

control of agammaglobulinemia in baby calves. Crude fractions of gamma globulin have been tested and toxic levels have been established. It appears unlikely at this point in our research that we can quantitatively inject into a calf sufficient gamma globulin to establish a "normal" level. The injection of 13 grams or more of gamma globulin in a single dose produces symptoms similar to anaphylaxis, and double that dosage results in death in just a few minutes. We are now testing the efficacy of smaller doses of gamma globulin and we are testing both crude and very pure fractions. We

are also testing the efficacy of oral gamma globulin fed to the calf for the first week.

It would appear that the immediate problem in improving the survival rate of agammaglobulinemic calves is to enlarge the immune system. It is hoped that bovine gamma globulin will sufficiently serve this purpose. If it proves to be inadequate, we plan to begin research on the rudimentary active immune system in the day-old calf. In the meantime, the most practical approach is to reduce the disease challenge as much as possible through good sanitation and good management practices.

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## An Epidemiologist Looks at Calf Disease

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The meeting theme, "The Present and Future of Bovine Practice," provides an ideal framework for discussion of an epidemiologist's view of calf diseases. The future belongs to those who prepare for it, but preparation for the future does not require clairvoyance. Facing the future challenge of calf health requires that we acknowledge the next decade will bring momentous changes in the feeding, housing and management of calves. Agriculture will advance far more rapidly than veterinary science and individual veterinarians will be effective advisors in calf health programs only if they can avoid "future shock" and cultivate a mentality for applying the age-old concepts of preventive medicine to increasingly complex situations.

*Epidemiology is that branch of veterinary science which records the distribution of animal disease in populations, attempts to explain the recorded distributions, and uses the knowledge thus obtained to control disease. In short, epidemiology is an approach which says: if we carefully tabulate who gets a disease and when and where they get the disease, we can likely learn why they get the disease and can hopefully control future occurrences.*

In over a decade of applying the epidemiologic

approach to cattle disease, we have observed certain repetitive phenomena which occur in all varieties of cattle diseases, in all aged cattle and under varying management conditions. Three of these "epidemiologic truths" or fundamental concepts of epidemiology are appropriate for discussion of calf diseases.

*Concept No. 1 — The apparent incidence of any disease is directly proportional to the intensity of the case-finding activities.*

The realism that "the harder you look, the more you find" is superbly appropriate to the diagnosis of calfhood diseases like white muscle disease (which becomes prominent when looked for), and salmonellosis which seems to be "on the increase" among calves when bacteriologic studies are intensified.

*Concept No. 2 — The consequences of infection with pathogenic organisms vary from mild inapparent infection to severe, sometimes fatal, disease.*

When an infection spreads through a population, many infected individuals remain healthy yet serve as the immediate source of infection for herdmates, some become sick and survive and others (frequently those with complicating disease) succumb.

**Concept No. 3 — Most disease is of multifactorial etiology.**

Rarely is observed disease the result of an infectious agent acting singularly on a susceptible host. Rather, many factors act in concert to produce disease and determine its outcome. Other speakers have discussed the influences of feeding practices, bacterial and viral infections and colostrally-acquired antibody on neonatal calf diseases. Recent work (1) suggests that prenatal infections (acquired *in utero* by the calf as a result of infection of its susceptible dam) may be decisive factors in neonatal calf diseases by causing debility or through a chronic infection resulting in early demise or a carrier state which is epidemiologically hazardous to herdmates.

If we accept that these concepts are operative in calf diseases, then we must acknowledge that ascertainment of the cause, source and extent of epizootics of calf disease is exceedingly difficult and readily influenced by bias of several kinds.

The epidemiologic approach to calf disease includes the tabulations of the age, sex, breed, feeding practices, vaccination status and multiple management and geographic factors imposed on calves experiencing neonatal disease and their healthy herdmates. In addition, it involves the study of the relationships of the onsets of cases to one another and to time (temporal distribution). In most eastern dairy farming areas, calves are born scattered in time and space. However, when large numbers of newborn calves are assembled and reared in confinement (in veal, dairy-beef or replacement heifer operations) opportunities for the study of distribution of disease with respect to time abound. Large calf rearing operations are an epidemiologist's dream because they provide opportunity for obtaining valuable knowledge from the distribution of diseases in populations. On the other hand, the movement of disease through populations of calves provides the perfect setting for drawing conclusions based on biased empiricism. It is our opinion that most decisions made by calf growers and their veterinarians are so derived.

A large proportion of this biased empiricism is unavoidable and perfectly understandable when one views the temporal distribution of deaths among calves in a veal operation as depicted in Figure 1. Full appreciation of the implications of Figure 1 requires that the reader first study the contrasting epidemiologic patterns portrayed by a point epidemic (2) (Figure 2), and the almost bell-type curve depicted by a propagated outbreak with animal to animal spread (Figure 3).

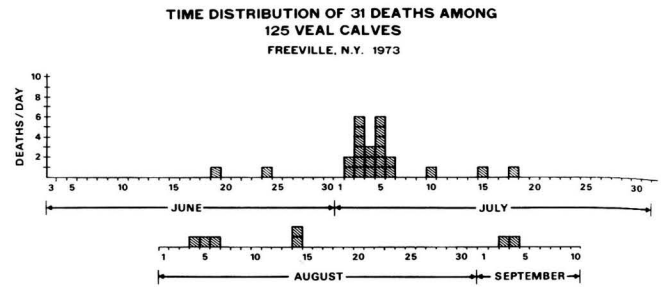


Figure 1. The epidemic curve of the temporal distribution of deaths in a veal calf operation suggests a propagated disease with animal to animal spread.

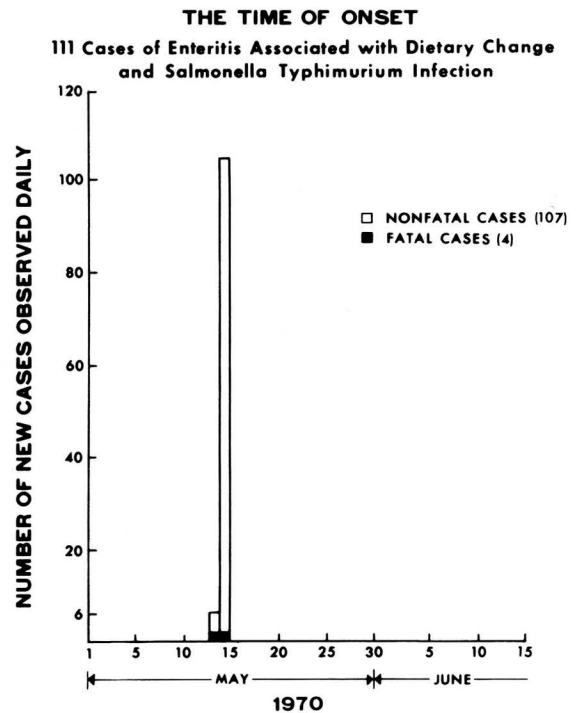


Figure 2. The time distribution of a point epidemic (all cases appearing at the same point in time and space) is highly suggestive of simultaneous exposure of all animals to a common source of infection by means of a common vehicle.

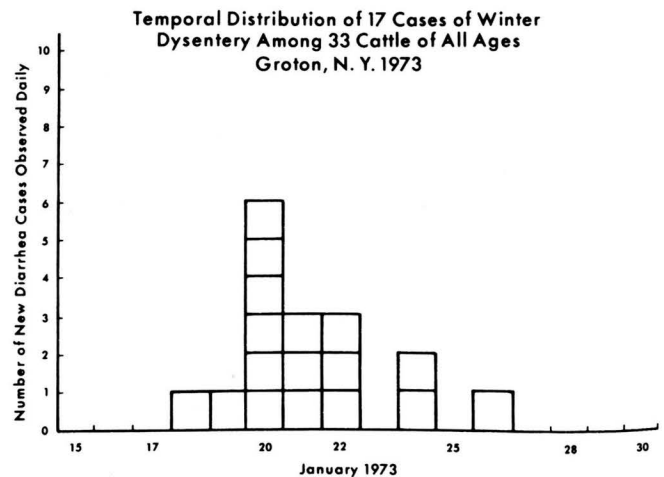


Figure 3. The almost bell-type curve results when animal to animal spread occurs, but can also be produced by variable incubation periods or differing times of exposure to a common source or common vehicle.

A point epidemic, like the one in Figure 2, is inevitably caused by simultaneous exposure to a common source (of infectious or toxic agent) via a common vehicle and usually results from waterborne or foodborne agents suddenly made accessible to a population in doses producing a uniform incubation period.

The almost bell-type curve can result from simultaneous exposure of many animals (with varying incubation periods) but usually such a configuration represents animal to animal spread.

With this background, reexamine Figure 1, depicting the distribution of deaths among 125 veal calves housed in wooden crates and fed all liquid milk replacer diet. Of the fatal cases depicted in Figure 1, 80% had pure cultures of *Salmonella typhimurium* obtained from the ileum at necropsy.

The time distribution of their deaths does not necessarily reflect the time relationship of exposures because of varying incubation periods. Neither does it truly reflect the times of onset of disease (diarrhea and anorexia) because the onset to death intervals vary. Nevertheless, the time distribution of the deaths is a crude reflection of the onset times and mimics the classic curve expected in a propagated epizootic with animal to animal spread. Evaluation of this time distribution explains the hazards inherent in prescribing medication, vaccination or management changes in intensive calf rearing operations. The procedure instituted immediately prior to the day of peak deaths is frequently considered a major contributor to losses (particularly if it is an MLV vaccine) while the procedure instituted at the peak of the curve (particularly if a medicine and sometimes if it is a vaccine) is credited with stopping the epizootic and is recommended as the procedure of choice for such situations. Usually however, in the next similar situation the procedure of choice is applied earlier in the epizootic and the mortality curve continues rising so another (newer) procedure is instituted. When the "newer" procedure coincides with the inevitable decline in the mortality curve it is assigned curative status.

In large calf production units this trial and error research is a continuous process from which emerge certain concepts of disease prevention and therapy. These concepts don't always hold but in the long run many are effective in controlling mortality. The age-old concept of hygiene is reinforced by combining the temporal mortality pattern with our knowledge that salmonellae have a fecal-oral mode of transmission.

Animal to animal spread is suggested by the

curve in Figure 1. Because salmonellae are excreted in the feces of both sick and healthy infected animals, the obvious weak link in the calf salmonellosis chain is to prevent ingestion of feces or licking of objects contaminated with feces. Since calves can't be housebroken and hygienic conditions comparable to human nurseries cannot be achieved, total freedom from exposure to salmonellae cannot be obtained. However, because acute salmonella gastroenteritis frequently involves a dose-response relationship, minimizing exposure by scrupulous hygienic procedures not only makes sense but seems to be substantiated by impressions that those veal growers with the cleanest operations have the lowest mortality from salmonellosis.

Even if it is eventually shown that calf diarrhea associated with salmonella infections is secondary rather than primary disease, and that salmonellosis develops insidiously in a non-pathogenic form or in subpathogenic doses until triggered by other factors, limiting the acquisition of salmonella by strict sanitation reduces the probability of salmonella-associated mortality.

The above discussion reintroduces the concept of multi-factorial etiology. While not evident from within-herd observations and time distribution, it is apparent from many observations that many other factors like ingestion of colostrum, the quality of milk replacer and the age at which grain rations are substituted for milk replacer are other determinants of calf mortality rates. The actual implementation of ideal nutritional, hygienic and management practices to calf health is not always feasible. Some contributors to calf disease are completely beyond the control of the calf grower or his veterinarian.

Thus, the future appears to offer another decade of biased empirical judgements applied to control of calf diseases. The veterinarian who appears at the right time on the epidemic curve is going to look very good and the same veterinarian appearing at a less fortunate point on the curve will appear to be rendering inadequate service.

The only alternative to another decade of biased empiricism is initiation of elaborate, expensive, carefully controlled field investigations of the management, dietary and microbiological influences on calf diseases. With some happy exceptions, it appears as if neither federal or state government, the pharmaceutical industry, the college of veterinary medicine nor the producers themselves are yet willing to pay the necessary price.

## References

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# Nutrition of the Baby Calf

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The successful rearing of baby dairy calves has become a prime target of many dairymen as well as all bovine practitioners because of the increased value of these calves. According to reliable estimates, over 15%, or more than one and one-half million dairy calves are lost annually at an estimated \$150-200 million. As herd size increases, these same surveys show a tendency for these losses to increase. Proper nutrition can be a big factor in reducing these losses.

**When considering the nutritional requirements of the young calf, it is important to consider the fact that the digestive system of the baby calf functions much differently from that of the adult bovine.**

The young calf at birth has a stomach with four parts like the adult animal; however, the various parts are much different in comparative size. In the young calf, the abomasum or fourth stomach, with a capacity of more than twice that of the other compartments, is the only functional portion. Whereas, in the adult animal, the abomasum or true stomach comprises only 8% of the total capacity while the rumen represents 80% of the total.

In the young calf, the liquid diet is shunted by the reticulo-rumen and is directed into the abomasum by flowing through a tube which is formed by closure of the esophageal groove. The groove which extends from the cardia to the reticulo-omasal orifice is closed by a reflex reaction which is stimulated by consumption of liquids. Milk is the best stimulant, glucose and sodium bicarbonate solution usually are effective stimulants also. Water will effect closure of the groove in the very young calf, but become less effective as the calf becomes older. It has been suggested that the reflex stimulation was evoked by sucking.

However, the stimulation can be evoked by drinking from an open bucket. Stimulation of the glossopharyngeal nerve will evoke the closure reflex also.

Before the milk reaches the abomasum, the only enzyme activity is that of salivary lipase. This enzyme works on butterfat releasing butyric acid.

Minutes after ingesting milk, clotting takes place due to the enzyme, rennin. Pepsin activity starts quite early in life also. As early as five minutes after feeding and continuing for three to four hours, whey is released from the clot and passes into the duodenum, followed thereafter by partially digested casein.

The enzyme activity of the intestine is largely that of lactase. This is why in the very young calf the only digestible carbohydrates are glucose and lactose. Until the calf is about seven or eight weeks of age, there is little maltase, amylase, or sucrase activity. Thus, for the young calf, only milk carbohydrates or simple sugars can be utilized.

The age at which ruminal digestion begins is largely dependent upon the diet the calf receives. The longer the calf receives a plentiful milk supply, the less inclined he is to supplement nutrient intake with other food. Veal calves full fed milk will go in to slaughter at 13 to 15 weeks with little or no ruminal development. However, if liquid diet is limited, the calf will start to consume dry food at a few days of age. These dry feeds pass into the rumen where bacteria and protozoa flora begin to develop. The bacteria flora may be started by backflow from the abomasum at feeding while the protozoa flora development is started by the ingestion of roughage, especially hay. When the ration of concentrates to roughage exceeds 1:1, the presence of protozoa declines markedly.