, Johnstone (37) reports that while mucous in the mouth was seen in the first Victorian outbreak it was not prominent in the second. There may also be conjunctivitis (8) or a discharge from the eyes (1, 5, 60). As a result of mucus in the mouth the bird may exhibit frequent or continued swallowing (5, 17, 22). Picard (5) looks upon this as an early sign. Or, the bird may shake its head to dislodge or expell mucus (5, 14) Wilting (22, 39) or cyanosis of the comb and wattles is also a very common symptom (2, 5, 8, 22, 29, 37, 39, 48, 49, 60, 61). Picard (5) observed cyanosis in 50 per cent of the cases and Farinas in the majority of cases.

Edema of the head (5, 48), neck and breast (5), eyelids (5) or comb has been reported.

In more protracted cases the bird may exhibit a great variety of nervous symptoms. Weakness of the leg or legs, progressing to paralysis, appears to be very common (1, 2, 5, 14, 17, 21, 22, 23, 24, 29, 37, 39, 48, 49, 60, 62, 72). Similarly, drooping or paralysis of the wing is seen (14, 17, 23, 29) and Gomez (17) saw one case of paralysis of the body muscles. The bird may pick at its tail or breast feathers or suffer epileptiform attacks (5). The head may be drawn down (5, 29) or over the back (5, 62) or merely drawn in (5). Albiston and Gorrie (72) report that torticollis is common and an S-shaped distortion or double twist in the neck has been
NEWCASTLE DISEASE

described (22, 37). Periodic shivering (37), convulsive movements (37), incoordination (1, 2, 24, 72), chronic spasms of head, neck and body (5, 22, 24) the latter accompanied by a hiccup-like sound, walking in circles, forward or backward (5) and twitching of head (2, 8, 22, 23) or tail feather (72) have been described. Farinas (22) mentions cloudiness of the cornea. Kaura and Iyer (62) described a bird that turned somersaults—it finally made a complete recovery. Some birds suffer alternating periods of excitation (5, 23) and depression (5, 29). Standing motionless or with head drawn over back and eyes fixed has been mentioned by Picard (5) who claims that if the latter type of bird is disturbed it dies at once. Turning movements are mentioned by Farinas (22) and Albiston and Gorrie (72).

Farinas (22) states that recovery requires about one week if paralysis does not occur. If it does death occurs in about 20 days. If one leg is affected the toes curl inward and there is atrophy (22), or it drags and causes the bird to stumble (5). Gomez (17) also reports atrophy of the affected member. If both legs are paralyzed they are stretched backward (22).

Shirlaw (59) expressed the belief that the paralytic type of disease was increasing in India.

Rodier (6) observed that 99 per cent of the birds which recovered develop paralysis and of these only 5 per cent finally recovered.

According to Picard (5) recovered birds (male and female) are sterile or they may retain a hoarse voice or a squint. Recovered birds are regarded as useless by some (5, 37).

Picard (22) noted cold feet, and coma is mentioned by some (29, 39, 49). The carcass has a dry, stiff appearance according to Kylasamaier (29).

There is general agreement that the symptoms in pigeons, ducks and geese are predominately of the paralytic type and rarely of the exudation. That is to say, the gasping symptom and collection of mucus in the head is rarely seen. This would at least suggest that the paralytic cases in chickens identify themselves as the more resistant members of the flock. Furthermore, if Shirlaw’s (59) observation be correct then it may mean that a more resistant population is being built up in India, where, because of the prevalence of the disease, a selection process would certainly have an opportunity to make itself apparent.

MORTALITY

Kraneveld (1) first reported (Dutch East Indies) that the mortality was frequently 100 per cent and so it is stated to be by many workers. The outbreak at Newcastle had killed most of the 700 and others were dying when the flock was destroyed. The mortality was 100 per cent of the 90 birds in the Somerset outbreak as well as in 3 of the 5 affected farms visited in Staffordshire. It was already 90 per cent in another 500 bird flock and deaths were continuing while in the fifth flock the disease was just beginning. It was mentioned also that 2 birds recovered out of 80 in another flock (2).

Picard (5) (Dutch East Indies) recorded 136 dead out of 144 that contracted the disease by contact. Of the 8 recovered 5 showed persistent nervous symptoms. Kee (8) lost 2700 from a flock of 3000. Gomez (17) recorded figures for two flocks in which 334 died out of 350, and in another 168 died out of 181 (Philippines).
Farinas (22) (Philippines) reported a mortality of 80 per cent in a large flock, and 100 per cent in many flocks. He adds that when the disease reappeared in 1928–30 fewer fowls (50 per cent) contracted the disease and 30 per cent recovered.

In Ceylon, Crawford (23) stated that mortality was about 100 per cent, a figure Sahai (57) gives for India. In northern India Shah (52) states that in some localities half or a large portion of the flocks is annihilated.

Reporting on small Indian village flocks Naik (56) cites 87 deaths out of 88 with the one still sick, and in another village 115 deaths out of 123 with 8 still affected.

In Kenya Hudson (60) reported 22 dead out of 25 in a Mombasa flock and in the laboratory 27 died out of 28 infected with this strain. The laboratory tests with the Narobi virus killed 100 per cent.

Albiston and Gorrie (72) killed 221 in 4–13 days or 98 per cent of the 225 inoculated with the Australian virus—the 4 which survived had received filtered virus. They say an exact estimate of mortality is impossible because of the slaughter policy, but it ranged from 50 to 100 per cent when the disease was allowed to run its course.

Some idea of the damage done by the disease is gained from the figures compiled by Picard (5) from reports of veterinary officers in the Dutch East Indies as of September, 1927. A total of the definite figures given amounts to 1,821,760. And this does not represent the total because many officers reported “Thousands,” “not to be estimated,” “200 per village” or “two-thirds of each flock.”

Farinas (22) estimated that for the year ending in September 1928 a loss of 75,000 occurred around Manila and that by November, 1930 this mounted to 140,000. In many flocks 90 per cent died.

The first Victorian outbreak had involved 31 flocks with a loss of 6824 fowls at the time of Johnstone’s report (26) and a final accounting showed a loss of 22284 birds on 72 farms.

Konno et al. (14) claim never to have seen a naturally infected bird recover. But the mortality is not always nearly 100 per cent as evidenced by the Hertfordshire outbreak in 1933 in which the old hens escaped entirely.

POST MORTEM FINDINGS

On the basis of over 400 autopsies, Picard (5) made certain general statements which tend to express the views of others. According to him, a certain number are wholly negative. Alterations in acute cases are more definite than in subacute or chronic, yet no single pathological change served as a guide to the intensity of the clinical disease. Chronic cases generally exhibited a total lack of morbid changes. However, in more than 65 per cent of the cases hemorrhages were found on the mucous membrane of the digestion tract.

External appearance:

Acute cases are well fleshed but chronic cases show emaciation (5). The carcass presents a dry, stiff appearance according to Kylasamaier (29), but Farinas (22) claims that there are no external changes to be used for condemnation purposes. The feathers are untidy and ruffied according to Hudson (60) and there is cyanosis of the comb and wattles (17, 60) or edema (60). The feathers around the vent are soiled with excreta (5, 17, 60). Hemorrhages in the skin are reported only by Albiston and Gorrie (72) and darkening of the skin is mentioned by Johnstone (37).
Subcutaneous tissues:

Edema is mentioned by many in the region of the head neck and entrance of thorax (22, 23), and this occurs in 30 per cent of the cases according to Picard (5). But Konno, et al. (14) seldom saw edema and Albiston and Gorrie (72) saw it in the first Victorian outbreak but not in the second. Congestion of the subcutaneous vessels is mentioned by Kylasamaier (29) and Acevedo (39), hemorrhages in the tissue (5, 72) and peritracheal edema (72).

Digestion tract:

1. Mouth and pharynx: A collection of foul-smelling mucus in these parts in regularly reported (5, 6, 14, 17, 22, 24, 55, 72) with congestion of the mucosa (15, 28) and occasionally hemorrhages in the pharynx (5, 22). Hudson (60) described pitting of the mouth mucosa due to ulcers, and there is desquamation of epithelium of the mouth and pharynx according to Acevedo (39). Excoration of the epithelium of pharynx and esophagus with an appearance of being sprinkled with bran was seen once by Gomez (17).

2. Esophagus and crop: Rodier (6) described a periesophageal inflammation accompanied by petechial or streak hemorrhages in all cases. Hemorrhages in the serous coat (72) and extending as far as the crop were seen in 20 per cent of the cases by Farinas (22). Kuppuswamy (49) reported hemorrhages over the the lower esophagus. Serous infiltration and hemorrhagic extravasation in connection tissue surrounding crop and esophagus has been reported once (17). Picard saw no changes anterior to the crop. Suffusions in the serous coat are reported by Acevedo (39) as well as desquamation of epithelium of esophagus and ecchymotic submucus hemorrhages in the esophagus (22, 39).

The crop itself is invariably reported to contain gas, brownish or dirty, four-smelling or nauseating liquid (2, 5, 6, 8, 17, 22, 72). Farinas (22) reported stasis if crop was full, and in the empty crop a whitish semi-transparent liquid.

3. Proventriculus: Hemorrhages on the mucosa of this organ constitute a valuable diagnostic finding (2, 5, 14, 17, 22, 23, 24, 29, 37, 57, 72). These are usually located on the papillae or near the entrance of the gizzard (5). In some cases the whole surface is hemorrhagic (5). Picard states that in the total absence of hemorrhages on the mucosa, blood clots the size of a pin-head may be visible from the exterior. This reviewer ventures the opinion that these were Tetrameres and not blood clots. According to Hudson (61) hemorrhages are present in 80 per cent of the cases. Kuppuswamy (49) refers only to congestion.

4. Gizzard: Here again hemorrhages beneath the cuticula are highly significant. Sometimes they are not seen and Konno et al. (14) seldom observed them.

Regarding these hemorrhages in the proven triculus and beneath the cuticula of the gizzard-changes likewise suggestive of fowl plague—it is claimed that in Newcastle disease they are less constant, less profuse and less sharply delineated.

5. Intestine: Authors invariably describe the presence of a catarrhal enteritis (2, 22, 29, 57). In some cases this is confined largely to the duodenum, in others it extends throughout the tract. Mention is made of hemorrhages in the mucosa (6, 8, 39, 60, 61) of the duodenum (5, 14, 17, 22, 61, 72), jejunum (5, 22), ileum (5, 22), ceca (in 50% of the cases according to Hudson 61) (5, 61), colon (5), cloacae (57, 61)
and rectum (5, 61). Picard (5) and Farinas (22) report the cecal mucosa reddened. The rectal mucosa has a streaked appearance according to Farinas (22). Actual hemorrhage in the lumen of the duodenum is reported by Gomez (17) and Albiston and Gorrie (72). Severe inflammation of cloacal mucosa is mentioned by Acevedo (39) and Kuppuswamy (49).

Crawford (23) was the first to describe ulcers in the intestine and he reports them in over 50 per cent of the cases throughout small and large intestines. These were from pin-head size to ½ in. in diameter, while others were elongated to 2 inches. They were covered by a tough necrotic membrane of greenish color and when removed they exposed a red surface. These may be seen through the serous coat. The presence or absence of these was not associated with any change in the symptomatology.

In following the literature as it appeared one is rather struck by this finding so different from previous descriptions. But, Crawford himself remarks that ulcers were not seen in earlier cases.

And, as if to confirm Crawford’s report in Ceylon of 1930, Kylasamaier (29) in India reported pin-point ulceration of the cloacal mucosa and bran-like deposits in the intestine and cloaca. Then Acevedo (39) in the Philippines described follicular ulcers of pinhead size throughout the intestine. Likewise, Kuppuswamy (49) described diphtheritic deposits in the large intestine and minute ulcers of the ceca. The largest ulcer was ½ inch long. Finally, elongated caseous ulcers are mentioned by Hudson (61) in Kenya.

6. **Liver:** According to Picard (5) this organ is not visibly affected. The parenchyma is slightly reddened according to Gomez (17) and Farinas found only focal necrosis in some cases. Konno et al. (14) and Acevedo (39) mention hyperemia but the latter states that it is pale and friable in most parts. The organ was normal in 50 per cent of Hudson’s (61) cases, rarely enlarged and when abnormal showed mottling with fine, diffuse, pale dots.

7. **Gall bladder:** Generally normal (5). The mucous membrane showed a slightly striated congestion in a small number of cases.

8. **Pancreas:** Not visibly altered (5).

**Respiratory tract:**

1. **Nasal cavity:** Mucus in the nasal cavity or sinuses is mentioned by several reporters (5, 6, 14, 21, 72), although some of it may have originated in the mouth. Farinas (22) states that the nostrils are not clogged as in a cold.

2. **Larynx, trachea and lungs:** Hudson (60) reports hemorrhages constantly present in upper larynx, which, in acute cases, tended to ulcerate. In less acute cases he found more necrosis and ulceration in the larynx. Or, the larynx may contain a loose plug of yellow cheesy material or a mass of adherent necrotic tissues on the floor. In another place (61) he describes cheesy material in the sinus or beneath the eye lids, and considers the laryngeal lesions characteristic since they occurred in 90 per cent of the cases. He also reports that hemorrhages rarely extend into the trachea. Since such marked changes have not been reported by others the question of an intercurrent disease naturally presents itself.
Konno et al. (14) mention slight hyperemia of tracheal and lung mucosa, but Kraneveld mentions a croup-like pneumonia.

Doyle's (2) observations based on over 200 experimentally or naturally infected birds showed the trachea usually unaffected, but with occasional slight congestion. The lungs were generally normal, occasionally congested.

Picard (5) reported congestion of larynx and anterior trachea with slight congestion and emphysema of the lungs, and Albiston and Gorrie (72) mention only slight injection of tracheal mucosa.

Cooper (24) was struck by the absence of lung lesions in spite of the extreme respiratory distress. He found hemorrhages in the lungs but rarely.

Gomez (17) reported congestion and edema in the lungs and Farinas (22) mentions occasional congestion or emphysema.

Reproduction and urinary systems:

1. Kidneys: In general these are normal according to Picard (5), sometimes they are swollen and dark (5, 14, 22, 39). Gomez (17) found only reddening of parenchyma. Farinas (22) found urate deposits in protracted cases and Hudson (61) mentions that in some the tubules were prominent.

2. Ovary, oviduct and testes: The ovary and oviduct are congested from a slight to a deep red and fully developed ova showed hemorrhages in the capsule, distention of follicular vessels with occasional ruptures so that yolk substance entered the abdominal cavity (5). In fact, Picard considered the latter as pathognomonic of the disease. Farinas (22) found no changes of importance except an obnoxious characteristic odor and marked inflammation of ovaries with congestion of oviduct. Konno et al. (14) and Acevedo (39) reported congestion of ovary and oviduct.

Farinas (22) and Hudson (61) reported the testes normal while Picard (5) and Konno et al. (14) found a slight hyperemia.

Circulatory system:

Hemorrhages are frequently reported on the heart (2, 8, 14, 17, 21, 57, 72). Picard (5) found petechiae on the epicardium in 45 per cent of his cases. They were more common in fat birds according to Farinas (22). Acevedo (39) mentions petechiae on the epicardium and aorta and ecchymoses on the endocardium. Hudson (61) found hemorrhages in half the cases. Hemorrhages in the heart muscle have also been reported (2, 14).

The pericardial sac is occasionally reported to contain small or large quantities of fluid (3, 22, 37, 39, 72). According to Picard (5) and Albiston and Gorrie (72) the quantity may be considerable and the fluid clots on exposure. Acevedo (39) refers to a sero-fibrinous exudate. According to Picard (5) the blood is normal and coagulates well.

Nervous system:

Rodier (6) found no gross changes in the brain in a limited number of paralytic cases, nor did Picard (5). Acevedo (39) found none in the cerebrum, but the hind brain, medulla and meninges were congested. Hudson (61) also reported congestion of meninges and scattered, fine petechiae in one case.
Muscles:

Johnstone (37) found the muscles darker than normal. Hemorrhages in thigh and pectoral muscles are reported by Acevedo (39). These are also reported by Albiston and Gorrie (72) as rare.

Bones—marrow and spleen:

Farinas (22) found the occipital, frontal, and parietal bones highly congested in a number of cases. According to Picard (5) the bone marrow is not visibly altered. The spleen has been reported as not altered (5, 61) slightly swollen (39, 61), occasionally soft (61) or rarely showing petechiae (22) or ecchymoses (37).

Fukushima et al. (36) studied normal spleen weights in 477 birds and expressing this in relation to a kilo of body weight found a variation from 0.8 to 3.6 with an average of 1.58. Then spleen weights of 8 effected birds were taken and found to vary from 0.5 to 2.0 with an average of 1.44. Of the 8 birds 3 had been killed before the disease ran its course and the weights were 1.7 to 2.0 or an average of 1.58. But, in the 5 dead birds the weights were 0.37 to 1.8 or an average of 1.2. They then showed that the spleen weight decreased in direct relation to the duration of illness.

Cavities and serous membranes:

A sero-fibrinous peritonitis is mentioned once. Hemorrhages on the visceral surface of sterum are occasionally reported (60, 72). Hemorrhages on the gizzard fat were found in about 20 per cent of the cases by Doyle (2) and were also seen here by others (5, 17, 22, 37, 39, 57, 60, 72). He did not see them on the sterum. Rodier (6) found hemorrhages on all serous membranes. Petechiae of the peritoneum in the region of the ileo-cecal valve are mentioned by Albiston and Gorrie (72).

Histopathology:

Picard (5) found no changes of importance, but in the lungs there was a distention of the alveoli and laceration of septa owing to the interstitial emphysema. Gomez (17) found desquamation of duodenal mucosa. The goblet cells were numerous and prominent. In some cases the intestinal glands had disappeared and in their places were areas of cellular infiltration. The capillaries of villi were engorged and some cells were extravascular. There was a serous infiltration of submucosa and distention of vessels in the muscular coat. The vessels of the liver, spleen and kidneys were engorged, particularly in the liver. The hepatic cells were indistinct in outline and showed a granular protoplasm. Farinas (22) found no changes except hemorrhage in sections of the lungs, esophagus, proventriculus, liver and spleen. He found no inclusion bodies in the brain. Farinas had sent brain and cord material to Pappenheimer who reported cord vessels surrounded by a mantle of lymphoid cells and wandering cells scattered irregularly through the central grey. Intense degeneration was found in many anterior horn cells, sometimes complete necrosis. Perivascular infiltrations were sometimes found in the brain. The peripheral nerves were either normal or showed purely degenerative changes which he thought secondary to cord lesions. In some cases sparse infiltration of lymphoid and plasma cells occurred between the fibers as in fowl paralysis.
Topacio (46) examined fragments of spleen, liver and brain from infected fowls grown in hanging drop preparations of normal chicken plasma, and also skin, liver, and intestine of embryos infected in vitro without detecting intracellular changes of significance. The nuclei of epithelial cells appeared pyknotic.

The most extensive studies have been made by Fukushima et al. (36) who used material from 33 cases of which 8 were spontaneous and 25 were produced by subcutaneous, intramuscular, or intravenous inoculations and by smearing virus in the mouth. Spleen, bone, marrow, liver, brain and saliva were used as sources of virus. The course of the disease was 5 to 9, except in 9 cases in which brain virus caused death in 2 to 4 days.

Briefly, the brain showed regressive changes of the glia cells, hyperplastic processes of the glia cells, perivascular lymphocytic infiltration and hemorrhages.

Spleen showed necrosis, hyalin degeneration, anemia and an increase of leucocytes in the spleen pulp. The experimental cases showed a noteworthy inflammation and necrosis or hyalin degeneration.

The bone marrow showed only microscopically visible necrosis in cases infected by strong virus.

The intestines showed catarrhal or pseudomembraneous inflammation. The proventriculus showed in spontaneous cases hyalin-fatty thickening of the intima of the vessels, regressive changes in the epithelium as well as the propria, round cell infiltration, and hemorrhages in the interstitial interglandular tissue.

The liver and heart showed acute parenchymatous degeneration.

BACTERIOLOGICAL EXAMINATION

No one has ever isolated an organism from cases of Newcastle disease with which the disease could be produced. Cultures have been taken at different stages of the disease from saliva (14), feces (6), blood (2, 5, 23, 14), brain (2, 5), liver (5, 17, 23, 39, 61), spleen (2, 17, 23, 39), bone marrow (2, 5), heart (5, 17, 39), kidney (5), cord (5), and "organs" (1, 22, 14) and these have been tested aerobically (2, 22) and anaerobically (5, 22). Stained smears from blood and organs have been negative. Naturally, birds injected intravenously with such contaminated material as saliva occasionally show organisms in the blood, but Doyle (2) was unable to reproduce the disease with a gram-positive coccus found in such a case.

Picard (5) isolated 2 types of gram-positive staphylococci and a gram-negative bacillus from saliva but fowls did not react to subcutaneous or intravenous injections. Hudson (61) occasionally found the colon bacillus which, of course, is to be expected.

FILTRATION OF VIRUS

Kraneveld (1) failed to obtain an active filtrate through a Berkefeld filter of mouth washings.

The disease was first shown to be caused by a filterable virus by Doyle (2) of England. A 1–100 saline dilution of mouth exudate from 2 recently dead and 2 moribund birds was filtered at room temperature under a negative pressure of 15 in. of mercury. Filtration was controlled by the addition of the cholera organism and 3–5 cc. of filtrate planted in broth and agar. Filtration required less than 15 minutes. The Seitz and Chamberland L5 filtrates were inactive and the birds were
later shown to be susceptible. However, the Chamberland L3 and the Berkefeld filtrates produced infection and death on the 12th and 7th days, respectively. Repeated with a 1–500 dilution of mouth exudate from a spontaneous case and injected intravenously and intramuscularly in 1 and 2 cc. doses respectively the Seitz filtrate was again inactive and the birds later proved to be susceptible while the Berkefeld and Chamberland L3 filtrates produced the disease and death on the 5th and 8th days, respectively.

However, Doyle did obtain an active Seitz filtrate from mouth exudate which in a dose of 4 cc. intramuscularly produced illness on the 5th day and death on the 7th day. Emulsions were made with blood, mouth exudate and various organs of this bird, filtered through glass wool and then Seitz discs, but only the mouth exudate was active by intravenous injection. About 8 other Seitz filtrates proved inactive, but the virus was passed serially through 7 chickens by means of Berkefeld filtrates.

Rodier (6) in the Philippines reported infection of 10 out of 11 fowls by the subcutaneous injection of a Berkefeld N filtrate of a suspension of intestinal mucous membrane. In another test 10 of 13 were infected with a Berkefeld N filtrate of crop content.

Picard (5) in the Dutch East Indies made 1–100 dilutions which were filtered through paper or flannel and then through various filters. With saliva as a source of virus and a dose of 1–3 cc. one of 2 birds receiving Chamberland L3 filtrate was infected and recovered. Three of 4 birds receiving a Seitz EK filtrate sickened and one recovered, and both birds inoculated with Berkefeld filtrates sickened but recovered. Using feces as a source of virus one of 2 birds given Chamberland L3 filtrate; one of 3 given a Berkefeld filtrate, and both Seitz EK filtrate-inoculated birds contracted the disease. A brain virus suspension was filtered and one of 3 birds receiving the Seitz, 2 of 3 receiving the Berkefeld, and both receiving the Chamberland L3 filtrates contracted the disease. Finally yolk was filtered through a Seitz EK disc and 4 birds injected. The 2 receiving 1 cc. doses were negative but doses of 2 and 3 cc. were infective. All negative birds were later shown to be susceptible and all recovered birds resistant to a test inoculation.

A saline suspension of duodenum filtered by Gomez (17) in the Philippines through a Berkefeld and given in a dose of 1cc. intramuscularly killed the bird in 6 days. The esophagus and duodenum of this bird supplied material for two more filtrations. The esophageal material in a 1 cc. dose intramuscularly, and the duodenal filtrate in a like dose were active. Likewise, a Berkefeld filtrate of crop content was infective. A duck given 4 cc. of duodenal filtrate intramuscularly died in 5 days but 1 cc. intramuscularly failed to infect a pigeon.

Farinas (22) in the Philippines obtained several active filtrates through Berkefeld V and W candles using crop content or a suspension of duodenal scraping. Injections were made subcutaneously.

Crawford (23) in Ceylon reported a positive Berkefeld filtration of a 1 to 4 saline dilution of mouth exudate. A bird that received 2 cc. intravenously of a blood filtrate did not sicken and was later found susceptible. But, the material had stood at room temperature for 20 hours before filtration.

In 1929, Cooper (11) of India reported serial passage of virus from Nov. 1, 1928 on—by subcutaneous injections of filtered mouth washings. Apparently a Chamberland L3 was used (24).
In Japan, Konno et al. (14) reported positive filtration through Berkefeld and Chamberland candles.

Acevedo (39) filtered a spleen emulsion (1 g. and 75 cc. of saline) through gauze, a Buchner funnel with paper pulp, Berkefeld V and N filters in succession and 3 cc. intramuscularly produced symptoms on the 3rd day and death on the 13th.

Dobson (47, 64) filtered liver, spleen and bone marrow emulsions of the second outbreak in England through Seitz discs, and 3 birds injected intraperitoneally with 3 cc. each died within 7 days. In 1934, Picard reported that virulence was considerably reduced by filtration through the Chamberland F Kerzen.

In spite of the unusual laryngeal lesions reported by Hudson (61) in the Kenya outbreak he obtained positive filtrates of a 10 per cent suspension of diphtheritic laryngeal exudate and pharyngeal mucus. This was passed through L5 and L3 filters in about 1 minute with negative pressure. Each of 2 birds got 2 cc. of each filtrate intravenously and a few drops in the larynx. The L5 birds died on the 9th and 10th days, and the L3 birds died on the 4th and 6th days. The non-filtered material killed one bird on the 5th day but the other recovered after showing a temperature from the 2nd to the 6th day.

**Virus Particle Size**

Burnet and Ferry (44) filtered Newcastle and fowl plague viruses through Elford's graded colloidion membranes and found that the former is definitely larger although the difference is not great. Fowl plague passed 0.16\(\mu\) A.P.D. membranes which retained Newcastle virus, and Newcastle virus was largely retained by 0.20\(\mu\) membranes which placed the particle size between 80–120 m\(\mu\) as compared with 60–90 m\(\mu\) for plague.

In the tissue culture of Newcastle virus Topacio (46) occasionally found it necessary to filter contaminated cultures through Berkefeld N or W candles and Seitz discs, and concluded that the particle size was about the same as that of foot and mouth virus, possibly smaller.

**Cataphoresis**

Farinas (22) claims that the virus carries a negative charge.

**Tissue Affinity**

Kaura and Iyer (62) on the basis of nervous symptoms expressed the view that the virus was neurotropic. But Topacio (46) found the virus as viable in tissue cultures of skin, visceral organs and brain as in whole embryo.

**Tissue Culture of Virus**

The first tissue cultures were made by Topacio (46). The spleen of an infected bird was removed aseptically 4–6 days after inoculation and ground in 4 cc. of Tyrode's solution of pH 8.4. It was extracted 1–2 hours in a Frigidaire, centrifuged at low speed for 1 minute, and the supernatant used for initial culture. Subculture virus was prepared from 1 or more Carrel flasks suspended in 1–2 cc. of Tyrode's solution. Plasma was obtained by centrifuging heparinized blood (1 cc. of 1–1000 heparin-saline to 10 cc. blood). Eight to 10 day old embryos were minced and washed in Tyrode's.
One part of plasma and two of Tyrode's were placed in Carrel D-flasks in amounts of 2.5 cc. One cc. of virus suspension added to each whole minced embryo, stirred, and allowed to stand ½ hour. Then a dose of 0.5 cc. of the tissue-virus suspension was inoculated into each flask, shaken to distribute tissue, and allowed to become firm. The tissue-virus mixture inoculum contained 0.1 cc. of virus and 0.4 cc. of tissue. Incubation was done at 37°C and subcultures made every 3-5 days. In making each transfer the virus underwent a dilution of about 1-30.

Topacio used 3 different strains and found differences in their adaptibility to propagation by this method. A laboratory strain underwent 31 successive passages in 112 days of which 17 were tested by bird inoculation in a dose of the contents of 1 or 2 flasks. All were found to be active.

Accidental bacterial contamination did not appear to hinder growth because a filtrate of such cultures gave growth in the next passage. However, the succeeding passage showed a lower virus concentration as judged by milder symptoms and recovery of inoculated birds. The reduction of concentration was attributed to loss of virus during filtration. Here it would be interesting to inquire into the antibiotic effect of bacterial growth.

**PROPAGATION OF VIRUS IN EMBRYONATED EGGS**

The virus was first propagated in eggs by Burnet and Ferry (44). They began with a portion of spleen in 50 per cent glycerin received from Doyle. The injection of a 1-100 dilution of this killed embryos in 30-48 hours. Rarely death was delayed to the third day and once it was within 24 hours.

The serosa of the infected egg is moist, thickened with edema, and may show some degree of cloudiness from cellular infiltrations. Small, irregular flecks of whitish opacity 1-2 mm., and often dendritic in form, may be seen and represent areas of ectodermal proliferation. Small petechial hemorrhages occur along the neck and ventral aspect of the embryo.

Histologically, the serosa is described as showing a patchy ectodermal proliferation followed by, or associated with, gross vacuolation of the epithelial cells and eventual necrosis. Also present in the mesoderm are edema, and accumulations of extravasated red cells. There is a variable degree of endodermal proliferation with occasional vacuolation. The ectodermal proliferation never exceeded 3-4 cells in thickness of which the outer cells are grossly vacuolated with pale-staining nuclei. In some vacuoles structures were found which were considered to be true cytoplasmic inclusions. However, Burnet (75) later reported that such bodies were not seen when an Australian strain was cultivated. Moreover, early sections of membranes infected with laryngotracheitis and psittacosis viruses showed the same structures so it was concluded that they represent a form of mucoid degeneration and, in reality, are droplets of mucin.

The virus is apparently present throughout the embryo as liver and brain were tested and found infective in a dose of 0.03 cc. of dilutions equivalent to 1-200,000 and 1-2,000,000 of the original tissue, respectively.

The virus carried through at least 12 passages was still pathogenic. Each of 2 fowls received 0.5 cc. of filtrate intramuscularly and both sickened on the 5th day.

The Brescia strain of fowl plague virus, in contrast, was found to kill embryos
within 14–18 hours, and with high dilutions, by the second day. The serosa was moist, sometime showed edematous thickening, a few hemorrhages, and occasional slight opacities. The embryo showed congestion, occasional diffuse hemorrhages in the skin, but never the multiple sharply-limited hemorrhages as in Newcastle disease. Histologically, there was usually complete absence of ectodermal cells. The virus was also found in liver and brains.

Burnet (75) reported the Australian strain rather more virulent than the English, and ectodermal proliferations were never clearly defined.

Iyer and Dobson (68) initiated their first egg passage with a Berkefeld V filtrate of a 1 per cent liver and spleen emulsion. Eggs were 9–13 days old and were harvested on the 2nd or 3rd days. For subsequent passages, a dose of 0.5 cc. of a 10 per cent suspension of serosa in saline or broth was used. They passed the virus through 58 generations in 12 months. In titration tests with eggs they found that a dilution of $0.5 \times 10^{-6}$ g. still killed embryos in 48 hours. The pathogenicity of the egg-passed virus for fowls will be discussed below.

Lush (74) used 11–12 day-old embryos, and gave a dose of 0.1 cc. of a 1–100 or 1–1000 dilution of allantoic fluid. Eggs were incubated at 36 C. and harvested after 24 hours.

In Burnet’s report of 1942 (73) 12 day-old eggs were used and, dropping the membrane with the aid of a saline drop, found that a hemorrhagic encephalitis was a conspicuous feature. With high dilutions of virus it was possible to produce well-marked specific foci on the chorioallantois. Satellite foci were more common in eggs showing only 1 or 2 primary lesions because these eggs lived longer. On the other hand, concentrated virus killed in so short a time that lesions did not appear. A small dose produced a purple hemorrhagic encephalitis and multiple hemorrhages in pectoral region muscles and less severe in skin, while a large dose produced less encephalitis but more diffuse hemorrhages in pectoral muscles.

Using virus agglutination of red cells titre as a measure of concentration of virus the end point for various fluids and organs was: allantoic fluid 20, ammotic fluid 2, liver 1600, lung 1200, and brain 300. However, if the egg was inoculated amniotically the concentration of virus here was 1–800 as compared to 1–200 for the allantoic fluid. And, inoculated into allantoic cavity the concentration here in 3 embryos was over 1–320 while the amniotic fluid in 2 showed no agglutination in 1–10 dilution and the third was active at 1–40.

**RED CELL AGGLUTINATION**

Burnet (73) tested the ability of Newcastle virus to agglutinate chick red cells. The “stock virus” was composed of a pooled mixture of allantoic and amniotic fluids of eggs harvested 44 hours after inoculation. Tubes were set up containing 3 parts of 0.25 cc. each (cells, virus and saline) and incubated in the refrigerator for 2 hours. The red cell suspension was a 2 per cent suspension of washed cells. Dilutions from 1-10 to 1-1280 were made. He found the virus capable of agglutinating the cells of man, guinea pig, mouse, fowl, sparrow, and frog and unable to agglutinate those of monkey, ferret, sheep, horse, rabbits, marsupial mouse and tortoise. He also demonstrated that the red cell agglutination titre gave comparable results to the foci counting method of titration or titration by amniotic injection.
Lush (74) also tested Hirst's method in the agglutination of chick red cells by the viruses of fowl plague and Newcastle disease, but employed direct reading of the amount of agglutination to accommodate the reading when Newcastle virus was used. She showed that an immune Newcastle serum with a titre of 1–2048 against its homologous virus had a titre of less than 1–16 against fowl plague virus, and reciprocally, an immune plague serum of 1–2048 titre had a titre of 1–32 against Newcastle virus.

PORTALS OF ENTRY

1. Per os: Whenever infectious material is placed in the mouth or given in food or drinking water and referred to as per os infection it should be understood that actual entrance may be through either the digestive or the respiratory tract.

Picard (5) swabbed mouth exudate in the mouths of 4 birds of which 2 had had the mucosa scarified. All became sick in 3–5 days and died in 7 to 9 days. He also gave 5 cc. doses of mouth exudate diluted 1–100 to 1–10,000 per os with the result that those receiving (2 each) the 1–100 and 1–1000 dilutions sickened in 4–7 days and died in 7–10 days. The 2 receiving the 1–100,000 dilution sickened on the 4th day and one recovered while of the 2 receiving the 1–100,000 dilution one escaped and the other sickened but recovered.

Rodier (6) infected birds by feeding washed tissue from the intestinal wall. Fukushima et al. (36) produced infection by smearing saliva on the mouth and Konno et al. (14) states that infection is by way of the digestion tract. Gomez (17) and Farinas (22) had positive results by feeding infectious material.

Doyle (2) gave 5 cc. of material with a pipette and the bird sickened on the 5th day and died on the eighth. He and Dobson (64) have infected birds by swabbing the mouth with saliva.

However, Haddow (51) states that virus given in a gelatin capsule failed to produce the disease or induce immunity. And finally, Picard (34) gave up to 50 cc. of virus into the crop without producing infection or immunity.

Whether by respiratory or digestive tract the fact remains that virus placed in the mouth is certain to infect.

2. Cloaca: Farinas had one positive infection out of 4 trials.

3. By coitus: According to Farinas this is not a means of transmission in itself except that head infection is apt to occur.

4. Intra-ocular: Farines injected 0.1 cc. into the anterior chamber of the eye and produced the disease with rapid-like symptoms.

5. Conjunctive sac: Doyle (2) placed 4 drops of mouth exudate in the conjunctival sac and caused death on the 7th day.

6. Intravenous: According to Doyle this is one of the most reliable means of inducing infection, especially for the detection of small amounts of virus, and the period of incubation is usually short 4–5 days.

Picard (5) injected saliva in doses of 0.5 cc. or 0.00005 cc. The 0.5 cc. failed to infect, the 0.05 and 0.005 produced death on the 8th and 6th days, respectively. The bird receiving 0.0005 cc. sickened on the 7th day and recovered while the bird receiving the highest dose escaped infection.

Fukushima et al. (36) successfully infected birds intravenously with mouth exudate, liver, and bone marrow suspensions. Positive results have also been reported by Farinas (22), Crawford (23), and many others (60).
7. **Subcutaneous:** This is also one of the common methods of artificial infection. According to Doyle (2) the results are similar to intravenous inoculation except that the period of incubation is somewhat longer. Rodier (6) infected 10 of 11 birds with a Berkefeld N filtrate of intestinal mucosa and 10 of 13 with a filtrate of crop content. Picard (5) injected 0.5, 0.05 and 0.005 cc. doses of saliva and the 2 larger doses produced infection. Farinas infected birds subcutaneously with 0.5 cc. of a 1–10 dilution of crop content.

Nakamura et al. (35) tested the pathogenicity, by subcutaneous inoculation, of various generations of brain to brain-passed virus and found that both incubation period and course were shortened. Thus, with 1st–14th generation virus the incubation period was 4 days or more and death on the 7th day, or later. But, with injections of the 32nd, or later generations, the incubation period was reduced to 2 days, and the course to 4 days. Also, the 15th generation was avirulent in a 1–5000 dilution, the 38th and 48th generations were virulent in a 1–100,000 dilution, and 100th or later passages gave a virus virulent in a 1–10,000,000 dilution.

Farinas (22), Crawford (23), Cooper (24), Fukushima, et al. (36) and many others have used subcutaneous injection as a means of producing artificial infection.

8. **Intramuscular:** This is likewise a common method used to induce artificial infection. And yet, Doyle (2) originally reported that some birds failed to contract the disease, and that the incubation period was longer than when virus was introduced intravenously.

Picard (5) injected birds with saliva in doses of 0.5, 0.05, and 0.005 cc. intramuscularly, but not with 0.0005 cc. Positive results have also been reported by Farinas (22), Crawford (23), Acevedo (39) Hudson (61) and many others (36).

9. **Submucosa:** Picard (5) using saliva as a virus injected 2 birds in the submucosa of the mouth with 0.5 and 0.0005 cc., respectively. The first sickened on the 4th day, and recovered after the 9th, while the second sickened on the 3rd, and died on the 6th.

10. **Scarified skin:** Doyle (2) applied mouth exudate to the scarified combs of 2 birds with the result that they died in 8 and 9 days, respectively. But, Picard (5) placed one drop of saliva on the scarified skin with negative results. Farinas (22) failed to infect birds in four attempts and the result was negative when the skin was pricked with beak of a sick chicken.

11. **Intracerebral:** Picard infected birds with doses of 0.5, 0.005, and 0.00005 cc. of saliva virus which caused illness in 5, 4 and 2 days respectively, and, curiously enough, death after a course of 6, 3 and 1 days, respectively.

Nakamura, et al. (36) as mentioned above, carried a strain of virus serially by brain passages with resulting shortening of the incubation period and course of the disease as determined by subcutaneous injections.

12. **Intraperitoneal:** Dobson (47) produced illness and death in 5–7 days with intraperitoneal injections, and filtrates of organs (liver, bone-marrow) produced death in 7–12 days. Later he (64) reported that intraperitoneal injection of each of 5 pigeons with a dose of 1 cc. of liver and spleen emulsion caused death in 7 days. However, Picard (5) injected 2 birds with 0.5 and 1 cc. of saliva and neither became infected.

13. **Intralaryngeal and intratracheal:** Hudson (61) reproduced the disease by swabbing the larynx. Many others have referred to swabbing the throat which is too
indefinite as concerns the respiratory tract specifically. Dobson (47) produced death in 7-10 days by intratracheal inoculation.

14. Feather shaft: Large doses of virus were injected into the quill of primary wing feathers by Picard (34) without producing fatal infection. However, injections occasionally produced transitory illness followed by a detectable immunity.

**DISTRIBUTION OF VIRUS IN THE BODY**

1. Saliva: In the natural disease the virus is consistently present in mouth exudate and is to be found here in more concentrated form than in any part of the body.

   Doyle's first report (2) concerned the distribution of virus in a natural case killed in extremis on the 7th day. Organs were removed aseptically, ground and filtered through paper and 1 cc. given intravenously. Brain and cord were washed 3 times. Results of inoculations of organs will be dealt with later. The mouth exude intra-venously killed on the 6th day.

   Virus in mouth exudate has been demonstrated by Kraneveld (1), Picard (5), Konno et al. (14), Gomez (17), Farinas (22), Crawford (23), Cooper (24), Kylasamaier (29), Fukushima et al. (14) and others. The latter charged cotton swabs with throat material of birds in the mofussil and these arrived at the laboratory in an active state. Nakamura et al. (35) reported 28 positive inoculations by swabbing, and 10 by the subcutaneous method with saliva. Hudson (61) found that a swab from a Mombasa bird was infective 2 days after storage.

   Doyle found the infectivity of virus in saliva to vary between 1-1000 and 1-200,000. The minimal infective dose reported by Farinas (22) was 1 cc. of a 1-1250 dilution given intravenously. Picard (48), presumably referring to saliva, states that a 1-10,000,000 dilution is infective.

2. Feces: Picard (5) mixed feces with food and fed it whole and in 1-100 and 1-1000 dilutions in 5 cc. doses. The three birds became sick in 5, 6, and 7 days respectively, and the first 2 died. The third was killed on 11th day when it had nearly recovered. Then 2 birds got 5 cc. intramuscularly of a 1-10 dilution of feces filtered through paper and both got the disease and died on the 8th and 9th days, respectively. Doyle (2) demonstrated virus in the feces by intravenous injection which produced death on the 6th day.

   Farinas (22) found the feces infective in only one instance even when collected at different stages of the disease. Kraneveld (1) reported feces positive.

   Gomez (17) states that the feces do not contain the virus and when mixed with food or water do not transmit the disease.

3. Liver: Doyle (2) reported this organ infectious by intravenous inoculation and death on the 6th day.

   In Picard's (5) study of the distribution of virus in the body organs were collected aseptically and a 1-10 suspension prepared. His birds 26, 55, 169, 175 and 184 received liver suspension subcutaneously and in no case sickened. The inoculum had come from a fresh cadaver, the cadaver of a laying hen, a bird killed during the febrile period on the first day, and a bird killed 3 days after infection during the incubation period.

   Gomez (17) found virus in the liver and Kylasamaier (29) in a mixture of liver and spleen. Hudson (60) reported it less concentrated in organs than in saliva. Virus
is also reported in the liver by Rodier (6), Farinas (22), Cooper (24), Fukushima et al. (36), Acevedo (39).

4. **Spleen**: In Doyle's (2) study spleen emulsion killed the fowl inoculated intravenously in 6 days.

Others reporting virus in spleen include Farinas (22), Johnstone (37), Acevedo (39) (by intramuscular inoculation), Kylasamaier (29) (mixture of liver and spleen) and Fukushima et al. (36).

But again, in Picard's (5) early report, spleen from a fresh cadaver given subcutaneously, and intramuscularly, failed to infect.

5. **Lung**: In Doyle's (2) report lung emulsion given intravenously failed to infect, but in other tests 3 out of 6 trials were positive.

In Picard's (5) early report lung suspension from a bird killed on the first day during the febrile stage given subcutaneously, and from a donor killed on the second day of the disease given intramuscularly, and subcutaneously, failed to infect.

Lungs also reported to contain virus by Rodier (6) and Farinas (22).

6. **Pancreas**: According to Doyle (2) the pancreas suspension given intravenously produced death on the 4th day. In Picard's (5) early report pancreas from a bird killed on the 3rd day of illness, and given subcutaneously failed to infect.

7. **Bone marrow**: In one experiment of Doyle's (2) bone marrow given intravenously failed to infect, but the virus was demonstrated in this tissue in other experiments. Fukushima et al. (36) reported positive results with inoculation of bone marrow.

8. **Kidney**: The presence of virus in this organ has been reported by Rodier (6) and Farinas (22). But again Picard's (5) early report shows that 2 fowls inoculated subcutaneously with a kidney suspension from a bird killed on the 3rd day of illness failed to infect.

9. **Egg yolk**: According to Picard (5) egg yolk from the cadaver of a laying bird given subcutaneously, intramuscularly, or orally, produced infection in every case. Farinas (22) reported the ovary infective.

10. **Testes**: Reported virulent by Farinas (22).

11. **Intestine and content**: Kraneveld (1) reproduced the disease with intestinal slime. Gomez (17) found virus in the duodenum (by feeding). Kylasamaier (29) produced the disease by drenching a bird with 5 cc. of intestinal content. Intestine reported virulent by Farinas (22).

12. **Bile**: An intravenous injection killed the bird on the 5th day according to Doyle (2). Rodier (6) found the bile to be infectious only once. Picard (34) killed fowls with a dose of 1 cc. of 1-50,000 dilution of bile.

13. **Crop content**: Like mouth exudate, crop content has been reported infectious by all workers who have employed it as a source of virus. Farinas (22) made the interesting observation that the concentration of virus was greatest when the crop was empty in which case it contained a semi-transparent substance. A piece of esophagus fed by Gomez (17) produced the disease.

14. **Edematous material**: Collected from the thoracic inlet failed to produce infection according to Farinas (22).

15. **Brain and cord**: In Doyle's (2) examination of the distribution of virus in a single bird the brain, and portions of cord from the cervical, dorsal and lumbar re-
gions were found to contain the virus except in the case of the cervical portion. In Picard's (5) report brains from a fresh cadaver, from a bird killed on the 4th day of illness, from the cadaver of a laying hen, and from a moribund bird showing paralysis injected subcutaneously, produced the disease in every instance. And, brain from a dead laying hen produced the disease when given orally. However, the brain of a bird killed 3 days after infection, that is, during the incubation period, failed to infect 2 birds injected subcutaneously. Two birds injected subcutaneously with spinal cords of different donors failed to take the disease. In both cases the brains of these birds were shown to contain the virus. One donor showed nervous symptoms and was killed on the 4th day of the disease, and the other was paralytic and moribund.

Virus also reported present in the brain by Konno et al. (14), Kuppuswamy (49) (by pigeon inoculation of 1 cc. of a 1-10 dilution from living and dead birds), Fukushima et al. (36) and others.

Hudson (61) injected brain emulsion from a spontaneous case intramuscularly after 2 days storage, and produced death in 5 and 11 days, respectively. The brain of a pigeon produced death in a fowl in 7 days, and, the brain of a white domesticated Madagascan guinea infected, by throat swabbing, a bird which died acutely.

Nakamura et al. (35) made 150 serial passages with brain virus which was also infectious by subcutaneous injection, but in the latter case less readily.

The concentration of virus in the brain may be very great as shown by Nakamura et al. (42). They showed that the minimal infecting dose of the Korean strains to be $10^{-6}$ to $10^{-7}$, of the Sato strain (Japan) $10^{-6}$ and of the Philippine virus $10^{-7}$ to $10^{-4}$. Rodier (6) failed to find virus in the brain of birds that had died of the disease.

16. Blood and pericardial fluid: In Picards (5) first report pericardial fluid given intravenously was non-infectious. Blood was likewise non-infectious, even when drawn at different stages of the disease and injected in various ways, even 3 cc. of fresh whole blood given intravenously. Failures were also had with diluted blood, washed cells and serum from dead and living birds. Gomez (17) failed to infect 2 birds injected intramuscularly with 1 cc. of freshly taken blood from a typical case, even though the duodenum of the donor was shown to contain virus. Kraneveld (1) was unable to demonstrate an infectious agent in the blood.

Farinas (22) used freshly drawn whole blood, blood serum and washed red cells and made injections subcutaneously, intravenously and by mouth and was unable to produce infection. Whole blood freshly drawn and transfused did infect 2 chickens. Pooled serum from several chickens caused a mild infection but the birds recovered and were immune. Pooled serum obtained by centrifugation did not produce the disease.

Doyle (2) bled 3 birds during the febrile stage (5th day), defibrinated the blood and pooled the sample which was diluted. The whole blood was infectious in a 1 cc. dose intravenously in 1-100 to 1-2500 dilutions but the 1-5000 dilution was negative. Duplicated with the blood of 2 fowls and only the undiluted blood was infectious. Blood cells which were infective in a dilution of 1-100 (not end point) were washed 4 times and 1 cc. was infectious intravenously. The corpuscles of blood infective in a 1-25,000 dilution were washed 10 times and 1 cc. was inactive. The serum from 2 fowls was infective in a dose of 1 cc. intravenously.
Rodier (6) stated that the virus is apparently present in the blood in the early stages of the disease.

Topacio (46) infected a bird with 1 cc. of blood from a natural case diluted 1–25,000.

Hudson (60) found the virus less concentrated in the blood. Two birds inoculated intramuscularly died on the 6th and 8th days, respectively.

Crawford (23) reproduced the disease in pigeons with blood from a fowl.

Kylasamaier (29) injected 2 cc. of a mixture of blood and saline and produced death, and typical lesions, but when broth was inoculated with blood and incubated 24 hours, the inoculated fowl died in 2½ days and showed lesions suggestive of pest!

Acevedo (39) reproduced the disease by intramuscular injections of blood. A 4 cc. dose of a $10^{-5}$ dilution produced the disease, but the bird recovered.

Cooper (24) found that subcutaneous injections of blood sometimes failed and Konno et al. (14) were not always able to find virus in the blood.

Emulsions of the organs found to be infective by Doyle (2) as reported above were also filtered through Seitz discs, but no filtrate was active except that of mouth exudate.

In 1932, Manninger reported that by rapid passage of Doyle's virus he was able to shorten the incubation period and course of the disease, and also produce lesions simulating those of fowl plague. Doyle (50) attempted to duplicate Manninger's results and bled inoculated chickens at 72 hours and inoculated 1 cc. intravenously to the next bird in the series and so on for 15 passages. With each passage a control bird was inoculated, and the disease was allowed to run its course. Likewise a pigeon was injected with each passage to determine any change in the virus. The results showed that the control bird died in the usual period of 5–7 days with two exceptions, one in which the course ran 16 days, and in the last passage in which the bird sickened but recovered. Likewise, of the 15 pigeons inoculated, all but 3 died in from 6 to 12 days, the three sickened but recovered.

Manninger's (55) criticism of the above was that Doyle had not bled the birds at the onset of fever, that after this, the virus gradually disappears from the blood remaining in a less potent form only in some organs.

Now, it will have been noticed above that, for the most part, Picard's results stand apart in that he found virus only in mouth exudate, feces, brain and yolk, and never in the blood or other internal organs regardless of the stage in the disease when the inoculum was collected. But, in 1933 (38) and 1934 Picard (48) reported that while the above had been true up to 1931, in that year a change was observed which has been constant for 4 years. The change was first noted in the laboratory strain inoculated as brain virus and the only change till then was that the disease was produced after a shorter incubation period, but then it was discovered that the blood, spleen, liver and kidney of inoculated birds now contained the virus. Even more interestingly, the same change had occurred in spontaneous field cases which were checked periodically in the laboratory.

**ACTIVE IMMUNIZATION**

In the course of experiments on this disease, fowls, either through deliberation or accident have been actively immunized. Sometimes valuable information can be
gained by noting the circumstances under which fowls were accidentally immunized, and, of course, planned experiments serve an equally valuable guide to future work.

1. Chloroform:

Picard (15) concluded from his experiments with this type of vaccine that the development of an effective immunizing agent was unlikely.

Cooper (24) found that organs treated with chloroform proved valueless.

Farinas (22) used liver, spleen, lungs, kidney and brain ground in salt solution and added chloroform to make 0.75 per cent. The material was stored at 0–4°C. and used in from 1 day to 1 month later in doses varying in number and quantity, and at intervals (in multiple dosing) of 4–9 days. The birds so treated were tested 4–14 days later against 0.5–1.0 cc. of virulent virus. Of 9 injected only 1 remained well and immune, 2 sickened and recovered and 6 died either from the vaccine or the test inoculation. Negative results were also obtained with brain and spleen, or with spleen alone treated with chloroform.

Nakamura et al. (58) found that saline suspensions of brain, spleen and liver treated with chloroform and held at 37°C for varying periods and administered subcutaneously, or intramuscularly, gave disappointing results when immunity was tested 10–14 days later.

2. Toluol:

Farinas (22) used a 5 per cent toluol tissue vaccine held 21 days at 0–4°C. Given in a dose of 1 cc. and 10 days later a dose of 2 cc. gave the chicken the disease. A single dose of 1 cc. did not protect against a test dose later.

Nakamura et al. (58) also reported negative results when toluol was used as an attenuating agent in an emulsion of brain, spleen and liver.

3. Tricresol:

A 0.5 per cent tricresol-treated spleen suspension gave negative results according to Farinas (22) and Nakamura et al. (58) reported disappointing results with the same agent.

4. Phenol:

A 0.5 per cent of phenol-tissue (spleen, liver and kidney) vaccine held in the ice-chest 21 days was used in 1 cc. and 2 cc. doses at intervals of 16 days. The single fowl treated remained well and resisted a test inoculation 14 days later. A fowl given a single 1 cc. dose died when tested with active virus 8 days later. A 50 per cent tissue vaccine containing 0.5 per cent phenol and 0.75 per cent of chloroform held for 21 days at 0–4°C. was injected subcutaneously in a dose of 2 cc. but 7 days later the bird died of a test inoculation. Likewise, the same vaccine in a 1 cc. dose followed 10 days later with 2 cc. of a 33⅓ per cent glycerin tissue suspension that had been held 21 days at 0–4°C. resulted in death of the bird from paralysis. A brain and spleen suspension treated with phenol was ineffective (22).

Another phenolized vaccine tried contained 49 parts of virulent crop content, 25 parts each of M/15 Sorensen’s potassium and sodium phosphate and 1 part of
phenol. The mixture was shaken, and stored 1 day at 0-4°C. Before inoculation
the dilution was doubled and was given in a dose of 1 cc. with 3 cc. a week later.
The injections either produced the disease or failed to immunize. After holding the
mixture 7 days longer and injecting it in varying doses it failed to protect a single
bird (22).

In a modification of the above, a 30 per cent tissue suspension in 50 per cent
glycerin containing 2 per cent phenol was held 8½ hours at 2-3°C. and then diluted
4 times with Sorenson's solutions (1 part) and water (2 parts) so as to contain 0.5
per cent phenol and 12.5 per cent glycerin. Birds receiving 0.5 cc., the 0.5 cc. and
2 cc., or 0.5, 2 cc. and 4 cc. took the disease, but the single dose of 0.5 cc. was not
fatal and the bird was immune (22).

5. Glycerin:

A 50 per cent glycerinated vaccine held about 5 months failed to confer immunity
to 2 fowls in a dose of 1 cc. subcutaneously, and a 33½ per cent glycerinated sus-
pension of spleen, liver and kidney held 21 days at 0-4°C. did not confer immunity
to 2 fowls in a dose of 1 cc subcutaneously according to Farinas (22).

6. Phenolized glycerin:

Farinas (22) also tried Todd's method (used in plague) except that 6 grams of
solution (60 per cent glycerin and 0.5 per cent phenol) instead of 4 were used to each
gram of tissue (brain, liver, spleen and kidney). Doses of 0.5, 1 and 3 cc. at weekly
intervals were given but only 1 out of 12 fowls remained well and was found to be
immune. Repeated, 3 sickened and died and 3 had a mild attack, recovered, and
were immune. Picard (15) used a phenol-glycerin-saline vaccine in dilution of 1-10
to 1-1000 and found the highest dilution infective in a dose of 1 or 2 cc.

7. Hydrochloric acid:

Virus treated with N/1 HCl failed to immunize (22).

8. Saponin:

Haddow (54) reported a measure of success with virus treated with saponin but
the inflammatory reaction to the agent was variable.

9. Formalin:

One per cent spleen emulsion in 1–1000 formalin kept at 37° for 3 days and there
after in a darkened room only lengthened the incubation period according to Craw-
ford (23). The dose was 1–7 cc. depending on size.

Haddow (54) reported that formalinized vaccines were the most helpful but the
reaction was too irregular to permit field trials. Experiments along this line had
been mentioned in an earlier report (40).

Nakamura et al. (58) used formalized saline suspensions of brain, spleen and liver.
Of 4 birds inoculated intravenously 2 became immunized, whereas 2 birds inoculated
intramuscularly with the same material were not immunized. This result led to the
intravenous injection of 68 birds with 1 or 2 doses with the result that 37 or 54 per
cent resisted a subsequent virus inoculation.
Then 20 lots of brain, spleen and liver saline suspensions were prepared and to each volume was added a volume of fowl serum diluted 1–3 and the whole formalized to 0.2 per cent. The mixtures were held at 37°C. for 72 hours and thereafter in the cold. With each batch several fowls were injected with 2 intravenous injections (interval not stated) and the birds were tested 10–14 days later. Of 87 vaccinated, 54 or 62 per cent resisted a test dose which killed all of the 52 controls. The vaccines showed a marked difference in spite of great care used in their preparation. They found that in 4 lots of vaccine the intravenous method of inoculation gave better results than the subcutaneous or intramuscular routes. Thus, 13 or 81 per cent of 16 injected intravenously, 4 or 17 per cent of 24 injected subcutaneously, and 3 or 21 per cent of 14 injected intramuscularly were immune. The virus used to check immunity was pathogenic in a dilution of $10^{-7}$ to $10^{-8}$.

Nakamura et al. (42) had earlier used injections of formalinized virus to protect fowls for hyperimmunization.

10. Sulphuric ether:

Bailly's method of rabies immunization was adopted by Picard (34) for pseudo fowl plague. Brains were exposed to ether for 25, 20 and 15 hours then each ground in 10 cc. of 50 per cent glycerin-saline, filtered through gauze and refrigerated for 24 hours. There were injected subcutaneously in a dose of 1.0, 2.5 and 5 cc. of brains exposed to ether for 25, 20 and 15 hours. Then in about 3 weeks the birds were injected with an M.L.D. of standard virus or exposed if the reactions were indefinite, with the result that no bird was immunized.

11. Feather shaft inoculation:

Picard (34) injected 0.05 to 0.1 cc. of a virus suspension (500,000–1,000,000 M.L.D) into the shaft of a primary wing feather and repeated the inoculation 6 days later with another feather. The injection produced no symptoms and the birds resisted 1 M.L.D. injection 3 weeks later which killed a control in 5 days, but when exposed to contact infection 12 days later they died in 7 and 6 days, respectively.

The test was repeated on 8 males 1 year old and 8 five months old, with virus diluted 1–10 and 1–1000. Three birds reacted slightly and 12 of the 16 were protected against a subcutaneous injection of 1 M.L.D. a month later. But, when 10 days later a second injection of varying doses of virus was given subcutaneously (1 to 100) only the 3 birds which received 1 M.L.D. survived.

In another trial 2 birds reacted slightly when 2 feathers were inoculated at one time but they resisted 100 M.L.D. of virus about a month later which killed the 2 birds that had had 3 feathers each inoculated at weekly intervals. Likewise, 4 birds which had had one feather inoculation died when 3 days later an attempt was made to fortify the immunity by giving 100 M.L.D. of virus subcutaneously or intravenously. Again 4 birds with 5 feathers injected simultaneously and 4 with 10 feathers injected, resisted 100 M.L.D. subcutaneously which killed 2 controls in 5 days. Then, the 8 were exposed to contact infection for 2 weeks and seemed to resist this, but when given a second subcutaneous dose of 1 cc. of standard virus all died.
One hundred M.L.D. of virus subcutaneously 1–4 days after a preliminary feather injection failed to kill birds, but given simultaneously with the feather injection killed, as it did a control. But these birds were not protected enough to resist 500 M.L.D. given subcutaneously 3 weeks later. A preliminary feather injection did not protect any bird against 500 M.L.D. given the same to 4 days later.

Picard also found that while 1 cc. of virus subcutaneously killed 2 birds in a test even though they had had a feather injection 3 days previously, the same dose or 5 cc. given orally failed to infect while virus applied to the scarified skin produced illness or death whether preceded by feather inoculation or not. In any event the birds surviving oral administration of virus were not immune even if the dose had been 50 cc.

12. Bile virus:

The presence of virus in the bile was easily established by Picard who tried injections of serial dilutions of this material and found it active even in a dose of 1 cc. of a 1–50,000 dilution. However, one bird survived (1–10,000 dil.) and was thereafter shown to be solidly immunized. Bile injection was resorted to as it had been used by Koch in cattle pest immunization. Bile held 1–4 weeks and injected in a 1–10,000 dilution was still active and lethal while that held 6 weeks to 2 months was inactive and non-immunizing. Finally, a combination of feather and bile inoculation proved ineffective.

13. Phage (?):

An attempt to isolate a phage for the virus was made by Picard (34). Ten cc. of a filtrate of droppings from an immune bird (phage?) was added to 100 cc. of broth, incubated 24 hours, and 1 cc of brain virus added and again incubated 24 hours, and finally passed through a Chamberland F candle to serve as inoculum for the next passage. After three passages the phage-bearing (?) broth was tested for immunizing properties. Each of 14 birds received 1 cc. subcutaneously and 2 weeks later half of them got 1 cc. of brain virus subcutaneously, and all died promptly 5 days later. The other 7 birds exposed to contact infection likewise died within 13 days.

An injection of virus also infected when followed a day later by phage (?) injection, and, diseased birds were not benefited by 5 to 10 cc. doses of phage (?)

14. Serum (as attenuating agent):

Farinas (22) observed that when the virus in incubated serum-virus mixtures was not fully neutralized the birds sickened but recovered and were immune. Haddow (54) mentions some success with injections of incubated serum-virus mixtures.

Sirlaw (59) also used graded serum-virus mixtures incubated 24 hours before injection. The results were uncertain, but some birds so treated later resisted 50 infecting doses of virus.

Nakamura et al. (42) prepared some birds for hyperimmunization with preliminary injections of serum-virus mixtures. A simultaneous vaccination with virus and immune serum prepared from a turkey or goose was tried by Picard (15) with results that were somewhat encouraging.
15. **Drying:**

Haddow (40) reported satisfactory results with one lot of tissue dried in vacuum at 0°C, but these could not be duplicated. Picard (15) had no marked success with vaccines attenuated by drying.

16. **Aging:**

Doyle (2) mentions 8 birds which survived an inoculation of stored virus and were found to be immune to contact infection which killed 16 healthy birds. Fifteen days later they resisted 1 cc. of mouth exudate and still later 1 cc. of mouth exudate intravenously. He (50) was unable to kill 2 birds 17 and again 31 days after they had received an intravenous injection of brain and spleen suspensions received from Rodier in the Philippines. Aging of the original inoculum apparently served as the attenuating agent.

17. **Dilution:**

Haddow (51) pointed out that immunity can be produced by doses slightly lower than those which produce death, but that the standardization of such a vaccine for use with safety in the field has not proved possible. As might be expected, accidental immunization in the course of experimental inoculations of virus-bearing material occasionally happens in consequence of low concentration of virus, aging or deterioration resulting from autolysis or bacterial growth. Nakamura et al. (42) used such birds for hyperimmunization.

18. **Tissue cultured virus:**

While making virulence tests on tissue-cultured virus Topacio (46) observed recoveries which were subsequently shown to be immune. Here, the attenuation may have been due to the artificial cultivation. But, in testing the activity of cultures at various stages of growth he observed that cultures of the “laboratory” strain 6–14 days old were active but some fowls recovered, that 4 day cultures of the Farinas strain produced mild symptoms and recovery, and that 6 day old cultures of the Statsenberg strain produced neither infection nor immunity. Topacio pointed out that culture virus gives promise of a potent vaccine. In these cases attenuation may have been due to a combination of factors, that is, artificial cultivation plus autolysis, aging, etc.

19. **Modified virus** (through egg-propagation):

By far the most promising report is that of Iyer and Dobson (68) who found that continued passage through embryonated eggs resulted in a modified virus. In spite of 58 passages in 12 months the virus still killed the embryo in the usual time, that is, 48 hours, and injections subcutaneously into fowls of 1 to 5 cc. doses of a 10 per cent suspension of the whole embryo in saline (up to the 33rd passage) produced the disease and death in the usual time (3, 10, 12 and 19th passages tested). But, fowls inoculated with the 33rd to the 56th passages (33, 37, 41, 48, 50, 51 and 56 tested) remained well, and when tested for immunity in from 14 to 17 days later resisted 1 cc. of a 1 per cent suspension of fowl-passed virus that was known to contain 100,000,000 minimal lethal doses.
Then a second line of the same strain of virus was started, and while the 12th passage was fatal, the 14, 16 and 21st passages had no effect on the fowls which were shown to be immune 14 or 15 days later.

That the changed virus did not revert was shown by inoculating a fowl with a 41st passage virus and 5 days later reinoculating the spleen of this fowl into another fowl which did not take the disease but which was shown to be immune to the usual test dose.

20. **Crystal violet vaccine:**

Iyer and Dobson (71) have also experimented with this type of vaccine. Two or more typical embryos were used to make a 10 per cent saline suspension and centrifuged at 500 r.p.m. Then 8 parts of the above were used with one part each of a 3 per cent aqueous solution of dibasic sodium phosphate and 0.5 per cent aqueous solution of crystal violet. The mixture was attenuated at 37°C. for 72 hours, and, as a rule, injected subcutaneously in 5 cc. doses. Fowls varying in breed, age and sex were used and tested for immunity at different periods by inoculation or exposure.

In all, 6 lots (12, 23, 33, 45 and 56 passage and the 23 passage of a second line) were tested. The 12th passage failed to immunize the single bird injected. The 23rd passage immunized both birds injected. The 33rd generation was injected into 7 birds whose immunity was first tested by contact infection 7, 14 and 21 days later with the result that 2 died. It is interesting to note that of these 2, one had been exposed to contact infection only 7 days after vaccination. Three of the immunized birds resisted an injection of 100 million fatal doses 36 days after vaccination. Six of 12 birds survived contact infection 21 to 32 days after being vaccinated with a 41st passage. The 56th passage failed to immunize any of the 10 birds exposed to contact infection 14 days later. The 23rd passage of the second line strain was tested on a mature hen, 5 cockerel (20 weeks old) and 14 four-week old chicks. All were exposed to infection 13 days later with the result that the hen, 2 of the cockerels and 9 of the 14 chicks died.

The authors conclude that of the 51 birds injected 30 died (their table shows 52 injected and 31 died), and that because of the irregular results further experiments were not justified.

21. **Tissue vaccine:**

The method used by Bergeon and Cébe for rinderpest was not successful when adopted by Picard (38) for pseudo fowl plague. Ground tissues submitted to the action of 9 per cent saline to free them of virus were, when injected, still virulent or entirely lacking in immunizing properties.

**EFFECT OF PASSAGE IN A HOST OTHER THAN CHICKENS**

1. **Pigeon:** A pigeon infected by throat swabbing and an intravenous injection died on the 5th day, but its mouth exudate failed to infect a fowl and a pigeon by intravenous injection (2). A pigeon which developed paralysis and died on the 6th day after an intramuscular injection of mouth exudate failed to transmit the disease to a pigeon in the same cage (2). Another pigeon infected by an intravenous injection of mouth exudate and dead on the 7th day failed to transmit the disease to a pigeon
and a fowl in contact. The same was true of a pigeon infected intramuscularly. Two sick pigeons failed to infect a pigeon and a fowl in contact yet the blood and spleen of these pigeons, by intravenous injection, killed fowls on the 8th and 9th days, respectively (2).

Hudson (61) infected 2 pigeons with blood and for 8 days before their death a contact fowl did not contract the disease. Then, the fowl was inoculated with the brain of one of the pigeons and died on the 7th day. Serial passage of the virus through pigeons as a means of attenuating or modifying it for purposes of immunization was not successful according to Picard (15).

Shirlaw (59) attempted to attenuate the virus by passage through pigeons and mice but found that the virus died out suddenly or is not changed.

2. Duck: A duck infected by intravenous injection of mouth exudate was able to transmit the disease to a fowl in contact, but a fowl inoculated with mouth exudate of the duck was unaffected (2).

3. Guinea: It has been cited above (61) that the brain of an infected guinea was infectious by instillation in the throat of a fowl.

**RECOVERED BIRDS DEVELOP IMMUNITY**

Birds that recover natural or artificial infection develop an immunity, but how early this develops or how long it lasts has not been determined. In the case of laryngotracheitis, for example, it could be observed that immunity lasted for at least a year because on farms where the disease was enzootic the old hens regularly escaped infection. In the case of Newcastle disease, however, so few birds survive that the literature fails to record their reaction when the disease reappears.

In any event, immunity appears to develop rather early and seems to be quite durable as might be expected. Picard (5) tested birds recovered from contact or inoculation (saliva and brain) infection at various periods afterwards either by further contact infection (1½ to 2 months) or by 1 cc. injections of saliva intravenously, or brain subcutaneously. All birds remained healthy even though tests were made as early as 18 days or as late as 190 days after recovery.

**COMPLEMENT FIXATION**

Farinas (22) prepared 6 types of antigen from spleen, liver, and brain and crop content and in no case demonstrated fixation of complement with positive serum.

**PROPHYLACTIC VALUE OF IMMUNE SERUM**

Immune sera have been collected from birds that have resisted inoculation, recovered after a mild attack, or hyperimmunized. Cooper (24) was unable to produce an immune serum in a donkey with filtered mouth washings even though the animal showed a distinct reaction.

Picard (5) injected 4 birds with 0.5, 1.0, 1.5 and 2 cc. of serum and exposed them with 2 non-treated birds to 4 sick birds. The non-treated birds took the disease on the 7th day and died on the 10th, while the treated birds sickened on the 8th day and died on the 11th, 12th and 14th days.

Cooper (24) gave 2.5–10 cc. of serum to 10 birds and a day later injected them and 4 controls with virus with the result that the serum-treated birds elicited a slight
temperature on the second day but all survived, whereas the 4 controls sickened in 3–5 days and died on the 4th–7th day.

Four hens in one flock and 8 in another were each given 3 cc. of serum intravenously by Farinas (22), and exposed to flock infection and all lived, whereas in a third flock 2 remained well while 2 got the disease 2 weeks later in spite of the treatment above. In still another flock 1 rooster and 12 hens each received 3 cc. of serum intravenously, and one got the disease as did 4 non-treated birds (number of the latter not given). In a final flock, it is difficult to distinguish between prophylactic and therapeutic effect. In any event, 46 birds were treated with serum and 4 were not with the result that 21 sickened and 19 died. Then, in a laboratory test, 8 birds received 0.5 to 2 cc. of serum intravenously, and a day later 125 infective doses of virus subcutaneously with the result that only one escaped infection and one showed a delayed reaction.

Nakamura et al. (42) actually studying the relationship between the Korean, Sato and Philippine viruses (all Newcastle disease) and the Chiba strain (fowl plague) showed that hyperimmune sera of any one of the first three protected fowls against the other two but not against the Chiba strain, and conversely, a hyperimmune serum to the latter failed to protect fowls against any of the first 3 strains. They used 2.5 cc. to 4 cc. of serum per kilo of body weight and gave the virus 24 hours later.

Kylasamaier (29) failed to protect 2 fowls with 3 cc. of serum against a virus injection a day and a week later, respectively. He also prepared a 10 per cent suspension of liver from a recovered bird and half of it was heated one hour on each of 3 successive days at 60°C. then filtered through paper and chloroform added to make 1–500 after which it was incubated 24 hours at 37°C. It was not sterile. To the other half toluol was added to 1 per cent and incubated 72 hours at 37°C. and filtered. Then formalin was added to 1–1000 dilution which resulted in a sterile product. He states that the results of controlled immunity tests were so good as to justify use, and that the mortality in birds exposed to infection was negligible.

Keogh (66) made the interesting observation that embryos given a minimal dose of virus and some serum later usually survive 3–4 days, whereas non-protected embryos die within 48 hours. The protected embryos showed focal necrosis of the serosa but petechiae were rare or absent at 48 hours. With larger doses of serum embryos were protected for 7 days or more, appear normal, and show no visible petechiae. Still, the virus could be found in such embryos in as high concentration as in non-protected dead or dying embryos. Keogh believes that the circulating antibodies prevent the establishment of secondary lesions.

**Therapeutic value of immune serum**

Picard (5) used serum from recovered birds in 0.5, 1.0 and 2 cc. doses administered the first and second, second and third or third and fourth days of illness with negative results.

Farinas (22) used 3 to 5 cc. of serum intravenously on 23 birds at various stages of the disease with the result that 19 recovered and of these 5 developed nervous derangements and finally died thus leaving 14 completely recovered. He reported that intramuscular and subcutaneous injections were not effective. No curative effect was observed in 4 birds each given 5 cc. intramuscularly of an emulsion of
interval organs from an immune rooster. While sheep serum appeared to neutralize the virus in vitro test it was not effective in a sick bird. In a field trial, a rooster given 5 cc. at the beginning of the disease recovered and in another flock only one of 3 birds recovered after 3 cc. of serum intravenously.

Cooper (24) gave 2 fowls 5 cc. each and two 2 cc. each of serum and had 2 as controls. The controls died on the 4th and 7th days and in the end only 1 of the 4 treated birds lived, however, the course of the disease was prolonged.

Kylasamaier (29) failed to save a spontaneous case with 3 cc. of immune serum.

NEUTRALIZATION TESTS

In neutralization tests Doyle (2) used serum from 8 birds that had survived (without reacting) an inoculation of stored Newcastle virus, and which had subsequently resisted contact infection as well as two infections of virus. Immune plague serum came from birds that had received formalinized virus and later active virus. In the tests, peritoneal fluid was used as a source of plague virus and mouth exudate as a source of Newcastle virus. Equal quantities of serum-virus mixtures were held 4 days at room temperature and injected in a dose of 0.5 cc. intramuscularly in the case of plague, and 1 cc. intravenously in the case of Newcastle disease. Controls were set up with virus and saline. The plague control died in 40 hours, the Newcastle control on the 9th day. Newcastle serum neutralized its virus but with plague virus permitted the bird to die in 40 hours. Plague serum did not completely protect against its own virus which killed, but only on the 8th day, while with Newcastle virus the bird died on the 9th day.

Farinas (22) used 0.25 to 1.5 cc. of immune fowl serum with 125 minimal infective doses of virus and held the mixtures at 0-1.5°C. for 4 hours, and injected chickens intravenously with the result that those receiving 1 or 1.5 cc. of serum with virus lived while those receiving 0.25 and 0.5 cc. of serum with virus sickened but recovered and were later proved immune. He also reported that mixtures of avian pest-immune serum (Manila) and Doyle's virus (in saliva or organ emulsions) were neutralized when held over night in the ice box.

Later Doyle (50) reported the results obtained by Rodier who used Doyle's virus and serum immune to the Philippine strain. The serum-virus mixtures were held a day in the ice chest before inoculation. Two birds that had each received 1.5 cc. (2 cc. Philippine serum and 1 cc. Doyle's virus) intramuscularly remained well and were later shown to be immune, while of 2 that received the same dose of virus and saline one died.

Other tests were made with organ suspensions filtered to remove particles and tissue. In this case 4 birds that got Philippine-immune serum and Doyle's virus lived while 2 that received normal serum and virus and 2 that received virus alone died.

In another series equal quantities of serum and virus were held at 14°C. for 4 days and 1 cc. of the mixtures given intravenously. The results showed that Newcastle serum and virus produced no reaction while this virus with normal serum and saline produced death on the 6th day. Reciprocally, Philippine virus plus Newcastle serum produced no reaction while this virus and normal serum did not protect the bird which eventually recovered. And the virus with saline caused death on the 5th day.
Manninger (55) states that he immunized "to a high degree" a goose against fowl plague and another against Newcastle disease and that in cross neutralization tests the two sera neutralized uniformly both infective agents. However, his protocol shows that either serum neutralized only 2 fatal doses of the corresponding virus. Nakamura et al. (42) made neutralization tests with the Korean Hühnerseuche virus, the Manila avian pest virus, the Sato strain (from Japan) and Doyle's virus as well as the Chiba strain, which is considered to be plague, and hyperimmune sera. Equal quantities of serum or serum diluted 1-10 and virus were stored for 2 hours and injected subcutaneously. The virus dilution was adjusted somewhat to the known M.L.D. of the strain in question. The results showed that there was cross neutralization with all strains except the Chiba. Moreover, the Chiba strain always killed in a shorter time. In quantitative tests it was shown that the Korean antiserum in a dilution of 1-120 was active against its virus in a 1-50,000 dilution. As pointed out elsewhere, this virus was active in a $10^{-6}$ to $10^{-7}$ dilution.

Sirlaw (59) reported that it requires at least 2 cc. of hyperimmune serum to neutralize 10 M.L.D. of virus.

As listed elsewhere, Keogh (66) found that neutral mixtures of serum and virus were non-lethal for the chick embryo, but that the virus could be demonstrated in the embryo.

Farinas (22) added 1 cc. of normal sheep serum to 5, 25, and 125 M.L.D. of virus and found that only the fowl which received the 25 M.L.D. sickened, but it recovered.

An immune serum diluted 1-800 neutralized the red cell agglutination of a given virus diluted 1-5, that is, in a ratio of serum to virus of 1-160. But, a 1-10 dilution of this serum would neutralize the lethal effect of only a $10^{-4}$ dilution of virus as determined by amniotic inoculation. Therefore, serum, according to Burnet (73), is about 50,000 times as effective in neutralizing red cell agglutination as it is in inactivating the virus in amniotic inoculation.

CROSS-IMMUNITY TESTS

I. Immune to Newcastle Disease:

1. Tested with fowl plague virus.

The 10 birds which supplied immune serum for Doyle's (2) neutralization tests, as detailed elsewhere, were injected intramuscularly with 0.25 cc. of a 1-1000 dilution of plague blood. After they had resisted contact infection and two injections of Newcastle virus, and all died of plague within 40 hours.

Likewise, in pen exposure experiments 6 Newcastle-immune birds and 4 controls contracted plague from 4 birds inoculated with the English strain of plague. The plague-inoculated birds and 3 controls were dead by the fifth day and the remaining control by the 8th day. Tested against a Dutch strain of plague the 2 Newcastle-immune birds died in 6 and 8 days, respectively.

Two birds recovered from the Staffordshire outbreak of Newcastle disease were exposed, one to the English, and one to the French strain of plague, and died in 6 and 4 days, respectively.

Picard (5) tested 3 birds which had recovered 4-6 weeks previously from the
pseudo fowl pest (Newcastle disease) of the Indies and found that they died of plague in 20, 33 and 40 hours.

Purchase (28) demonstrated that a Newcastle-immune bird died in 5 days after injection with an Egyptian virus (an atypical plague) as did 4 healthy controls.

Three birds recovered from the 1933 English outbreak of Newcastle disease and 2 controls were injected with fowl plague by Dobson (64) and all were dead on the third day.

Nakamura et al. (42) showed that birds hyperimmunized against various strains of Newcastle virus (Korean, Japanese and Philippine) were susceptible to the Chiba strain which they consider identical to the European plague. In these tests the virus was injected in a dose of 1 cc. of a 1–10 dilution subcutaneously.

2. Tested with other strains of the same virus:

Farinas (22) sent his own Manila virus to Picard in Java who could find no difference between it and his own Java virus. In turn, Farinas inoculated 9 birds immune to the Manila virus, and 3 controls, with Picard’s virus by intramuscular, subcutaneous, intraperitoneal, intravenous and oral routes with the result that 2 immune birds died instantly as a result of the intravenous injection (mixture of organs and saliva) and the remaining 7 resisted while the 3 controls died in 5, 6, and 7 days, respectively.

Farinas had sent his Manila virus to Doyle who found it immunologically identical to Newcastle virus. In return Farinas inoculated an emulsion of spleen from Doyle into 4 Manila-immune birds and 2 controls with the result that only the latter died of the disease. In a similar test a pooled emulsion of spleen and saliva from Doyle was inoculated intramuscularly in a dose of 1 cc. into 8 Manila-immune birds and 2 controls. The immunes resisted while both controls sickened on the 7th and 8th days, respectively, and died.

Finally, Farinas sent his Manila virus to Cooper of Muktesar, India who could find no difference immunological or otherwise, between it and the Ranikhet virus of India. In turn, Farinas inoculated 6 Manila-immune fowls with Cooper’s Ranikhet virus and none sickened. Then 2 immunes resisted the Ranikhet virus while the 2 controls died.

Rodier of the Philippines also made test with Doyle’s virus which Doyle (50) was permitted to publish. Two control birds died in 6 and 10 days, respectively after an intramuscular injection of 1 cc. of a spleen emulsion of Doyle’s virus while 4 Philippine-immune birds resisted the same dose. In another test, 2 controls sickened and 1 died after a 1 cc. subcutaneous injection which had no effect on 8 Philippine-immune birds. Finally, 2 Philippine-immune birds resisted a dose which killed 2 controls in 7 and 8 days, respectively. Reciprocally, a Newcastle-immune bird resisted a 1 cc. spleen emulsion intravenously which killed 2 controls in 5 and 6 days and a similar immune bird in contact with 3 birds affected with the Philippine disease remained well, while a control died on the 7th day. Finally, a Newcastle-immune bird resisted a 1 cc. spleen emulsion injected intravenously which killed a control on the 4th day, and while a control in contact died on the 11th day.

Nakamura et al. (42) used fowls hyperimmunized to the Korean Hühnerseuche, the Sato Japanese strain and the Philippine avian pest strain and found that there was reciprocal cross-immunity. The test dose was 1 cc. of a 1–10 dilution given
NEWCASTLE DISEASE

subcutaneously which killed control birds in 5–6 days. They also obtained Doyle's strain and by subcutaneous, intramuscular or subdural inoculation it killed 4 normal birds within 8 days, but failed to produce disease in 2 Sato-immune and 1 Korean-immune birds inoculated subcutaneously.

According to Albiston and Gorrie (72) virus from the 1932 Australian outbreak was found by Doyle to cross immunize with Newcastle virus. The Australians also reported that a fowl which was naturally immune to the 1932 virus remained normal after a subcutaneous injection of virulent 1930 virus.

Likewise, Doyle (2) showed that a bird immune to Newcastle virus resisted the virus from another English outbreak (Staffordshire), and reciprocally, a bird recovered from this outbreak resisted Newcastle virus.

An atypical avian pest appeared in the Philippines and Acevedo (39) injected blood from these cases intramuscularly into 4 avian pest-immune birds (Newcastle disease) without effect while a control died in 5 days. Then the same 4 immune were reinoculated with 3 cc. intramuscularly of a liver spleen emulsion of the new virus and resisted while 2 controls receiving 2 and 1 cc. respectively, died on the 4th and 5th days. Reciprocally, a bird which had recovered after an injection of 0.00004 cc. of blood virus of the atypical disease resisted 125 M.L.D. of typical avian pest virus which, in a dose of 1.25 M.L.D., killed a control on the 7th day.

II. Immune to fowl plague:

1. Tested with Newcastle virus:

Doyle (2) immunized a fowl against plague with formalinized vaccine and proved its immunity by pen exposure for three weeks (23, normals died in this time) as well as by an injection of plague which killed a control in 40 hours. This bird died on the 8th day after a 1 cc. intravenous injection of mouth exudate Newcastle virus. Another plague-immune bird which had resisted pen exposure as above to plague, died on the 10th day, and a control on the 8th after pen exposure to Newcastle disease.

The effect of pseudo plague (Java) virus on plague-immune birds could not be tested by Picard (5) because all attempts to immunize failed.

Nakamura et al. (42) demonstrated that birds hyperimmunized to the Chiba strain (fowl plague) were susceptible to the Korean Hühnerseuche virus, the Sato virus, and the Philippine avian pest virus all of which are considered identical to Newcastle virus.

A typical plague occurring in Egypt, and described by Lagrange (9, 10, 13, 16, 18), was for sometime considered to be Newcastle disease. But Purchase (28) studied the Egyptian virus and concluded that it was an atypical fowl plague. Among other things he showed that 3 birds, each immune to a different strain of fowl plague, were also immune to the Egyptian virus which killed the 4 controls in 4–5 days. Likewise, he showed that 2 birds immune to the Egyptian virus were also protected against separate strains of plague, while controls died in 48 hours. Thus, having established the identity of the Egyptian and plague viruses by reciprocal cross-immunity tests, he showed that a bird immune to the Egyptian virus died in 7 days after an inoculation of Newcastle virus which killed a control in 5 days.
NATURAL IMMUNITY

The question of natural immunity was raised by Farinas (22) who found 2 chickens that resisted an inoculation that killed others. They were retested and found to be solidly immunized. Since these birds were only 3½ months old, the possibility of an earlier attack seemed unlikely, the more so, because they had come from a place where the disease had not been present for 2 years previous to their purchase. The question was unanswered.

However, in the course of experiments Iyer and Dobson (70) found 2 hens which resisted subcutaneous injection of virus, and were therefore presumed to be naturally immune. They were mated to a cockerel (later shown to be susceptible), and 3 female and 2 male chicks were hatched. At 3–4 weeks of age, the chicks were injected subcutaneously with 50 million M.L.D. of virus and, except for one which died of other causes 2 days later, all lived. As a control 11 chicks hatched from susceptible parents (as shown later) were tested at 1–4 weeks of age with 20 to 50 million M.L.D., or exposed to contact infection with the result that all died. They concluded that resistance may be inherited in a general manner, and that since male and female chicks made up the population, that resistance is not sex-linked. Breeding was suggested as a means of controlling the disease.

DIFFERENTIVE DIAGNOSIS

I. Fowl plague:

The similarity of Newcastle disease to fowl plague has been pointed out by many (2, 4, 5, 6, 12, 14, 17, 22, 35, 41, 42, 43, 44, 58, 63, 69) and its identity with plague only by Manninger (33, 55).

Doyle (2) early suspected the difference because of occasional failure of the blood to infect, whereas in plague it never fails. The highest concentration of Newcastle virus found in the blood by Doyle was 0.00004 cc. as an infective dose, whereas in plague it may reach a dilution of $10^{-9}$ according to Krumwiede et al. (A. V. M. A. May 1925). The longer period of incubation, the longer course, and the presence of respiratory symptoms further characterized Newcastle disease. Moreover, this disease always infected by contact which sometimes failed in the case of plague, and it was transmissible to pigeons of all ages.

The complete absence of virus in the blood of pseudo fowl plague (of the Indies) as it was at the time of Picard's (5) early reports gave him a good means of distinguishing his disease. Also, he pointed to an incubation period of 4–8 days in 4 birds inoculated with pseudo plague in contrast to 13–24 hours in 4 birds injected with plague. Likewise, the course of the disease in the former was 3–10 days in contrast to 18–30 hours in the latter. Symptomatically, pseudo plague birds showed mouth discharges, dyspnoea, diarrhea, cyanosis, paralysis and nervous disturbances in contrast to somnolence, cyanosis, absence of diarrhea, edema and prostration in plague. Autopsy findings in the 2 diseases were similar, but the hemorrhages were more predominant in plague which also showed acute peritonitis and a hemorrhagic transudate in the abdominal cavity.

Gomez (17) used the absence of virus in the blood of his disease as well as its infectivity for pigeons as features distinguishing it from plague.
NEWCASTLE DISEASE

But, when Manninger (33) claimed to have changed Doyle's virus by rapid passage so that symptomatically, anatomically and even immunologically it was identical to plague, Doyle and others attempted to duplicate these results. As pointed out elsewhere, Doyle and others failed in these attempts. Against Manninger's argument that Newcastle virus is merely an attenuated form of fowl plague Doyle (43, 50) cites the fact that the mortality is nearly 100 per cent. And, against considering it merely as another strain of fowl plague (immunologically distinct) is the fact that in other diseases in which this occurs the diseases produced are at least alike whereas here the period of incubation, symptoms and lesions are different.

Lagrange's (9) Egyptian disease presented a more difficult problem in differential diagnosis for a while. The disease had a longer course than plague (4–8 days), and the low infectivity of the blood (1–200 cc.) caused many to consider it as Newcastle disease. But, Lagrange never concurred in this belief. In fact, he cited these differences, plus its failure to infect ducks and pigeons, as features distinguishing it from plague. He distinguished it from Newcastle disease by the absence of respiratory symptoms, the lower infectivity of the blood (1/500 never killed as compared to Doyle's 0.0004 cc.) and its non-infectivity for pigeons. Lagrange also distinguished his disease from Picard's which was transmissible to ducks, geese, and pigeons.

The question of the position of Lagrange's disease was disposed of by Purchase (29) who found that salivation was not a symptom, that it was not pathogenic for pigeons, showed plague lesions, and that, in contact infection experiments, it approximated plague, that it was in fact, as pointed out elsewhere, immunologically identical to plague even though the period of incubation was longer and the infectivity of blood low (highest 0.001 cc.).

Carpano (41) agreed with Purchase that Lagrange had no real basis for distinguishing his disease from fowl plague. He states that the differences cited by Lagrange are within the limits of variation capable of being exhibited by classical plague, that in the matter of the course of the disease birds inoculated for the diagnosis of plague between May 2, 1930 and Dec. 31, 1931, died after varying periods, that is, 2 died 2 days after infection, 26 after 3 days, 22 after 4 days, 5 after 5 days, 3 after 6 days and 1 after 7 days.

Ochi and Hashimoto (12) and Konno et al. (14) differentiated their disease from plague by its longer course and incubation, presence of respiratory symptoms, ease of contact infection, and a difference in the distribution of hemorrhages, that is, in the Korean disease these were confined largely to the digestive tract—in the duodenum in 90 per cent of the cases—whereas in plague they were clearly present, but not in the duodenum. They could not differentiate their disease from those described by Doyle and Picard. They had no opportunity then to make cross-immunity tests.

But, as pointed out elsewhere, the immunological identity of various viruses (Korean Hühnerseuche of Ochi and Hashimoto) to Sato's Japanese strain, Avian Fowl Pest of Farinas and Rodier's virus (Philippine), Cooper's Ranikhet disease (India), Picard's Pseudo Plague (Dutch East Indies) and Albiston's Victorian Virus (Australia) has been well established with Newcastle virus.
Summing up all the differences between Newcastle disease and fowl plague we find that on the basis of:

1. Species susceptible, Newcastle infection is readily reproduced in pigeons and ducks, whereas these are highly resistant to plague. Newcastle virus cannot be carried serially in mice by subdural inoculation while plague can.

2. Geographic distribution, Newcastle disease has not appeared in Europe (except England) where plague has been reported for years, nor in the United States where two plague outbreaks have occurred. And, the areas now stricken with Newcastle disease have not been notable for outbreaks of plague.

3. Period of incubation and course, the period of incubation in Newcastle disease averages 4–5 days and the course from one to several days in contrast to death in 2 days from infection in the majority of plague cases.

4. Infectivity by contact, Newcastle disease is highly infectious while plague may fail in laboratory tests.

5. Symptomatology, Newcastle disease is characterized by dispnoea, diarrhea, mucus in the mouth, disagreeable odor and paralytic or nervous disturbances while in plague these are lacking and many birds show edematous infiltrations about the head.

6. Post mortem findings, Newcastle disease shows less abundant hemorrhages on the heart, mucosa of proventriculus and beneath cuticula of gizzard or sometimes their absence from one or more of these places plus hemorrhages in intestine and charges in the crop, while in plague profuse and widely distributed hemorrhages on serous membranes are characteristic with a general absence on the mucosa below the level of the gizzard. Also charges in the crop are lacking.

7. Histopathology, Newcastle disease has not yielded inclusion bodies while in rare cases of plague Klein’s corpuscles have been described.

8. Virus particle size, Newcastle disease virus was calculated to be 80–120 mμ as compared with 60–90 mμ for plague.

9. Egg propagation, Newcastle virus requires somewhat longer to kill the embryo and causes ectodermal proliferation which is not seen in plague.

10. Immunity, Newcastle and plague do not cross-immunize nor do the immune sera cross-neutralize the lethal effect in chickens and embryos, or the agglutination of red cells by virus.

11. Susceptibility to photodynamic inactivation by methylene blue, Newcastle is the more resistant.

12. Distribution of virus in the body; Newcastle virus is concentrated in secretions of the mouth and may be absent in the blood, whereas plague virus is more evenly distributed in the body and never absent from the blood (in chickens).

13. Modification of virus by continued egg passage, Newcastle virus has been modified to the point of not producing apparent disease on inoculation which provokes active immunity, while to date, this has not been demonstrated with plague virus.
II. Other diseases:

Several infections have been mentioned by various authors discussing differential diagnosis. Some of these disease bear some similarity to Newcastle disease but others seem quite remote to this reviewer.

1. Fowl cholera is mentioned by Doyle (2) who points to negative cultures and insusceptibility of rabbits.

2. Typhoid is mentioned by Shah (52) who proposes only microscopic examination of blood (?). The same author also mentions pullorum disease.

3. Spirochaetosis is mentioned by Shah who proposes microscopic examination of the blood.

4. Blackhead and Coccidiosis are also mentioned by Shah and here again the microscope is resorted to.

5. Fowl paralysis is mentioned by Rodier (6) and Farinas (22) because of the paralytic symptoms in both diseases. This hardly requires comment.

6. Laryngotracheitis, is mentioned as similar by Doyle (2), Rodier (6) and Konno et al. (14) on the basis of symptomology, but the presence of blood in the trachea readily distinguishes this disease.

7. The respiratory-nervous disorder of Stover (76) or avian pneumoencephalitis of Beach (77) not mentioned by writers on Newcastle disease, deserves consideration by virtue of gasping, nervous phenomena, distribution of virus in the body and infectiousness for pigeons. It differs from Newcastle in a low death rate, difficulty of transmission from field material in the laboratory, and the failure of the virus to kill embryos in early passages.

COMPARISON WITH OTHER VIRUSES

In a comparative study Burnet (73) observed certain similarities between Newcastle and influenza viruses. Both produced small chorioallantoic foci and provoked identical-appearing hemorrhagic encephalitis. Multiplication takes place freely in allantoic and amniotic cavities, and infection can spread over the whole respiratory epithelium. In both cases chorioallantoic inoculation resulted in death of the embryo with hemorrhagic lesions, but without specific lung lesions, or appearance of virus in the amniotic fluid. Both viruses agglutinated fowl red cells of the same general character and similar titre.

Because of these resemblences Burnet believes that the viruses are related although serologically different. He raises the question as to the likelihood that members of the group are derived from viruses which developed most of their characteristics in birds!

The relation of Newcastle disease to rinderpest was considered by Kylasamaier (29). One cc. of liver emulsion of rinderpest virus subcutaneously killed a fowl in 16 days, with lesions suggestion of pest. Likewise 1 cc. of rinderpest blood killed another fowl in 15 days with pest-like lesions. Another lot of 4 chickens given pest blood died in from 4-10 days, and 2 given 3 cc. of pest liver subcutaneously died in 10 and 16 days, respectively.

However, two calves given virus from a fatal case of the fowl disease were not affected, and were later shown to be susceptible to rinderpest. Moreover, a fowl
was not protected against 5 cc. of liver virus from an infected fowl administered with 5 cc. of class 2 antirinderpest serum.

Turkhud (27) mentioned the similarity between the fowl disease and psittacosis.

SPREAD OF THE DISEASE

A discussion of the manner of dissemination would seem to concern three questions, namely the manner of spread from one country to another, the manner of spread from one flock to another, and finally the manner of spread from bird to bird, or more properly the portal of entry.

Thus far, no country, having its first outbreak, has been able to discover the source of infection, but it is interesting to note that the first outbreak usually involves a port or at least a coastal city (Batavia, Newcastle, Manila, Melbourne, Mombasa). This would suggest that infection comes in with overseas traffic. Hudson (60) in Kenya does mention India. Moreover, the present distribution of the disease concerns adjacent land areas, and even though they may be far apart in distance they are adjacent. On this basis, if the disease ever reaches the United States in normal traffic it is most likely to appear on the west coast.

The means of spread from one flock to another within a country is, obviously, largely a question of traffic in live birds, Doyle (2) pointed this out with reference to the Somerset and Staffordshire outbreaks in England. Farinas (22) mentions spread by the purchase of market birds as does Johnstone (26) in Australia. Markets should perhaps always be looked upon as sources of infection because they so often serve as an outlet for healthy appearing birds from a stricken flock.

Kuppuswamy (49) mentions the dealers in India who carry birds in baskets and so spread infectious droppings. Konno et al. (14) also mentions bird dealers, and traffic in live birds. Farinas (22) condemns the practice of borrowing roosters (Philippines).

The feeding of offal appears in the histories of the Newcastle and Cheltenham outbreaks.

Free-flying birds have been mentioned frequently as possible means of spread (14, 22, and others), and of these crows have been listed most often. But, as cited elsewhere, the only examination made on crows dead or dying near an outbreak gave negative results.

But, of the various means of spread, traffic in live birds stands as the most important. The proof is that where quarantine measures were instituted the disease was controlled (England and Australia).

As regards the actual means of spread from bird to bird within the flock the question is not settled even though the majority of authors refer to digestion tract infection. But, as suggested elsewhere, infection by feeding contaminates digestive as well as respiratory tracts. In fact, the one investigator who gave the virus in a capsule failed to produce infection or immunity.

However, most workers are agreed that rather close contact is necessary for the spread of infection. Picard (5) put 8 healthy birds in a pen, in which 10 had died and which had not been cleaned 4 days after the last death and only 3 birds died on the 4th, 5th and 8th days afterwards, the others remained well. Two fowls were put in a small cage 14 days after the last bird died and they remained healthy during 6 weeks. Dobson (47) placed a bird in a recently vacated and infected cage and the
exposed bird sickened on the 8th day. Hudson (60) showed that infection still remained in a box for 1 or 2 days after a sick bird had been removed. But, the same box stored in a cool place (Kenya) from Dec. 20, 1935 to Jan. 3, 1936 was then not able to infect 2 fowls placed in it for 5 days. Still, it should be remembered that when chick were introduced on the depopulated and disinfected (?) farm in England 7 weeks later, they contracted the disease in a week.

Farinas (22) claims that expired air is not a factor, and that if birds are kept apart the disease does not spread. To show that the disease was spread by contaminated water Kee (8) cites that healthy birds kept in cages near sick ones failed to get the disease.

Flies and mosquitoes are not considered as of any consequence in the D. E. Indies (48).

**EFFECT OF VARIOUS AGENTS ON VIRUS**

**I. Disinfectants:**

The effect of a number of disinfectants on the virus was tested by Doyle (2). Equal quantities of virus (saliva-active in $10^{-6}$) and disinfectant were mixed and held 1 hour at room temperature and then injected intravenously in a dose of 1 cc. with the following results:

<table>
<thead>
<tr>
<th>Agent</th>
<th>Virus active</th>
<th>Virus inactive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potassium permanganate</td>
<td>1-10,000</td>
<td>1-5000</td>
</tr>
<tr>
<td>Lysol</td>
<td>1-5000</td>
<td>1-1000</td>
</tr>
<tr>
<td>Azol</td>
<td>1-1000</td>
<td>1-500</td>
</tr>
<tr>
<td>Cresol (R-W coef. 18-20)</td>
<td>1-1000</td>
<td>1-500</td>
</tr>
<tr>
<td>Carbolic acid</td>
<td>1-100</td>
<td>1-20</td>
</tr>
<tr>
<td>Bichloride of mercury</td>
<td>1-100</td>
<td>1-20</td>
</tr>
<tr>
<td>Oil of cloves</td>
<td>1-100</td>
<td>1-20</td>
</tr>
<tr>
<td>Sodium salicylate</td>
<td>1-50</td>
<td>1-20</td>
</tr>
<tr>
<td>Copper sulphate</td>
<td>1-50</td>
<td>1-20</td>
</tr>
<tr>
<td>$\text{H}_2\text{O}_2$ (20 vols.)</td>
<td>1-10</td>
<td>1-2</td>
</tr>
</tbody>
</table>

(With virus active in 1-500,000)

<table>
<thead>
<tr>
<th>Agent</th>
<th>Virus active</th>
<th>Virus inactive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antiformin</td>
<td>1-500</td>
<td>1-100</td>
</tr>
<tr>
<td>Formalin</td>
<td>1-50</td>
<td>1-2</td>
</tr>
<tr>
<td>Acetone</td>
<td>1-5</td>
<td>1-2</td>
</tr>
<tr>
<td>Ether</td>
<td>1-4</td>
<td>1-2</td>
</tr>
<tr>
<td>Methyl and Ethyl alcohol</td>
<td>N/100</td>
<td>N/50</td>
</tr>
<tr>
<td>NaOH</td>
<td>N/25</td>
<td>N/100</td>
</tr>
</tbody>
</table>

One batch of vaccine treated with chloroform (Kelser method for rinderpest) remained active (24).

According to Farinas (22) Clenzal at 2.5 per cent kills the virus in 30 minutes, 1 and 2 per cent formalin kills in 30 minutes, but 0.1 and 0.5 do not, 1 per cent chloroform kills in 30 minutes, but 0.1 per cent failed, and N/1 HCl killed in 1 hour, but N/10 and greater dilutions were ineffective. Attempts to test the action of sodium hydroxide were not successful because even an N/2500 solution by intravenous injection in itself killed the fowl. Dilutions of potassium permanganate from 1-2000 to 1-10,000 containing 10 per cent of crop content as a source of virus
and given to fowls to drink failed to infect in all dilutions, while such a suspension without the permanganate infected.

One ounce of sodium hypochlorite per 4 gallons of water was found to be the most effective agent of those tested by Albiston according to Johnstone (37).

II. Heat:

According to Farinas (22) virus was inactivated in 30 minutes at 75°, 62°, and 55°C. while that held at 50°, 42° and 37°C. caused infection. Crop content dried on cotton was inactive after 3 days at 37°C. Virus was active after 48 hours at 37°C. but not after 72 hours.

III. Cold:

1. Dry: Picard (5) dried brain in 48 hours at 27°C Celsius and stored it over CaCl₂ and found it active after 35 days. However, the 3 fowls injected subcutaneously recovered. Cooper (24) found that sterile organs dried and held at 4°C. were active for 125 days and probably 169.

Haddow (45) reported that dried vaccine was shown to be virulent after about 2 years.

Doyle (2) ground organs, pressed them between sheets of sterile filter paper in a dish and dried them in a desiccator over CaCl₂ in the cold. Activity was tested by an intravenous injection of 1 cc. of suspension except in the case of brain which had to be given intramuscularly. Lung, liver, spleen and brain were active at 40 days but not at 100, kidney was active at both periods and ovary at neither. Strips of organs dried over CaCl₂ or H₂SO₄ and held 5 months and 18 days were still virulent (22).

2. Moist: Virus in 50 per cent glycerin-saline was active at 197 and inactive at 259 (2). Farinas (22) found heart and spleen in pure glycerin at 0–2°C. still active at 175 days. A 1–10 brain suspension in 50 per cent glycerin was active after 5½ months. The 3 injected fowls died in 15 days. (5). Crop content in saline and 33½ per cent glycerin was inactive in 2 mos. and 26 days at 0–4°C. In 50 per cent glycerin it was inactivated in 4 mos., 20 days. Berkefeld filtrate in water was virulent 1 month and 22 days, and dilute crop content in water plus 30 per cent Sorenson’s phosphate mixture of pH 6.8 (final pH 7.2) remained virulent for 320 days (22).

Brain and spleen in 50 percent glycerin and held at 1°C. for 239 days produced death on the sixth day after laryngeal inoculation (61). Virus in saline active at 86 days, dead at 100 (2).

Saliva in a 1–10 dilution after 6 months caused the disease in only 2 of 3 birds injected with 2 and 3 cc.; the third bird had received only 1 cc.

Whole spleen active after 80 days, dead after 150 (2).

Blood in sealed pipettes active after 109 days (2).

Crop content in water at 0–4 C. active after 5 mos. and 11 days. Organ emulsions in water active after 2 mos. and 18 days (sealed) and with cotton plugs active for 1 mo. and 5 days.

Doyle (43) found bone marrow and muscles of chilled carcasses active after 6 months.
IV. Direct sunlight:

Diluted virus was placed in Petri dishes by Farinas (22) and floated in water to prevent heating. Exposures were made between 10 A.M. and 1 P.M. (Philippines) for 30 minutes, 1 and 3 hours, but even the shortest exposure killed the virus. A 1–10 dilution of saliva in a cotton-plugged tube was inactive after 24 hours exposure to daylight (5).

V. Room temperature:

1. Dry:
   Crop content dried on cotton and held for 5 days was inactive (22).

2. Moist:
   Berkefeld filtrate active after 1 day. Liquid crop content in cotton stoppered tube active after 15 days. Spleen and liver in Petri dishes covered with dry sandy earth was inactive by feeding 12 days later as was crop content after 4 days when mixed with garden soil and covered with rough glass to diffuse light. Chickens on two occasions failed to become infected from drinking water infected with crop content (50 cc. to 2 l.) after it had stood for 4 days (22).

   Farinas (22) also tested the infectivity of the spleen of dead animals by an injection of 0.5 cc. of an emulsion subcutaneously. Tests were made from 36 to 168 hours, and only one bird became infected and this from a spleen removed 72 hours after death.

PHOTODYNAMIC INACTIVATION BY METHYLENE BLUE

Burnet and Ferry (44) employed the photodynamic inactivation by methylene blue as a means of distinguishing between Newcastle and fowl plague viruses. A dilution of 1–50,000 methylene blue was used and filtered virus mixed with this and exposed in layers 2 mm deep at 15 cm. below a 300 c.p. filament lamp. Heating was prevented by a glass dish containing 3 cm. of water through which a flow of water was maintained. They found that Newcastle virus exposed 30 minutes had its titre reduced from 1–100,000 to 1–100 (inactive at 1–1000), while plague virus after only 10 minutes had its titre reduced from 1–100,000 to 1–10.

The method could be used to separate a mixture of viruses. Thus, a mixture of equal parts of the two viruses was exposed 10 min., and then eggs were inoculated with undiluted irradiated material which died in 24 hours (from plague) while those inoculated with a 1–100 dilution died after 2 days and showed the typical ectodermal proliferative lesions peculiar to Newcastle disease.

Shirlaw (59) attempted to attenuate Newcastle virus by this method with the view of producing a suitable immunizing product, but with no conclusive results.

THERAPEUTIC AGENTS

Kee (5) fed native onions and green peppers 3 times a day, and quinine bisulfate with water at 2 grains 3 times a day with the result that 6 died in 4 days and 14 recovered.

Picard (15) tested the prophylactic and curative effect of permanganate, Zonite, neosolvarsan and urotropine and concluded that once the virus is in the tissue it could not be affected by any of the agents tested.
Khan (20) opened the crop and flushed it with Lugol’s solution containing potassium permanganate 1-1000 and Lysol 1-5000. This was also injected in the rectum and placed in drinking water, but the results were negative. Results are not given when a mixture of Quin. sulf. gr. 5, dil. sulf. ac. m. 3, and water dr. 4 was used or when a mixture of Bis. subn. gr.5, oil terab. m.4, oil eucalyptus m.2, Tr. card. comp. m.10, Tr. asaf. m.8, ar. spts. am. m.16 and water dr. 4 was employed. Lugol’s solution subcutaneously and intravenously was also tried in 2-4 cc. doses. Of 4 birds treated at the height of temperature, 3 recovered after 5 days treatment, and the other died of paralysis a month later. At another time 2 treated birds died, while “those (2) left to the mercy of God died on the second day.” Because of these encouraging results 80 cases were treated at Lyall-Pur (India) by intravenous injections with the result that 65 recovered.

Farinas (22) tested the therapeutic worth of a number of agents including: Hizons’ phenamethylene, Clenzol or electrolytic hypochlorite of soda, sodium cacodylate, creolin, arsenisetin, arsenic iodide, indol, mercurochrome soluble-220, glucose, strychnine sulphate, quinine and urea hydrochloride, gold chloride, Intracid, acriflavine, neo-salvarsan and Fowler’s solution of which only four gave promise. Hizons phenamethylene diluted 1-3 with water and given intravenously to 3 fowls resulted in their recovery and later they were found to be immune. A 0.05 per cent solution of gold chloride in a single intravenous dose of 2 cc. to 9 fowls at height of disease resulted in 4 recoveries which were later shown to be immune.

A 10 per cent aqueous solution of Intracid intravenously in a dose of 2 cc. to 2 birds resulted in one recovery. Large doses of Fowler’s solution per orem in field cases seemed quite successful, but not in artificially infected birds. Of 5 field cases treated with 3 daily doses of 10 cc. and 2 daily doses of 5 cc. 3 recovered in 4 weeks, and 2 developed nervous derangements which disappeared in about 2 months. Servilla (53) also used Fowler’s solution in a dose of 10 drops daily to experimental birds with apparent favorable results, but suggested further trials. Kuppuswamy (49) found that potassium permanganate did not prevent the disease in birds exposed to contact infection, and treated birds died.

Naik (56) had no results with intravenous injections of formalin or Lugol’s solution, nor with potassium permanganate solutions in brandy or 0.3 per cent HCl in milk. However, he claimed good prophylactic results in a field test with intravenous or subcutaneous injections of 1-40 cc. of a 1 per cent saline solution of trypanblue. In an experiment, birds were treated with 2-3 cc. and then exposed in parallel to contact infection or injection 7 days later, 24 hours later, and simultaneously. Those simultaneously exposed received intravenous, and all others subcutaneous injections of the drug. The result was that all birds got the disease and died or were killed within 12 days. His field results showed that in 3 flocks of 406 birds 33 or 8.1 per cent died, but only 4 or 11 per cent died excluding those having pre-injection infection.

But this agent in the hands of Shirlaw (59) gave opposite results, that is, treated birds were more susceptible and suffered more severely. On the basis of reported success in the use of camphor as a prophylactic in rinderpest this agent was used for
the fowl disease with encouraging results in preliminary trials but in large scale experiments the results were not so encouraging.

HANDLING AN OUTBREAK

Recognizing the infectious nature of the new disease Kraneveld (1) (D.E.I.) laid down the usual measures such as destruction of sick birds, digging out the runs, isolation, and disinfection of coops. He especially warned against buying kampong birds because in these the disease rages uncontrolled.

Cooper (24) (India) brought the disease under control by destruction of affected birds, the use of potassium permanganate or iodine in the water and feed, and phenyle compounds in the pens.

Kretzer (21) (Philippines) recommended destruction of the sick, slaked lime in the runs, and permanganate in the water which was to be changed twice a day, while Farinas (22) of the same place suggested 2–3 per cent creolin for the ground. He cited a flock of 61 chickens, 3 geese, 6 turkeys and 15 ducks in which 2 fowls had died, and 3 were showing symptoms. The flock was divided into 3 parts, sick, suspicious, and well, with the result that only 15 chickens sickened of which 13 died.

The measures instituted to bring the second Victorian outbreak under control were detailed by Johnstone (37). Auction rooms were set up to handle poultry from non-infected districts. Users had to get permission from the Chief Veterinary office, and had to declare that the birds had been in their possession for 28 days, and that no disease had appeared. The written permit thus issued had to accompany the birds. Poultry forwarded on a declaration could be used for restocking, but birds forwarded on a statement had to be slaughtered.

In order to facilitate the sale of poultry from disease-free farms within the quarantine district certain auction rooms were provided with facilities for slaughtering and dressing. Owners had to forward a declaration of health of the poultry on their farms, and on receipt of this, a written permit covering the movement from farm to auction room was issued. The permit had to accompany the birds and all birds entering the rooms were slaughtered. The slaughtered birds were fire-branded as a guarantee that they were free of disease at the time of slaughter. All crates, vehicles, etc., used in conveying the birds were disinfected before leaving the premises.

There were no restrictions on the sale of eggs from clean farms in Victoria.

Infected farms were quarantined and birds were removed only for immediate slaughter, and eggs had to be disinfected before removal.

Owners were instructed to notify the Chief Veterinary Inspector immediately of any disease with symptoms resembling Newcastle disease. Visiting of poultry farms was advised against, and if unavoidable, the clothing was to be washed with a solution of sodium hypochlorite solution (1 oz. to 4 gals. of water). Garbage from hotels was to be fed only if cooked, and egg shells were to be kept out of the feed. The flock was to be managed so that it would be unnecessary to enter pens to feed the birds or collect eggs and permanganate (1 oz. to 64 gals.) was suggested for the water. An impervious partition 2–3 feet high between pens was recommended.

Evidence that the regulations were enforced is found in a note following Johnstone’s article. A Mrs. A. A. Y. having failed between Oct. 1 and Nov. 28 to re-
port to the Dept. of Agriculture that pseudoplague existed in her poultry was fined 5 pounds and costs of 5 pounds and 5 s. One G. C., Stock Inspection, testified that on Nov. 24 he visited the farm and in one pen found 2 dead fowls, and in another, 4 or 5 sick. These were examined by the Veterinary Research Institute. The birds had been purchased innocently on the market, and disease did not appear until after delivery.

Outbreaks in England in 1926, and 1933, were controlled by the slaughter method. The 1933 outbreak began on one farm in Aug. 1933 and never spread further. Operations were resumed on this plant the following November (64).

PREVENTION

Since transmission of the disease requires rather close contact as indicated already, and since infection can be spread by infected equipment, droppings and offal, avoiding these means of spread appears to be all that is necessary to prevent the disease, especially traffic in live birds.

Crawford (23) in Ceylon warned owners to keep their birds at home and also exclude straying birds. He advised 2 weeks quarantine of purchased birds. In India, Kuppuswamy (49) advised that poultry dealers be kept out of the villages where the disease is known to exist. New purchases as well as birds from poultry shows should be quarantined 21 days. Birds purchased for table purposes at the market should be dressed there and offal buried. Also recommend confinement of birds and exclusion of visitors.

During the 1930 outbreak in Victoria, South Australia protected itself under the Stock Disease Act and prevented the importation of any poultry, duck, geese, turkeys, etc. as well as their eggs. A close watch was kept on overseas steamers so that foreign birds were not admitted except by proper certificates. Poultrymen were instructed to report all disease so that an investigation could be made (25).

At the same time N. S. Wales instituted certain measures as reported by Henry (31) when informed on Dec. 18, 1930 of the Victorian outbreak. Here finches, parrots, pheasants and quail were declared to be stock within the meaning of the act. Traffic into the state was controlled by the border control so that introductions of eggs were prohibited unless accompanied by a certificate from an Inspector of Stock that they were produced on a holding not less than 15 miles from any holding on which an outbreak had occurred. Moreover, the containers had to be new or else cleaned with hot water and soda. The introduction of carcasses was absolutely prohibited. The last outbreak in Victoria was reported March 6, so that restrictions were gradually lifted until July 1 when freedom of traffic was fully restored.

The measures appear to have been effective because the disease never spread beyond the state of Victoria.

Thanks are due Prf. H. M. Biekart for translation from the Dutch.

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NEWCASTLE DISEASE


NEWCASTLE DISEASE


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In the enforcement of the Federal Food, Drug, and Cosmetic Act the Food and Drug Administration has had occasion to evaluate several remedies which were represented by means of labeling for the prevention and treatment of coccidiosis in poultry. While these coccidiosis remedies varied widely in their composition, the method or methods by which they were to be administered were somewhat similar.

To understand better the problem of evaluating a coccidiosis remedy it will be necessary, first, to understand the life history of the organisms which cause coccidiosis, and second, to know the effect or the effects of the various stages in the life history of the coccidia organisms on the chicken.

At the present time there are recognized 8 or 9 species of coccidia which attack the chicken. Of this number, 4 or 5 species may cause considerable damage. It is a well known fact, among investigators in this field, that each species of coccidia has a preferred place in the intestinal tract where it enjoys optimum development. From this particular characteristic, the species of coccidia infecting the chicken have been roughly divided into the cecal type and the intestinal types. In speaking of the cecal type of coccidiosis, reference is made to that species which inhabits the ceca or blind pouches, these structures being located near the posterior end of the intestinal tract. Those species which are designated as intestinal are found in various parts of the gut from the gizzard down to the cloaca.

Without question, the type of coccidiosis which causes the greatest damage in the United States to the chicken industry, is the cecal or bloody type. Because of this fact, the major part of this brief discussion will be confined to cecal coccidiosis.

The cause of cecal coccidiosis is a protozoan or one-celled organism known scientifically as *Eimeria tenella*. When this organism is passed from an infected chicken it is in the droppings in the uninfective cyst stage also called the unsporulated oocyst. A coccidium in the oocyst stage is egg-shaped and is approximately one-thousandth of an inch in diameter. All other stages in the life history of this organism are microscopic also. After 36 to 48 hours, exposure to the proper conditions of temperature and moisture, this unsporulated oocyst develops into the infective spore-containing stage, also called a sporulated oocyst. When a sporulated oocyst is eaten by a chicken there begins immediately the development of a new generation of coccidia. The time required for the development of this new generation within the chicken is approximately 7 days. The increase in the number of coccidia is tremendous. Provided there is no loss or destruction in any phase of the life cycle there would be developed as a new generation, from a single oocyst, more than one million new individual oocysts.

The life history of the coccidium which causes cecal coccidiosis has been worked out by several investigators and is very complicated. Starting with one ripened
Coccidiosis Remedies for Poultry

or sporulated oocyst which has just been eaten by a chicken, there are liberated, in a very short time, 8 much smaller bodies called sporozoites. These small bodies which are motile, immediately penetrate the epithelial cells lining the ceca or blind pouches. Each sporozoite starts to grow at once, and grows in the cecal cells for approximately 2 days, at the end of which time its size may be as large or larger than the cell which it occupies. This stage is called Schizont Generation I. This Generation I schizont now bursts and liberates almost one thousand very small active bodies called Generation I merozoites, which immediately penetrate other cecal cells and start to grow in the same manner as the sporozoite. In less than 2 days these Generation I merozoites have developed into a second series of large bodies called Generation II schizonts. On the fourth or fifth day the Generation II schizont bursts and liberates from two hundred to three hundred Generation II merozoites. At this time or shortly before, the first blood appears in the droppings, and it may continue for 2 or 3 days increasing or decreasing in volume according to the degree of the infection. Most of the mortality usually occurs, in acute cases, during this period of hemorrhage. As previously stated, with the passage of blood, there are liberated in the ceca large numbers of Generation II merozoites. These Generation II merozoites, which are also motile, immediately penetrate other epithelial cells lining the ceca. The development from this point on enters the sexual stage. The Generation II merozoites differentiate in their development into male and female cells. Within 3 days the female cells are fertilized and the new generation of coccidia begin to appear in the droppings as the uninfective cyst-stage. After proper exposure to temperature and moisture this new uninfective oocyst becomes infective, which completes the life cycle.

Special attention is called to the fact that coccidiosis is a self-limiting disease. In other words, when the life cycle is complete, the cause of the disease is automatically eliminated and thereafter the chicken enjoys immunity or protection from reinfection with this particular species of coccidia in proportion to the intensity of its infection.

In most cases the first tangible evidence of an outbreak of cecal coccidiosis is the passage of blood. At this time much damage has already been done and the life cycle is more than half completed, deep in the epithelial cells of the ceca. As a rule most of the mortality occurs during the next 2 days, after which a rapid recovery usually takes place unless there is interference from other factors. Without question, you will recognize that even pink lemonade, if given at this time, will appear to have been responsible for the recovery of a chicken suffering from cecal coccidiosis.

To evaluate a coccidiosis remedy, experiments, properly controlled, must be performed which will give the remedy every opportunity to show merit, if any exists, as well as the presence or absence of toxic action. These facts must be shown conclusively without taking advantage of the life history or any other natural condition. Controlled laboratory tests can supply these facts.

There may be other ways in which these fundamental facts can be obtained but based on several years of experience it has been found that the 4-pen test supplies those essential facts needed for the evaluation of a remedy which has been recommended for the treatment of coccidiosis. The chief criteria used are: (1) Pas-
sage of blood, (2) variations of body weight, (3) variation of food consumption, (4) variation of water consumption, and (5) mortality. Some of the necessary conditions for a test of this type are: (1) Uninfected healthy chicks of the same age, breed, strain, etc., (2) the same general care and feed for all chicks with a continuation of this unless changed by the directions on the labeling as part of the treatment, (3) same culture of coccidia and the same size dose of coccidia to be used for all infected chicks. Under these conditions the 4-pen test is arranged as follows:

Pen I. Chicks to receive a standardized dose of coccidia and no treatment.
Pen II. Chicks to receive a standardized dose of coccidia and treatment according to the directions on the labeling.
Pen III. Chicks to receive no coccidia but to receive treatment only in the same manner as Pen II.
Pen IV. Chicks to receive no coccidia and no treatment. This pen to be normal in all respects.

Many coccidiosis remedies have been tested according to the 4-pen plan, but the time allotted for the presentation of this subject will permit only a discussion of one of these remedies. To illustrate, a remedy belonging to the acid-type and recommended for both treatment and prevention, will be called remedy-A. That part of the test covering its value as a treatment, appears in graphic form on page 181. Chemical analysis of remedy-A shows it to consist of: sulphuric acid 47%, hydrochloric acid 3%, tannic acid 2.3%, formalin 1.1%, glucose 33.2% and the remainder was water. This medication was given in various amounts according to the age of the bird and the intensity of the coccidial infection. For 100 chicks, 4 weeks of age, 3 ounces of remedy-A was diluted with sufficient water to moisten slightly that quantity of ground corn which they would eat in 30 minutes, this to be given on 4 successive days with epsom salts in the drinking water for 3 hours on the fifth day. Treated chicks were to be fasted 3 hours before and 3 hours after eating the medicated grain. Attention is called to the undesirable feature in these directions which required that young growing chicks, attempting to recover from the ravages of coccidiosis, be fasted for 6 hours during the day.

The experiment on the treatment phase of remedy-A was a carefully controlled laboratory test consisting of 4 pens of 25 uninfected Rhode Island Red chicks, 4 weeks of age. These chicks were kept on wire floors and all conditions of management were the same for all 4 pens. Infection and treatment were the variable factors in this experiment. The length of this experiment was approximately 1 month. Referring to page 181, in Pen 21 there were 25 chicks, each of which received 10,000 sporulated oocysts on each of 5 successive days as indicated by the 5 arrows on April 1 to 5 inclusive. The multiple inoculations of oocysts were used to copy, as closely as possible, field conditions where the chicks would be exposed daily to infection until blood appeared in the droppings. The procedure of discontinuing inoculations on the sixth day would simulate very effective sanitary measures which are represented by the labeling in connection with remedy-A. The upper curve represents the average daily water consumption, the middle curve, the average daily weight and the lower curve, the average daily food intake of the birds in this pen. The effect of this coccidial infection is apparent in the
weight curve from April 6 to 9 inclusive, as well as the mortality during the same period. Pen 23 was similar in all respects, i.e., number and age of chicks, size and kind of coccidial inoculation, freedom from extraneous infections and all other features except one. That one exception was treatment with remedy-A according to the directions on the labeling. By comparing Pen 21 and Pen 23 the effect of remedy-A on coccidia infected chicks can be observed. In Pen 23 there was a definite decrease in water and food consumption with a greater interruption in
the weight curve than occurred in Pen 21. Both Pen 21 and Pen 23 had received the same size dose of coccidia. Pen 24, at the start of this experiment contained 25 chicks of the same breed, age, etc. as Pens 21 and 23. Pen 24 acted as the normal control pen and received neither coccidia nor treatment. Pen 22, also at the start, contained 25 chicks of the same breed, age, etc. etc. as the other 3 pens but received remedy-A only (no coccidia) according to the directions on the labeling. As will be noted in Pen 22 there was a definite disturbance in the water and food consumption as well as in the weight curve during the period of treatment. Pen 22 compared with Pen 24, shows the effect of the treatment alone on normal uninfected chicks. This observation, when examined with the findings from Pens 21 and 24, explains the intensified adverse effect on water and food consumption as well as the greater reduction in weight during the period of illness for Pen 23.

Attention is called to the fact that the coccidial infection in Pens 21 and 23 was light. The purpose of this procedure was to give remedy-A every possible chance to show real benefit if any existed. With a light single-species infection, genuine beneficial results could be more quickly detected than if the birds were suffering from heavy coccidial infections. One of the great values of the controlled laboratory test is that all extraneous factors are eliminated as much as possible and the remedy under test has only one job to perform and that job is to fulfil the label claims. However, it must be remembered that once a remedy shows itself to be of value under controlled laboratory conditions, it must still prove its worth under field conditions where there exist numerous factors many of which are unknown. If no value can be demonstrated under the controlled laboratory test, there is no reason to believe that the remedy in question would be of any value under any other condition.

The labeling in this instance specified that additional remedy-A should be given in the following quantities should the intensity of the coccidial infection justify it: (1) The medicated grain should be given twice a day from 1 to 3 days during the regular 4-day treatment, (2) remedy-A to be added to the drinking water at the rate of 1 ounce per gallon and placed before the chicks during the forenoon, (3) if necessary, this medicated water to be placed before the chicks for the entire day, (4) if conditions justify it, continue the treatments.

For the prevention of coccidiosis, remedy-A was to be given in grain 2 days in every 10 days to chicks 7 weeks of age or older. No explanation has been found in the directions as to why remedy-A is of value for 2 week old chicks as a treatment and is not effective as a preventive until the chicks are 7 weeks of age. In this connection time will not permit a discussion of immunity and the possibilities of its development and presence in a 7 week old chick. Remedy-A was not effective, in the slightest degree, for the prevention of coccidiosis in chicks of any age.

In the testing of remedy-A almost one thousand chickens were used. In the laboratory, the experiment reported here was repeated and, other experiments were performed by use of various quantities of remedy-A as directed by the labeling. In addition to this experimental work several hundred chickens, in different sized groups, were placed in outside poultry yards where the coccidial infection was acquired from naturally contaminated ground and consisted of 4 species of coccidia instead of 1. Various quantities of remedy-A were given to the poultry yard experi-
ments, also. All experiments, both those performed in the laboratory and those performed in the poultry yard, confirmed the results obtained in Pens 21, 22, 23, and 24. Conclusions to be drawn from this test are:

1. Remedy-A is worthless for both the prevention and treatment of coccidiosis in chickens.

2. Remedy-A treatment was not only worthless but caused a marked disturbance in those chickens receiving it.
SAVE A HEN AND FEED A SOLDIER

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Because of a shortage of labor, equipment, transportation, and certain essential feed ingredients, the poultry war goals may not be met, unless adequate measures are instituted promptly.

To support the war effort, the poultry industry must produce more—but with less. It must market more poultry meat and eggs with less labor, inadequate transportation, less equipment and less feed. For the duration of the war, at least, and thereafter until critical food shortages in the invaded countries have been met, we must maintain the health and productiveness of our flocks. A higher percentage of the chicks and poults must be hatched than during the past few years. For the good of the industry and the successful prosecution of the poultry war effort, the poultryman, the hatcheryman, the feed manufacturer, the processor, the veterinarian, the educator, the research worker and the extension specialist should be able to agree on the fundamentals of good poultry husbandry.

It was this realization which prompted the Bureau of Animal Industry to establish a poultry viability program. At the call of Dr. John R. Mohler, then Chief of the Bureau of Animal Industry, 32 representative leaders of the poultry industry met in Chicago last March and formed the National Poultry Advisory Council. The aim of this organization was to establish a fundamental program of management, nutrition and disease control for the poultry industry which would aid in reaching the astronomical poultry, meat, and egg goals for the war effort. The country was divided into seven districts, each headed by a director from the industry. These seven directors constitute the Executive Committee of the Council.

Meeting with representatives of state colleges, experiment stations, and federal agencies, it was quickly agreed that this emergency program should avoid all controversial subject matter and concentrate on well recognized practices so that all branches of the industry would tell the same story at the same time to the same people—the 54 million farm flock owners who should be reached with such a program. This association was represented by its President, Dr. Hendricks, and its Secretary, Dr. Welsh. Later Dr. Stafseth, Chairman of the Committee on Transmissible Diseases of Poultry was added.

Three programs were prepared, namely Chicken Brooding and Rearing, Laying Houses, and Turkey Rearing. Breeding supplements later were added to both the chicken and turkey programs. The industry council sponsored the preparation, printing and distribution of booklet material, and to date more than 2,000,000 copies have been distributed to the industry and to educational agencies on a cost basis.

An official emblem, symbolical of participation in this program, has been created and is now used by more than 50 organizations, publications and firms in the poultry and veterinary fields; 20,000 posters have been prepared; and an educational fund...
of nearly $3,000 has been established to provide poultry conservation material for 10,000 4-H Club leaders, 8,600 teachers in vocational agriculture, county and home demonstration agents, and veterinary students.

State poultry conservation organizations have been set up in approximately 35 states. Producer groups, hatchery organizations, veterinary organizations, state poultry improvement associations and other farm organizations have exhibited splendid leadership and demonstrated excellent cooperation in furthering this important work. It has been stated frequently that the poultry industry has rallied to its food production goals to an extent not equalled by other livestock production groups. The work of the National Poultry Advisory Council, in cooperation with the Bureau of Animal Industry, has contributed its share in achieving these goals.

It may be pointed out that, while the work of the Council was created as an emergency war measure, poultry conservation is as profitable in peacetime as it is necessary in wartime. Capable management may be expressed as good judgment plus prompt action, and when combined with good breeding, adequate nutrition and proper disease control, flock owners will be able to increase their production significantly, and with even less layers.

Our annual laying house mortality is equivalent to 50 pounds of meat and 52 dozens of eggs for each man in an army of 10,000,000, and one-half of this loss could be prevented if all flock owners adopted the practical recommendations contained in the Council's booklets. The Council's slogan, "Save A Hen And Feed A Soldier" well typifies the poultry industry's challenge during these critical times, and all veterinary agencies are concerned with meeting successfully this patriotic challenge.
REPORT OF COMMITTEE ON TRANSMISSIBLE DISEASES OF POULTRY


The poultry industry has been requested to produce more—but with less: less labor, less equipment, less feed, inadequate transportation, and, perhaps, less stock. This extraordinary request has been met remarkably well.

Under the present difficult conditions one should expect that disease would become seriously prevalent. However, the disease situation has not assumed alarming proportions. To be sure, a few diseases have increased in prevalence in some sections of the country, if not generally.

BACTERIAL DISEASES

Fowl cholera and fowl typhoid were encountered in Kansas after a lapse of 10 years. Dr. Robert Graham, in a letter dated Nov. 23, 1943, reports several outbreaks of fowl cholera in Illinois. These two diseases have been absent from other localities for many years. Their reappearance in Kansas and Illinois suggests that the same thing may happen elsewhere.

Pullorum disease is becoming increasingly prevalent in turkeys. Efforts should be made to eradicate this disease before it becomes too widely disseminated in this species of birds. It should be remembered that as long as there is a single infected bird in a breeding flock one must be prepared for an outbreak of pullorum disease in poults. The low tolerance provision may be a halting step in the right direction, but any amount of pullorum infection in a flock is dangerous. Hatching from flocks which prove to be clean on the first test is, by far, the best preventive measure. Repeated testing with the tube agglutination test, supported with prompt removal of reactors and a strict program of sanitation, covering ranges, yards, houses and incubators, may also be effective.

There has been a moderate increase in pullorum disease in chickens, perhaps due to less care in selection of hatching eggs, which, in turn, is due to the extraordinary demand for chicks. The much too general lack of understanding of the real meaning of the term "pullorum clean" may also be partly responsible. Many so-called clean flocks are evidently far from clean. In New Jersey evidence has been found suggesting spread by itinerant sexers.

In Utah a law has been passed prohibiting the importation of hatching eggs, baby chicks or turkey poults unless they come from U. S. Pullorum Clean breeding stock.

As reported by the Kentucky Agricultural Experiment Station two new species of Salmonella have been isolated from turkeys, Salmonella madelia and Salmonella amherstiana. Salmonella newington infection in turkeys is reported to be important in California by Hinshaw and McNeil (1).

Avian tuberculosis is still prevalent in certain midwestern states and is responsible for a great deal of retention and condemnation of pork. The annual cost of this waste of pork has been estimated to amount to $2,500,000. It is hoped that the
new mycobacterial rapid agglutination antigen developed by Moses and Feldman (2) will aid in the eradication of this disease.

Swine erysipelas in turkeys was reported in the State of Washington.

PROTOZOAN DISEASES

Coccidiosis has been rather serious this year, perhaps due to the unusually wet weather. A new species, Eimeria brunetti, has been found in chickens in New York. Sulfaguanidine, if used in time, has given very satisfactory results in combating this disease, but is difficult to obtain.

Hexamitiasis has been found in turkeys in Connecticut, New Jersey and Kansas. Traffic in live turkeys, especially breeding stock, is the main avenue of spread.

Leucocytozoon smithi infection has been found in Michigan, California and Texas.

VIRUS DISEASES

Fowl pox has been unusually prevalent in many localities this year. In turkeys it has been very serious in southern Michigan, northern Ohio and elsewhere, affecting particularly breeding males with consequent lowered fertility of eggs. These losses need not occur in view of the possibility of successful vaccination of both chickens and turkeys.

Infectious bronchitis has been prevalent among baby chicks in some regions and has been found to affect laying hens, causing some loss in production but only a low rate of mortality.

The leucosis complex is still the number one disease in some localities and is increasing in prevalence in turkeys. The U. S. Regional Poultry Research Laboratory, East Lansing, Michigan, reports progress in breeding for resistance to this disease. Furthermore, stock free from lymphomatosis has been raised to at least 300 days of age.

The ornithosis virus has been isolated from chickens and pigeons in the United States, and neutralizing antibodies for this virus are reported to occur rather frequently in the blood of these species.

Pneumoencephalitis is reported to be of considerable concern to poultrymen in California and Utah.

DISEASES OF UNDETERMINED OR COMPLICATED ETIOLOGY

The roup syndrome is recorded as the number one disease of chickens in Los Angeles County, California, and is considered to be very serious in Utah. Progress is being made in unravelling the etiology of coryza, which may be in the same class as pneumonia with respect to multiplicity of causes. The latest addition to the agents, which have been found to play a role in the production of this syndrome, is a virus reported by Delaplane and Stuart (3). Sinusitis in turkeys has been an important problem in several sections of the country during the past year.

Pullet disease has been the most important problem in Connecticut during July and August, 70 per cent of the adult chickens, submitted to the Storrs Agricultural Experiment Station for diagnosis during this period, having been affected. Losses due to this disease in Connecticut during the summer of 1943 have been estimated
at a quarter of a million dollars. This disease is also very prevalent in Michigan
and now appears to be becoming a serious problem to turkey growers.

THE NATIONAL ADVISORY COUNCIL

In order to assist the poultry industry to produce more—but with less, the
National Poultry Advisory Council was organized in Chicago, Illinois, March
16, 1943, with Doctor Cliff D. Carpenter as executive secretary. A part of the tangible
evidence of the activity of this council is the publication and distribution of over
two million copies of three effectively illustrated booklets dealing respectively with:
1. Chicken Brooding and Rearing Program, 2. Laying House Program, and 3. Tur-
key Rearing Program.

DISINFECTANTS AND REMEDIES

Some of the disinfectants used by poultrymen are practically worthless. The
blame for this situation rests partly with manufacturers, who do not make adequate
tests to determine the microbicidal power of their products and partly with research,
educational and regulatory agencies, which have been rather indifferent with
respect to attention to scientific facts when it comes to recommending disinfectants.
For example: hot lye solution is claimed to kill coccidia and worm eggs when applied
to floors of poultry houses. It is doubtful whether lye solution, as commonly used,
destroys coccidia and worm eggs; it may serve well as a cleansing agent, thus aiding
in their mechanical removal. Furthermore, no hot solution can be poured on a
cold surface without quickly cooling below an effective germicidal and parasitocidal
temperature.

Worthless remedies, sold and distributed by people who are willing to capitalize
on the misfortunes of their fellowmen, still constitute the greatest drain on the
income of our poultrymen. Much education is needed to combat this great evil.

RECOMMENDATIONS

1. Turkey growers should be urged to secure new stock from flocks that are
found to be free from pullorum disease on the first test.
2. The continuance of the “tolerance” provision in the pullorum disease eradica-
tion program is to be discouraged.
3. We urge that the fight against pullorum disease in general be intensified,
taking into consideration all possible means of spread, such as sexing and the
Eames-way method of culling, in which tumors, cysts and other abnormalities
in the abdominal cavity are detected by palpation after inserting a finger full
length into the rectum.
4. We also recommend that a greater interest be taken in the eradication of
avian tuberculosis.
5. All traffic in live birds, with the exception of baby chicks and poults, is one
of the main factors in the spread of many diseases such as the protozoan and
parasitic diseases, as well as others. This practice should be discouraged.
6. We recommend greater attention to the prevention of fowl pox in chickens
and turkeys by proper vaccination.
7. In view of the fact that chemotherapy has now ascended to a state of respecta-
bility it is hoped that efforts will be made to find effective chemotherapeutic agents for diseases of poultry.

8. We urge increased efforts toward the education of poultrymen as to the relative value of sanitation, in all its ramifications, and medication in the control and eradication of disease, giving due attention to the proper use of biological products, approved disinfectants and therapeutic agents of proven value.

9. We wish to go on record favoring better support of our present State educational, research and regulatory agencies in their efforts to combat poultry diseases.

REFERENCES


THE PRESENT STATUS OF CANINE RABIES VACCINATION

BY HARALD N. JOHNSON

The purpose of this paper is to present data as to the effectiveness of vaccination for the prevention of rabies under field and experimental conditions. There is still much work to be done but the information at hand indicates that vaccination may be the most practical method of rabies control in view of the difficulty in obtaining and enforcing strict quarantine regulations. The basic requirements necessary to the operation of an efficient rabies control program are discussed.

FIELD REPORTS OF CANINE RABIES VACCINATION

The single injection method of vaccination with chemically inactivated tissue virus suspensions has had extensive use in the United States, but the lack of pertinent information as to the total number of dogs, the number vaccinated, and the number of vaccinated and unvaccinated dogs dying of rabies makes it difficult to draw any statistical conclusions regarding the effect of the procedure.

In 1937 the Alabama State Legislature passed a Dog Control Act for the suppression of rabies. This law requires that all dog owners must have their dogs vaccinated annually with a rabies vaccine approved by the State Health Officer and the State Veterinarian, except for animals kept constantly confined. The law also provides for the impounding of unvaccinated dogs running at large and confinement of dogs suspected of having rabies. Enforcement of the law is relegated to rabies inspectors who are to be appointed annually by each county board of health and they are vested with full police power.

During the period 1932 through 1936 from 836 to 1,017 animal heads were found positive for rabies each year in Alabama. The compulsory vaccination program was begun late in 1937. A total of 177,038 dogs were vaccinated. During that year 927 animal heads were found positive for rabies and 3,794 human rabies treatments were distributed. There was a rapid reduction in the state wide prevalence of rabies during 1938 and 1939. In 1939 only 237 animal heads were found positive for rabies and the number of human rabies treatments distributed dropped to 1,230. Whereas the disease had previously been widespread over the state, it soon disappeared from most of the counties. Some counties, however, failed to appoint rabies inspectors to carry out the control work.

In 1942 only three counties of the sixty-seven in the state had more than isolated cases of rabies and forty-five counties reported no rabies. The three counties mentioned above accounted for 181 of the 220 cases of animal rabies reported during the year. None of these three counties appointed a rabies inspector to carry out the vaccination program. Eight counties reported only one case of rabies and the

1 The studies and observations herein reported were conducted with the support and under the auspices of the International Health Division of the Rockefeller Foundation and the Alabama State Board of Health.

To be presented at the 47th annual meeting of the U. S. Live Stock Sanitary Association, December 2, 1943.
other eleven counties had from two to four reported cases of animal rabies. During the period 1937 to 1943 Mobile County had an average of 108 reported cases of animal rabies a year. In this instance there was active opposition to vaccination and only a small proportion of the dogs were vaccinated on a voluntary basis. During 1942 this county submitted 157 of the 220 animal heads found positive for rabies in the entire state.

As the result of an outbreak of fox rabies in this county early in 1943 many dogs were vaccinated on a voluntary basis and with the aid of educational work through civic and sportsman’s organizations it now appears likely that the vaccination law will be enforced. In 1943 there has been a further decrease in the number of cases of dog rabies in Alabama. Only seventy-three positive specimens have been reported to date. Of these thirty-seven were from Mobile County. There were only sporadic cases of rabies in other counties and these occurred predominately along the Georgia and Tennessee borders.

The State Health Department is located in Montgomery County and here there has been excellent cooperation with the vaccination program. Table I shows the dog control activities for the years 1936 to 1943. All unvaccinated dogs found at large are impounded. No quarantine was enforced at any time and vaccinated dogs are free to roam the streets. In April 1940 a dog developed furious rabies in the city of Montgomery. This dog had been brought to this city from Florida. Two persons were bitten and several dogs were exposed. No secondary cases of rabies developed despite the fact that the salivary glands of this dog were found to contain a high concentration of rabies virus. In 1943 a cat was found positive for rabies in the city of Montgomery. This cat belonged to a family that had moved to Montgomery from Florida. They brought a young chow dog with them when they moved and this dog died of an undiagnosed illness shortly after arrival. The cat, which was obtained in this city, developed furious rabies a few weeks later. The cat attacked several dogs and persons before it died. Examination of the brain was positive for rabies. No secondary cases of rabies developed among exposed dogs though none were killed. Except for these two cases of rabies, the county has been free of the disease for five years while in previous years the disease was constantly present.

### Table I.—Rabies control activities, 1936-1943, Montgomery County, Alabama

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<thead>
<tr>
<th>YEAR</th>
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<th>DOGS IMPOUNDED</th>
<th>DOGS KILLED</th>
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<tr>
<td>1943*</td>
<td>5,873</td>
<td></td>
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<td>1 (cat)</td>
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</table>

* To November 1, 1943.
Another example of an efficient vaccination program is the experience of Burke County Georgia during the fox rabies epizootic of 1940-1941. Rabies was introduced among the foxes in that county by a fox hound. Two foxes were bitten by this hound and escaped. The next day the fox hound developed typical symptoms of furious rabies. Soon thereafter rabid foxes began appearing in farm yards in day time, attacking people and domestic animals. Positive proof of the existence of rabies in foxes was soon obtained. Many domestic animals and dogs developed rabies during the first few weeks of the epizootic. The Federal Wild Life Service began a program of reducing the number of foxes by hunting and trapping. Local authorities passed a regulation that all dogs were to be vaccinated. A total of 2,900 dogs were vaccinated within three weeks after the ordinance was passed. Four dogs developed rabies within thirty days after vaccination, evidently due to exposure before vaccination. Thereafter there were no further cases of rabies among the vaccinated dogs although a considerable number of rabid foxes were killed subsequently by dogs and found positive for rabies virus in both the brain and salivary glands. In a recent communication Doctor Hirleman, who so ably carried out the vaccination program, states that to the present time no vaccinated dogs have developed rabies. One young unvaccinated dog developed rabies in Burke County during 1942. This dog was evidently exposed in an adjacent county. Fox rabies continued for several months after the dog vaccination program was completed. A total of 159 foxes were found positive for rabies by laboratory examination. There are a number of cities that have enforced licensing and vaccination of dogs. Where properly administered this type of rabies control program has given very satisfactory results.

EXPERIMENTAL STUDIES OF CANINE RABIES VACCINATION

The first study at this laboratory of the single injection method of canine rabies vaccination was made with a commercial phenolized vaccine of ovine origin containing 20 per cent brain tissue. Each of the vaccinated dogs received 5 ml. of vaccine subcutaneously and the test virus inoculation was given one month later. A 0.5 ml. dose of a 1:10 suspension of street virus dog brain was injected into each masseter muscle. Of 105 vaccinated dogs twenty-six, or 25 per cent, died of rabies when tested as noted above. The mortality for the control dogs tested in parallel was thirty-four of fifty-five animals, or 62 per cent. The level of immunity to experimental infection was thus raised from 38 per cent to 96 per cent.

The experience of Webster (3) and ourselves that a commercial chloroformized vaccine was more effective than most of the phenolized vaccines for immunizing mice stimulated our interest in testing this product in dogs. Of fifty dogs given a subcutaneous injection of 5 ml. of this product only two, or 4 per cent, died of rabies when tested as noted above. The mortality for the control dogs tested in parallel was thirty-four of fifty-five animals, or 62 per cent. The level of immunity to experimental infection was thus raised from 38 per cent to 96 per cent.

We then tested the effect of prolonged storage on the antigenicity of the chloroformized vaccine. A single lot number of commercial vaccine was tested after 4.5, 10.5, and 16.5 months storage in an ordinary refrigerator. At least thirty vaccinated and thirty control dogs were used in each test. In all three tests the vaccinated dogs developed a high degree of resistance to experimental infection.
Discounting the test of the expired vaccine 16.5 months after preparation, a total of 120 dogs have been vaccinated with this product and tested for immunity. Only eight dogs died of rabies, 7 per cent, compared to eighty-one rabies deaths among the 132 control dogs, or 61 per cent. In other words, 93 per cent of the vaccinated dogs were resistant to infection.

The chloroformized vaccine contains 33\% per cent brain material and to determine whether the superior results obtained with the chloroformized vaccine was due to this added concentration of brain material we tested a phenolized vaccine containing 33\% per cent brain material. Of thirty-one dogs vaccinated with a single 5 ml. dose of this product and tested as above, only three, or 10 per cent, died of rabies compared to twenty rabies deaths among the thirty-six control dogs, or 56 per cent. It therefore appeared that the phenol vaccine was essentially as good as the chloroformized product.

**MOUSE POTENCY TEST**

Our studies have shown that it is extremely difficult to immunize dogs or mice to intracerebral inoculation, even with live virus vaccines. Large groups of dogs that have survived intramuscular inoculation with street virus have been tested subsequently by intracerebral inoculation. Even where we have used a 1:1000 dilution of street virus salivary gland, only an occasional dog survived the intracerebral test. When tested by intracerebral inoculation dogs that have been given fourteen injections of live virus vaccine develop rabies with about the same frequency as the dogs surviving intramuscular inoculation.

We have repeatedly tested commercial and experimental rabies vaccines in mice, using both the intracerebral and the intramuscular method of test inoculation. Large groups of mice have been used in each test. None of the vaccines have protected more than 50 per cent of the mice when tested with 100 intracerebral MLD standard rabbit fixed rabies virus. In contrast, we frequently get 90 to 100 per cent protection to intramuscular inoculation where less than 20 per cent of the control mice survive.

**DISCUSSION**

It is easy to see why criticism has been directed at canine rabies vaccination. Several reputable investigators have reported that rabies vaccine failed to protect animals from developing the disease under experimental conditions. There are several reasons for their negative results. In order to secure a uniform fatality for small control groups they have usually resorted to cisternal, intracerebral or intraocular test inoculation. Furthermore, when the test inoculation was given intramuscularly, young animals were used so as to obtain a high mortality for the controls. We have found that most of our failures for vaccination occurred in four to six month old dogs. Similarly, mice over six weeks of age are more readily immunized than those under four weeks of age. This emphasizes the importance of using fairly mature animals in vaccine potency test studies. In order to obtain significant results it is necessary to use large groups of animals.

It is reasonable to expect more consistent protection for vaccinated dogs exposed to rabies under natural conditions than where large doses of street virus are injected.
into muscle tissue. It is difficult to infect dogs with rabies by puncture and abrasion of the skin with instruments dipped in a 10 per cent suspension of salivary gland virus of high titer. The mortality for a group of 29 dogs exposed by this method was only 15 per cent. Where the exposure involves muscle tissue the mortality is much higher.

In the absence of asymptomatic carriers, it is not necessary to obtain 100 per cent herd resistance in order to eradicate a disease. This is especially true for rabies where infection depends on the virus entering a wound. Not all rabid dogs have the virus in the salivary glands, others develop paralytic rabies and do not bite. A considerable number of mature dogs also have a natural resistance to infection. Therefore, if the general herd resistance is materially increased by vaccination, the chain of infection will soon be broken.

The commercial phenol inactivated vaccines containing 20 per cent tissue virus now on the market are undoubtedly superior to the one we first tested. New methods of production have been adopted resulting in higher and more consistent antigenicity. As might be expected, the antigenicity depends on the amount of virus in the brain tissue used for vaccine production.

We have recently tested vaccines inactivated with phenol, chloroform, and formalin which were prepared from the same pool of tissue virus. Each contained 20 per cent brain tissue. The phenolized and chloroformized vaccines were approximately equal in antigenic potency when tested in mice and dogs. If anything, the phenolized vaccine was slightly superior. The formalinized vaccine was definitely inferior to the other two vaccines.

Rabies will remain widespread over the United States until control work is coordinated on a national basis under a single agency. The relative freedom of transport of dogs from one community to another is the main reason for the continual development of new foci of rabies. Where rabies is present the public must be informed about the disease and the necessary control procedures through radio programs, newspaper articles and pamphlets giving the salient information about the disease. Rabies in animals should be made a reportable disease so that the State Veterinarian will be notified immediately of new foci of the disease. Control work could then be started before the disease has time to spread.

If canine rabies vaccination is to be effective in the field it must be under the supervision of qualified veterinarians. The prescribed dosage must be given and the vaccine should be massaged into the subcutaneous tissue. We recommend that the dose be divided and injected into two different areas. Rabies vaccine must be kept in a refrigerator when not in use otherwise it will lose its potency. It is evident that vaccination will not eliminate rabies unless a constantly functioning program of picking up unvaccinated dogs found on the street is maintained. There are two alternatives in the financing of this type of program. One is to license all dogs and use this revenue for maintaining a dog pound and the necessary personnel and equipment. The other is to make the vaccination fee high enough to cover both the cost of vaccination and other dog control activities. The latter has been found more practical in Alabama as the dog owner pays only one fee, the vaccination tag differentiates the dog from ownerless strays and the fee is large enough to make it worth while for the veterinarian to assume the other control activities of a rabies inspector.
SUMMARY

Field reports of canine rabies vaccination are presented, together with the results of experimental studies of the single injection method of vaccination. Examples are given of how compulsory vaccination and collection of unvaccinated dogs has brought about suppression of rabies without quarantine.

Rabies cannot be eradicated from the United States until a uniform program under the supervision of a single agency is obtained. The control work will of necessity be done by the veterinary profession. The success of a rabies control program depends on prompt reporting by veterinarians of all suspected cases of animal rabies. Adequate facilities for collecting ownerless dogs must be made available.

BIBLIOGRAPHY


REPORT OF THE COMMITTEE ON RABIES


Under date of September 30, 1942, the Bureau of Animal Industry of the United States Department of Agriculture furnished the National Rabies Committee information on the additional legislation that would be necessary to enable the Bureau to participate in a national rabies program. The latest meeting of the National Rabies Committee was held on April 25, 1942, in New York. From the information now available, it is expected that another meeting will be held shortly. Just what action can be expected from the committee is problematical, as progress since the formation of this committee has been slow. It should be recognized, however, that the problem is a complex one and will require time to work out all the necessary details.

In reviewing statistics collected by the Bureau of Animal Industry since 1938, listed above in Table 1, it will be noted that there has been little change in the incidence of the disease in the United States. It appears that the disease is largely restricted to certain areas, but there is a fluctuation of the incidence within these areas. However, in the aggregate, the number of cases remains about the same. The attached map shows the number of cases of rabies reported by the states for the calendar year 1942. It will be noted that the New England states and the Northwestern states have a low incidence or absence of the disease. In the remaining states, the disease is quite prevalent.

Both the public health and livestock sanitary authorities of each state have been furnished with reports of this committee annually since 1938. In these reports, methods are described which should bring the disease under control, if properly carried out. In so far as the committee is aware, not one state has put into effect all the recommendations made. Until these measures are put into effect properly, there can be little hope that the incidence of the disease will be appreciably reduced in the United States.

<table>
<thead>
<tr>
<th>YEAR</th>
<th>DOGS</th>
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<th>SHEEP</th>
<th>SWINE</th>
<th>CATS</th>
<th>GOATS</th>
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<td>12</td>
<td>160</td>
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In so far as the committee is aware, not one state has put into effect all the recommendations made. Until these measures are put into effect properly, there can be little hope that the incidence of the disease will be appreciably reduced in the United States.
Number of cases of rabies reported in the United States in 1942. Total 7,165
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Not listed as to species; probably mostly dogs
Your committee strongly urges that authorities in states where the disease is prevalent take the necessary steps to insure that the recommendations of this committee for the control of rabies are carried out in their states. If each state were well organized to take care of any rabies outbreak within its borders, it is believed that the disease could be readily brought under control. At least great progress could be made without waiting for the machinery to be set up to control the disease on a national basis, which may be some years in the offing. Concerted action by groups of states in an area where rabies exists would be an effective way of controlling the disease on an area basis, and it is urged that the state authorities give this serious consideration and take necessary action.

Recommendation:

It is recommended that the committee on rabies be continued and that it be empowered to coordinate its activities with the National Rabies Committee, looking to ways and means of controlling rabies on a national basis.

Your committee again has been furnished material on the incidence of rabies in the various states in 1942 through the courtesy of Dr. A. W. Miller, chief of the Bureau of Animal Industry, U.S. Department of Agriculture. According to the reports received for the calendar year 1942, there were 6,332 cases in dogs, 288 in cattle, 15 in horses, 48 in sheep, 32 in swine, 250 in cats, 12 in goats, 160 miscellaneous and 28 in man, making a grand total of 7,165 cases.
A REPORT OF THE SPECIAL COMMITTEE
ON RULES AND REGULATIONS

WILLIAM MOORE, Chairman, Raleigh, N. C.; R. L. CUFF, Kansas City, Mo.;
C. H. CLARK, Lansing, Mich.; A. J. De FOSSET, Columbus, Ohio; DON E. KENNEY,
Salt Lake City, Utah; A. L. FELKER, Concord, N. H.

In making up the report of the Committee this year, your Chairman has gone
over the Committee reports of this or a similar committee for the past few years.
In checking over these reports, I find that this Committee has made a number of
suggestions and recommendations, especially in connection with more uniform
regulations by the several states; yet it would seem that very little has been
accomplished along this line.

On conferring with other members of the Committee, one suggestion perhaps
worthy of note has been made as follows: That the federal government promulgate
a regulation governing the shipping of cattle interstate to provide for better control
of Bang's disease, and to offer protection to those areas that have expended a lot
of time, money and effort in eliminating Bang's disease from their cattle population.

It would seem that it is not necessary nor advisable at this time to make further
recommendations, but I would refer the members of this Association to the report
of this Committee for the past several years.
PANEL DISCUSSION ON DISEASES OF CATTLE

If Dr. Boyd is here, we will arrange our panel discussion on diseases of cattle. Dr. W. L. Boyd, of St. Paul, Minnesota, is Chairman of this part of the program. The men whom he is going to ask to assist in this discussion are the following:

Dr. C. H. Case, Akron, Ohio
Dr. R. R. Dykstra, Manhattan, Kansas
Dr. W. R. Krill, Ambulatory Clinic, Ohio State University, Columbus, Ohio
Dr. R. B. Little, Rockefeller Institute, Princeton, New Jersey
Dr. Herbert Lothe, Waukesha, Wisconsin
Dr. C. K. Mingle, U. S. Animal Disease Station, Beltsville, Maryland
Dr. Paul H. Phillips, Madison, Wisconsin

Dr. W. L. Boyd assumed the Chair.

THE CHAIRMAN: Will the gentlemen whose names have just been read please come to the platform.

THE CHAIRMAN: As in past years, we shall attempt to make this meeting a most informal one. We believe we have a very interesting program. We are attempting to present to you problems that are very acute and important today.

I know you all realize that a number of men cannot cover all of the various topics that are of interest to each and every person in this room. We have gathered here at the table men who have had many years of experience both in the field of teaching, in the field of practice, and in the field of research. I think these men will do a very good job of answering any and all questions you may put to them.

We have a number of questions that we have prepared, and to start this off it falls to the lot of Dr. Paul Phillips, Professor of Biochemistry in the University of Wisconsin College of Agriculture.

There is a tremendous amount of interest in nutritional diseases at this time, both in human and in domestic animals. We are indebted to a great many men working in his field, for the information they are bringing to us, and the help they are giving us in solving many of our problems. Dr. Phillips has made some very excellent contributions in nutrition. We all know what he did in connection with making artificial insemination more effective. Later his work with vitamins, particularly in connection with the feeding of calves, also the adult animal, is very well known.

I am going to ask that Dr. Phillips take approximately ten minutes to give you a background of the vitamins in the newborn calf, of vitamin requirements to maintain normal health in the calf, and vitamins as related to certain of the diseases, and vitamins also as they may be related to the use of sulfonamide therapy.

Dr. Phillips, I am going to turn this microphone over to you.

DR. PAUL H. PHILLIPS (Madison, Wisconsin): I would like to spend just a little time, if I may, at the beginning of this discussion, to picture for you the normal blood picture of a newborn calf.

When the newborn calf arrives, if you were to take a sample of blood from that calf and make an analysis for vitamin A, you would find on the average that it contains about 4 or 5 micrograms of vitamin A per 100 cc. of blood. If you were
to analyze the blood of that calf again at the end of 24 hours or 48 hours, you would find that it had climbed about threefold, that it would be at least 12 and more likely 16 micrograms of vitamin A per 100 cc. of blood.

If you examined the blood stream of an A-deficient animal, you would find that the blood contains on the average about 15 or more micrograms of vitamin A per 100 cc. of blood.

We have worked out, as carefully as we can with the technics at hand at the present time, the requirements of the calf, and where the values of vitamin A have to be if he is to be a healthy one, the dividing line comes between 8 and 10 micrograms per 100 cc. of blood.

If vitamin A values drop below 8 and remain there for any appreciable time, you can be sure that you will have an A-deficient animal, that will exhibit a variety of symptoms that you have all seen in your daily practice or as you have gone about the countryside and just casually looked at herds.

I might just run over those symptoms for you for a moment, because there is a train of symptoms that are associated with this disease which would tell you at once what might be done in the way of therapy.

The first thing you notice is that the calf may have a slight cold, a nasal discharge, excessive lacrimation. He may have intermittent scours, soft feces. These symptoms increase in severity until toward the end you have a well defined case of pneumonia. That is about the sequence of symptoms that attend vitamin A deficiency in either grown cattle or calves.

If you take this newborn calf with a vitamin A content in his blood ranging around 4 or 5 micrograms, and allow it to go ahead without any vitamin A, you will find that it will quickly develop all of these symptoms, develop pneumonia and die before you can do anything for it. Death may ensue on the second day, it may happen on the tenth day after birth.

One thing that is absolutely essential is to get some vitamin A into that calf and do it as quickly as possible.

Mother Nature has provided a way of giving vitamin A in that the colostrum usually is three to five times as rich in vitamin A as normal milk. There are circumstances, I believe, without much evidence to date, that this colostrum milk may vary considerably in its vitamin A content, and therefore the giving of colostrum milk will not always supply the vitamin A that we need.

If we can boost the vitamin A content of the blood up into the normal range or above, you can be sure that your calf is going along in pretty good style regardless of what you do to him. I hope no one will take offense here and assume that by making such a remark I do not believe in sanitation, because I do, and I do know that bacteria causes disease. But I am not going to deal with bacterial diseases because I have a limited time in which to cover my subject.

There are circumstances under which you cannot get vitamin A into the bloodstream, and that is where nicotinic acid or niacin comes into the picture.

In our early work, which we attempted, we knew the B complex would work, and we attempted to determine which one of the complex factors was most essential. It simmered down to nicotinic acid in the final stages of those experiments, and if you gave the same diet with pantothenic acid, for instance, you could not raise
the vitamin A content of the blood. As a result the calves died from scours and pneumonia. You never, or very seldom ever, in my experience at least, got death from scours directly. It is usually associated with pneumonia in the terminal stages.

If you put in nicotinic acid the blood vitamin A comes up. The explanation of that lies in one of the functions of nicotinic acid. That function is to keep the digestive system working properly. In the dog, if you have a nicotinic acid deficiency you not only get black tongue but you get a marked congestion throughout the entire digestive apparatus of the dog, and you may have excessive hemorrhage into the stomach and into the intestinal lumen itself.

So in summing up this work we say that the vitamin A and nicotinic acid content of the newborn calf is extremely important, and that it has a direct relationship to the general health of the calf, and it will also take care of most scours. We have not gone far enough in our experiments to say it will take care of all cases of scours. We have met some conditions in the newborn calf that we have been unable as yet to correct with vitamin A and nicotinic acid.

I have a few remarks that I want to devote to vitamin C. Again, if you go to the newborn calf to determine the normal picture, you will find that blood collected the minute the calf is dropped, will show about 7 milligrams per cent vitamin C. This was the average of some 50 to 100 calves.

Within twelve to twenty-four hours vitamin C has done just the reverse of vitamin A—it has dropped from this value down to .3 milligrams per cent. If you watch the blood stream carefully over three to six weeks, you will find that this vitamin C drops slightly but steadily down to the end of the second week or thereabouts, say the fifteenth to the seventeenth day. It will drop from .7 to begin with, down to around .2.

Vitamin C is one of the vitamins that has a very active part in normal physiology. It has a function in oxidation and reduction in the body. It has a great capacity for reduction. We think it is necessary, in all the physiologically active cells of the body. Certainly it is associated with disease in no uncertain way, and we believe this drop in vitamin C from .7 to .2 during the early weeks of the calf’s life represents largely the change from dietary vitamin C over to a synthetic process within the calf’s body.

There is considerable vitamin C in milk, and the calf can obtain vitamin C from the diet for the first ten days of life. By the end of the twelfth day he no longer can obtain vitamin C from the diet. Apparently the paunch begins to destroy vitamin C, and it has to be injected after the twelfth day. So there is a shift from a dietary source to a synthetic source within the tissues of the calf.

Now, it happens that in cases like naval infection, peritonitis, pneumonia, the vitamin C disappears from the blood very rapidly. We believe it has a function in combating bacterial infections, and for that reason we assume we should put vitamin C into the calf to support these blood levels in the calf diseases. When we did that we found that vitamin C was a very effective agent in recovery from diseases.

In pneumonia, for example: The ascorbic acid or vitamin C practically disappears from the blood stream, and if you inject enough vitamin C into some of these animals
that are already on their way out, you will find there is a marked response. So vitamin C has a place in calf nutrition, and particularly the effects some of these diseases have which might be termed in that class of idiopathic origin.

I want to mention in closing that there is another effect of vitamin C which has to do with sterility. As you know, and probably a lot of you think, as have others, that we have stuck our neck out quite a way on vitamin C and sterility, I just want to relate to you one experiment which is not under way at our station, where we have used chlorobutanol. It has been referred to in the literature as chloretone; that is a trade name of one particular company.

This chlorobutanol, if fed to a sterile bull, will do the same thing as the injection of ascorbic acid. Either one raises the concentration of ascorbic acid in the blood, and for some reason which we have been unable to explain as yet, it stimulates the sexual behavior as well as the sperm, and has some effect on the ovary. If we had plenty of time I could show you the data upon which these statements are made.

In the case of this chlorobutanol we recently had a bull which went into one of our artificial insemination rings.

He had been rented from his owner and he was a vicious animal. It took three men to lead him out of the stall. He almost killed our herdsman on one or two occasions.

We persuaded our dean to buy that bull for us; then we hid him in the barn for three or four weeks until we got some flesh on him. The bull lost 500 pounds in the time he was in the ring; so you can see his nutritional care was very poor. He came to us emaciated and absolutely infertile. We bought the bull because we believed we could bring him back.

We put him on a good ration, and in addition we gave him about 5 grams of chlorobutanol per day, fed on his feed. At the end of about six weeks he had gone from no motility and a watery semen to a good sample of semen which could be stored and which had fertility and which could be used to breed cows.

At the same time one of my men, who knows more about this subject than I do, ran experiments on this semen sample and found that the oxygen uptake of the sperm had increased from zero to 19 millimeters of oxygen per 100,000,000 sperm per hour. That is quite an increase. The normal is about 25. So you can see that the administration of chlorobutanol which raised the ascorbic acid did a job in this bull. It also works in cows.

Thank you. (Applause)

THE CHAIRMAN: Thank you, Dr. Phillips. I know there are men in the room who have some questions. Let's get right along, and have any of those questions. We will keep Dr. Phillips on his feet for a while to answer any questions. Please keep in mind that he has been talking about nutritional problems, not infectious problems. He is talking about nutritional infertility and not pathologic infertility; nutritional diarrhea or scours in calves, and not specific infectious scours, but perhaps relating to that. So let's have some questions.

Dr. A. H. QUINN: Dr. Phillips, could I ask you just for a brief elaboration on the international unitage or the vitamin A unitage to be recommended as a prophylactic in calf scour complex, and what unitage you would recommend daily as collateral treatment in calf scours in baby calves?
DR. PHILLIPS: In answer to that question, I would like to state a recent development which is coming into the field, and I believe now the veterinarians can obtain these capsules. The Gelatin Products Company of Detroit is making capsules directly suited to calf feeding. That capsule, upon our recommendation, will contain 5,000 units of vitamin A daily, 50 milligrams of niacin or nicotinic acid, and in addition there will be 250 milligrams of ascorbic acid.

We realize the ascorbic acid will not be used after the tenth or twelfth day, but it may be highly important during that first interval, and it doesn't cost much to put it in. There will also be 500 units of vitamin D in that capsule. They can put up in this gelatin capsule the vitamin A oil. The vitamin A is very easily destroyed. They can put this material up in this gelatin capsule under any gas or in the absence of any gas, and guarantee the vitamin A potency for at least a two-year period.

I have tried to get specific names of companies that would handle these capsules. They are very convenient to give, and they do the work. We have had some of them on an experimental basis at our station, and in farm hands and in our own hands they have given us excellent results. The only company whose name I have at the present time, who handles such a product, is the Jensen-Salisbury Company of Kansas City.

THE CHAIRMAN: While we are still on nutritional problems again I would like to ask Dr. Phillips to take the microphone and talk to us relative to the contents of wheat germ oil and the possibilities of wheat germ oil when given to certain forms of infertility in cattle.

DR. PHILLIPS: In reply to that question I may state that thanks to Merck & Company we were able to use pure vitamin E or alpha-tocopherol in some experiments we carried out several years ago, and we had a series of young bulls that we tried to develop sterility in. We fed them a low vitamin E diet and were absolutely unable to change the character of semen or to improve the fertility of these young bulls by administration of vitamin E.

We get vitamin E, as you know, from wheat germ oil, but if there is any merit in wheat germ oil it lies in something other than the vitamin E content of the product.

We have also tried it on cows, and I believe that confirms the experience of the Minnesota Station, also the Iowa Station, that vitamin E has no direct relationship to fertility except in the case of the rat. In other species, such as the rabbit and others that could be named, vitamin E is specifically related to what the chemist calls phosphorylation of muscles. We have done some of that work in our own laboratory, where we know that vitamin E is definitely tied up to the transfer of phosphorus in muscles.

In the absence of vitamin E you get a failure primarily in the muscle, and in the rat it hits the reproductive organs first. Therefore you have a disturbance in reproduction in rats, but you do not get it in other species.

THE CHAIRMAN: Dr. Phillips, if one engaged in dairy cattle practice, seeking a source of vitamin A, could he take colostral milk and freeze it and then refrigerate it and keep it as a source of vitamin A? If so, how would he go about doing it?

DR. PHILLIPS: Yes, that is entirely possible. I believe under normal circum-
stances it would be a lot more convenient to use these capsules that I spoke of, because you have a definite quantitative amount of vitamin A in each, and it can be stored under ordinary room conditions without the expense of refrigeration.

I have in my own barn a bottle of these capsules just sitting there, and if a calf begins to show signs of scours, or if the herdsman does the wrong thing at the right time, or the right thing at the wrong time, and we get scours, we simply use these capsules. They are so convenient that I don't think it pays to bother with colostrum milk.

I would rather divide that milk up among the rest of the calves in the herd and resort to a product of known potency. We have not done this experiment, but I expect and I hope we will have data on it, for sure, by this time next year.

I believe colostrum milk varies considerably in its vitamin A content. I think probably in the months of February and March, after the long winter feeding season, that we are very apt to have a different colostrum milk, or if we have a high producing cow we may have a difference in vitamin A content of that colostrum milk as compared to one freshening on pasture or in the early autumn.

The Chairman: As we proceed along with the program it becomes more and more apparent to each and every one of us that diagnosis is a very, very important thing if we are to meet these problems fairly, and be successful in combatting them.

I would like to call on Dr. Fincher, of Cornell University, to elaborate on the necessity of diagnosis. I have not said anything to him, but this is very informal and I hope he won't feel that I am taking any liberties by calling upon him. He has had a rich experience, and I am sure he can make a contribution.

Dr. Fincher: This is no time to worry about a speech you are going to make. (Laughter)

I feel rather unprepared on this particular subject. I think we have a lot of very acute scours, however, in New York State that is not accompanied by pneumonia. At least I don't hear reports from our pathology laboratory that pneumonia is present, and I wonder if we have missed that or if this is a different disease from the vitamin A scours, the vitamin A and B deficiency that Dr. Phillips talked about.

We have used a great deal of concentrated cod liver oil, so-called, as a source of A and D, with good effect. We would not think of trying to keep a good Guernsey herd going without it, and we feed it as a preventive and not as a curative to those calves.

We haven't used any niacin as niacin. We have used Brewers' yeast in the grain ration, and I wonder whether Dr. Phillips would feel that brewers' yeast incorporated in calf pellets, or in some grain ration given to calves as young as 24 or 48 hours of age (as I have seen it done places, where they force them to eat it and force it into their mouths), would answer the purpose.

I think we have used with good effect, also, the sulfa drugs, notably sulfapyridine, sulfathiazole and sulfaguanidine, with a little preference for sulfapyridine; but all have failed sometimes.

I think that is all I would care to contribute, but I would like to ask for an answer to the questions, Dr. Boyd, the two points I raised.

The Chairman: Dr. Phillips?
DR. PHILLIPS: In reply to that question I would say that if you can get yeast in the calf it would do just as well as the B vitamin in pure form. We have tried yeast, but the difficulty with yeast is that sometimes we have difficulty with inexperienced men trying to get it into the calf.

We have used the pure niacin or nicotinic acid in capsule form, which is really easy to administer. The reason why niacin is needed in the calf, I think, is this: Milk is borderline in its nicotinic acid content, this is true on all analyses of colostrum that we have made.

We were puzzled at first to understand why we should need the B complex in view of work from other stations, until we began to get a response from certain of the B complex and then pinning it down to nicotinic acid. From that step we went back to check milk, because we had been led to believe that milk was a source of nearly all of these factors, and we found out that nicotinic acid is quite low in milk, and until the calf can get up to the point where he manufactures his own in the paunch, nicotinic acid may be the limiting factor.

We have fed vitamin A alone without nicotinic acid in our early experiments, and we had partial success. I would say 75 per cent of our cases responded to vitamin A alone, but there was an undetermined percentage that did not respond, say 25 per cent or more, which failed to respond until we gave them nicotinic acid.

Yeast is high in nicotinic acid, and feeding yeast will do the job just as well.

From the point of view of the operator it is a little less convenient to feed.

Going now to the sulfa drugs, our concept of scours at the present time (at least from the standpoint of nutritional scours) is that sulfa drugs will do a good job after the bacteria are fully established. I think the sequence of events coming into this calf scour picture is about like this:

The calf is nutritionally deficient to start with, and in that state bacteria develop, and the normal defenses of the animal are lowered. As a result, bacteria develop, and I think there is no quarrel, at least from our point of view. We do not see any conflict between our researches and the bacterial theory of infection.

I think the lowered nutritional state of these animals permits the bacteria to assume the ascendency, and in those cases you have then in the terminal stages not a nutritional disease exactly, but a nutritional disease complicated by an overwhelming invasion of bacteria; and in those cases the sulfa drugs will do much to relieve the problem.

Again it is like feeding vitamin A. You do not get 100 per cent response because you have not corrected the causative factor, which is primarily nutrition and involves mainly vitamin A, conditioned, as I have pointed out, by nicotinic acid.

We have made one or two observations where we have used sulfa drugs. We can take a Holstein calf, put him on skim milk, and raise him in good style. He will gain a pound to a pound and a half a day on skim milk alone from birth, without any colostrum if he is given vitamin A, nicotinic acid and ascorbic acid.

We have tried that on Guernseys and have failed, but recently we have upped the vitamin C to levels above that present in the newborn calf, which would mean .7 milligrams per cent or more. If you put in sulfa drugs along with these three vitamins you can have 100 per cent success in Guernseys. Apparently the Guern-
sey cannot combat disease on equal terms with the Holstein calf, and for that reason we believe there is quite an important part to be played in calf scours control by the sulfa drugs.

We do know that the sulfa drugs change or at least affect some of these vitamins present in the blood stream. Sulfasuxadine, for example, will change the level of vitamin C in the blood. Why, I do not know. That is a preliminary observation and I give it to you for what it is worth.

The Chairman: Are there other questions?

Dr. Fleming (Louisville, Kentucky): I would like to ask Dr. Phillips this question: Dr. Phillips has given us the blood content of vitamin A in calves at birth, and then I believe he found that there is a definite rise in that vitamin A content in the blood of these calves within a few days after birth.

I would like to ask him if he has found any difference in the vitamin A content of the blood of these calves in cases where the dams were fed what we would call adequate or large amounts of nice green late-cut alfalfa, and yellow corn which should contain and furnish a goodly supply of vitamin A.

What difference, if any, does Dr. Phillips find in the vitamin A content of the blood of these calves at birth, as compared with dams which were fed an inadequate supply, and also how does the incidence of infection in calves compare in the case of calves from those respective groups of dams?

Dr. Phillips: There is some correlation, I believe, between diet and the amount of vitamin A in calves. We did try such an experiment with a group of cows which, I think, were three years old. We used the shark liver oil as our vitamin A carrier rather than alfalfa or corn.

I would just like to mention in passing that sometimes yellow corn does not carry enough of these carotenoid pigments to carry enough vitamin A.

In support of that I would like to cite the case of a boar, a very fine breeding animal, that developed an abscess, had lacrimation similar to that of a calf, on a ration containing 70 per cent corn. The boar was about ready to go out of the picture as far as we were concerned (we were about to give up hope for him) when we noticed that lacrimation and thought perhaps vitamin A might help. So we put him on shark liver oil and there was a phenomenal response within three or four days.

We are now given to believe that yellow corn, as far as a source of carotene is concerned, may or may not be adequate. We used to feel that 40 per cent corn would give enough carotenoids to furnish vitamin A, but we know now that is not so. It may be due to the storage of corn or other factors.

You cannot change the blood stream of the calf appreciably by feeding the dam. I think the answer lies not in the blood stream of the calf but in the colostrum of the dam. The highest level we have ever had in any calf, regardless of how the cow was fed, even though she had many, many times her requirements of vitamin A in the form of shark oil, we have never been able to raise that above 8 micrograms, which is still borderline, by feeding the cow; but it does effect the vitamin A in the colostrum itself, and the calf getting that colostrum will be better fortified within twenty-four hours than with the milk from a borderline fed animal.
I think that is the answer, if I have made myself clear. Animals fed suboptimal or minimal levels of carotenoids or vitamin A will produce a colostrum milk which will not come up to that of the animal which is well fed.

THE CHAIRMAN: Someone has very ably said that these are vitamin and hormone days. There are plenty of good reasons to feel that these substances may be interrelated.

The subject of infertility, or sterility, is a very big problem and is one which is a barrier toward increasing our goals for more meat and milk.

At this time I want to call upon Dr. Herbert Lothe to tell us something about the use of stilbestrol, that very powerful synthetic estrogen, and the dipropionate, relative to the treatment of genital diseases of the cow, and some of the pitfalls encountered in the use of stilbestrol. Dr. Lothe.

DR. HERBERT LOTHE (Waukesha, Wisconsin): I have used stilbestrol and stilbestrol-dipropionate in a limited way, and it does seem to have a place in the treatment of anaphrodisia in the cow. It is especially useful in cases of pyometra.

The injection of 80 milligrams of stilbestrol-dipropionate intramuscularly into cows with pyometra produces a prompt evacuation of the pus from the uterus. The same also has some value in pyometra following retained afterbirth.

In cases of deep-seated corpora lutea that cannot be removed by any rectal or vaginal manipulation, the injection of 75 to 80 milligrams of dipropionate suspended in oil will bring on estrus in from three to five or six days. It has this advantage over manual removal: You do not take the chance of internal hemorrhage following the manual removal of the corpus luteum from the ovary.

One thing that may be brought on by the administration of the dipropionate is the condition of over-stimulation with the production of nymphomania. The follicles become cystic, and the cow may remain in heat for a long period of time, sometimes as long as two and three weeks.

Recently we have been using a preparation, an endocrine product, experimentally in conjunction with some of the people at the University of Wisconsin. It is an anterior pituitary extract, a white powder that is dissolved at the time of use and injected intravenously. This will stop the nymphomania in from one week to ten days, usually bringing on estrus in about eighteen, nineteen or twenty days, with a recurrence of estrus in about twenty-one days when the cow is bred. A larger percentage of animals so treated conceived to a single service.

I believe steps are being taken now to have this product or preparation produced commercially so it will be available to the profession.

I believe that is all I have to say.

THE CHAIRMAN: Before Dr. Lothe gets away from the microphone I would like to ask him about the use of stilbestrol in this rather common condition called anestrus. Some of the farmers in the middle west plan their farming operations so that the breeding is done at a particular month or two during the season; let's say that in the middle west many of our farmers like to carry on the breeding program during the months of December and January. Many of these farmers call veterinarians with the statement that their cows are not showing estrus, and as a result they are unable to get them bred. They say this does not only occur among those who are using a sire on an owned farm or the user of sires, but those engaged in artificial insemination.
It becomes a problem as to what can be done to bring these animals in estrus, and if these animals are used would there be any dangers, Dr. Lothe, that they should be on guard against? Does ovulation take place when the heat appears, or does it not?

DR. LOTHE: Apparently ovulation does not take place the first estrum following the first injection of the stilbestrol, and it is well to check them afterwards to see whether or not you have a cystic condition of the ovary. If you have, it should be corrected, and oftentimes just a simple rupturing of those cysts will quiet the animal, and then at the next estrum she will oftentimes ovulate and can be bred and conception will take place.

But the production of a cystic condition of the ovary by the injection of stilbestrol is not at all uncommon, and possibly I have been using too large a dosage. Maybe 80 milligrams is too large a dose. Possibly 25 or 30 or 40 would be better.

THE CHAIRMAN: Dr. Lothe. We are going to turn for a moment away from the functional forms of infertility, and I want to ask you about trichomoniasis, and whether or not you feel that a disease of cattle due to the trichomonas is widespread, and how about the management of a herd of cattle in which this disease has made itself known.

DR. LOTHE: The condition known as trichomoniasis is common, but not as prevalent as, for instance, Bang’s disease; but it is prevalent enough so that it is quite a problem, and you will run across cases of it every now and then. I probably run across four or five herds affected with it in the course of a year.

The handling of it is not too difficult in some herds, and in other herds it is quite difficult. As you know, the condition is spread by the use of an infected bull. The majority of these bulls do not recover. Occasionally some do recover.

The first thing to do is to eliminate those bulls from service. When a herd is found to be infected, the breeding operation should be stopped, and then every animal should be checked periodically over a period of four or five months, at about two-month intervals, to see which is pregnant and which is not.

Some of the animals, if you diagnose them pregnant at 60, 80 or 90 days and check them later, will show that instead of being pregnant will have pyometra, that liquefaction of the fetus has taken place, and proper measures should be taken to evacuate it.

If those cows that have already been infected seem to develop a certain amount of immunity, or at least tolerance, they may be bred back to your infected bull. Virgin heifers should be bred to a non-infected bull, and if a non-infected bull is to be used upon cows that have been served by an infected bull he should be used artificially, otherwise you will take a chance of infecting the non-infected bull.

I used to think that if an animal had delivered a live calf subsequent to her being infected with trichomoniasis, that she was a safe animal to use on non-infected bulls. I am sort of beginning to doubt it. I know of one instance of an animal that delivered a full-time calf, that had previously been infected with trichomoniasis. A smear of the secretions from the nose and mouth of the calf showed viable trichomonas upon microscopic examination. It is well within the realm of possibility that such a cow might transmit the infection to a non-infected bull.

Some of these infected bulls seem to be fertile, others seem to be hopelessly barren.
after they have harbored the infection for some little time. Some of the work done at the University of Wisconsin on postmortem showed that the trichomonads had even gained entrance to the seminal vesicles and other sex glands. A bull infected like that, of course, is hopelessly barren.

You don't have a great deal of trouble in some herds, but in other herds you have considerable difficulty. The sterility rate sometimes runs quite high.

The Chairman: Thank you, Dr. Lothe.

We have a gentleman in the room whom I think of as being on the trail of the trichomonad, a man who is engaged in research in the field of parasitology who has had considerable experience with trichomoniasis. I would like to ask Dr. Morgan, of the University of Wisconsin, some of the more recent developments in the field of diagnosis, and possibly immunization. Dr. Morgan.

Dr. Morgan (Madison, Wisconsin): I am a little bit better in this job than Dr. Fincher because I was warned ahead of time that Dr. Boyd might call upon me. I might say a word or two about Dr. Lothe's statement concerning carrier cows. We have found a total of about sixteen cows that have been bred by infected bulls and have calved normally. Two of those have become carrier cows and on the next time around for calving the normal calves, the trichomonads were found.

It appears that about 1 to 2 per cent that have been infected by an infected bull, that calved normally, will remain carrier cows.

Concerning diagnosis, the problem is rather a difficult one at best. The methods we have now will give approximately 60 per cent results. We have developed a diagnostic medium which will pick out about 60 per cent of the infections. However, that is not good enough. We need something that will pick out between 85 and 95 per cent.

We have a diagnostic media in which we use Ringer's solution, dextrose, cattle serum, and Haematin, in which the trichomonad will remain viable from four to five days at room temperature. They do not multiply in this medium.

A great amount of bacteria will not hinder the trichomonads being motile so if they are there you will pick them up. That gives only 60 per cent results.

We have been working on a complement fixation, precipitin test, agglutination test, and various allergy tests. The complement fixation test gives us too many nonspecific reactions, so we can't use it as yet.

If you inject animals with living trichomonads long enough you will build an immune substance in their blood which will react or agglutinate. However, we have worked on over 400 cows which have been infected, and only three of those reacted to the agglutination test. Those were cows out in the field, infected with trichomoniasis, and only three of them reacted to our test. So we can't use the agglutination test because it is not good enough.

In the precipitin test we have failed to make a good antigen which will not react to normal serum. It is very, very hard to grow a trichomonad and powder it or make it in any particular suspension so it will not react to ordinary normal serum. However, we have grown trichomonads and gotten them out into a particular suspension, and have isolated a polysaccharid. This particular polysaccharid is showing some promise, as it will react with infected cattle and not with normal serum. However, a lot remains to be done in that particular field.
In the way of allergy tests of trichomonad suspensions we have used every type known that we can make in the laboratory, both dead and living, treated with various chemicals, also with polysaccharid, and injected subcutaneously. Under no circumstances do we get any reaction. So in this field of diagnosis, as far as trichomoniasis is concerned, it is really difficult, because the protozoa are just not antigenic. They are very, very low in antibody-producing powers.

As far as immunization is concerned, we have experiments under way now on vaccination that I am not at liberty to disclose as to details, but very good promise is being shown that out of a total of 18 cows, 10 of which were vaccinated, 9 of those cows are carrying calves at the present time after being vaccinated, and were heavily infected. The control cows, eight of them, have all come down with the infection and only one is pregnant at the present time.

In the way of immunization, it also seems that there is some immunity. Some cows once infected are harder to infect the second and third time, and so on. The immunity is either partial or solid, depending upon sterility of the cows.

In the way of using infected bulls, we are working on various methods of treating semen from infected bulls that can be used in artificial insemination. We have a particular biological method that we are using now, which we use to test the fertility of the sperm, mixing this particular material with semen which kills the trichomonads, but we are now trying to find out how it hinders the fertility, and that will be done in the future.

THE CHAIRMAN: Thank you, Dr. Morgan. Are there any questions or is there anyone who would like to add something to this discussion?

DR. DURANT (Columbia Missouri): I am very much interested in this matter of trichomoniasis, but it has reference to fowls and not to cattle.

I would like to mention particularly one thing in regard to the medium. At Missouri we had great difficulty in getting the trichomonads to grow in artificial media. We used Dr. Allen's egg medium, and Locke's egg medium, but we found that the trichomonads died very rapidly even in the best media we could concoct.

We discovered later that if we added a fairly pure form of vitamin A to the culture media, the trichomonads would not only live but to multiply in a most remarkable manner.

I realize that the trichomonad of cattle is much more resistant to unfavorable environment than the trichomonad of turkeys, but it seems to me that it might be helpful if you could add to the culture media a few drops of vitamin A on the surface of the culture media, and you might get them not only to live but to multiply at a rapid rate.

Thank you.

DR. MORGAN: I might mention that in our particular medium in which we can grow trichomonas fetus in cattle, we have developed a medium in which we can grow the organism approximately twelve to fifteen million organisms per cc, which is a very, very heavy concentration, sufficient to make any amount of antigenic material we want.

With regard to growing of trichomonas fetus, our particular medium will make the trichomonads grow approximately twelve million per cc. Of course, the various nutrients that we add include some vitamin A. This particular medium which we
use, in a week's time if we used $125 worth of eggs, we could grow enough tricho-
monads to isolate our polysaccharid from it.

The Chairman: Anybody else? This is a very important topic, and we wel-
come additional discussion.

If there is nothing further we will pass on to the next topic, to be presented by
Dr. Walter Krill. We are coming back to that old familiar subject, milk fever.

The first question I would like to ask of Dr. Krill is this: What are the causes of
milk fever, and can milk fever be prevented?

Dr. W. R. Krill (Columbus, Ohio): I think first of all we should recognize
that the term "milk fever" is a misnomer. Normally fever does not constitute
one of the symptoms of the ordinary case of so called milk fever. The work of
Dryer and Gregg in around 1925, in which they published the work they did on
the study of the blood in cases of milk fever, showed that in all cases of milk fever there
was a definite hypocalcemia, a definite decrease in the calcium content of the
blood of those cows. That symptom is quite constant in all cases of milk fever.

Naturally, with the initiation of milk flow there is a considerable drain of the
blood calcium. It is estimated that a half gallon of colostrum milk contains calcium
equivalent to that found in the blood of an animal at any one time.

It is interesting to consider, however, why some cows develop milk fever and
others do not. I don't think that question has ever been definitely solved. I
don't know whether we will ever solve it. I still think the actual cause of milk
fever, the basic cause, is yet to be found.

There has been some work done at the Ohio Station by Krause and some of his
co-workers, on a study of the use of irradiated yeast supplying these cattle with
vitamin D, since vitamin D does play a part in the utilization and metabolism of
calcium in the animal's body.

Their work at first showed considerable promise. The first report they gave
seemed to indicate there was a marked reduction in milk fever in all cattle that were
fed vitamin D. Later results showed that it does seem to have some definite effect
on reducing the incidence in Jersey cattle. We know that Jersey cattle exhibit a
definite breed variation, and are more prone to milk fever than are other breeds.

But just why should we have the symptoms of milk fever developing, except
that it is a calcium deficiency, why this calcium deficiency should occur in the
blood stream, we do not know. It has been supposed, of course, that the para-
thyroids which control the calcium in the blood stream are functioning improperly,
and are responsible for this. However, the use of parathyroid extracts in the

treatment of milk fever do not seem to be very satisfactory.

We also notice in milk fever that there are a lot of seasonal variations. During
certain seasons of the year, particularly during the fall and winter months, there
are a greater number of cases of milk fever developing than in other seasons of
the year. Of course that is during the periods of greatest numbers of parturitions.
But I don't think that entirely accounts for the greater incidence of milk fever
during those seasons.

Some have also recognized—and it has been reported by numerous practitioners,
and I have made this observation also—that in periods in which there is a high
degree of humidity in the atmosphere there are a greater number of cases of milk
fever than at other times. Possibly that is due to a lesser amount of available oxygen for the tissues.

We also notice this condition in the same herd from year to year: There are definite variations in the incidence of milk fever in those herds, which would lead one to think that possibly nutrition may play a part. There may be some variation in the quality of the roughages which these animals get from year to year which may play a part in the cause of milk fever. But as yet we do not know what the basic cause of milk fever is.

As to whether or not it can be prevented, we don't know the basic cause and naturally we don't know any true prevention for milk fever. As a prophylactic measure in some of these cows that have milk fever regularly from year to year, we can use certain prophylactic measures such as administering calcium, particularly at the time of parturition, and daily for a few days after that, and it does seem to abort the symptoms of milk fever.

It seems to me there must be some endocrine disturbance which affects the calcium controlling mechanism in the blood stream. Whether or not they are basically nutritional we don’t know, but I feel certain that nutrition plays a very definite part in the cause and prevention of the disease.

THE CHAIRMAN: Dr. Krill, what are the advantages and disadvantages of calcium therapy and udder inflation treatment? And why do some of these animals fail to respond to either form of therapy?

DR. KRILL: I think we will all have to recognize that there are very definite advantages to the calcium line of treatment. In the first place it is much more professional. It is supplying the thing which is lacking in the animal's body to correct the symptoms which the animal manifests. Supply the calcium and you have a rather rapid recovery in most instances.

Another big advantage is the elimination of the danger of introducing infection into the udder, with the resultant mastitis. It is almost impossible to inject air, regardless of how careful we may be, without introducing some infection, and even though we might be ever so careful, most of these cows may have some low-grade infection in the udder and if, following the inflation of the udder, mastitis developed, responsible or not, we would get the blame for it. The calcium treatment obviates that incrimination.

Another big advantage is that there is no decrease in milk production with the calcium treatment as results from air inflation. One of the complaints with the old air inflation method was that these cows never did as well at lactation as they did normally when they didn't have milk fever. There is a marked diminution in the milk flow when the udder is inflated. With calcium treatment they go along very nicely, and with normal production.

I think most of you will realize there are some disadvantages. We will have to admit there are more relapses with calcium therapy than there were with the air inflation method.

Also, in my experience at least, there are some cases that will not respond to the calcium treatment, which will respond to air inflation, but I do want to make this statement, that air inflation should only be used as a matter of last resort, we prefer the calcium treatment in these cases.
THE CHAIRMAN: Then I would like to have you follow up with the following questions: What is acetonemia or acetonuria, and what may be done to prevent the occurrence of this problem?

DR. KRILL: Acetonemia is a disease which sometimes has been referred to as chronic milk fever, acetonuria, ketosis, and various other names. It is a disease which occurs more often in high producing dairy cattle at or within a few months following parturition.

It is thought to be caused by an impaired carbohydrate metabolism, and this results in an improper breakdown of the fats, and leaves certain ketone and acetone bodies in the blood stream.

The disease manifests itself in various ways. We recognize several forms of the disease. We think of the milk fever type of the disease, one which is frequently associated with milk fever. We think of the nervous form of the disease, and also the digestive form.

In the digestive form we find that there is a gradual impairment of the appetite and a gradual decrease in milk flow. The animal may show or give evidence of having acetone in the breath, and the odor of acetone in the milk and urine. Personally my sense of smell has never been developed to the point where I would want to diagnose a case on that basis. Some people have a keener sense of smell than others, and possibly they can. In this form we find that it occurs in high producing cattle and usually occurs within a few weeks to maybe several months following parturition.

The nervous form, in my experience, may show various symptoms, all the way from mild irritatatability to very marked symptoms of nervous disease, and in some cases marked delerium. I have encountered most of the cases of the nervous form in the spring of the year, when cattle have been turned out to graze, especially during the cold, damp spring days, and where they are not given any supplemental feeding. These cases become very nervous, show marked acetonemia reaction, and will respond very nicely to the acetonemia treatment.

We also find numerous cases in which they show symptoms of licking. I have seen those frequently occurring within a few days following parturition. The treatment for acetonemia will stop the symptoms very suddenly.

Those associated with milk fever or close to the time of parturition—I always like to make a rather careful check of the reproductive tract to make sure that there is not some involvement there, an atonic condition of the uterus in which the uterus is not properly involuted, and in many of those cases we will and that the uterus contains a considerable amount of debris.

I have a case in mind right now that came into the hospital just before I left, that had been treated for about a week for acetonemia, was not responding, and when it was brought into the hospital upon examination we found a definite metritis. So we want to keep that in mind in connection with those cases.

In so far as the prevention is concerned, I think in many cases of true acetonemia, especially of the digestive form, and many of these nervous types of acetonemia, we are dealing with a definite dietary deficiency or unbalance in the diet, feeding heavy protein diet with insufficient amount of roughages. I think the dairy man in many instances has forgotten that the dairy cow is a ruminant and is capable of converting large quantities of roughages into essential food.
Possibly it is essential that they receive a large amount of roughage. When we are feeding large amounts of grain, heavy grain feeding markedly decreases the roughage consumption of these animals, and I feel it is very necessary that these cattle have large quantities of roughages to supply the various elements which they need, and by feeding too heavy grain diets they are not consuming the necessary amount of roughage.

I think the proper balancing of the diet will go a long way in the prevention of acetonemia.

THE CHAIRMAN: Gentlemen.

The next subject is a tremendously interesting one. The speaker is Dr. Dykstra, and the first subject that I would like to quiz him on is anaplasmosis.

The first question is, "How can a definite and accurate diagnosis of anaplasmosis be established? How is the anaplasma transmitted? Are infected animals curable? What are the most commonly employed methods of treatment during the acute or clinical stage?"

Dr. Dykstra.

DR. R. R. DYKSTRA (Manhattan, Kansas): I know of no other means (of diagnosing anaplasmosis) than by the inoculation of a susceptible bovine. Other methods have been suggested, such as that of Boynton and Woods, in which two drops of suspected serum are added to 2 cc. of distilled water, with resulting cloudiness and precipitate. This test, however, lacks accuracy.

In nature, anaplasmosis is transmitted very largely by biting ticks. A few of these ticks are biological vectors in that the causative factor is passed through their eggs. Biting flies are also thought to serve as mechanical carriers; non-sterilized or incompletely sterilized surgical instruments may serve as vehicles of transmission.

Infected animals are curable only in the sense that they no longer exhibit outward evidences of the disease. Such so-called "cured" animals are immune carriers, and are potentially dangerous to susceptible cattle. The immune carrier stage usually persists throughout the remainder of the animal's life. The disease can be made to flare up in immune carriers by surgical removal of the spleen.

Sodium cacodylate solution is standard treatment, and is made by dissolving four half-grains of this chemical in each cc. of a 5 per cent solution of dextrose. The dose is from 25 to 30 grains of the sodium cacodylate per 100 pounds of live weight, administered intravenously.

From 12 to 15 grams of sulfathiazole sodium dissolved in 30 to 40 cc. of distilled water and administered intravenously as a single dose has been reported as beneficial in the treatment of acute cases of anaplasmosis.

A recommended step in regard to these so-called carrier animals is to dispose of them for slaughter purposes before the next crop of biting insects makes its appearance.

THE CHAIRMAN: Please remain here, Dr. Dykstra. There are some men in the room who I am sure would like to ask some questions about anaplasmosis. There are pretty good reasons to feel that the disease is spreading throughout certain sections of the country. Let us have your questions quickly, gentlemen. We have a lot of material yet to cover. Please come to the microphone and give your name.
DR. BUTLER: Dr. Dykstra, you failed to mention a blood smear. Is a blood smear not to be relied upon in making a diagnosis to find the anaplasma marginale?

DR. DYKSTRA: There are times in the development of the anaplasma, Dr. Butler, when it cannot be detected in a blood smear. As far as we are able to tell by microscopic examination, the microparasite has disappeared entirely from the blood.

DR. BUTLER: But is you find it you would consider that a positive diagnosis, would you not?

DR. DYKSTRA: Reasonably accurate. There is a lot we do not know about the anaplasma marginale. There are still a good many investigators who have difficulty in positively identifying that parasite simply from a microscopic examination.

DR. BUTLER: One other question please, Doctor. Are all apparently recovered cases carriers?

DR. DYKSTRA: To the best of our knowledge they are.

DR. BUTLER: All?

DR. DYKSTRA: Yes.

DR. W. H.LYTLE (Salem, Oregon): I might say we have found that by burning off the range we can exert considerable control over the spread of the ticks.

DR. DYKSTRA: The State Veterinarian of Oregon said he feels they can control the disease in a measure at least by burning off the range. We have not tried that, Dr. Lytle, so I cannot comment upon it.

Dr. Boyd has suggested (and I believe it is a good point) that it should be emphasized that in those sections of the country where anaplasmosis has made its appearance, that great care should be taken in removing blood samples from those cattle for Bang's disease testing, or in the use of the common surgical instrument.

The disease is spread quite readily from animal to animal by the common use of needles or other instruments, and certainly those who are engaged in practice in sections of the country where the disease is prevalent should take the proper precautions to see to it that they do not spread the disease by those methods.

THE CHAIRMAN: Are there any other questions?

DR. RAY (Omaha, Nebraska): I would like to have Dr. Dykstra tell these men what he would recommend if a case or two of anaplasmosis is found in a bunch of feeder cattle that have been shipped into his territory and will probably go out to the packers later on, but who definitely have anaplasmosis on the farm, in the territory where it is not ordinarily recognized.

DR. DYKSTRA: Certainly, Dr. Ray, those cattle of the type you mention usually come onto a place in the fall, and biting insects at that time of the year are disappearing.

I don't believe there is very much danger in a situation such as you mention, when biting insects are disappearing, but they should get rid of those cattle before the next crop of biting insects comes along.

THE CHAIRMAN: The next topic is that of actinomycosis. Here again I would like to group a number of questions so that Dr. Dykstra may move along more rapidly. I will not interfere by popping up here with a question every moment or two.

Is the disease that usually, in an offhand manner, is diagnosed as actinomycosis, actually this condition?
Is it of great importance to distinguish clinically between the two conditions?

Can animals be immunized so as to give them some degree of protection against these ailments?

How is actinomycosis spread?

Dr. Dykstra: The disease which usually is diagnosed as actinomycosis may in general be either actinomycosis or actinobacillosis. The latter condition is due to the actinobacillus Lignieresi—a gram negative organism—and it is the condition that is usually called lumpy jaw and wooden tongue. Laboratorians state that when the soft tissues are involved it is usually actinobacillosis, and it is actinomycosis when caused by the so-called ray fungus, which, by the way, is polybacterial and not a fungus, when a hard tissue such as bone is affected.

Is it of great importance to distinguish clinically between the two conditions? No, because the causative factor in each of the two conditions is iodine-sensitive. Local surgical treatment, followed by iodine applications, or the intravenous use of a solution of sodium iodide, are the approved methods of handling. Bone cases and those with intensive induration never respond so well, and honeycombed bone cannot be made normal.

There is no known immunizing product so animals can be immunized so as to give them some degree of protection against these ailments.

Concerning how actinomycosis is spread, the generally accepted theory is that the causative organism is an obligate parasite of the animal body, and that it gains entrance to the tissues through abrasions. Therefore, abrasive roughages should be withheld, and abrasions should be iodine treated. This is most applicable when there is an extensive outbreak of the condition, or when it is persistently present.

The Chairman: Are there questions?

Mr. DeNormandie (Massachusetts): I would like to ask a question about the danger to humans and other cattle.

Dr. Dykstra: There is danger, of course, but not so very great in view of the fact that it is a wound infection. I think from the sanitary standpoint it is an excellent practice, when actinomycotic abscesses are lanced, not to permit that to flow out on the ground or into the corral or wherever the animal happens to be.

It is better to catch it in a pan of antiseptic and avoid promiscuous distribution of that material. The danger is not particularly great.

Dr. Butler: Is there any evidence obtainable or available with reference to its direct transmission from one animal to another?

Dr. Dykstra: I doubt whether there is, Dr. Butler. I know of no such evidence.

The Chairman: I am very glad to hear that question by Mr. DeNormandie. I might relate that just a short time ago I was in the University of Minnesota clinic, where I had an opportunity to observe five human cases of actinomycosis. It happened that all five individuals had had some contact with farm animals. I think Dr. Dykstra's answer to Mr. DeNormandie is a very good one, that it is not to be looked upon as a disease readily transmissible to man, but all five individuals who appeared in that University clinic had had contact with farm animals.

I was also interested in the type of surgery being used on those cases, and the surgeon brought out the point that all of these lesions must be exposed to the at-
mosphere, given plenty of exposure to the air, as the organism does not do well under those conditions.

Are there any other questions? If not, we will go on to the next subject, which is to be discussed by Dr. Dykstra, and that is the subject of listerellosis.

This condition of listerellosis is a problem that is coming to the attention of many of us. It would seem to some of us that it is a disease of recent occurrence or a disease in which we are just beginning to learn something about it. Certainly it is one that is causing more or less confusion. Just how well distributed it may be we will probably bring out in the discussion.

What is listerellosis?

What are some of the observed clinical symptoms in cattle?

Can the disease be positively diagnosed on the basis of clinical symptoms?

From the standpoint of differential diagnosis, what cattle diseases bear some clinical resemblance to listeriosis?

Can animals be immunized against this disease?

Dr. Dykstra: Listerellosis, also more correctly and commonly designated as listeriosis, is generally in the nature of an encephalitis in sheep and cattle. In swine the nervous symptoms are not so marked. Affected chickens appear weakened and emaciated, with focal necrosis of the liver. In sheep, because of the principal clinical symptom, it has also been designated "circling disease."

The causative organism, the *listerella* or *listeria monocytogenes*, is the rabbit organism. It is believed that the species in other animals is at least very closely related to the rabbit organism.

Field and laboratory studies indicate that cattle are much more resistant than either sheep or swine. The symptoms are of a nervous nature such as depression, weakness, incoordinated gait, walking in circles, pushing against objects, progressive paralysis, and, in the more severe cases, death in a few days.

Aggressiveness has been observed in some affected cattle. Mild cases usually recover. The disease has been observed most frequently in feeder cattle.

Answering the question of whether the disease can be positively diagnosed on the basis of clinical symptoms: No, an accurate diagnosis demands recovery of the causative organism from the central nervous system, with reproduction of the disease following its intracranial inoculation, or that of brain material, into rabbits and guinea pigs.

The question is asked concerning what cattle diseases bear some clinical resemblance to listeriosis. Ketosis, milk fever, rabies, sporadic bovine encephalomyelitis, so-called wheat poisoning or grass tetany, infectious bulbar paralysis (mad itch), and various infections, toxemias and nutritional disorders. It is claimed that in the laboratory the introduction of young cultures into the conjunctival sac in rabbits is followed by a very severe purulent conjunctivitis.

Answering the question as to whether animals can be immunized against this disease, the production of agglutinins can be stimulated, but because of the sporadic nature of the disease this is of no practical significance. Clinical treatment is symptomatic. Sanitary steps are good general steps.

The Chairman: I think Dr. Dykstra has done a most excellent job in giving us the characteristics of this disease, including the symptomatology, diagnosis, immunization, and so forth.
Those of you who have questions—and I am sure you do have—please come right up to the microphone and make your contribution. Some of you have observed this disease in sheep; some of you have observed it in cattle.

It has been recorded as having been recovered from the fetus of a cow that had aborted, let's say, somewhere midway during gestation. From this aborted fetus listeriae were recovered. Probably it was accidental. Maybe it is not an organism that often effects embryonic tissue, but in this instance it was thought to have been the cause of the abortion.

DR. RABSTEIN: As an aid in diagnosis isn't it a fact that there is a seasonal incident?

THE CHAIRMAN: Did you ask about a seasonal occurrence which might apply to diagnosis?

DR. RABSTEIN: Yes. Wouldn't that be a help in diagnosing it?

DR. DYKSTRA: Not very much, I should think, because of course the disease is observed in cattle, mostly those that are in the feed lot, and some of these other diseases that have been mentioned from the standpoint of differential diagnosis occur under comparable conditions.

THE CHAIRMAN: Thank you, Dr. Dykstra. Perhaps we will be calling upon you again before the session is over.

Now we are coming to a gentleman who is pinch-hitting for Dr. Port of Wyoming, who is unable to attend. I am very happy to present this gentleman, because while he was presented a while ago, this will probably be the only opportunity I will ever have to introduce you to the President-elect of the American Veterinary Medical Association, Dr. James Farquarson, Professor of Surgery, Colorado State College of Veterinary Medicine, Fort Collins.

Dr. Farquarson is going to talk to us on a subject which some say is a specific entity. Some question it, and some say there is no such disease. I am referring to shipping fever, stockyards pneumonia, and hemorrhagic septicemia.

I want to ask him if this disease does affect cattle maintained under range conditions; if so, what time of the year is it most prevalent, and at what age are cattle most frequently affected, and what measures he recommends for the prevention and treatment of animals thus infected. Dr. Farquarson.

DR. JAMES FARQUARSON (Fort Collins, Colorado): This is a golden opportunity to stick out my neck.

There is a disease entity, call it what you may. We call it shipping fever. First of all I want to say that shipping fever is not necessarily hemorrhagic septicemia. The term "hemorrhagic septicemia" should be limited to a disease only after laboratory procedures have confirmed that diagnosis.

The majority of cases that we see in Colorado or in the western ranges we are now calling shipping fever, because in the majority of cases we fail to demonstrate the hemorrhagic septicemia organism. I say in the majority of instances that happens.

This disease does occur under range conditions, and it occurs in the fall of the year. It occurs when the cattle have been gathered together and driven down usually to the meadows or to the home ranches.

The animals which are infected are usually the weaning calves, yearlings, and
some two-year-olds. Occasionally we do see hemorrhagic septicemia, if that is a
disease, and we are diagnosing the disease by the recovery of the organism, in
range cows.

The last question here is one that is controversial: "What measures do you
recommend in the prevention and treatment of animals affected with this malady?"

The prevention, I think, is the main thing in our method or in trying to educate
the ranchers to our method in the prevention of this disease. The cattlemen in
our part of the country are syringe-crazy. They vaccinate for black leg, they
vaccinate for hemorrhagic septicemia. Undoubtedly there is a place for it, yet
the main thing in the prevention of the disease is when these cattle are collected
on the ranges, to ease them down and not drive them for long distances without
water or feed. Drive them slowly; let them eat as they go, and drive them for
short distances.

The old method of collecting them, rounding them up and driving them for
twenty-five or thirty miles a day without any feed, letting them lie down at night
and then moving them along at the same pace the next day, materially lowers
their resistance.

In shipment the main thing is to protect them, as far as we can under range
conditions from storms and to see that they are not shipped in inclement weather.
These animals have been ranging over a large area, and now they are confined and
bunched together. They are suddenly exposed to mass infection in railroad cars;
they are denied water and feed for long periods of time, and when going through
public stockyards there is danger of mass infection, which materially lowers their
resistance. Then they are shipped to feed lots.

By exercising care in the transportation of these animals I think you have one
of the greatest measures of conserving their resistance and preventing the disease
we call shipping fever. I don't care how often they have been vaccinated—these
animals will come down with shipping fever when they receive rough treatment,
when they become tired, either at the shipping point or at the point of destination.

Many stockmen believe that their cattle do not contract the disease. They say
they don't contract the disease because they have learned to ship them to their
initial shipping point, get rid of them, and then say the cattle didn't have the
disease. But if you follow the cattle through the shipping yards you will see that
they come down with this respiratory condition in the course of the next week.

As far as treatment is concerned, as they reach their point of destination for
feeding, these cattle are tired. They are wild, and any individual handling and
vaccinating of them is absolutely contraindicated at that time. We believe they
have been on rather meager rations—buffalo grass, mountain grass. Then we
suddenly subject them to a heavy nutrition ration the first day they are in, and
that is a bad practice. They should be allowed to rest, given plenty of shelter,
good drinking water, a little alfalfa, or allowed to range over the farms on stubble
for a few days.

Concerning treatment, I have little to offer. Under our conditions, again, these
animals are wild. Many times when we see an entire group that is affected with
so-called shipping fever we should attempt, first of all, to go through them and
find those cattle in which the infection is confined to the upper respiratory tract,
isolating them from those which show more severe infection, tracheitis, bronchitis or even pneumonia, and then through sanitation, hygiene and resting, most of those animals will get along without any treatment. You will do more damage than good otherwise. I am talking now of western cattle.

The individual cattle should be segregated, placed in comfortable quarters. They may be treated with expectorants, blood transfusions, salines at times, and the sulfonamides. We find sulfapyridine possibly is the best agent. On high-priced cattle you may use serum.

DR. GLENN NYE (Elgin, Illinois): I think you are a little modest about expectorants. Will you elaborate on that a little bit?

DR. FARQUARSON: I don’t think there is any specific treatment for this condition. We have found that the most satisfactory expectorant is the sulfonamate of glycol, and that is combined with sodium iodide, a little alcohol, and seems to be a rather effective stimulant; we give that intravenously.

I realize that must be used very early in the course of the disease. It is worthless in pneumonia, but in an earlier course of the disease the results which we see—and, remember, we see them earlier than you do because we are at the back door of the shipping port—are rather spectacular in many instances. I think you should also combine the sulfapyridine or sulfathiazole.

DR. O. L. CAMPBELL (Astoria, Illinois): What about mixing these cattle that come in with native cattle?

DR. FARQUARSON: I haven’t seen very many cases of evidence of animals that have been there for some time contracting the disease. If you bring them in, unless it is possibly hemorrhagic septicemia, the majority of our shipping fevers are not transmitted to the animals that have good resistance and that have been in those places for some time.

THE CHAIRMAN: We will pass on to the next topic, coccidiosis in western feed lot cattle, and I would like to ask Dr. Farquarson if he regards this as of considerable importance; I would also like to have him elaborate upon the treatment of animals thus affected.

DR. FARQUARSON: Coccidiosis in range cattle is not a serious condition. We have made fecal examinations on range cattle. We find that we can recover on one examination, the coccidia in usually 85 per cent of the animals. We feel if we take it from day to day for a few days that probably all animals are carriers of coccidia.

Under range conditions it is not a clinical entity. It is a sub-clinical condition. Therefore, as far as I know we don’t see the acute conditions there; we see them after they come into the feed lot.

Our treatment in coccidiosis in cattle is to remove them from all concentrates and put them on a native or timothy hay, cut out all grains, barley, corn, beet pulp, and again you are dealing with cattle that you can’t treat, lots of times, individually.

I wonder how much we would gain by treating them, knowing the nature of the disease. It is cyclic and self-limiting. When we see coccidiosis we use sulfaguanidine, also suxadine (and we have used many other agents in the past) only to give the agent credit for the results, when, as a matter of fact, the disease is on
its way out anyway. If we could use these agents three or four days previous to the clinical evidence, the passing of blood, then we might have some reason to believe we have more or less of a specific agent against coccidia.

Treat individually those animals that have become badly dehydrated, that are constantly straining and that lose a lot of blood. Keep them warm and administer blood transfusions. Of course we usually try to support that with some of the sulfaguanidine or suxadine. Keep them warm during cold weather, blood transfusions seem to be most effective.

**The Chairman:** We have just one more question, Dr. Farquarson. Will you tell us the nature of the ailment regarded as an acute sporadic pulmonary emphysema of cattle.

**Dr. Farquarson:** Acute pulmonary emphysema—if that is the name we are going to give to it—is quite common in western cows. Again it is a condition that we observe in the fall. These animals are driven in from their range conditions, and in the mountainous areas we usually see it. They are driven down to the home meadows or barns. They were perfectly all right when they started. Some of them will die en route, and some of them will die shortly after their arrival.

The symptom is that of a very high temperature, 106 to 107, tongue protruding, mouth breathing, and a picture of toxemia. The temperature is undoubtedly due to the toxemia. The autopsies reveal cultures are negative, and the pulmonary emphysema is the outstanding thing.

We have nothing to say about the etiology of it. One might think of allergy, but we don't know. Occasionally we see it in the feed lots. Certainly one or two will develop this condition.

I have no explanation as to its cause. Dr. Butler, I am sure, sees it in Montana. If I were to offer a cause it would be along some form of allergy. We hear also that it comes from eating water hemlock, scrub water hemlock, poisonous plants, and other things like that, but water hemlock does not cause pulmonary emphysema as we see it.

**The Chairman:** Dr. Butler's name was mentioned. Dr. Butler, would you care to discuss this topic?

**Dr. Butler:** I have nothing much to say that Dr. Farquarson hasn't said.

In Montana we have noticed this disease for about seven years. Previous to that time we had never observed it. I believe Dr. Scofield, of Canada, has reported this condition in that country. The history in our country is practically always the same. It is a lactating cow coming in from short grass, and brought into a pasture generally where there is alfalfa or clover, in the early fall after we have had a frost. The animal shows symptoms that Dr. Farquarson has described, and we presume it is due to an allergy, some complex plant sensitization whereby the residual air builds up in force. You get this emphysema; we call it asthma.

The residual air gradually develops until the pressure becomes so extreme that we get what we call a “blow-out.” We will get a “blow-out” two or three inches in size in the lung tissue, and there is hemorrhage, of course, and under these conditions the animal will die.

We have used atropine and morphine and epinephrine with apparent success in some conditions. In other cases we have to use calcium gluconate. In some
cases it works with miraculous results, and in other cases with absolutely no results whatever other than the death of the animal. And so calcium gluconate is not a specific in this particular condition, although it has been given credit for cures in a number of cases.

The animal is not able to exhale afterwards the residual air or the amount of residual air it should exhale, with the result that these bronchi become more or less restricted, and you get this build-up of residual air with the resultant "blow-out" —and believe me, when I say "blow-out" I mean "blow-out."

The Chairman: Thank you, Dr. Butler. We are very glad to have this discussion. The condition is absolutely new as far as I am concerned. It does add to the list of those diseases that seem to be associated with the termination of pregnancy.

Are there any other questions? Is there anyone who would like to contribute further to the discussion? If not, we are going to take up the subject of mastitis.

This subject will be approached from the standpoint of the bacteriologist and from the standpoint of the practitioner. We think as the years go by that we are gradually accumulating more and more information relative to the etiology, pathology, and therapy of mastitis.

The disease is a very stubborn foe. It is world-wide in distribution. It is one of the most important barriers toward milk production. It is a disease of greater importance at this particular time, with greater demands being made for milk, increased consumption and lower production, and of the various factors responsible for a lower milk production mastitis is a most important one.

From the bacteriological standpoint the subject will be presided over by Dr. Little, who has spent a great many years in the study of this disease from the research standpoint, having made a very notable contribution relative to etiology.

The practitioner's side will be taken care of by Dr. Case of Akron, Ohio, who is interested in all diseases of the cow, particularly the dairy cow, and who has had a rich experience in the treatment of mastitis.

I do want to say at this time that this is not a disease that is of interest only in the dairy cow. Perhaps we see more of it in the dairy cow, but it is also a problem among beef cattle. Even in the Angus cow, a cow that sometimes does not have much more milk than is needed to raise her young, still the man breeding those black cattle will realize that mastitis is a foe which he meets, and not infrequently.

In starting off with the subject I want to approach it first from the bacteriological side and I want to ask Dr. Little this question, because it is an oft-repeated question: Is mastitis caused by microorganisms other than streptococci and staphylococci? Is it caused by a virus, and is it reasonably feasible to feel that the secretions from the various endocrine glands are in any way responsible for mastitic conditions of the udder?

Dr. Little.

Dr. R. B. Little (Princeton, New Jersey): In answer to the first question, yes, coliform and coliform pyogencs infections, both acute and chronic, are frequently observed in certain herds. Under exceptional conditions microorganisms not usually associated with mastitis in the cow may be responsible for rather severe infections.
In answer to the second part of the question, "Is mastitis caused by a virus?", this is a very important question. The reports of Broadhurst, Peterson, Hastings, Beach and Hadley strongly suggest that a non-specific agent, possibly a virus, may be the precursor in mastitis, and that streptococci, staphylococci, and other microorganisms, are usually only secondary invaders.

I do not question these results; however, I have been unable to demonstrate a virus from the udders of mastitic cows in varying phases of the disease. Perhaps in some herds a virus may be a causative factor in the disease. There are, I believe, certain extraneous factors that may cause the same syndrome of symptoms in the udder as have been described for non-specific mastitis. These factors are:

1—Overworking the udder in an attempt to maintain a high milk yield, with little rest between lactations.
2—Milking cows in advanced lactation.
3—Improper use of milking machines.
4—Improper milking by hand.
5—Possibly an overabundance of oestrogen in the blood.
6—Intensified breeding programs to produce large secreting udders.
7—Presence in the udder of unidentified bacteria.

It would seem most logical that before non-specific mastitis can be ascribed as being caused by a virus, the influence on the udder of the factors just mentioned should be thoroughly considered. They may be responsible for the development of an increased permeability of the udder, resulting in the production of a low grade induration and changes in the character of the secretion.

Answering the third question, it would seem that the hormones from the ovary, adrenal cortex, and placenta, are all concerned in the control of the mammary gland. However, little if anything is known about their influence on the udder as regards bovine mastitis.

It is possible that in udders working at a peak capacity, an excess or a low concentration of these secretions in the blood might either predispose the gland to infection or make it more resistant to infection.

This is simply a suggestion, but I feel that when more is known about the influence of hormones on the udder it may help to clear up some of the confusion now existing in regard to the transmission of mastitis.

The Chairman: Dr. Little, has nature provided the udder with natural barriers to protect the gland? Also, how do bacteria gain entrance to the udder?

Dr. Little: Although nature placed the udder in a position where it is continually subject to injury and filth, it also provided the gland with natural barriers for its protection, namely, the teat sphincter and the presence in the udder of a natural bactericidal substance.

A function of the teat sphincter is to retard the ingress of bacteria to the duct of the teat, while the bactericidal or bacteriostatic substance tends to inhibit the development of bacteria which have passed by this barrier until they are flushed out by the outward flow of milk.

It should be realized that occasionally microorganisms in the active phase of growth become established in the udder, and they multiply so rapidly that the inhibitory substance is unable to hold them in check. When this occurs it results in infection.
In answer to the question "How do bacteria gain entrance to the udder?" in bovine mastitis transmission experiments have been confined largely to streptococcus agalactiae and staphylococcus aureus. At present there is no conclusive evidence, substantiated by careful experimental observations, that would indicate that these organisms gain entrance to the udder by any common route other than the teat canal.

The Chairman: When streptococci or other microorganisms are detected in the fore milk from an apparently normal quarter on two or more cultural examinations, should it be considered that an infection exists in the quarter?

Dr. Little: Yes, if it can be shown by cultural tests that the streptococci are slowly multiplying in the quarter, and if sufficient examinations have been made to rule out external contamination.

The Chairman: What portion of the milk for cultural study is the most satisfactory for the detection of the mildly infected cases?

Dr. Little: The fore milk; that is, the first 2 or 3 cc. of milk expressed from the quarter, none discarded.

The Chairman: Of the bactericidal agents now being used for udder therapy, are certain ones more effective than others?

Dr. Little: The selection of the cases to be treated is of importance in this connection. Apparently some bactericidal agents are more effective than others in the treatment of the mildly affected cases, whereas there is little difference in the effectiveness of many of the preparations in the treatment during the dry period of advanced cases.

In lactating udders, either gramicidin or tyrothricin may be preferable, since usually fewer injections are required and the reaction may be so mild that the cow is out of production for only a short period.

Since the cost of udder medication is an item to be considered, the agents that are most effective in either lactating or dry udders on a single treatment are preferred.

The Chairman: Before treatment is begun, should the veterinarian inform the dairyman as to the future usefulness of the cow as a dairy animal if cured of the existing infection?

Dr. Little: Yes, especially in sections where regulations prohibit the dairyman from maintaining in the milking line cows with slack or blind quarters.

If the udder is fibrotic and the secretion scanty or of a poor quality, the practitioner may be able to predict with a reasonable degree of accuracy whether or not these conditions will be improved following the elimination of the streptococci. It is rather discouraging for a dairyman to pay for a series of treatments on an animal and then have an inspector eliminate the cow from the herd on account of udder induration and scanty secretion.

The Chairman: Should the administration of udder medication be restricted to the veterinary practitioner?

Dr. Little: Yes. Chemotherapy of the udder with any bactericidal agent is a surgical procedure and should be administered by a veterinarian.

If udder medication is carried out in a careless manner it may result in damage to a quarter, even in complete destruction of the secretory tissue. It was recently
reported from England that in a number of cases of tuberculosis of the udder the primary lesions were found at autopsy to have originated in the udder. All these cases occurred in herds under a tuberculosis eradication program, and in which the affected udders had been previously irrigated for the treatment of mastitis infections.

The Chairman: With the bactericidal agents now available for udder therapy, what percentage of cures can be anticipated in the average dairy herd?

Dr. Little: It has been reported that in special herds, maintained under exceptionally strict sanitary conditions, the percentage of cures may be high, possibly 80 to 90 per cent of the affected cases being freed of their infections.

Usually in such herds the advanced cases of the disease will be eliminated as they occur, so the existing infection on the whole will be mild and more receptive to treatment. In the average dairy herd, on the other hand, the percentage of cures may be much lower, a conservative figure being below 50 per cent.

The Chairman: Is it possible to eliminate streptococcus agalactiae infections from a dairy herd by segregation and disposal alone?

Dr. Little: Yes. Although recent reports from England and Australia throw doubt on the possibility of developing and maintaining herds free from chronic mastitis caused by streptococcus agalactiae, the results of such experiments conducted in this country are very promising.

The authors of these reports attribute their failures to three factors:
1—High incidence of infection in first calf heifers;
2—Inability of present laboratory or cultural tests to detect the mildly infected cases;
3—The possibility that there may be other reservoirs for streptococcus agalactiae than the udder, and that this organism may gain entrance to the udder by some route other than the teat canal.

To develop herds free from streptococcus agalactiae infections, all the infected cows in the herd must be identified, and either evacuated from the herd or maintained in a separate unit. The replacements should be free from this infection, and strict sanitary control measures must be adopted for the care of the milking cows and the dry cows. Treatment is merely an adjunct to the economical eradication of this infection.

The Chairman: In the diagnosis of streptococcus agalactiae infections in the udder, what test is the most informative?

Dr. Little: A bacteriological examination of the fore milk (2 to 3 cc.) plated in nutritive horse or ox blood agar, is the most informative and delicate diagnostic test that can be used. The most satisfactory results are obtained when the milk is cultured soon after the samples are collected.

A bacteriological examination, however, is often impractical in large-scale field work, and it is necessary to resort to simpler methods, such as the Hotis test. I consider that this test when properly conducted is more informative than the examination of stained milk films from incubated samples, especially when the negative Hotis samples and the suspicious reactions are filmed.

Some workers who are making their diagnosis from films prepared from incubated samples may take exception to this statement. Both tests are simple to use. I prefer the Hotis test, if the samples are properly collected, because it saves some labor after one is accustomed to reading the reactions correctly.
The Chairman: Should mastitic cows milked by mechanical milkers be stripped out by hand?

Dr. Little: Yes. From the recent reports of Schalm it would seem that mastitis cows on mechanical milking fare better when stripped out by hand. In other words, it is beneficial to strip out the residual milk.

The Chairman: At parturition are first calf heifers frequently infected with streptococcus agalactiae, and if so, how did the infection occur?

Dr. Little: This is a very important question. The success or failure of any mastitis control program depends upon the freedom from disease of the heifers used to replace the older, infected animals. Without question, first calf heifers are occasionally infected at parturition with streptococcus agalactiae. The incidence of these infections is likely to be higher in badly infected herds where little attempt is made to control the disease in mature cows.

It seems logical to believe that first calf heifers are infected through the teat canal, for if infection occurred through the blood stream, or any other common route, the incidence of these infections would probably be much higher than it actually is. Infections in first calf heifers apparently can be reduced or avoided entirely by feeding pasteurized milk from infected herds. If unpasteurized milk from infected herds is used, the calves should be restrained from suckling one another for at least an hour after feeding.

The Chairman: Thank you, Dr. Little.

Before we open the subject for discussion, I wish to present Dr. Case, who will discuss mastitis from the standpoint of the clinician. If, after Dr. Case has concluded, there are any questions, we will open it up for general discussion.

Dr. Case, is the veterinarian of today in a position whereby he can render a highly satisfactory service in the treatment of mastitis in the dairy cow.

Dr. C. H. Case (Akron, Ohio): Yes. Our dairymen today are begging for somebody to help them, and the veterinarian is certainly trained to do this. He should be able to collect samples of milk in test tubes that are sterile, and mark the tube with the number of the cow and the teat from which he collected it, and take it to the laboratory. It doesn't take much work to establish a laboratory in which you can have an incubator, even a homemade one.

Leave the milk in it for twelve hours, take one loop of the milk, make a slide, dry it, stain it, and examine it under oil immersion. It is quick, and any veterinarian can do it. Then you will have a true picture of mastitis.

The Chairman: Can a high percentage of acute forms of mastitis be satisfactorily treated by intra-mammary infusions? I refer to those very acute forms or acute attacks, Dr. Case.

Dr. Case: We do not try to treat the very acute cases with injections into the udder. Our method has been to get a sample of the milk from the quarter, take it back to the laboratory and check it for the bacteria. It includes incubation of the milk, making a slide and finding out. Then we treat that animal at the same time by having the udder bathed with hot epsom salts, and if the cow is carrying a high temperature—which a lot of them do—we give her 800 grains of sulfanilimide in a capsule or in a paper bag, and generally we leave several doses with the owner so he can give one or two doses a day until her temperature is down.
The following trip, on the monthly examination of these herds, if we have streptococci or any other kind of infection, we inject the udder at that time.

The Chairman: Do you feel, Dr. Case, that we have probably saved the lives of a great many cows by large oral doses of sulfanilamide?

Dr. Case: I feel very sure that the reaction you get the next day—the way the cow responds to sulfanilamide—is almost a miracle, the temperature dropping to 102 or 103.

The Chairman: I am glad Dr. Case brought out that point. We don't regard we have disposed of infection or sterilized the udder at all, but we do feel we have saved the lives of a great many cows by giving sulfanilamide by way of the mouth.

Dr. Case: Should mastitis be handled from the standpoint of a herd problem?

Dr. Case: That is right where I come into the picture. I have tried to work out a method whereby I could take care of the herds for the dairymen, using all the methods we know of to protect the cows from spreading mastitis, and get them so they will give good milk, learn which quarters of which cows are infected, and treat them.

In our practice we are examining sixty-four herds every month. We make the rounds and have certain herds every day except Sundays which we examine. We examine the milk from each quarter of those cows with a bromothymol test to see if there is any trouble in those quarters. If there is any reaction we take a sample of their milk to the laboratory and examine it for long or short chain streps, or whatever infection we can find.

On the following trip we are able to treat that quarter for the owner; or, if it is possible, if the animal is located anywhere nearby, we sometimes treat the animal before the next regularly scheduled trip.

In the first ten months of this year we examined 5,252 samples of milk in our hospital, taken from our herds. We have tabulated the results. Out of the 5,252 samples, 59.5 per cent showed leukocytes alone; 22 per cent showed streptococci; 1.2 per cent showed long chain cocci, and 9.2 showed staphylococci, and 9.4 per cent showed tissue cells only.

To help that owner, if we find only leukocytes alone, we write back and tell him that there are leukocytes present. He doesn't have to worry about that cow, but the ones that have strep are the ones whose milk we tell him not to use.

If our report to the owner is that there are only a few bacteria, we tell him to discard the first cup of milk and use the remainder. If the report shows many bacteria, strep or staph, we tell him to discard all the milk from that quarter. If the animal has twenty million bacteria he is not to use that milk except for calf feeding or pig feed or some other way. In that way the owner can eliminate the quarter that is infected, and can get a premium for his milk which he otherwise could not get.

Calves seem to have a good way of getting rid of this milk. They can drink it and grow fat on it.

The Chairman: Dr. Case you regard bromothymol blue as being a fairly efficient method in the hands of a clinician. I wish you would speak on that a little more fully.

Dr. Case: Regarding bromothymol blue, the reason why we use that is because
it is quick; it also has quite an effect upon the owner. If he milks the four tubes, and if you put the bromothymol in the milk and the milk turns blue or green, he says, "Look at that! What is it?" He remembers that quarter and knows which quarter that is, and knows something is wrong. So I appreciate the psychological effect on the owner. He does not want any green milk to drink—he wants milk that is all right.

The Chairman: I have another question, Dr. Case. Should a veterinary practitioner attempt to differentiate between the different forms of mastitis before initiating treatment or medication?

Dr. Case: Absolutely. Find out what you have before you start to treat or inject any treatment into the udder.

With our method of collecting samples and taking them to the laboratory, and then checking them to find whether the animal has streptococci or staphylococci or leucocytes or maybe colon bacillus, we know what to do next and how to treat the animal. If we had gone out in our business and treated every cow that reacted to the bromothymol test, we would have had 59.9 per cent of them with leukocytes only, and we would have had wonderful results when it came to a cure—but it wouldn't have done any real good.

There is one thing about this that I might bring out: In our examinations for leukocytes, the most leukocytes were found in milk samples in June, July and August. In August we had about 550 samples with leukocytes only. That shows flies and mosquitos. The cows are more restless; they injure their udders, and they get leukocytes.

The Chairman: Dr. Case, from time to time we have practitioners telling us that by attempting to give intramammary infusions, especially with those forms of therapy in which oil is a vehicle, that the oil may remain in the udders for a long while and that some men have considerable difficulty in the introduction of this oil up into the gland. It gives them trouble with the syringe, and then we hear that the sulfanilamide crystals may separate themselves and come down to the end of the teat canal, and it may take quite some time before it becomes diffused.

They seem to have a great deal of difficulty in finding methods of satisfactory introduction of the agents. It is a syringe problem. Have you anything that could help at this time?

Dr. Case: Yes. Dr. Boyd, that was the greatest obstacle we ran into. We could examine this milk and find what we had, but how could we get the sulfanilamide and oil into the udder?

I have with me (and you can come up and look at it later) an ordinary alemite gun. I happened to see one of them in one of the stores the other day, which they were selling out. This is the one you start with, a small one. We have one now that is much longer, that will hold more sulfanilamide solution.

The top comes off. We have fixed this with a milking tube so it will fasten on and will not slip off. Five turns of this handle will deliver 10 ccs of sulfanilamide. On our laboratory guns, each revolution delivers 10 ccs. These guns facilitate the administration of sulfanilamide.

In this treatment you must use every possible precaution to keep from carrying other infections into the udder. We clean the teat off with a 4 per cent chlorine
solution in a soda base. We take a piece of cotton to do this, and the skin of the udder will be white and clean. This solution will dissolve all particles. At the last we dip the end of the teat into chlorine. We wipe off the teat tube with chlorine solution before we insert it into the udder.

Going from one cow to another, always wash the teat tube off with chlorine solution. In that way, with the number of cases we have treated, in following up and examining the cows, we haven't had any bad effects. That is a system we have developed, and it is working wonders for us.

The Chairman: A great deal of this mastitis control problem rests with the man in the field who is charged with the actual medication of these animals. Some cows don't take too kindly to the treatment, and one needs to be more or less acrobatic at times to succeed in the introduction of these agents. I might say that after having learned what Dr. Case was using—this alemite gun—I proceeded to buy one. Then we went out to meet with one of our practitioners, to show him how easy it would be to use this pump. In that instance it was sulfanilamide and oil.

To make sure we were going to do it correctly, we put this gun in an autoclave, Dr. Case, and left it in there until we were sure everything was sterile. But when we unwrapped it and started to demonstrate to the practitioner, he was just about as disgusted as the friend of mine who ran through a delousing station in World War I, in which they took his trousers and his belt and put them in an autoclave, and when they came out the belt fell into a dozen pieces. So did the washers in the alemite gun. (Laughter)

Certainly, if you are using this gun, don't put it in an autoclave to sterilize it. I know that now. That practitioner was mildly disgusted with the laboratory men, because we had told him we had the solution to his syringe trouble.

Dr. Case, I interrupted you. I know you have a lot of other points to bring out. I know more and more practitioners are following in the footsteps of Dr. Case and are attacking this from the herd angle.

We know better than anyone else here that these things are time-consuming, and men are so busy today that it just seems impossible to enter into this from a herd standpoint. But once you get the herd under way, it is going to pay dividends—big dividends—and, as Dr. Case says, then we are going to render the dairy industry a greater service. I believe we have sufficient weapons at hand with which to put all the information at work that we have. The information is here, but we haven't put it into use. If we do, then we will do as much to enable our people to reach increased demands for increased milk goals or higher production, and we will do just as much as the men who are advocating improved methods of feeding. We will greatly assist in increasing milk production, which is so greatly needed at this time.

Dr. Case and Dr. Little are here. You have an opportunity to ask questions. You have an opportunity to add to the discussion, so don't be backward. We still have a few minutes. We are told there are no further papers to be presented, so we are not encroaching on another speaker's time. We want you to make the best of this opportunity that is presented to you. We don't often have this opportunity.
DR. GLENN (Illinois): We speak often of the streptococcal infection and how to control it and how to treat it. Occasionally we lose an animal from a streptococcal infection if it isn’t properly handled. But we are passing over this staphylococcal infection, which is really a killer of cows sometimes, despite treatment.

Do you have any new ideas or new wrinkles with regard to the treatment of acute staphylococcal infection in cows, where the animal presents an inflamed udder, a watery solution, and the udder turns purple? You may be called to treat such a condition. The cow has fever and is down and can’t get up.

Do you have any suggestion to make in such a case?

DR. CASE: That is what we call gangrene of the udder.

DR. GLENN: That is staphylococcal mastitis.

DR. CASE: Absolutely, not streptococci. There is one thing that I didn’t mention here, and that is that streptococci grow on the walls of the well of the udder and on the cells. Staphylococci grow in the tissues. Dr. Schaum opened my eyes to that years ago when he sent me some sections of an udder.

There were those numerous staphylococci centers in the tissues that developed. Going over to another point for a minute, the man who tells you he has a cow that laid down on the cold cement, or laid down on the cold ground, and the next morning had her udders swollen, those cows are generally not acute cases—they are acute at that time, but they are old chronic cases of staphylococcal infection in the udder, and they have had an abscess in the gland tissue. It breaks down into the well. In the morning they have thick, watery, yellow-colored milk. I have proved that by getting samples and finding staphylococci.

In gangrene, the staphylococci grow very fast, destroy the circulation, and often in a few hours the animal is dead. I haven’t found anything that could stop that condition. In some of them the udder sloughs and falls right out, some recover but many die.

DR. GLENN: Have you checked any hemograms on streptococcal infection as compared to staphylococcal infection in dairy cows?

DR. CASE: No.

THE CHAIRMAN: Are there any questions? If not, I want to take this opportunity to publicly thank the men who have appeared on the program this afternoon. All of these gentlemen have spent considerable time and effort in preparing the subject material. They have been a great deal of help and assistance, and if it hadn’t been for their help we would not have had this type of session.

I also want to thank the retiring Secretary, Dr. Mark Welsh, because he has given us a great deal of assistance; also our present Secretary, Dr. Hendershott, and Dr. Hendricks, the President of our Association. They have all worked hard to help make this part of the program a success.

I thank our audience for being so attentive and so willing to sit through a rather lengthy discussion.

I will now turn the program back to the President.
REPORT OF COMMITTEE ON POLICY


Your committee on Policy has not received any requests or recommendations from members for any change in Policy of the Association as heretofore established. Your committee recognizes we are living in times of an evolutionary nature and at this time recommends that the Policy of the Association remain status quo.
RETAINING VETERINARIANS IN DISEASE CONTROL

BY R. W. SMITH, D.V.M.

State Veterinarian, Division of Animal Industry, Concord, New Hampshire

History tells us that a hundred years ago human beings followed the wild herds of cattle using them and their products for food, clothing and shelter, and not until man attempted to domesticate these wild cattle, did civilization advance. It has been said that man is his own worst enemy in the spread of communicable diseases, but the lower animal must be voted a close second. As far back as history is recorded in writing, mention is made of some of the diseases which we have since learned to connect with the lower animals. Cattle have been the most indispensable animals of all domesticated creatures, furnishing not only labor, but meat, milk and by-products which include hundreds of manufactured articles such as clothing, shoes, suit cases, handbags etc. Added to this are many important drugs used in both human and veterinary medicine and more than one hundred pharmaceuticals derived from by-products of cattle and other domesticated animals.

The average person has little conception of the magnitude of the cattle industry in all its phases in the United States. It has been carefully estimated that approximately one-half of the average American's annual food intake is from cattle.

When Columbus discovered America, there were no native cattle on the continent. There were, however, millions of American bison which are probably a near relative. History tells us that the American Indians followed the buffalo herds over the plains using the meat for food and their hides for shelter and clothing, but as white man and civilization spread from the east to the west the American buffalo was ruthlessly exterminated. In the August issue of the Readers Digest, it was claimed that at one time there were more than 50,000,000 buffalo in the United States. Soon after Columbus' voyage to America, Spanish cattle were taken to the West Indies and later transported to what now is Mexico. Here the animals flourished to the extent that the cattle became so numerous in South America that there was not a sufficient food supply to maintain them. Large numbers were slaughtered and according to history, approximately one million hides were shipped to Europe from South America alone as early as the year 1796. Up until the time of the Civil War, millions of cattle in this continent were slaughtered for their hides only, but as civilization advanced and human populations began to grow, these wild herds of American cattle, like the buffalo herds, were thinned out to make room for more modern farming. With the advance of agriculture came the necessity for better livestock.

The first importation of cattle from England to this country was in 1624 and as might be expected, these animals were brought to New England where they were later transported to other seaboard states and from there westward to Ohio, Kentucky and other states. The first Guernsey cows were brought from the Guernsey Islands to Boston. From there they were driven over the road to Lake Winnepesaukee, New Hampshire and taken on a boat to an island since known as

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Cow Island. The owner labored under the belief that, inasmuch as this breed was bred and raised on an island in the old country, they would thrive much better in this country if still maintained on an island. To some of you westerners, it will be interesting to know that in 1937 by an act of Legislature the name of Cow Island located in Lake Winnipesaukee, New Hampshire was changed to Guernsey Island, and every Guernsey breeder in the United States was solicited for funds to resurrect the old wind mill on this island. This island is about five miles from my present home in Laconia, New Hampshire. From these early shipments have grown a cattle population and an industry second to none in the world. Records of contagious diseases of cattle of the world certainly do not extend back to their beginning. However, if we read history we find that during the 18th century diseases of domestic animals were rampant in the European countries causing famine and ruin wherever they spread. In the early seventeen hundreds, three million cattle were killed in England because of Rinderpest and at about the same time Germany lost over 30 million from the same disease.

We could go on mentioning several of the contagious and infectious diseases that have been more or less prevalent in this country, but this is not necessary as you are all familiar with them. The one point that I wish to emphasize is that without healthy livestock this, or any other country, could exist only for a short while.

Our Secretary of Agriculture, Claude R. Wickard, in the 1942 Agriculture Year Book had the following to say, “Few persons not engaged in the livestock business realize the number, variety, and seriousness of the diseases and parasites that attack domestic animals or the care and skill necessary to keep them healthy under the conditions of intensive production that prevail in this country. Few outside of those familiar with the situation as a whole realize the extent of the losses caused by diseases, parasites, and insect pests; in the United States they amount to well over $400,000,000 a year in spite of an enviable record in working out and applying control methods. Finally, few except those acquainted with public health problems realize how closely many animal diseases are associated with similar or identical diseases in human beings.”

“There is some satisfaction in knowing that the United States is widely regarded as the safest country in the world in which to conduct stock raising. We have a well-developed veterinary service, and with the protection of quarantines and other safeguards the United States is free from several animal diseases that cause great losses abroad.”

Let me repeat the Secretary's last statement as quoted above, “We have a well-developed veterinary service.” This leads us to review the early history of the veterinary profession in the United States and the part it has played in the disease eradication and control programs in this country.

In Europe, as would be expected, the first veterinary school was instituted in 1761 at Lyons, while in the United States the initiation of systematic veterinary education was the granting of a charter in 1852 by the Legislature of Pennsylvania and the securing of a subscription of $40,000 to serve in the organization of a veterinary school in Philadelphia. No students responded until 1859, when two were secured. From then on the history of our veterinary schools was what one might
call spotty until the 'eighties when the Bureau of Animal Industry was formed to meet a national necessity. The horses, cattle, sheep and hogs on the farms of the United States at that time were valued at more than two and one-half billion dollars. This large industry had been neglected both by national and state governments. Destructive animal diseases were spreading, and unorganized veterinary opposition was not making satisfactory progress in coping with them. Farmers were constantly asking for information and advice which the Department of Agriculture was not in a position to furnish. Our cattle had been denied admission into Great Britain since early in 1879 on the ground that contagious pleuro-pneumonia existed in the United States, and the markets of Continental Europe had been closed to our pork products since 1879 with the explanation that our swine were infected with trichinae.

The extension and improvement of transportation facilities and the rapid development of our export trade in animals and their products from 1870 to 1879 gave an impetus to the live stock industry, and it became essential to find an outlet for the surplus. The closing of foreign markets to our animals and their products aroused considerable indignation among our livestock producers and allied interests, and there was an insistent demand for assistance from the national government. Thus the attitude of foreign governments toward our export trade hastened the action of Congress in establishing the Bureau of Animal Industry.

The new Bureau proceeded immediately to meet the objections of foreign governments. Contagious pleuro-pneumonia was eradicated and a system of microscopic examination of pork for export was established, which resulted in reclaiming much of our export meat trade.

Since its establishment, the Bureau has been called on from time to time to assume many duties varying widely in character, and its responsibilities have been increased, until it has become one of the most important organizations of the national government.

If, as stated above, the extension and improvement of transportation facilities in the early 'eighties gave impetus to the livestock industry, what is to take place after the present great conflict is over? What will be the rehabilitation program in countries where live stock has been slaughtered—in some instances, to the last animal? In the rebuilding of the European countries, the control and eradication of contagious and infectious diseases of domestic animals will play an important part.

Livestock is the foundation of America's agricultural wealth and the veterinarian is its greatest safeguard. Last year in this very room we celebrated the twenty-fifth anniversary of the bovine tuberculosis eradication campaign, a project that no other country, save one, has ever dared to start, to say nothing about completing. While this campaign alone cost approximately $241,000,000, the economic savings because of the clean-up are now returning big dividends on the original investment. Add to this the thousands of humans who have been saved from contracting bovine tuberculosis. With justifiable pride, we may claim this project as one of the greatest achievements in the agricultural history of our country. While we recognize the valuable assistance of all cooperating agencies, to the veterinary profession must go the lion's share of the credit for the achievement. It was the veterinarian who applied the tests, it was the veterinarian who condemned the diseased animals,
and it was the veterinarian who inspected the carcass at time of slaughter to determine whether or not it was fit for human consumption. It has been the veterinarian, under the able leadership of our state regulatory officials and the Bureau of Animal Industry, who has been on the firing line wherever a contagious disease has threatened our live stock industry.

Will the veterinarian be retained in the control of livestock diseases? In this day and age, anything can happen. Many of us in this room never thought that we would see the day when the meat inspection service of our country would be taken from the Bureau of Animal Industry and placed under a lay organization. Politicians choose queer bed-fellows and politicians rather than statesmen seem to be running our Government. In my opinion anything can happen in the readjustment which certainly must come when the present emergency is over. Judging the future by what has taken place in the past, it is my judgment that the veterinarian will always be retained in all disease control programs. We must, however, be alert and forge ahead at a pace that cannot be overtaken, if we expect to retain our place in the sun. The United States Live Stock Sanitary Association, the American Veterinary Medical Association, the several state and county Associations should work together in all matters pertaining to disease control and never forget that old saying "It is the banana that leaves the bunch that gets skun first."

There are a few here who have lived to see the Bureau of Animal Industry, the Veterinary profession, the United States Live Stock Sanitary Association, the American Veterinary Medical Association grow and develop down through the years, and the wonderful record they have made. It is for us of the present and for those who are to follow to see to it that the control and eradication of animal disease remain in the hands of those who are trained for this work. The millions of dollars already spent must be protected by trained men. The veterinarians are those trained men. They have demonstrated their ability to meet an emergency in the past. They will not fail in the future.

The price of continued freedom from a return of the ravishing bovine white plague which has been practically eradicated from the livestock of our country, as well as the pressing need to wage increasing warfare against other dangerous and economic livestock diseases, is constant watchfulness and service. In the spirit of those who have fought and fallen, and who left to us the slogan, "Carry On," we must not fail.

They spoke it bravely, grimly, in times of fear and doubt;  
They spoke it when the tide of faith and hope seemed ebbing out;  
But they buckled on the armor in the troubled days now gone  
And left to us their slogan, "Comrades, carry on."  
"Carry on," when critics scoff and scorn you, "Carry on" and doubt despair.  
"Carry on," you'll win the battle though the burden's hard to bear  
'Twas the slogan that they gave us as they fell beside the way  
And for them and those who follow, we must "Carry on" today.
THE IMPORTANCE OF CONTINUING ACTIVITIES IN TUBERCULOSIS ERADICATION IN LIVESTOCK

By A. E. Wight, D.V.M.

In Charge, Tuberculosis Eradication Division, Bureau of Animal Industry, Agricultural Research Administration, United States Department of Agriculture

It has been thoroughly demonstrated that follow-up work in the control and eradication of tuberculosis in livestock is very important. At the meeting of this Association last year, it was our privilege to listen to a most interesting paper by Dr. John R. Mohler, who was then Chief of the Bureau of Animal Industry, covering many of the activities in connection with the cooperative tuberculosis program during the previous 25 years. That paper rounded out, in a very complete and pleasing manner, the voluminous information that has appeared on this subject during that period.

TUBERCULOSIS IN CATTLE

Owing to the shortage of veterinarians, the volume of tuberculin testing has been considerably reduced. Altogether, about 9,309,000 tuberculin tests were applied to cattle by the cooperating veterinarians during the fiscal year ended June 30, 1943. These cattle were contained in about 563,000 herds, and only 17,167 reactors were disclosed, or 0.18 per cent. This is the lowest degree of infection reported for any year since the work was taken up cooperatively with the various states in 1917. The expenditures, both state and federal, for this work, have, of course, been greatly reduced because of the smaller number of reactors found, as well as on account of the necessary curtailment of tuberculin testing. However, during the last fiscal year, the federal funds expended amounted to about $1,230,000 for operating expenses and indemnity. The combined state, territorial, and county expenditures amounted to about $2,706,000, of which approximately $2,330,000 was for operating expenses and the remainder for indemnity. The average appraisal of the reactors was $135.19, and the average salvage received by the owner, $65.03. The owner also received an average of about $27.50 from the state and $18.75 from the federal government for each head of cattle condemned. At the end of the fiscal year, there were 6,317,670 herds, containing 63,846,496 cattle, under official supervision for the eradication of this disease.

In a pamphlet that is available at this meeting, there is a table showing the number of cattle tested and reactors found since 1917, by fiscal years.

The greater part of the tuberculin testing done during the last fiscal year was conducted under the area plan. It was possible to remoldify practically all the counties due for such action, although some difficulty was encountered on account of lack of manpower and other conditions existing because of the war.

There is considerable interest in accrediting herds of cattle as tuberculosis-free in some of the states, especially where there are quite a number of herds of registered purebred cattle. According to our records, on November 1, 1943, approximately 244,332 herds, containing 3,829,918 cattle, were in this classification.
In order to comply with state requirements for moving cattle interstate, during the last fiscal year approved veterinarians tested 183,373 dairy or breeding cattle, and only 16 reactors were reported.

The reports received from slaughtering houses operating under federal inspection concerning tuberculosis found among cattle on post-mortem examination, continue to be very helpful in discovering centers of infection. It is surprising in some instances to note the severity of the disease in some of these herds. However, the reports from the packinghouses show continued reduction in the incidence of this disease in both cattle and swine. During the last fiscal year, there were slaughtered at these establishments about 11,560,000 cattle, not including those that had previously given a positive reaction to the tuberculin test. Only 5,630 or 0.04 per cent showed any evidence of tuberculosis. You will recall that when the cooperative work was first taken up, the number retained was more than 2 per cent. In 1919, when about the same number of cattle were slaughtered under federal inspection as were in the past year, 176,250 cattle showed some evidence of this disease. Of the 5,630 that were retained for further inspection during the last fiscal year, only 1,440 were considered to be either unfit for food or were allowed to be used for food after the affected parts were removed and the meat thoroughly cooked. This is only 0.012 per cent, compared to 0.38 per cent in 1919.

TUBERCULOSIS IN SWINE

Concerning hogs killed under federal inspection, the situation is somewhat different. Last year, about 56,867,000 hogs were slaughtered at plants operating under federal inspection, and, of this number, about 4,057,000 showed some evidence of tuberculosis upon post-mortem examination. Twenty years ago, the number of hogs slaughtered under federal supervision was about 8,000,000 less, but over 3,000,000 more showed evidence of tuberculosis. The per cent retained last year was about seven, whereas 20 years ago, it was about 14, or twice as high.

AVIAN TUBERCULOSIS

There continues to be considerable tuberculosis of the avian type, which is very readily transmitted to swine, in many localities in the Central and North Central States. Fourteen Bureau veterinarians have been devoting their entire time to this branch of the work in eight states during the last fiscal year. They visited 7,536 farms and observed about 1,312,000 fowls. Tuberculosis infection was found on 1,248 of these farms. They applied the tuberculin test to 323,847 fowls, disclosing 11,355 reactors, or 3.5 per cent.

Publicity on the importance of placing this disease under control and eradicating it as far as possible, has been made available. A poster, prepared by the National Committee on Boys and Girls Club Work, Chicago, Illinois, in cooperation with the United States Department of Agriculture, state agricultural colleges, and the livestock and dairy industry, has been widely distributed and should be very helpful in this connection.

Professor H. R. Smith, Chairman of the National Poultry Advisory Council has, within the last few months, as an aid in the educational program to control and eradicate tuberculosis in poultry and swine, prepared a Certificate of Cooperation,
which is to be awarded to owners of flocks who dispose of the hens at the end of the first laying year. It is intended for flocks producing eggs for food, not purebred hatchery flocks.

JOHNE'S DISEASE

Of the 4,877 cattle tested for Johne's disease in 11 States, 247, or 5.0 per cent, were found to be reactors to either johnin or avian tuberculin. These reactors were condemned, and the owners received state and federal indemnity for them, in addition to the salvage.

Some very valuable work in connection with the study of this disease has been conducted at the Federal and State Regional Animal Disease Research Laboratory at Auburn, Alabama. It is fortunate that provision has been made for this work, and it is hoped it will continue.

As usual, this paper contains some statistical information on tuberculosis eradication in livestock, but, as previously mentioned, a pamphlet is available here containing more statistics on this subject, together with results of work in the control and eradication of bovine brucellosis (Bang's disease).

The employees who have been identified with the tuberculosis eradication project during the last year are to be highly commended, and it is hoped their efforts and good work will continue to be effective in making a still greater reduction in the incidence of both bovine and avian tuberculosis in this country. All the organizations that are cooperating have been very helpful. The United States Live Stock Sanitary Association has, of course, been an important factor in the continuation of the work.
PROGRESS IN THE ERADICATION OF TUBERCULOSIS IN POULTRY AND SWINE

By H. R. Smith

General Manager, National Live Stock Loss Prevention Board

More than a hundred years ago our country was invaded by a foreign enemy. It gained a beachhead, then infiltrated through our lines almost unmolested, to become eventually so great a destroyer of human lives and property that we despaired of its conquest. That enemy was the bovine type of the tubercle bacilli. For years we were unaware of its approach and its method of attack, but finally, when our people became fully informed of the damage, there came the will to combat and a strategy that succeeded beyond our expectations.

All identified with our livestock industry are proud to have had a part in this cooperative effort to crush an enemy that had gained large proportions, but is now at our feet. What were those proportions and to what extent have we succeeded?

The enemy had crossed our continent from east to west, and had spread from breeding herds to market cattle, gaining momentum each year. Federal meat inspection records, which give us a true picture of conditions, show that in 1916, the year before the national campaign of eradication was started, 2.35 per cent of all cattle slaughtered had tubercular lesions as compared with 0.96 per cent in 1908, increasing two-and-one-half times in eight years. If nothing had been done, and if it had continued at the same rate of increase since 1916 as before, today 50 per cent of our cattle would be infected with the disease. But the situation was brought to the attention of legislative bodies and something was done. With adequate appropriations from Congress, state legislatures and county boards, and with an efficient army of veterinarians ably directed by federal and state sanitary officials, so thorough a job of tuberculin testing has been done that by 1943 only 0.048 per cent of all cattle slaughtered under federal inspection showed lesions and were retained for the disease—a reduction of 98 per cent in proportion to number slaughtered. The number of beef carcasses condemned has been reduced from 40,746 in 1917, or 0.44 per cent of total kill to 1,248 or 0.01 per cent in 1943, also a reduction of 98 per cent. In Chicago, each has been reduced 99 per cent.

The fact that retentions and condemnations have decreased at the same rate proves conclusively that tuberculosis in poultry, while it is occasionally transmitted to cattle to cause a sensitization and reaction, does not produce lesions visible under the usual methods of inspection.

HOGS SUSCEPTIBLE TO AVIAN TUBERCULOSIS

On the other hand, hogs are very susceptible to the avian as well as the bovine and human types of the disease. With all cattle in the United States 99.8 per cent free, and with the incidence of the disease relatively low in people, we know that practically all of the tuberculosis in hogs is now of the avian type. Further-
more, the exposure is considerable. Tuberculin tests in the north-central states in recent years disclose that on the average, 50 per cent of the poultry flocks in that area are infected to some degree, and this is where the great bulk of hogs is produced. With poultry having the run of the premises on the average cornbelt farm, the droppings of tuberculous chickens are deposited in the feed troughs or on the soil, to be picked up by other birds and by hogs to spread the disease.

While the percentage of cattle retained for tuberculosis has gone down consistently since 1917, the retentions in hogs from this disease increased from 9.9 per cent in 1917 to 13.5 per cent in 1927. Obviously, the increase in the prevalence of the disease in poultry during that period more than offset the decrease in exposure from cattle. Since 1927, the decrease of the disease in cattle and to some extent in poultry has reduced swine retentions to 7.08 per cent in 1943. Even so, there were 4,030,207 hogs retained for tuberculosis in 1943. On each hog retained there was an average loss of 65 cents at current wholesale values, or a total waste of pork and pork products with a value of approximately $2,600,000 for the year. If we add the loss under state and city inspection, it would approach $4,000,000. The loss to the poultry industry from premature deaths and lowered vitality is still greater.

At one packing plant in the heart of the cornbelt, 10.5 per cent of all hogs slaughtered during the past year were retained for tuberculosis. As there were only seven beef carcasses sterilized and 12 condemned during the entire year, we know that practically all cases were of the avian type. The losses itemized were as follows:

<table>
<thead>
<tr>
<th>Description</th>
<th>Quantity</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>8,602 complete sets of viscera at $1.50 each</td>
<td></td>
<td>$12,903.00</td>
</tr>
<tr>
<td>12,417 single sets of viscera at $1.10 each</td>
<td></td>
<td>13,658.70</td>
</tr>
<tr>
<td>72,606 heads sterilized at 35¢ each</td>
<td></td>
<td>25,412.10</td>
</tr>
<tr>
<td>3,817 heads condemned at 65¢ each</td>
<td></td>
<td>2,481.05</td>
</tr>
<tr>
<td>251 hog carcasses condemned at $33.50</td>
<td></td>
<td>8,408.50</td>
</tr>
<tr>
<td>604 hog carcasses sterilized at $17.50</td>
<td></td>
<td>10,570.00</td>
</tr>
<tr>
<td><strong>Total loss</strong></td>
<td></td>
<td><strong>$73,433.35</strong></td>
</tr>
</tbody>
</table>

While this was a direct loss to the packing company, it was indirectly a loss to producers.

**PRACTICAL METHODS OF ERADICATION**

Why do we permit this waste of 14 million pounds of meat in one year in the United States, when there are practical methods of prevention? The small reduction from 7.9 per cent of all hogs slaughtered under federal inspection in the United States retained for tuberculosis in 1942 to 7.08 per cent in 1943 effected a saving of pork and pork products with a value of $257,667, but this decrease can be made much greater if we intensify our educational program, coupling this with the more general application of the tuberculin test to breeding flocks. It would be impossible and impractical to apply the test to the 90 per cent of the farm flocks producing eggs for food purposes only, but it can be done with the relatively few valuable breeding flocks that should be kept for more than one year of egg production.

Twenty years ago when we found that pullets had a light infection as compared
with older birds, the program adopted by sanitary officials and others of selling all birds at the end of the first laying year, proved both effective and economical. It presupposes cleaning and disinfecting the houses before the pullets, grown in a nearby field on clean soil, are placed therein. In flocks, and in entire communities where such flock management has been followed, the incidence of the disease has been reduced to a minimum in both poultry and swine. In fact, the increase in this practice has been largely responsible for the gradual reduction in losses during recent years. It is a practice of long standing in the East where we have reason to believe that early importations of tuberculous breeding poultry from Europe were just as great as in the Middlewest. However, poultry raising is more commercialized there, and this has been done to increase profits. In the cornbelt, where poultry is secondary to cattle and hogs, many older chickens are kept. Flock owners are frequently unaware that trapnest records, compiled from experiment stations, show that, on the average, hens lay 30 per cent more eggs during the first twelve months of production compared with the second year.

Under date of November 23, 1943, Roy E Jones, Extension Poultryman of the University of Connecticut, Storrs, writes:

"Tuberculosis is a very uncommon disease on Connecticut poultry farms. I have been in this work over 30 years and I think I have encountered only three cases of tuberculosis in poultry. For a number of years, we have recommended disposing of all layers at the end of the first year because of economic reasons. The pullets lay on an average of 50 eggs more per year than hens, and those 50 eggs are the profit."

Under date of November 24, 1943, H. O. Stuart, head of Department of Poultry Husbandry, Rhode Island State College, Kingston, writes:

"I would say that in this state farmers dispose of all birds at the end of the first laying year to the extent of replacing 96 to 98 per cent of the yearlings with pullets. There is no more practical way of eliminating tuberculosis in poultry that I know of. You are correct in your assumption that this has been the general practice in the northeastern states for, lo, these many years; and it is usually the reason we ascribe for the low incidence of tuberculosis in poultry in this area."

Under date of November 23, 1943, J. R. Smith, Professor of Poultry Husbandry, University of Maine, Orono, writes:

"The general practice in Maine of those flocks producing only market eggs is to dispose of all of their old birds, keeping only the pullet flock each year. I am of the opinion that this is the practice in at least 95 per cent of the cases. Some of the breeders do keep a few old birds, but probably not in excess of 10 per cent of the flock."

The same general practice is followed in the west coast states and they have very little tuberculosis in poultry and swine. Why there is so little in the south I am unable to say. No doubt the shorter winters there, permitting poultry to be outdoors in open fields for a longer time, is partly responsible. Only 0.8 per cent of all hogs slaughtered under federal inspection in the State of Georgia were retained for tuberculosis during the fiscal year ended June 30, 1943. As we go farther north, the percentage retained increases. During the past year, 3.3 per
cent of the hogs slaughtered at Kansas City were retained for tuberculosis, 4.3 per cent at all plants under federal inspection in Indiana, 4.5 per cent at St. Louis, 5.0 per cent at Omaha, 5.3 per cent at Cincinnati, and 6.3 per cent at Chicago. In the northern states from Michigan and Ohio through Wisconsin, Minnesota and Iowa, to the Dakotas on the west, the hogs retained for tuberculosis the past year ranged from 10 to 12 per cent of the total kill.

COMPLACENCY

The greatest obstacle to progress in bringing about a still greater decrease in losses from tuberculosis in poultry and swine is complacency on the part of the flock owner. The fact that relatively few infected chickens and hogs show physical symptoms gives the owner a false sense of security. Though hens die occasionally, he is not unduly concerned.

How can we overcome this complacency? In the fall of 1936, four states tuberculin tested a majority of the flocks in one township of each of five counties in Illinois, five in Indiana, four in Iowa and three in Nebraska. At that time, 54 per cent of the 832 flocks tested disclosed one or more reacting birds. A sufficient number of federal and state veterinarians were engaged in this work to complete the township in about two weeks. At the end of this period, a poultry clinic was held in each of these counties at which flock owners were invited to bring in diseased specimens. At these clinics, which were well attended, the records showed the number of flocks tested, the percentage infected, and the percentage of young and old birds reacting. As each of these were typical townships, it gave the flock owners of the county an accurate picture of local conditions which was usually surprising because many of the flocks that disclosed reactors did not show physical symptoms. The results of the testing was given publicity in the local papers, and many flock owners in each county adopted the plan of keeping pullets only, as recommended by those in charge of the work. We attended many of these clinics and helped in the publicity. It is hoped that this type of work can be repeated next fall. Too much time and expense is required for testing all poultry flocks in any one county, but if a relatively small area can be tuberculin tested, it will reveal the true conditions and make all flock owners more anxious to eliminate the disease.

The testing of hatchery flocks in recent years in Michigan, Indiana, Ohio, Nebraska and South Dakota has accomplished much to reduce the infection from pullorum disease as well as tuberculosis in such flocks. This has also improved vigor and livability in baby chicks from such hatcheries, which encourages more farmers to keep pullets only in flocks producing eggs for food purposes as distinguished from breeding flocks. There should be a clear distinction between these.

CERTIFICATE OF COOPERATION

Recently we have had a "Certificate of Cooperation" printed to award to owners who keep pullet flocks. It reads as follows:

CERTIFICATE OF COOPERATION

In the Eradication of Tuberculosis from Poultry and Swine
Decrease in losses from tuberculosis in cattle and hogs
Table showing the number of cattle, exclusive of reactors, slaughtered under Federal
Inspection, the number and percent.age retained, sterilized and condemned for
tuberculosis. Compiled from records of the United States Division of Meat
Inspection
FISCAL

I

PER CENT
STERILIZED
AND CONDEMNED

NUMBER
:ONDEMNED

1917
1920
1927
1930
1933
1936
1939
1941
1942
1943

2,160,899
2,155,913
2,079 ,362
1,535 ,099
1 344,373
1,467,408
1,306,526
1,397,595
1,571,659
1,363,566

93,896
72,551
50,375
17,820
5,181
2,001
921
751
566
454

4.34
3.36
2.42
(1.16
0.39
0.13
0.07
0.05
0.036
0.033

4,757
4,805
1,592
758
287
144
52
18
21
21

0.22
0.22
0.07
0.05
0.02
0.009
0.004
0.001
0.001
0.001

20 ,293
13, 937
12,435
4,388
1,031
538
253
209
163
159

0.94
0.65
0.60
0.29
0.08
0.036
0.019
0.014
0.010
0.011

1.16
0.87
0.67
0.34
0.10
0.045
0.023
0.015
0.011
0.012

1917
1920
1927
1930
1933
1936
1939
1941
1942
1943

9,276,049
9,666,188
9,810,797
8,119,760
7,554,258
10,215,227
9,515,754
10,102,594
11,743,465
11,559,167

195,488
157,016
112,924
61,192
31,971
19,076
10,090
8,029
7:255
5,636

2.10
1.62
1.15
0.75
0.42
0.18
0.10
0.07
0.063
0.041

8,468
8,610
4,342
2,772
1,563
829
403
284
272
198

0.08
0.08
0.04
0.03
0.02
0.008
0.004
0.002
0.002
0.001

40 ,746
30,695
27,413
12,715
6,430
4,027
1,992
1,584
1,593
1,248

0.439
0.31
0.28
0.15
0.08
0.039
0.020
0.015
0.013
0.010

0.51
0.39
0.32
0.18
0.10
0.047
0.024
0.017
0.015
0.011

)

Hogs slaughtered under Federal Inspection, the number and percentage retained,
sterilized and condemned for tuberculosis
Chicago hogs
1917
1920
1927
1930
1933
1936
1939
1941
1942
1943

7,550 ,530
6,772,692
5, 655,764
6, 964,441
6,269,045
3,481,236
4,345,549
5,339,433
5,066,766
5,732,018

16.28
15.04
16.09
12.85
10.70
11.35
8.99
7.94
7.06
6.34

1,229,297
1,018,632
910,463
895,500
671,277
395, 315
390 ,898
424,148
358,154
363,690

51,274
34,825
15,024
13,038
7,478
2,845
2,131
1,928
1,410
1 210
)

0.41
0.51
0.26
0.18
0.12
0.08
0.04
0.036
0.027
0.021

25,791
18,076
14,850
11,073
6,584
2,506
2,186
2.589
2,038
1,950

0.34
0.26
0.26
0.16
0.10
0.07
0.05
0.048
0.040
0.034

0.75
0.77
0.52
0.34
0.22
0.15
0.09
0.084
0.067
0.055

76,807
65,609
59 ,656
42,381
35,680
15,195
13,190
15,317
13,357
13,051

0.19
0.17
0.14
0.09
0.07
0.05
0.034
0.031
0.026
0.022

0.41
0.41
0.31
0.20
0.16
0.10
0.073
0.064
0.054
0.046

United States hogs (all markets)
1917
1920
1927
1930
1933
1936
1939
1941
1942
1943

40,210, 847
38,981,914
42,650,443
46,688,860
45,698,053
28,506,019
38,656,537
48,710,059
50,133,871
56,867 ,080

3,978,168
4,260,719
5,777,708
5,321 ,352
4,820, 152
2 ,925,593
3,418,805
4,014,021
3,991,367
4,030,207

~

9.89
10.92
13.54
11.39
10.54
10.26
8.84
8.24
7.96
7.08

91,543
94,264
73,232
53,783
40,769
16,389
15,160
15,907
14, 447
13,660

246

0.22
0.24
0.17
0.11
0.09
0.05
0.039
0.032
0.028
0.024


ERADICATION OF TUBERCULOSIS

ALL PULLET POULTRY FLOCK

This Certificate is Awarded to........................................for cooperating with National, State and County Agencies to eradicate tuberculosis from poultry and swine by disposing of the entire poultry flock each fall at the end of the first laying year, thereby producing more meat and eggs from feed consumed.

(Signed)........................................
Chairman County Committee

(Signed).......................................
Chairman State Committee

In a number of the north-central states, state and county committees have been organized to undertake more intensive educational campaigns using this certificate as a supplement. The reverse side explains advantages in following this practice as a means of eliminating tuberculosis from poultry and swine, and to increase egg production from feed consumed. This material has also been printed in the form of an illustrated folder for general distribution and each recipient of a certificate is given a supply of these folders to hand to his neighbors as a means of disseminating information in the hope that others will adopt the practice. In the counties where this is being undertaken, the vocational teachers of agriculture, as well as the county agents, are cooperating in having flock owners fill out questionnaires to determine who is eligible to receive the Certificate. While the program has been operating for several years, the use of the Certificate is new, and to date has given encouraging results.

If many more of those engaged in commercial egg production keep pullets only, and the tuberculin test can be applied to the relatively few valuable breeding flocks kept over, there is certain to be a pronounced reduction in poultry and swine losses, and more eggs produced from feed consumed. It is a project that merits aggressive work not only because of the losses in poultry and swine, but because the avian type does occasionally cause cattle to react temporarily, showing no visible lesions on postmortem, and valuable breeding cattle may be slaughtered unnecessarily.

Such a thorough job has been done in the eradication of bovine tuberculosis, it is hoped that this same energy can be given to the avian problem in which, when well understood, producers will cooperate to the fullest extent to complete the conquest of tuberculosis in our livestock industry.

We all regret that our good friend Dr. John R. Mohler who served as Chief of the U. S. Bureau of Animal Industry through the entire cattle campaign, has retired because of ill health. We know that his successor, Dr. A. W. Miller will continue the same policies and that the entire federal staff, which has given such fine leadership in tuberculosis eradication, will continue its cooperation with state livestock sanitary officials to eradicate all tuberculosis from livestock, including poultry, as one means of eliminating waste in the livestock industry.
THE ERADICATION OF BOVINE TUBERCULOSIS AND ITS EFFECT ON THE HUMAN TUBERCULOSIS CONTROL PROGRAM IN MICHIGAN

BY GEORGE A. SHERMAN, M.D.

Director, Bureau of Tuberculosis Control, Michigan Department of Health

The distinguishing feature of the campaign against bovine tuberculosis in the United States has been the development of a program based on scientific knowledge and applied in a practical way, which has been the envy and admiration of the entire world. Tuberculosis control as carried out by official agencies, regardless of whether it is bovine or human disease, depends upon two fundamental influences. The first of these is the extent of the scientific knowledge that is available, and the second, our ability to apply this knowledge to the problem at hand. The control of human tuberculosis in this country has been characterized by a lack of uniformity in the application of the scientific knowledge available. The knowledge that we have had available for the control of human tuberculosis has been entirely adequate for its control if applied. However, as you very well know, it has been applied more here and less there, but never anywhere in full force. Notwithstanding, there was always some gain to the general public health as a result of these efforts. The eradication of bovine tuberculosis, on the other hand, has not suffered from these drawbacks to the same extent. However, the two programs fundamentally have much in common, and the veterinarians of the United States have taught tuberculosis clinicians much, as a result of the work that has gone on during these past 25 years or more.

INFLUENCE OF THE ERADICATION PROGRAM ON TUBERCULOSIS CONTROL

The influence of the tuberculosis eradication program initiated by this Association 26 years ago, fully justifies the tribute awarded it by J. Arthur Myers, M.D., in his book, "Man's Greatest Victory Over Tuberculosis." Possibly this influence is appreciated more clearly when one considers that a great nation like Great Britain believes that a victory of the magnitude that you have made possible and have now accomplished, would be entirely impossible in that country. It would be a waste of time to review before this audience the available known facts to show the part that your program has played in our national effort to eradicate this disease. However, medical literature indicates quite clearly that we are well aware of the contribution that you have made in this respect, and of its continuing influence. Recently the Public Health Reports for October 1, 1943, carried a very interesting analysis of mortality figures for the country as a whole, covering a period of the last forty years. The study shows that since the initiation of your eradication program in 1917, the nonpulmonary forms of tuberculosis have decreased much more rapidly than tuberculosis of the respiratory system. This has been particularly noticeable since 1930. Since that time, we have had a reduction of 35 per cent in the deaths from pulmonary tuberculosis, but during the same time the deaths from other forms
have decreased 54 per cent. Other forms claimed less than four people per hundred thousand in the year 1941. In the State of Michigan a study of our figures indicates that during the past 20 years, deaths from pulmonary tuberculosis have decreased 32 per cent but deaths from other forms of tuberculosis have decreased 54 per cent.

Doctor Carl V. Weller, Professor of Pathology at the University of Michigan and head of the Department, has had an opportunity to review all of the material that has passed through that large laboratory in the last 30 years. He tells me that he has seen far less tuberculous lymphadenitis in the past ten years than in the first twenty years in which he worked in the department.

Doctor Bruce H. Douglas, Commissioner of Health for the city of Detroit, and nationally known as a tuberculosis clinician and administrator, tells me that tuberculosis due to bovine sources has practically disappeared from that city. However, it is of practical interest to know that eternal vigilance is required to maintain this satisfactory state of affairs. During the worst years of the depression, when milk ordinances were evaded by bootleggers, the representative of one of the large clinical pathological laboratories in Detroit tells me that its workers were aware of a noticeable increase in the amount of tuberculous lymphadenitis, presumably of bovine origin, that was submitted to their laboratory for examination. Doctor M. P. Hunt of our State Bureau of Animal Industry told me an interesting experience in this connection which was of great interest to me, but obviously well known to you men. A fairly large herd of cattle supplying milk to Detroit had been negative from the years 1927 to 1937. Following that there was a four-year lapse when no tests were made, and at the time of the next examination, 52 reactors were found in a herd of 79. There have been no further lapses and the herd has remained clean. The significance of this observation is of fundamental importance in your program as well as in ours.

**CHANGING CONCEPTS IN TUBERCULOSIS CONTROL**

The program of tuberculosis control as conceived many years ago, consisted simply of finding the case and providing the necessary hospital care. Our great cry for many years has been for better case-finding methods. During recent years, tremendous advances have taken place in that field. The experiences now taking place in this country as a result of the enormous number of persons who have been X-rayed at the time of their induction into one of the armed services, or at the time of their employment in war industries, have caused us to give serious consideration to possible changes in our program of control as practiced for the past 20 years or more. It is conservatively estimated that in Michigan alone close to 50 per cent of all the people have been X-rayed by one method or another during the past three years. The new cases of tuberculosis discovered as a result of these screening methods are found to be chiefly cases of minimal tuberculosis. The common experience is that 60 to 80 per cent of the new cases found have not gone beyond the minimal stage. By that we mean that cavity has not developed. Surprisingly enough, approximately one half of these new cases show sufficient evidence of spontaneous healing when found, to permit them to continue with their work in industry while under observation, notwithstanding the fact that the armed services have rejected these cases for active service. The result of this enormous case-
finding program now going on is the addition of great numbers of new cases to our tuberculosis registers. These supposedly benign cases must be supervised and examined over a period of years at quite frequent intervals.

Advances in bacteriology and newer methods of detecting the tubercle bacillus have upset seriously our former concepts of the public health dangers associated with this disease. We are now able to demonstrate tubercle bacilli in 50 per cent of our minimal cases. Also we are finding that a great number of our supposedly well controlled cases have positive sputum when studied by these improved methods. The result is that many sound clinicians are now somewhat confused with regard to the possible public health significance of these positive sputum cases found by these methods, in contrast to the sputum that is positive by the simple direct methods. One of the senior workers in this country, Doctor F. M. Pottenger of Monrovia, California, feels that these people are not a serious menace to the community when they come from our better families who are reasonably intelligent. However, the tubercle bacillus is still the tubercle bacillus, and as has been established so well both by your Association in your work and by clinicians in my field, prolonged intimate contact is not necessary to bring about serious infection. We owe much to Doctor J. Arthur Myers for the emphasis that he has given to this important fact in the epidemiology of tuberculosis. I can simply say that we are at that point where we are trying once more to state our objectives in more precise terms.

Tuberculin testing. Tuberculin remains one of the most valuable of our diagnostic procedures. However, changing concepts have also entered into this field. About 10 years ago, workers from the United States Public Health Service, carrying out extensive tuberculin X-ray programs in some of our southern states, found that a perceptible number of people who had obvious evidence of tuberculous infection by X-ray did not react to full strengths of tuberculin. The published observations resulted in a number of conferences, but it was soon the concensus of opinion that these negative reactors were in most all cases well calcified cases. Clinicians were able to demonstrate conclusively that approximately 97 per cent of all patients ill with tuberculosis in hospital reacted to tuberculin. In recent years a commercial house has made available a so-called “tuberculin patch.” Many physicians, not realizing that a positive reaction to tuberculin depends upon the amount of tuberculin given, have placed too much reliance on this percutaneous method of testing. We were able to demonstrate that in a small group of approximately 300 patients ill with tuberculosis in one of our large state sanatoria, approximately 56 patients with positive sputum failed to react to a tuberculin patch. You men would not care to use a screen that was as unreliable as that. However, important, careful work is now going on in a number of clinical centers in this country under the observation of the National Research Council, which will undoubtedly result in substantial additions to our knowledge regarding the value and significance of tuberculin when used as a diagnostic agent. One of the most interesting bits of information in this connection is the study of the tuberculin tests given to nurses, in varying strengths and at fairly frequent intervals during their entire three years in the hospital, and frequently while working with tuberculosis cases.
ERADICATION OF BOVINE TUBERCULOSIS

PROBLEMS INVOLVED IN THE FINAL ERADICATION OF TUBERCULOSIS

The last stretch in the race against tuberculosis in the human family in contrast to that in the bovine family is the most difficult. The mortality rate from tuberculosis in this country was cut in half during the first 20 years of this century and then halved again by 1940. One would suppose that the need for beds would decline with the deaths. Unfortunately, this does not follow. Fifteen years ago Michigan had approximately 3100 deaths a year from this disease. At the present time we experience about 1300 less deaths per year, but we use almost the same number of beds. Recently I analyzed the work in a group of six counties in Michigan with an aggregate population of one million people and an average death rate of 18 or approximately one half of the state rate. These six counties were using 75 beds per one hundred thousand population or four beds per death, and the study indicated that only slight improvement downward had been made during the past decade, so far as beds were concerned. We have been aware for some years that tuberculosis in this country was killing a much larger number of males than females. For instance, the death rate for males for the last three years has been approximately 40 per cent higher than that for females. Also, and of more importance, is the fact that tuberculosis kills the male about 20 years later than it does the female. Another important fact is that almost half of the men who died of tuberculosis were more than 45 years of age. The fact is of great practical importance for the following reasons: first, many of the modern methods of treatment do not apply to a man past 45 for the reason that he is a poor surgical risk, and second, it is frequently much more difficult to persuade a man in this age group to accept sanatorium care promptly.

Education has done much to acquaint both the medical profession and the lay public with the fundamental problems involved in tuberculosis control. However, the oft-repeated statement that tuberculosis is now one of the less important killers: namely, seventh in the causes of death, overlooks the fact that it is still the first cause of death for both men and women up to the age of 35, and that for men tuberculosis remains one of the three leading causes of death up to age 54. For non-whites, tuberculosis is a principal cause of death for both men and women up to the age of 39.

FUTURE TRENDS

One of the few good things that has come out of this war has been the accumulation of a substantial surplus in the treasuries of most states. Michigan is taking concrete steps to improve her present position in the field of tuberculosis control as a result of this large amount of money that will be available for the post-war period. We hope to place sanatoria close to the people that need them. The experience that we have gained from the mass X-raying during the war will be applied to our entire population. Present trends seem to indicate clearly that there will be few counties in any state that will not have the benefit of modern public health organizations capable of carrying on a modern program of supervision and control. Governments are becoming impatient with slipshod methods and practices in this connection. The old slogan that health is purchasable really applies to the field of tuberculosis control.
REPORT OF COMMITTEE ON TUBERCULOSIS


During the past two months, letters were directed to all State Live Stock Sanitary officials, giving them the opportunity to submit recommendations for the control of tuberculosis in livestock.

Replies were received from 32 state officials and the United States Bureau of Animal Industry. Eighteen replies included recommendations, 15 offered no recommendations. Careful consideration has been given to all recommendations received.

Paragraph 17 of the Uniform Methods and Rules for the Establishment and Maintenance of Tuberculosis-free Accredited Herds of Cattle and Modified Accredited Areas makes provision for the continuation of areas in the modified status for six years under certain conditions set forth in part 2, paragraph 17. It is found that tuberculin testing of all the cattle in such areas is prevented under present conditions, one of which is the great shortage of veterinarians.

Under these circumstances, it is recommended that paragraph 17, a and b, part 2, be amended to read as follows:

"Modified Accredited Areas where the incident of infection on the last complete test did not exceed 0.2 of 1 per cent may remain in the modified status for a further period of three years, provided that all infected herds had been quarantined and tested as provided in part 1, paragraph 1. Other herds of cattle in such areas may be retested at the discretion of State and Federal officials."

This recommendation is made as an emergency provision, because of a shortage of veterinarians, transportation, equipment and farm labor, and is not to be construed for a permanent policy. This Committee believes that when economic conditions permit and manpower is available, testing in the areas referred to in this recommendation be resumed, according to the Uniform Methods and Rules adopted by this Association and approved by the Bureau of Animal Industry, United States Department of Agriculture on December 19, 1940.

Part 1, paragraph 7, of the Uniform Methods and Rules reads as follows:

"Calves shall not be fed milk or other dairy products unless such products have been properly pasteurized."

It is recommended that this paragraph be amended to read as follows:

"Calves in accredited herds shall not be fed milk or other dairy products from other herds not fully accredited, or from unknown sources, unless such materials have been properly pasteurized."

TUBERCULOSIS IN POULTRY AND SWINE

Tuberculosis in poultry and swine continues to impose an unnecessary burden on American agriculture. A definite program for control and eradication is needed.

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This is a matter that should be seriously considered. It is recommended that Federal and State officials and other interested agencies create plans, or improve existing plans which will steadily decrease the prevalence of infection.

NO VISIBLE LESIONS

Whereas experience with the tuberculin test has demonstrated that it is a highly efficient method of detecting tuberculosis in animals, it is well known that it is subject to errors which were not serious when the incidence of the disease in this country was high, but are more serious now that the disease is becoming less frequent. The number of no visible lesion cases in proportion to the total number of reactors, is too high. The skin lesion tuberculosis cases which apparently are not tuberculous, but nevertheless, often elicit reactions to tuberculin, present another problem.

It appears to the Committee that the time has come to recognize the fact that apparent reactions to the tuberculin test do not always indicate tuberculosis. We do not know of any practical manner of establishing the spurious nature of such reactions. We believe that these circumstances warrant the placing of greater responsibilities for making the diagnosis on the shoulders of local officials, who should consider not only the reaction obtained, but also the history of the animals and of the herds involved, the autopsy reports on previous reactors and all other factors which, in their judgment, have a bearing on the efficiency of the diagnosis.

POST MORTEM REPORTS AS AN AID IN LOCATING INFECTED HERD

It is recommended that the Food Distribution Administration require all inspectors in charge of federal meat inspection to obtain, in all cases possible, information which will reveal the location of owners of herds from which animals have originated and found tuberculous on post mortem. This information should be forwarded to the Federal Inspector in charge of Tuberculosis Eradication of the state of origin, and to the State Live Stock Sanitary official. State Sanitary official should obtain similar information on animals found tuberculous on post mortem in establishments not operating under Federal inspection, and from practicing Veterinarians. Infected herds so located should be quarantined and retested at required intervals.
REPORT OF THE COMMITTEE ON RESOLUTIONS

WILL J. MILLER, Chairman, Topeka, Kans.; WARD GILTNER, East Lansing, Mich.;
JOSEPH BARBER, Providence, R. I.

WHEREAS: It is recognized that the livestock industry of this country has sustained a great loss through the retirement of Dr. John R. Mohler, Chief of the U. S. Bureau of Animal Industry, since the greater portion of Dr. Mohler's active service, covering a period of more than 46 years, was devoted to the improvement of the livestock industry and especially to the control and eradication of livestock diseases, and

WHEREAS: The Bureau of Animal Industry has been recognized throughout the world as the most efficient organization of its kind, and since Dr. Mohler was largely instrumental in the advancement of the Bureau as a result of peerless leadership and keep administrative ability,

Resolved: That this Association, assembled in regular session, realizes the great loss of this leader in livestock sanitary matters, and be it further resolved, that this resolution be spread on the minutes of this Association and the Secretary be instructed to forward copies to the Hon. Claude R. Wickard, Secretary of the U. S. Department of Agriculture, Washington, D. C., and Dr. John R. Mohler.

WHEREAS: There is much diversity in the rules and regulations of the various states governing the movement of livestock interstate, and

WHEREAS: This condition is the source of much confusion, dissatisfaction and annoyance to everybody concerned in the shipping and handling of livestock moving interstate; therefore, be it

Resolved: That the National Livestock Loss Prevention Board, through its Executive Committee, heartily endorse the efforts being put forth by the United States Livestock Sanitary Association to secure a greater degree of uniformity in the rules and regulations of the various states governing the interstate shipment of livestock.

Be It Further Resolved, in line with the resolution adopted by the National Livestock Loss Prevention Board and other similar communications received by this body; that it is the sense of this organization that the President, as soon as practicable, appoint a small committee to prepare a questionnaire covering interstate regulations on Cattle, Swine, Sheep and Dogs, and forward same to all State Livestock Regulatory officials, in anticipation that a number of states may finally adopt uniform regulations which will result in great benefit to our livestock industry and allied interests.

WHEREAS: Phenothiazine is the only known effective remedy for the removal of nodular worms,

Be It Resolved: That this association commend the United States War Production Board for making available supplies of diphenylamine to supply the essential needs of the states of phenothiazine, and be it further resolved that the United States Livestock Sanitary Association urge the Livestock Sanitary officials of
each state, where nodular worms are prevalent, to organize and foster an effective campaign for the control of nodular disease in sheep.

Whereas: Cattle grubs annually cause a tremendous preventable loss, be it

Resolved: That this association commend the active control programs now in progress in several of the midwestern states and encourage further grub control, especially in cattle feeder producing states.

Resolved: That we extend to Mark Welsh our deep appreciation for his splendid efforts and services rendered the United States Live Stock Sanitary Association as our Secretary and Treasurer the past years; our best wishes for his success in his new field of endeavor.

Resolved: That it is the sense of this association that the best interest of the livestock industry of our country would be served if each state would establish and maintain a diagnostic laboratory under the direct supervision of the state regulatory official of the respective states.

Resolved: That all trucks used for the transportation of animals affected with communicable diseases be cleaned and disinfected under official specification before leaving public Live Stock Auction Sales and other vehicles before transporting other livestock.

Resolved: That the twenty-eight hour law be made applicable to all forms of transportation of livestock.

Resolved: That we express our sincere appreciation to the management of the Hotel LaSalle for the efficient manner in which they have accommodated this convention, and for the many courtesies extended to our members during the time it was in session.

Resolved: That the president and secretary-treasurer of this association be and hereby are authorized to supply copies of the foregoing resolutions to the appropriate persons and by letter direct their attention to these resolutions.
REPORT OF COMMITTEE ON LEGISLATION


Your Committee on Legislation in attempting to collect information concerning new legislation passed in the various states pertaining to livestock sanitary control work wrote to the 48 states and received a report from all but five. Eleven states reported no changes during the past year in their laws or regulations governing livestock sanitary control work.

Generally speaking, very few new laws were passed on this subject, although a number of new regulations have been adopted in the various states. A few additional states have passed laws and regulations aimed at the control of community sales. However, there is still a marked lack of adequate legislation found governing community sales.

It was noted that a law governing disease prevention among domestic animals in one state also authorized the constituted authorities to properly inform livestock breeders and dealers what is required of them in the shipment of livestock into their own state.

Such laws and regulations passed on Bang's disease control work show in most instances the inclusion of the word "vaccination."

Montana passed new legislation governing the testing of cattle for Bang's disease in areas, and it is noted that this law included the word "vaccination." This is one of the most timely laws on the subject that we found in going over the laws of the various states. Note the all inclusiveness of this law in Section 1, which reads, in part, as follows:

"Section 1. Upon receipt of a petition signed by not less than seventy-five per cent (75%) of the livestock owners of the species of animals to be inspected, tested, treated, or vaccinated, and representing not less than fifty per cent (50%) of such species in any township of any county in the State of Montana, petitioning for the area control, treatment, prevention, or eradication of any dangerous disease of livestock within such township, the Montana Livestock Sanitary Board is authorized and empowered to establish such township as a disease-control area and to enforce the inspection, test, treatment, or vaccination of all livestock of the species designated within such township in accordance with the rules and regulations promulgated by the Montana Livestock Sanitary Board for the inspection, eradication, treatment, or vaccination of such livestock and to reimburse the owners of livestock slaughtered by order of the Montana Livestock Sanitary Board or its authorized agent in accordance with the laws of Montana governing the payment of such animal or animals."

Laws and regulations from a number of states indicate that vaccination of cattle against Bang's disease is rapidly assuming importance. In some states the authorities have been forced by their state legislature to back down from their chosen program of test and slaughter only.
One state amended its law providing for the prevention of Bang's disease, giving the director or his agent the right to vaccinate cattle for the purpose of preventing Bang's disease with the approval of the owner. In order to make this workable, this particular state intends to vaccinate any animals that are unbred at the time of the annual tuberculin test. The director states that this will necessitate raising the age limit to approximately 18 to 20 months.

Another state, by regulations, provides for the vaccination of adult cattle in seriously infected herds.

Your Committee, in studying the regulations promulgated by the various states, is convinced that there is great need for clarity and brevity in the regulations of many of the states, and your Committee urges that livestock sanitary boards and commissions endeavor to so write their regulations that they may be easily understood, not only by the livestock sanitary officials of neighboring states, but by any shipper of livestock. This has reference particularly to interstate regulations.

The State of Nebraska passed a very unique law during its last legislative session regulating the distribution and use of live vaccines pathogenic to humans, with particular reference to erysipelas vaccine.

Your Committee again urges all states that have community sales to pass adequate legislation to control in so far as it is humanly possible the spread of transmissible diseases from such sources, and it particularly warns livestock sanitary officials in states where this curse is just beginning to take immediate steps to enforce such supervision before the community sales organize and become too great a pressure group to pass adequate legislation to control them.

Your Committee further urges that although the matter of controlling interstate shipment of biologics has been repeatedly presented to the Association and has just as often been rejected or dismissed that some uniform control measures be adopted and that this subject again be given the most serious consideration.