Mastitis — The Struggle for Understanding

James M. Murphy, V.M.D.

New York State Veterinary College, Ithaca

In 1954 Reid published the results and interpretations of mastitis workers in Pennsylvania. Coming after years of work, this article seriously presented a concept thought to be correct (Board's Dairyman, 99: 941. 1954).

Also in the northeast, New York and Connecticut conduct formal, organized mastitis control programs, which have been in operation for ten or more years. These two are the only such programs in the United States, and, although the concepts of mastitis overlap in important aspects, they still differ to a significant degree. Both concepts were built upon many years of experience, but both were contradicted extensively by the article mentioned above.

At the annual meeting held at Storrs, Connecticut, in 1956 the American Dairy Science Association committee on animal diseases reported that mastitis is the most costly dairy cattle disease not under satisfactory control. A representative of each of three states mentioned above was asked to present his views at a forum, the purpose of which must have been to achieve an understanding of the problem. Yet how can people in general hope to understand when there is little or no understanding among the men spending their lives on the problem?

The American Dairy Science Association has correctly concluded that mastitis is the most costly dairy cattle disease not under satisfactory control. It follows from this that some action should be taken to institute control where none now exists. But what action should be taken? Which of the three states has the proper concept on which to build? Surely such differences must ring warning bells in the heads of intelligent people. Something is wrong when, after all these years of trying to understand the problem, all we have are several concepts that appear to clash with each other.

An analysis of the situation yields only three broad possibilities: (a) mastitis is so amorphous that it cannot be reduced to a single concept, (b) one of the concepts presented by the three states mentioned (or some other not mentioned) represents the proper concept, or (c) all the concepts fall short of the mark because the problem is larger than any single effort so far put forth toward its understanding.

As one who has devoted his entire career to the problem of mastitis, the author contends that the third possibility is the correct one. Furthermore, it seems that the problem can be reduced to a simple outline by which a general understanding can be achieved. The following outline is proposed. It is arranged in eight points. Each succeeding point is dependent on those going before it. In using such an outline it is of the utmost importance to achieve a state of objectivity. The outline and our own experience must be studied in a detached, impersonal way, and understanding should be our objective.

**Point 1**

Much confusion arises from the many terms and classifications used in mastitis. For example, it is often stated that 20 or more kinds of infection have been found to cause mastitis. However, the truth of the matter is that only four of them account for at least 99% of all mastitis. These are streptococcal mastitis due to *Streptococcus agalactiae*; streptococcal mastitis due to other streptococci; staphylococcal mastitis; and bacillary mastitis (including coliform, *Pseudomonas*, etc.). These four are really separate diseases but, since the term mastitis is often used to include all forms of the disease they can be referred to either as forms of mastitis or as particular diseases.

Each of these forms of mastitis can occur in three degrees of severity. There is a nonclinical stage, in which an animal is negative to barn tests; a mild-clinical stage, in which an animal is positive to barn tests; and a severe-clinical stage, in which there is also swelling of the gland and possibly general illnes of the cow.

Both of these deliberate consolidations are presented in the first part of the table, with the additional notation of YES under each of the 12 combinations. This is the first fundamental point: each of the four forms of the disease can appear in each of the three clinical stages. For example, bacillary mastitis can appear as nonclinical bacillary mastitis, as mild-clinical bacillary mastitis, and as severe-clinical bacillary mastitis.

**Point 2**

Recognition of the three clinical stages is within the ability of most people once they have had a little experience. Of course there will be overlapping due to variations in the care used in observing the signs of disease, but, generally, the three clinical stages will not be confused. However, it is only by the use of laboratory testing (culture) of milk samples that the different forms of the disease can be separated, and only by culture can an animal in a non-clinical stage be distinguished from a disease-free individual.

Thus the second fundamental point is that the clinical stages of each form of the disease can not be distinguished from each other without laboratory testing. For example, a case of mild clinical mastitis could be the result of infection with *S. agalactiae*, other streptococci, staphylococci, or bacillary bacteria, but no one
running the barn tests could tell which one was causing the trouble.

Point 3

For reasons that have never been adequately explained, the three clinical stages do not occur with the same frequency with each of the four forms of the disease. For example, *S. agalactiae* mastitis can be nonclinical or mild-clinical but only rarely becomes severe-clinical, whereas bacillary mastitis very often is severe-clinical. This third fundamental point is illustrated in the second part of the table by replacing the YES entries with + marks. The distribution of the + marks in the table is a generalization from the examination of many herds, and, as such, may not represent the actual situation in any one herd at any particular time.

Point 4

Within each form of the disease, the clinical appearance of the disease varies from time to time and from herd to herd. This fundamental point is represented in the second part of the table by \[ \rightarrow \rightarrow \] marks between clinical stages to denote the possible change of the disease back and forth from one clinical stage to another.

At one extreme it is possible, in any given herd, to have practically all infected animals showing mild-clinical or severe-clinical mastitis. At the other extreme, it is possible for a given herd to harbor all forms of the disease but to have them all in the nonclinical stage. Changes toward one extreme or the other can take place within days or weeks in the same herd, leading to what is often called flareups of mastitis. The precise reasons for such changes are not known.

Point 5

The different forms of the disease have different shedding characteristics. These are expressed in the third part of the table according to the duration of the infection itself and according to whether one can expect cultures to be positive when samples are taken at infrequent intervals. For example, the *S. agalactiae* form of the disease is more or less permanent, and cultures made at almost any time are usually positive, whereas in the bacillary form cultures made during the disease are often negative, and it is not uncommon for the disease to disappear completely in a matter of weeks. It is particularly to be kept in mind that the more severe the clinical signs, the more chance there is of getting a negative culture at that time.

Point 6

The *S. agalactiae* form of mastitis, one of the four main diseases of the udder, differs from the other three in the fact that its habitat is limited to the udder. It exists in the environment only a short time. If all udder infections are eliminated, the organism disappears from the environment. This simple fact has been known for years.

In addition, it has been established that the *S. agalactiae* form is peculiarly vulnerable to properly applied antibiotics such as penicillin. Apparently this vulnerability is not just a simple sensitivity to penicillin but involves the ease with which the bacteria are reached by the drug. Most *S. agalactiae* infections are curable, but in eradication any incurable cows must be removed from the herd.

Although these two facts have not been exploited to any great extent in this country, they make the disease eradicable from herds. Eradication is followed by improvement in both the quantity and the quality of the milk produced. It should always be remembered, however, that the eradication of *S. agalactiae* mastitis is the eradication of just one of four main forms of the disease. On the other hand, in spite of many statements to the contrary, there is as yet no acceptable evidence that the eradication of the *S. agalactiae* form leads to a greater incidence of the other forms.

Point 7

Unlike the *S. agalactiae* form of the disease, whose habitat is the udder of the cow, the habitat of the bacteria involved in the other forms of the disease is the environment. These other forms are found in many herds and often cause a great deal of clinical mastitis. Their mode of operation is still unknown, and the complexity of the problem has led some to the false conclusion that they are not actual diseases. However, in the absence of the knowledge we need they will remain uncontrollable in the specific, precise sense in which we can control the *S. agalactiae* form. In some herds and areas where the *S. agalactiae* form of the disease has been greatly reduced or eliminated, these other forms naturally receive greater attention and may constitute a serious, and very frustrating, problem. The knowledge needed for their control will be obtained only from research. At present there is practically no scientific effort being spent in this area, and it will require many dollars to obtain the needed facts.

Point 8

Since the clinical stages of mastitis are relatively easy to observe, in contrast to the difficulties involved in detecting the forms of the disease based on laboratory culture, it is not surprising that when most people talk about mastitis they are talking about the mild-clinical and severe-clinical stages of the disease. They do not realize that, while about one-half of all cows are not diseased at all, many are diseased but are in the nonclinical stages. Thus they erroneously conclude that, if cows are not showing mild or severe signs, they are disease-free, and they do not fully realize the frequency with which shifting can occur between the non-
clinical, the mild-clinical, and the severe-clinical stages.

An overwhelming majority of the modern treatments applied by farmers and veterinarians are administered solely on the basis of the presence of mild-clinical or severe-clinical mastitis. A true cure, based on adequate laboratory examination, is rarely determined, and the success or failure of the treatment is judged on the disappearance of the clinical signs of disease. Since the four forms of the disease can not be distinguished by the clinical signs, it is necessary to include in the treatments various drugs thought to be effective against each of the forms. Often treatments which are thought to be successful when the clinical signs subside have served only to force the disease into the non-clinical stage from which it can, and often does, flare up at some future time.

With so many farmers and veterinarians observing only the clinical aspects in the barn, and with so many different management and environment combinations to be observed, it is not surprising that a complex group of diseases can come to be connected in our minds with almost every conceivable factor of management or environment. Two of the three states mentioned earlier stress at least a dozen such factors, ranging from mud and dust in the barnyard, through the construction of the stalls, the feeding and milking of the cows, to the ventilation of the barn. Nearly everyone seems to overlook the fact that mastitis exists, and that we have a serious problem, even in herds where the management and environment meet sensible standards.

The reasonable conclusion from this is really quite simple: The shifting between the clinical stages is the result of a battle going on between the infection in the udder and the cow. Thus two things must be considered: the ability of the infecting bacteria to injure the udder and the udder’s ability to withstand injury. Naturally, many things happen as a result of management and environment which might upset the balance between the infection and the cow. Treatment is known to be able to tip the balance in favor of the cow, even when the causative infection is not cured. Often deliberate changes in the management and environment aimed at mastitis control are followed by a reduction in the incidence of mild-clinical mastitis and severe-clinical mastitis; the disease is reduced, at least temporarily, to the nonclinical stage. So far, however, there is little or no evidence from controlled experiment to support or reject any of the dozen or more factors, and they must be considered on a trial-and-error basis.

The Laboratory Muddle

Three rather clear lines of action crystallize from the 8-point outline. However, the biggest obstacle to the general understanding of mastitis is the fact that there is no standard method for the laboratory culturing of milk samples. Probably not even two states use exactly the same method, although one state does operate six laboratories on a method standardized within their own organization. Without a standard test, many lines of thinking arise which are based not on differences in the thing being tested, but rather on the twists and side issues raised by the tests themselves. Under such conditions, even good tests can produce much misunderstanding.

Very little can be done without standardization, but who is going to bring about such standardization? So far nothing has been done by any source with authority enough to see the matter through. The Northeastern Mastitis Council, an unofficial group of mastitis workers in the northeastern states, has wrestled with the problem for the last five years, but without real success. It is not difficult to predict that success will not be achieved until some powerful coordinating group accepts the responsibility.

It is not within the scope of this discussion to delve deeply into the matter, but a brief analysis of this situation should help many to understand the muddle in which we find ourselves. A method of laboratory culturing suitable for widespread use should possess three characteristics: It should be one that can be performed without undue difficulty; it should be one that can serve for the diagnosis of all forms of the disease in control work; and it should be one that with little or no modification can be used in research.

At this time it is only the second of these three characteristics (serving for the diagnosis of all forms of the disease) about which we need to be concerned in order to arrive at a preliminary judgment of the method in use in your particular area, and which, therefore, has served as the basis for the understanding existing in each area. Laboratory culturing begins in one of three ways:

(A) Refrigerating the unaltered milk samples until they are cultured in or on a solid culture medium (blood agar) which does not contain chemicals inhibitory to bacteria;

(B) Incubating the unaltered milk samples themselves prior to further examination such as by the use of the microscope or solid culture media; and

(C) Incubating the milk samples with the addition, either to the milk sample or to subsequent culture media or to both, of chemicals deliberately intended to prevent or subdue the growth of “unwanted” bacteria.

Only a method incorporating the procedure involved in A can be used for diagnosis of all forms of the disease regardless of which clinical stage they happen to be in. Methods incorporating the procedure set forth in B excessively magnify all the bacteria present in the milk sample, contaminating as well as infecting, and lead toward the erroneous conclusion that most
cows are infected. (The true situation is that only about one-half of all cows are infected, and practically all clinical mastitis is caused by infection.) Methods incorporating the procedure set forth in C are most numerous and were designed primarily for the detection of \( S. agalactiae \). They magnify the presence of \( S. agalactiae \) while largely preventing the growth of other bacteria, including most of the organisms causing the other three forms of the disease. There are many variations of the C-type, and some (but by no means all) are capable of performing the specific job of detecting \( S. agalactiae \). However, none of them can serve for the diagnosis of all forms of the disease regardless of which clinical stage the disease is in.

Thus a B-type of test shows too much false “infection” and leads to a distortion of the true situation. A C-type of test fails to show how widespread is infection with the forms of mastitis other than that due to \( S. agalactiae \). Only on the basis of an A-type of test can the broad picture shown in the table be obtained. Once a satisfactory standardization of laboratory culturing is achieved, then, by means of an outline such as the 8-point one presented here, we should be able to speak the same language. It is then that three broad lines of action will appear.

First Action

The line of action suggested by point 8 comes first, not because it is most important but because it is the area into which most of our effort now falls. Most of our effort is in this area now because it involves the clinical signs which all can see. Our effort falls here also because proper laboratory culturing is not widely practiced and because drugs now exist by means of which we can attack this area.

When from a barn examination we say that a cow has mastitis, we mean that we see the signs of mild- or severe-clinical mastitis. When a drug manufacturer includes in his mastitis remedy several antibiotics and sulfas, he is doing so, even if he does not realize it, for the simple reason that we are trying to treat the clinical stages of up to four diseases without being able to tell them apart. When a long list of management and environment errors are mentioned to farmers as the cause of mastitis, it is because each one is believed to be related on some farms to either an increase or a decrease in the amount of clinical mastitis.

From the table, and from statements already made, the utter futility of thinking that mastitis can be controlled by the treatment of clinical mastitis only should be obvious. This is merely cutting the tops off the weeds and leaving the roots. On the other hand, it is also perfectly obvious that treatment helps hold the line. However, after about ten years of applying treatments in this way and considering that treatments are about as good as they are going to get, should we not give serious attention to the conclusion of the American Dairy Science Association that mastitis is the most important disease of dairy cattle which is not under satisfactory control?

Such an attack on the widespread use of treatment, and the futility of thinking that treatment alone can ever control the disease, usually is followed by a bombardment with many or all of the management and environment errors. The inference is that, although treatment alone will not control mastitis, the farmer can reach this desired goal if he will correct his errors. Few scientists working on mastitis will deny that there must be something of significance in this inference. However, there is probably less real information in this area of mastitis than in any other, and it would be difficult or impossible to find existing, adequate, scientific proof of the mastitis-causing effect of any of these so-called errors. It is obvious that good management would be good for cows, but the question is what is good management from the standpoint of mastitis? The problem is one of getting into operation, just as soon as possible, properly conducted and scientifically based research into the effects of various management and environment errors, while treatment holds the line.

Second Action

The second line of action is suggested by point 6: The \( S. agalactiae \) form of the disease can be eradicated from herds. Given the proper conditions, it is usually not difficult to eradicate, and its eradication is followed by a significant reduction in mild-clinical mastitis and a significant increase in milk volume. This action is the only precise, scientifically based, mastitis control action that it is within our power to take at present. Proper laboratory culturing of milk is absolutely necessary.

In general, there are three types of misunderstanding of this form of mastitis, and they should be easily corrected. The first stems largely from the use of B-type culturing, in which it is very easy to lose sight of the importance of the \( S. agalactiae \) form of the disease in the great mass of positive cultures shown by such methods. Unsound methods can lead only to unsound conclusions, and the simple correction is to change to an A-type method.

A second misunderstanding arises from the relatively mild nature of the \( S. agalactiae \) form of the disease. Since it is often found on farms in a nonclinical stage and at certain times on some farms may be entirely nonclinical, there is a tendency for people whose knowledge of the disease is superficial to question whether it really is a disease. However, the existence, nature, and importance of this form of mastitis have been adequately established over the years, and only in the face of very strong evi-
<table>
<thead>
<tr>
<th>Disease forms based on laboratory cultures</th>
<th>Clinical stages based on barn observations</th>
<th>Non-clinical</th>
<th>Mild-clinical</th>
<th>Severe-clinical</th>
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<tbody>
<tr>
<td></td>
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<td>Negative to barn tests*</td>
<td>Positive to barn tests only</td>
<td>Also swelling or general illness</td>
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**POINT 1.** Each of the four forms of the disease can appear in each of the clinical stages.

**POINT 2.** Without laboratory cultures the clinical stages of each form cannot be distinguished from one another.

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<th>Yes</th>
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<td>Streptococcal, <em>S. agalactiae</em></td>
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<td>Streptococcal, other</td>
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<td>Staphylococcal</td>
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<td>Yes</td>
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<td>Bacillary</td>
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<td>Yes</td>
<td>Yes</td>
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**POINT 3.** The clinical stages do not occur with the same frequency in each form of the disease.

**POINT 4.** All forms of the disease may fluctuate between the clinical stages, except that severe-clinical mastitis due to *Streptococcus agalactiae* rarely occurs.

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<td>Bacillary</td>
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**POINT 5.** The four forms of the disease have different shedding characteristics.

**POINT 6.** The *Streptococcus agalactiae* form of the disease is the only one that can be eliminated from herds. This action can be economically worthwhile.

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<tr>
<td>BY MEANS OF TREATMENT AND MANAGEMENT THE CLINICAL STAGES MAY BE CURED, OR FORCED TEMPORARILY INTO THE NONCLINICAL STAGE. AT PRESENT IT IS NOT KNOWN PRECISELY WHICH MANAGEMENT PRACTICES ARE OF TRUE VALUE.</td>
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**POINT 7.** The habitat of these bacteria is the environment. It will be a monumental research task to discover their mode of operation. Until then they cannot be eliminated from herds.

**POINT 8.**

By means of treatment and management the clinical stages may be cured, or forced temporarily into the nonclinical stage. At present it is not known precisely which management practices are of true value.

A third, and perhaps most common, misunderstanding tends to minimize the importance of the *S. agalactiae* form of the disease because this organism is not found as the cause of severe-clinical mastitis nearly as often as are staphylococci and bacilli. A thorough study of the outline should make it obvious that an examination of only the severe-clinical mastitis cases of all four forms of the disease is no way to judge the occurrence and true importance of any one of the four diseases. This can be done only by studying all the cows in a herd, using as a basis a suitable A-type of culturing method.

**Third Action**

The third action, suggested by point 7, lies entirely in the field of research. So little is known about the mode of operation of the three forms of the disease other than that due to *S. agalactiae* that it is usually foolish to impose any control measures unless they cost little or nothing to impose, or unless they are imposed under very carefully controlled conditions so that some knowledge will be obtained.

Since the bacteria involved are apparently part of the environment, some investigators maintain that we will never be able to achieve a satisfactory degree of control over them. However, even though we know very little about the other three forms, we know enough to make it virtually certain that they are specific diseases for which specific counteractions could be devised. We have hardly begun to study them by scientific methods and, although it will be a difficult and costly job, it is high time that the work is started.
Conclusion

There are many people in responsible positions today who believe that mastitis can be controlled and that we know enough right now to satisfactorily handle this number one disease of dairy cattle. These same people, quite naturally, feel that research on mastitis is not needed nearly as urgently as is research on some of our other diseases of dairy cattle. It is to be hoped that these people will study the outline for the understanding of mastitis and will see that only in one of the four diseases which comprise mastitis is there sufficient knowledge on which to base a precise, sensible, general control effort. With the other diseases comprising mastitis, information is lacking. Until we make the tremendous effort needed to correct this deficiency, the control of these other forms will remain in the empirical, trial-and-error area supported at best by testimonials.

Biochemical Aspects of Cheese Ripening

W. J. Harper and T. Kristoffersen
Department of Dairy Technology, The Ohio State University, Columbus

The cheese ripening process results from the changes that occur during the storage of cheese that alter it from a bland, hard, rubbery mass to a smooth-bodied and full-flavored product. Cheese ripening is a complex system that involves numerous chemical, physical, and bacteriological changes, which are controlled by altering several environmental factors.

The history of cheese ripening is obscured in antiquity, but for each given cheese variety the curing practices have been handed down from generation to generation. Each cheesemaker knows how to treat his product to obtain a recognized cheese of the variety he desires, although he often is puzzled by the unwanted changes that frequently occur.

Type of Microorganisms Important

The ultimate quality of any given cheese depends upon both careful manufacture and proper ripening. The manufacturing procedure determines the future of the cheese by establishing the proper physical and chemical conditions under which ripening will proceed. The type of microorganisms, either in the milk or starter or added to the cheese; manufacturing methods; the general curing room practices; and the curing temperature and humidity are variable factors that combine to determine the cheese variety and the quality of the cheese within its recognized variety. The variable factors are altered for each specific variety, and often only minor variations differentiate cheese varieties. However, there are general concepts of cheese ripening that are applicable for all varieties. These fundamental concepts will provide a background for later discussion of the interrelationships that exist between cheese making and ripening and will show how the different variable factors involved in ripening are related to each specific variety.

Generally, the cheese ripening process is considered to begin when the cheese is placed in the curing room. However, in the broader sense, cheese ripening actually may be considered to start at the time the milk is drawn from the cow. From the time of milking until the milk is in the cheese vat, a bacterial flora becomes established in the milk. In raw milk cheese all of the organisms originally present may contribute to the ripening process, whereas in pasteurized milk cheese only those surviving the heat treatment will affect the ripening. Milk with an extremely low bacterial count is not always suited for cheesemaking since flavor development in cheese is frequently dependent on the activity of naturally present organisms, even in pasteurized milk cheese. To date, insufficient knowledge of the specific roles of the various microorganisms has prevented the use of bacteria-free milk. On the other hand, improperly handled milk usually contains too many bacteria of the wrong species to make good cheese. Even after pasteurization such milk rarely makes high quality cheese since an improper flora often has initiated undesirable fermentations which may continue into the cheese ripening process.

The bacterial starter cultures are essential to provide in the green curd the physical and chemical properties that will favor desirable changes during ripening. Also, the starter organisms may contribute more directly than is generally realized to the development of desired flavor in the cheese.

Rennet Plays an Important Part

Rennet, which is used in the manufacture of most types of cheese, has as a primary function the coagulation of the casein, but its proteolytic activity continues during the ripening process. Except for the types of rennet con-