nent facts are that no available compound will reverse the life cycle of the coccidia once it has proceeded beyond the first generation of schizogony and that clinical illness does not develop at least until the second generation of schizogony or later in the life cycle. This means that treatment must be instituted before illness develops or, much better, before exposure even occurs if there is to be a chance of stopping the progression of the disease. Oral sulfonamides may help stop secondary bacterial invaders and fluid therapy will help combat dehydration.

Two compounds, Amprolium and Monensin Sodium, are effective in preventing bovine coccidiosis if calves are started on continous low levels prior to exposure.

Anthelmintic Failure

Occasionally there are reports of anthelmintics failing to remove nematodes against which they previously have been shown to be effective. While drug resistance to phenothiazine, thiabendazole and cambendazole has been reported in nematode parasites of sheep, no cases have been reported in cattle helminths. Many cases in which drug resistance is suspected because of high egg counts or numbers of parasites at necropsy following treatment are actually due to the maturation of inhibited larvae. In several species of nematodes larval stages may be inhibited in their development when large numbers of adult parasites are already present. Removal of the adults with an anthelmintic allows the inhibited larvae, which have been waiting safely in the mucosal glands, to develop quickly to adulthood. This is why repetition of treatment is recommended in two weeks in cases of heavy parasitism.

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Toxicology - Pinpointing the Toxic Agent

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While fundamental to all diagnostic problems the importance of a thorough history and examination of surrounding environment are essential to clinical toxicology. Comparatively few plants or chemicals produce pathognomonic signs or lesions. It seems to be impudent to speak of the approach to anamnesis to bovine practitioners who practice the art daily. But it is appropriate to emphasize the context in which accidental intoxication may occur. It is important to determine not only what chemicals, including drugs and feed additives, are in use but also to determine what substances are stored on the farm, to inspect the storage areas and to determine the method of disposal of unused chemicals and other trash.

The first discovery of aflatoxins was made in tracing back the events that led to massive deaths of turkey poults in England in 1961. The trail led to mold-contaminated peanut meal and had a significant impact on research and knowledge of mycotoxins. The history should not be limited to recent occurrences for it is possible to have intoxication resulting from events that took place months previously. Such events may include: application of pesticides on both crop and non-crop lands, painting of buildings, fences, bridges, pipeline construction, well drilling and perhaps such events that took place on adjacent property.

Human error in preparing or administering chemical substances may not be known but should not be discounted. A classical example of such an error is illustrated by the following report from the New Zealand Vet. J. 19, (March 1971): because of an error in preparation of sodium selenite solution 376 of 557 Aberdeen Angus calves weighing approximately

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300 to 450 pounds died after incorrectly injecting 20 mg of selenium when 2.4 mg was intended.

Physical examination or necropsy may yield clues to the discovery of toxicants. In particular, unusual findings such as blindness, muscle fasciculations, chocolate colored blood, brilliant red blood, pulmonary edema or generalized gastrointestinal hemorrhage may narrow the list of possible toxicants whereas slobbering, anorexia, depression or rumen atony are rather vague signs.

If there is a correlation of the signs or lesions with those known to be associated with chemical substances revealed in the history a possibility of poisoning by that substance may be added to the list of tentative diagnoses. This may or may not be confirmed with laboratory assistance. But certainly do not eliminate lead poisoning as a possible cause of blindness for example, because a source of lead exposure was not discovered. The association of blindness in cattle with lead poisoning occurs with such great regularity that it cannot be discounted.

Even one who is a toxicologist by profession cannot be knowledgeable about the signs and lesions of the thousands of chemicals in use today. Therefore, it should not be expected that a practitioner will have an encyclopedic knowledge of the toxic effects of plants and chemicals. Plant pathologists can assist in identification of non-crop plants. When seeking toxicologic consultation be prepared to give as much specific information as possible concerning chemical composition of the substance in question. Generic chemical names should be used and available information by percentage, weight, or volume should be obtained.

Laboratory aid in diagnosis of poisoning is often misunderstood or at least oversimplified. Laboratory studies should not be limited to toxicologic analyses but should include standard clinical pathology tests to assess organ function. Determination of erythrocyte and plasma cholinesterase activity is useful to confirm a diagnosis of poisoning by phosphate ester insecticides. However, poisoning by anticholinesterase insecticides is an example where one cannot await the results of laboratory studies before instituting treatment. Thus, on an individual animal basis the laboratory results may be used to confirm the diagnosis after treatment is begun and serve as a guide to preventive or therapeutic measures on a herd basis. There are few laboratories, if any, that are capable of identification and quantitation of the wide variety of substances to which cattle may become exposed in toxicologically significant amounts. Except for lead, there are not many analyses that are routinely performed. There are no useful standard screening procedures for chemical identification that are analagous to the drug abuse detection procedures used in man. Unless sufficient information is available in the history and physical or post-mortem diagnosis to suggest one or two specific substances or even chemical classes of substances, it is utterly futile to submit samples of feed, body fluids or tissues to a laboratory for toxicologic analysis. Furthermore, it is easy to "cop out" on a diagnosis by passing the buck to the diagnostic laboratory.

Where intoxication is suspected what specimens should be saved for for the laboratory? These are summarized in the following table.

Serum (clot removed)	10 ml
Whole blood (EDTA or citrate as anticoagulant)	10 ml
Urine	50 - 200 ml
Liver	200 gm
Stomach or rumen contents	1 pint
Perirenal fat (for pesticide analyses)	50 gm

The capabilities and kinds of services of state diagnostic laboratories vary among the states. Where such services are not available private laboratories may be consulted. However, the cost may be based on a sample preparation charge of $30-$50 plus an hourly rate for analytical procedures that are not part of the armamentarium for the laboratory. Other than the use of anticoagulants chemical preservatives should not be used and the samples should be frozen until analysis can be made. Blood is useful for determination of lead and copper or other agents that are slowly excreted. However, those that disappear rapidly from blood may be "hit and run" toxicants that may not be present when the sample is taken. For example, methemoglobin formation and resulting anoxia is a consequence of acute nitrite or nitrate poisoning. Yet extremely small amounts of nitrite are present in blood even after intravenous injection of sodium nitrite for the nitrite is rapidly oxidized to nitrate. The methemoglobin formed is enzymatically reduced to oxyhemoglobin by the erythrocyte. This may occur in the blood collection vial as well as in the animal. The half life for methemoglobin reduction appears to be about two hours, consequently the chance for hit and run intoxication is a problem with acute nitrate intoxication. From studies in sheep the disappearance of nitrate in blood occurred with a half life of approximately five hours. Consequently measurement of serum nitrate should receive additional consideration as a diagnostic aid in nitrite-nitrate intoxication. Normal serum nitrate concentration is less than 20 μg/ml and in the presence of nitrate poisoning serum concentrations of 20-100 μg/ml may be found.

In general the absolute concentrations of substances in urine do not exceed those in blood but urine specimens are of value in analysis for hit and run toxicants where the urine retained in the bladder may contain those substances cleared from blood by renal excretion over a period of several hours. It is possible also to concentrate the substances present in urine by extraction procedures to improve the detectability of chemicals present in low quantities in both blood and urine.

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There are few field tests with proven reliability for on-the-farm analysis. This is not to imply that field tests are of no value but reflects a lack of information and data concerning their usefulness. The diphenylamine test for nitrates will detect high concentration of nitrate (2%) in feed but is not sufficiently sensitive for nitrate concentrations (0.5%) that may lead to chronic nitrate intoxication. The reagent is made by adding 0.5 gm of diphenylamine to 20 ml water and concentrated sulfuric acid is added q.s. to 100 ml. This is a stock solution which is mixed with equal parts of 80% sulfuric acid for use. One drop of the reagent is placed on the cut surface of the plant and a color change from green to blue is indicative of high nitrate content (approximately 2%). Corn suspected of containing aflatoxin may be examined with an ultraviolet light. If aflatoxin is present the split corn kernel will have an area of blue fluorescence around the endosperm layer.

Laboratory procedures that may serve adequately for clinical diagnosis may not necessarily be adequate for evidence in a lawsuit. This should be recognized prior to rendering any opinion to the client concerning a basis for suing for damages or recovery of losses.

The future direction of clinical toxicology is dependent on greater specialization of veterinary toxicologists and subsidized support of university and governmental analytical laboratories to serve in a consultative capacity with the private veterinary practitioner. It is impossible to speculate on the impact of chronic intoxication on animal health because of our limited ability for diagnosis. Since we instituted a modest program of lead analysis in our laboratory there has been a significant increase in the number of diagnoses of chronic lead intoxication in both pet animals and cattle. It is unlikely, however, that laboratory toxicologic diagnosis will ever become as routine as the complete blood count.

Practical Fluid and Electrolyte Therapy and its Pathophysiological Basis

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Introduction

All of us who are involved in bovine medicine and therapeutics are aware of the need for fluid therapy in many disease conditions. Intimately associated with dehydration, toxemia, gastro-intestinal disorders, and the need for fluid therapy are derangements of electrolytes and acid-base balance.

Let me first acknowledge that a whole battery of laboratory tests is impractical and probably impossible in practice. The objective of this presentation is to discuss the pathophysiology of acid-base and electrolyte disorders as they occur in bovine disease. With a basic understanding of pathophysiology, fluid therapy can be approached in a rational manner even without the benefit of laboratory tests. If those of us who can use laboratory determinations in cattle succeed in communicating our findings to those who don't, we will have come one stop closer toward making the contribution to veterinary medicine that I feel we owe.

The bovine species is somewhat unique in that both acidosis and alkalosis are common. Acidosis is a common condition in disease in all species. Alkalosis, however, is rather rare in most species but very common in cattle. This is not to imply that acidosis and alkalosis *per se* are diseases of cattle which need to be treated. Rather, acid-base and electrolyte disorders are manifestations of disease which must be taken into account in designing a regimen of supportive therapy.

Many excellent articles have been written on the subject of practical fluid therapy. Included in many of these papers are various formulations of fluids and electrolytes for use in supportive therapy. Along such lines I really have nothing new to add. After all, the standard toward which each of these formulas strives is a standard upon which none of us can improve: that is plasma itself.

Instead, I would like to suggest that, as a starting point, two different kinds of corrective fluids are required: one for supportive therapy in cases of acidosis and the other for supportive therapy in alkalosis. In acidosis, Eltraad L. A.,* or a formula of similar com-

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