Megascorbic Prophylaxis and Megascorbic Therapy: 
A New Orthomolecular Modality in Veterinary Medicine

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Introduction

The term “Orthomolecular” in the title above may be unfamiliar to some veterinarians. By definition it means “right molecule” and refers to a new branch of medicine developed by Linus Pauling (1968 and 1974). He defines it as follows: “Orthomolecular Medicine is the preservation of health and the treatment of disease by the provision of the optimum molecular constitution of the body, especially the optimum concentration of substances that are normally present in the human body and are required for life.” In general, orthomolecular medicine excludes the use of substances that are not normally present in the body.

In this paper we apply the principles of orthomolecular medicine to veterinary usage and limit our clinical work to one phase - the use of ascorbate or ascorbic acid. This natural substance is normally required and produced in large multigram amounts each day in the mammalian liver. “Ascorbate” is the preferred terminology, because at physiologic pH’s the ascorbate ion is the predominating form. It is an historic error of human medicine and it is improper to refer to this metabolite as “vitamin C” in the physiology of most mammals.

Ascorbate and Comparative Physiology

Ascorbate is absolutely essential in the living process and all living forms either produce it endogenously, get it in their foods or they perish. The enzyme systems for the production of ascorbate in both plants and animals are basically similar and probably had a common origin in early life forms. The natural history of the enzyme system in the evolution of the mammals is not only interesting but essential to an understanding of ascorbate’s clinical effects in therapy (Stone 1965, 1972).

Ascorbate has many functions in vertebrate physiology. One of its important responsibilities has been to maintain biochemical homeostasis under stress. During the course of vertebrate evolution over the past 300 million years, from the amphibians to the reptiles, birds and mammals, there has been an increasing need and production of ascorbate. In the amphibians and reptiles the locus of this enzyme system is in the kidneys and their daily ascorbate ration is produced in this organ. Daily production of ascorbate in these cold-blooded species was size-limited because of the smallness of the kidneys relative to total body size. However, because of their sluggish metabolism they were able to survive on their small daily production. About 165 million years ago when the mammals arrived on the scene with their new fangled, highly active, warm-blooded metabolism, the daily amounts of ascorbate produced in the earlier cold-blooded forms no longer sufficed. This was a problem requiring an immediate solution if the mammals were to survive in that hostile environment. It was neatly accomplished by the transfer of the enzyme system from the small kidneys to the liver, the largest organ in the body. All present day
mammals capable of producing ascorbate, do so in their livers and the kidneys are inactive. Any early mammal that was unable to make this physiological organ transfer died from the effects of scurvy before reaching a reproductive age and thus the kidney producers were wiped out. Table I shows the daily amounts of ascorbate produced in present day reptiles and mammals.

**TABLE I: DAILY PRODUCTION OF ASCORBATE IN ANIMALS**

<table>
<thead>
<tr>
<th>Animal</th>
<th>Ascorbate Production (Milligrams/Kg Body Wgt / per day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Snake</td>
<td>10</td>
</tr>
<tr>
<td>Tortoise</td>
<td>7</td>
</tr>
<tr>
<td>Mouse</td>
<td>275</td>
</tr>
<tr>
<td>Rabbit</td>
<td>226</td>
</tr>
<tr>
<td>Goat</td>
<td>190</td>
</tr>
<tr>
<td>Rat</td>
<td>150</td>
</tr>
<tr>
<td>Dog</td>
<td>40</td>
</tr>
<tr>
<td>Cat</td>
<td>40</td>
</tr>
<tr>
<td>Monkeys, Apes, Man</td>
<td>0</td>
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</tbody>
</table>

**Suboptimal Compensatory Feedback**

Another mechanism that the mammals evolved is a feedback system that increases the liver production of ascorbate with increasing stresses (Subramanian, 1973). This protective mechanism had immense survival value and is still present in living mammals. A rat for instance increases its daily ascorbate synthesis, tenfold, when under stress (Conney, 1961).

The present day birds are living examples of this kidney-to-liver transfer. The older bird species are kidney producers and as we go up the evolutionary scale we find birds producing ascorbate in both their kidneys and livers and finally the most recent birds are solely liver producers. Some of these later birds, like Man, carry a defective gene for the fourth enzyme in this system and thus produce none at all.

For a long time the impression has existed among veterinarians and others that those mammals capable of synthesizing their own ascorbate produce enough each day to fully satisfy their requirements. This is
far from the truth especially during periods of stress. There are large species variations as can be seen in Table I. The mere endogenous production is no assurance that they are producing optimal amounts. Dogs and cats as noted in the table appear to be particularly low producers.

The classical signs of frank clinical scurvy, normally, do not appear in the mammals capable of making their own ascorbic acid. However, under very severe stresses, the signs of scurvy do appear. It was observed nearly 30 years ago that under the severe stresses of Antarctic cold, a heavy work load and without fresh food, husky dogs became listless, readily fatigued and developed swollen, hemorrhagic gums. These scurbutic symptoms readily cleared when fresh ascorbate-containing seal meat became available (Burton, 1972).

Other mammals under less stress may also produce suboptimal levels of ascorbic acid for all their needs and suffer from chronic subclinical biochemical scurvy throughout their lives. This may be due to poor diet or to errors or deficiencies in their enzyme systems for the liver production of ascorbic acid. The enzyme system may also be overwhelmed by the unfavorable biochemical effects of severe stresses or toxins and is unable to produce the ascorbate fast enough and in large enough amounts.

In addition to the possibility that the liver enzymes may not be producing optimal amounts of ascorbic acid in the stressed animal, there is a great deal of biochemical individuality in the optimal requirements for ascorbate in individual animals (Cameron, 1973). Yew (1973) recently showed that individual requirements for ascorbic acid in guinea pigs vary over a very wide range.

**A Subclinical Endemic**

The failure to recognize and differentiate between the premortal signs of frank clinical scurvy and the more insidious, less dramatic, but nonetheless serious, subclinical scurvy has been a stumbling block for the past 40 years in clinical research in both veterinary medicine and in human medicine. About four decades ago, our technology progressed to a point where pure ascorbic acid or sodium ascorbate, for the first time, became available for use in preventive medicine and in therapy. The record to date indicates that the full therapeutic potential of ascorbate has been neglected. The subject has recently been reviewed for human medicine (Stone, 1972).

As a result of nearly a decade of clinical experience in this area, it is our opinion that the incidence and morbidity of chronic subclinical scurvy amongst dogs and cats is much higher than is generally recognized. It amounts to an endemic condition afflicting most of these animals throughout their lives, unless simple steps are taken to correct this genetic condition.

**An Overview**

As far back as 1939 (Sure), it was shown that deficiencies of vitamins A, B1 and riboflavin caused a decrease of ascorbate in rat tissues. The nature of dietary carbohydrate affects the amount of urinary ascorbate excreted by the rat (Fredericks, 1940). Vitamin A deficiencies frequently occur in farm animals and it has been shown that the ascorbic acid levels of the blood plasma of calves can be raised by feeding shark liver oil rich in vitamin A (Linquist, 1943). Poor quality feed adversely affects the ascorbic
Acid levels of the semen of bulls and low blood plasma levels of ascorbic acid of both bulls and cows and is detrimental to their breeding performance (Sutton, 1942). Stallions with low breeding efficiency have lower plasma ascorbic acid values than normal stallions: Good breeding performance is associated with pasturing on fresh vegetation (Davis, 1943). Apparently these animals require “supplementation” of their diet with ascorbate-containing foodstuffs. This also applies to the breeding performance of mares. Strenuous exercise in horses also adversely affects their blood serum ascorbic acid levels (Axt, 1968). Thirty minutes of galloping produces a much lower and more persistently low ascorbic acid levels than 60 minutes of trotting.

Supplementing the ascorbate intake in the stressed animal has beneficial effects. In poultry the site of ascorbic acid syntheses is in the kidneys instead of the liver as in the mammals. Because of the smaller size ratio of the kidneys to total body weight, as compared with the liver, these birds when stressed suffer more from chronic subclinical scurvy because they are chronically producing suboptimal levels of this metabolite. In 1941 (Bell), it was shown that hens on a heavy egg laying program developed scurvy-like muscular weakness in the legs, inanition and depression of egg production. These symptoms were promptly relieved by injections of ascorbic acid. Birds subjected to the stresses of experimental bruising had lower ascorbic acid levels than the unbruised controls. Bruises injected with only 2 milligrams of sodium ascorbate daily healed much faster than the untreated bruises (Hamdy, 1961). Rats, turtles, chickens and guinea pigs experimentally infected with M. tuberculosis strains all showed drops in blood serum ascorbic acid levels (Axt, 1967).

In the mammals, cows of poor breeding performance conceived after injections of ascorbic acid. Ascorbate is intimately associated with early phases of reproduction and ascorbate injections are beneficial in certain types of sterility in the cow (Philips, 1941). Because of the complicated digestive system in the cud-chewing animals, ascorbate administration has to be done by injection; orally administered ascorbate is lost in the long digestive process before absorption.

Canine and feline distemper and other complications rapidly respond to injections of ascorbate as shown by Belfield (1967), Hamdy (1961), and confirmed by Leveque (1969). Edwards (1968) successfully used injections of ascorbate in treating feline rhinotracheitis. These three workers confirmed in animal therapy what has been known in human pathology for the past 25 years from the clinical experiences of Klenner (1949, 1952, 1953, 1971, 1974). Ascorbate when used at the proper high levels is a non-toxic, non-specific, therapeutic virucidal agent (Stone, 1972).

Ascorbate requires recognition as a versatile therapeutic agent when used at levels of a different order of magnitude beyond the trace dosage levels usually associated with its use as a “vitamin.” Ascorbate is not a true mammalian “vitamin” but is rather a liver metabolite produced and needed in large daily amounts to maintain biochemical homeostasis in the face of severe stresses. Progress in this nearly virgin area of Megascorbic Therapy requires as a first step the breakdown of the “vitamin” mental barriers that have arisen in the past 60 years.

Table I shows the daily levels of ascorbate synthesis in the livers of mammals and in the kidneys of two reptiles. All figures are calculated on the basis of milligrams of ascorbate produced per day per kilogram
of body weight of the animal (Chatterjee, 1973). It is seen that in the evolution of the vertebrates, from the reptiles to the mammals, there was a four to forty-fold increase in ascorbate production. It is also important to note that cats and dogs are relatively inefficient producers of ascorbate among the mammals tested. This may be due to their early domestication by Man and also their long term inbreeding which may have affected this genetic trait. It may also explain the very high degree of success obtained in the prevention and therapy of disease with ascorbate in these animals. Compared with other mammals, dogs and cats thus have a higher risk of suffering from chronic subclinical scurvy during their lifetimes. Therefore, this provides a logical rationale for the daily supplementation with at least, about 200 milligrams or more of ascorbate per kilogram of body weight per day to bring the combined endogenous synthesis and oral intake closer to the average for the other four mammals and to take care of mild stresses. This preventive correction of the subclinical scurvy should produce a healthier and longer life span among dogs and cats. The severe stresses of pathology will require higher intakes than these holding doses in order to obtain a good therapeutic and curative effect. Large daily doses of ascorbate can be given because of its very low toxicity and essential freedom from side effects.

Technique and Clinical Data

Having given the evolutionary background and rationale for the use of “mega” doses of ascorbate in veterinary preventive medicine and therapy, we now offer a report of the application of “mega” doses of sodium ascorbate in the successful treatment of a wide spectrum of diseases in dogs and cats. Many of the diseases are those commonly encountered in a small animal veterinary practice while others are not so common and for which no other useful therapy is available.

One of us (W.O.B.) has been using megascorbic veterinary therapy in the viral diseases for the past eight years although the rationale was not too clear in the early treatments. All that was known was that it worked and gave successful results where other treatments failed. We now know that we are merely duplicating and aiding a normal mammalian protective mechanism against disease stress by providing enough ascorbate to maintain biochemical homeostasis. This normally is the function of the mammalian liver but for some reason the liver enzymes do not seem to produce enough ascorbate, fast enough during disease stress in these small mammals. This successful therapy may be regarded as an application of veterinary orthomolecular medicine.

Materials

In our early treatments 100 ml vials of sterile injectable 25% sodium ascorbate were used.* This was satisfactory in most cases, but there were occasional side reactions, especially when very large amounts were used. This product contained 0.1% sodium sulfite as a preservative, which we suspected as being responsible for these side reactions, which resembled the neurological signs of beri-beri. Elimination of this unnecessary preservative from the medication** also eliminated our encountering these side effects. The preservative-free product is the only one to use.

* “Scorbate,” supplied by Burns-Biotec Labs, Inc., Oakland, CA 94621.
** A sulfite-free injectable 25% sodium ascorbate is “Cetane-Injection prepared by Fellows Medical Manufacturing Co., Inc., Anaheim, CA 92806.

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**Administration**

**Intravenous use:**

In all cases where this route is used, the cephalic vein is the one of choice. It is extremely important to realize that if the 25% sodium ascorbate solution is used undiluted, that it is strongly hypertonic and to prevent any adverse reactions, such as weakness or vomiting, it should be administered very slowly, taking approximately at least two minutes for the injection. The ideal strength would be that of an isotonic solution - 3%. For continued administration, we also give multivitamin and mineral preparations. Mega doses of ascorbate have a diuretic effect. Increased diuresis over long periods means increased losses of calcium, magnesium, potassium and the soluble vitamins which should be replaced.

In cases where cats and smaller dogs are being treated intravenously b.i.d., there sometimes is a problem of collapsed veins making it impossible to use this route. By diluting the 25% injectable ascorbate solution with about 2 parts of sterile water to give an approximately 8% solution, the required dosage can be injected subcutaneously. About 30 to 40 minutes are required for the injected ascorbate to be absorbed into the animal’s body.

**Oral Administration:**

Intravenous therapy may be supplemented with powdered sodium ascorbate* given orally. Sodium ascorbate is essentially tasteless and readily mixes and dissolves in the animal’s food, 1/2 to 2 teaspoons (1.5 to 6.0 grams) or more a day.

For the past year it has been our standard practice to give a token supply of powdered sodium ascorbate to the owners of our discharged patients with instructions on how to use it and its source of supply. The daily use of this being and good health [??], increases the mental alertness and improves their resistance to infections and other stresses. In an occasional animal, usually small ones, a tendency toward diarrhea may develop. If this be the case, the dosage is reduced until the digestive tolerance is reached. Diarrhea is not encountered on I.V. administration.

The following dosage schedule for powdered sodium ascorbate is being recommended for our discharged patients as a continuous prophylactic measure:

- toy and miniature breeds - 1/4 to 1/2 teaspoonful daily,
- medium breeds (ca 25 pounds) - 1/2 to 1 teaspoonful daily,
- large breeds (ca 50 to 100 pounds) - 1 to 2 teaspoonsful daily, extra large breeds (over 100 pounds) - 2 to 3 teaspoonsful daily.
Pregnant bitches should be administered the highest level for their size or even more. This should be continued after whelping so the pups receive adequate ascorbate levels in the milk. After birth the pups are given 50 milligrams of ascorbate daily,** for the smaller breeds and 100 milligrams for the larger. At about three weeks, when weaning, the pups should receive 1/4 teaspoon of sodium ascorbate with their food. When fully weaned the pups are put on the above dosage schedule according to their size.

The very good clinical results, thus far obtained, with this preventive medical use of sodium ascorbate has led us to experiment and develop an improved sodium ascorbate preparation containing other necessary vitamins and minerals. This preparation mixes readily with foodstuffs and is an abundant source of these materials for daily administration.

*Powdered sodium ascorbate is available at about a penny per gram from Bronson Pharmaceuticals, 4526 Rinetti Lane, La Canada, CA 91011.

**A convenient product for administering this dosage is Ce-Vi-Sol, Mead Johnson Labs., Evansville, Indiana 47721.

**Analgesia:**

Before discussing our main series of case histories, we would like to describe an effect of ascorbate that apparently has not been reported previously in veterinary medicine, and that is the pain relieving effects of "mega" doses of ascorbate. This effect was reported many years ago by Dr. F.R. Klenner (1949, 1952, 1953, 1971, 1974) in the treatment of severe burns and snakebite in human medicine and recently has been observed in relieving the pain of terminal human cancer (Cameron, 1973).

In hundreds of pectinectomies, performed here, for the relief of the discomfort of hip dysplasia (Burton, 1972), many patients develop at a later date, osteoarthritis which is manifested by severe pain. Upon the daily administration of powdered sodium ascorbate orally, 1/2 teaspoon to 2 teaspoons dissolved in their food, the pain disappears in a few days and the animal again walks normally.

During the winter months, many dogs are brought in limping from neuralgia. The condition has a tendency to rotate from one leg to the other. Sodium ascorbate at 1 gram per pound body weight is administered intravenously and the owner is given a supply of powdered sodium ascorbate with instructions to add it to the animal’s food before serving, 1/2 to 2 teaspoons daily. This regime usually eliminates the lameness in a few days.

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**Case Histories:**

The following case histories are typical of the utility of megascorvic therapy in a variety of small animal diseases. In the interest of conserving space, only one case in each type of pathology will be detailed and the number of similar successful treatments will be noted.

**Viral Diseases:**
The intravenous use of ascorbate is especially valuable in the therapy of the viral diseases as it appears to be an effective, non-specific, non-toxic virucidal agent. We have not seen any viral disease that did not respond to this treatment. Successful therapy appears to depend on using it in sufficiently large doses.

**Distemper:**

A 4-month-old, 25 pound Doberman Pinscher, non-immunized against distemper, and with a history of being impounded in the local humane society facility a week earlier, was brought in. Its temperature was 104° F, with mucopurulent ocular and nasal discharge, bronchitis, anorexia, listlessness and hard pads - diagnosis canine distemper. 12.5 grams of sodium ascorbate was administered intravenously at 10 A.M. At 10:30 A.M., the temperature was 102.5° F and continued steady at this level. A second injection was given at 5 P.M. At 8:30 A.M. the next morning, there was great improvement, a normal ration of food was eaten, general activity noticeably improved, ocular and nasal discharges slightly diminished, temperature 101.5° F. The two injections were repeated leading to marked improvement on the third day. Temperature 101.5° F, bronchitis and coughing diminished by half, digital pads not quite as hard as on admittance and only very slight trace of ocular and nasal discharges. The patient was lively, playful and barking, appetite excellent, eating voraciously. The fourth day, temperature 101.3° F, no nasal or ocular discharges, digital pads almost normal, excellent appetite and extreme activity and the I.V. sodium ascorbate medication repeated. The fifth day the patient was released and thereafter kept on 1 teaspoonful of sodium ascorbate added to its food daily. One month later, the patient returned for a distemper immunization and upon examination was found normal in all respects. This case is typical of results obtainable with I.V. megascorbin therapy and in the past eight years we have had about 150 cases.

It is our belief that in communicable viral diseases, the virus is incapacitated within a few minutes after the intravenous administration of a sufficiently large dose of ascorbate. We have exposed unvaccinated canines to and in continued close contacts with ascorbate injected distemper patients shortly after their initial ascorbate injection and we have never experienced any of the control dogs acquiring the disease.

**London Flu:**

In the winter of 1973, during the “London Flu” epidemic, a client brought in a 15 pound, 2-year-old poodle. She had taken the dog to an emergency clinic during our absence on holiday and was told that a heart murmur had been detected. The history comprised violent vomiting, listlessness, anorexia, 103° F temperature and prescription of 1/4 gram phenobarbital. The dog was brought in for examination the next day after administration of the phenobarbital. There was no vomiting, temperature 102.5° F, anorexia, listlessness and a slight ventricular murmur. The owner was asked if anyone in their household had the “Flu” and it was disclosed that the entire household had it for the last week or ten days. This information plus the patient’s symptoms, including the cardiac murmur helped in making my diagnosis of London influenza. In human medicine, this virus has adverse effects on cardiac muscle.

One gram per pound of body weight, per day, in divided doses, of sodium ascorbate as a 25% sterile solution, was administered intravenously. The client refused hospitalization for the patient but agreed
to return the next day. Sodium ascorbate was dispensed to the client and told to use 1 teaspoon daily in the dog food. The following afternoon, the patient was returned; temperature 101.5° F, eating subnormally but activity slightly improved. The I.V. ascorbate was repeated. The third day, the client phoned and said the dog was back to normal. The following week the heart was ausculated and was normal - no murmur. About 250 similar “Flu” cases have been treated this way in the past eight years.

**Rhinotracheitis:**

A 2-year-old, 8 pound, male Siamese cat was presented with classical symptoms for rhinotracheitis; 103° F temperature, profuse lacrymation, sneezing, anorexia, listlessness and laryngitis. At 4 P.M., 1 gram of sodium ascorbate per pound body weight per day in 2 doses was administered intravenously. The following morning; temperature 101.5° F, sneezing greatly diminished, appetite returned, laryngitis greatly improved and increased activity. After the second and third ascorbate injections (4 grams each) the patient was released. The client was given a supply of sodium ascorbate powder with instructions to use 1/2-1 teaspoon each day in the cat food. In the past eight years, we have similarly successfully treated about 100 cases.

**Paralytic nictitating membranes:**

Two, 2-year-old, male felines weighing 7 pounds each, were brought in with paralytic nictitating membranes. Examination showed they were otherwise normal or as expressed by the client, “normal except their third eyelids are at half-mast.” Each animal was given an injection of sodium ascorbate, 0.5 grams per pound body weight and released. Three hours later the client phoned to report that the nictitating membranes had returned to normal. In the past six months we have had six cases of this eye condition which have responded similarly well.

**Acute bronchitis:**

A 1-year-old, 15 pound active male miniature poodle with a long history of coughing, mainly in morning or evening or when excited. Temperature 101.5° F, appetite fair - diagnosis - acute bronchitis. Patient hospitalized (isolation) and sodium ascorbate was administered intravenously, 1 gram per pound of body weight per day in divided doses. In 72 hours the cough was suppressed. Chronic bronchitis appears to be not as quickly responsive to megascorbic injection therapy as some other conditions at this dosage level. Possibly higher levels or longer oral therapy with sodium ascorbate at 1 gram per pound body weight B.I.D. or T.I.D., may be better.

**Jaundice:**

A 5-year-old, 75 pound female German Shepherd was admitted with a very deep jaundice condition, being extremely weak, unable to walk, eat or drink. The prognosis is fatal according to all texts. One gram per pound of body weight of sodium ascorbate per day in divided doses was administered intravenously. The following morning, the animal began eating, drinking and was mobile. The injections continued for three days after which the animal was released with the client instructed to continue 1 teaspoon of sodium ascorbate in its food daily. It is likely that the known strong detoxicating properties
of ascorbate combatted the toxic effects of the bile and permitted repair of liver damage. The use of ascorbate in hepatitis has been reported in human medicine (Stone, 1972).

**Pregnancy and Whelping:**

In human medicine, the use of mega doses of ascorbate in pregnancy was pioneered by Dr. F.R. Klenner. In over 300 cases he obtained excellent results in both the health of the mother and the babies (Stone, 1972). He delivered and supervised the care of the Fultz quadruplets, the only ones to survive in the Southeastern United States.

Pregnancy is a severe biochemical stress that unduly taxes the liver enzyme system for ascorbate production. It is likely, in most cases, that inadequate amounts of daily ascorbate are present and the pregnant animal would benefit from abundant supplementation.

It has been our general practice to administer sodium ascorbate, 1-2 teaspoons a day in the food as soon after breeding as possible. This has resulted in uneventful pregnancies and whelping. A noticeable effect was a decrease in whelping time by about 50%, and very robust puppies, with no abnormally small and weak siblings. All the litters were free of any congenital abnormalities. It is suggested that additional clinical data be obtained in this important area of congenital defects because if mega doses of ascorbate are effective in their prevention, it would be especially valuable for breeders and the only available regime for doing this.

**Allergies:**

A 1-year-old, 15 pound mixed terrier female was presented with a history of sneezing and increased lacrymation. These symptoms only appeared when the patient was in the client’s backyard. Being springtime, it was felt that pollen or a similar allergen was causing the condition. Ten mg prednisolone, I.M., was administered and tablets were dispensed, 5 mg twice daily, given orally. One week later, and the condition had not improved. The patient was then given 10 mg pyralamine maleate twice daily for five days with no improvement and the patient becoming lethargic. The patient was readmitted and 5 grams of sodium ascorbate was administered I.V. and sodium ascorbate crystals dispensed, 1-2 grams in the food daily. The following day, the client telephoned stating that the patient was free of all symptoms.

Ascorbate is the natural antihistamine which has been used by the mammals for millions of years to detoxify the effects of histamine. It is part of the mammalian feedback mechanism which is utilized to increase ascorbate production in the liver when the animal is under stress (Subramanian, 1973). Apparently in most canines and felines under physiological histamine stress, the enzymatic response is not fast enough or sufficient enough to produce the required ascorbate, therefore, outside megascorobic supplementation produces these very beneficial results in these particular animals. We are merely helping the cat or dog in a natural physiological response in which it is not fully competent to complete by itself.

**Dermatitis:**
A German Shepherd breeder had a litter of eight pups, eight weeks old, with infantile pyoderma on the face and paws. Previously this condition had been treated with the more common antibiotics such as tetracycline, ampicillin with slow results. This litter was administered tetracycline orally, 100 mg, three times daily, and 1-2 grams of sodium ascorbate incorporated into the food daily. Within one week the incrusted areas showed vast improvement and the condition cleared in 12 days. Sodium ascorbate has also enhanced the healing in moist eczemas, parasitic dermatitis and sarcoptic mange.

**Respiratory Conditions:**

A 12-week-old, 15 pound Shepherd was admitted with 105° F temperature dyspnea, anorexia, diarrhea and listlessness. A diagnosis of pleurisy was made and the patient was given intravenously 7.5 grams of sodium ascorbate. this repeated in eight hours. The following morning, the patient was eating and was noticeably more active, temperature 103.2° F. Two more similar injections were given this day. The morning of the third day found the dyspnea greatly improved, temperature 102.5° F and the patient resented being caged, barking and pawing at the cage door. The injections were again repeated and the patient released on the fourth day with a supply of sodium ascorbate to be administered in food - 2 grams daily.

**Epilepsy:**

A 4-year-old, 20 pound, mixed male terrier had a history of epileptic seizures several times a month while on heavy doses of Dilantin. Two grams of sodium ascorbate were incorporated in the daily food given the dog. While on the ascorbate and Dilantin, no seizures occurred for a period of two months. When the ascorbate was discontinued, the monthly epileptic seizures returned. Since the ascorbate administration was resumed with Dilantin, the dog has been free of seizures up to the present time (about four months).

**Urinary Tract:**

When large doses of sodium ascorbate or ascorbic acid are administered and the ascorbate blood levels exceed the kidney threshold, the mammalian kidney is quite efficient in removing the ascorbate from the blood and excreting the excess into the urine. The ascorbate passage through the kidney appears necessary for the physiological homeostasis and efficiency of the kidney. High levels of ascorbate in the urine tend to lower its pH and endow it with antibacterial, antiviral, anticancer and healing qualities. High levels of ascorbate intake, because of this blood to urine transfer should be very beneficial in the prevention and treatment of pathology of the urinary tract. Excretion of high levels of ascorbate in the urine is not “wasted” as many authors in human medicine contend.

A typical case is that of a 3-year-old female miniature poodle with a history of recurring cystitis. Previously she had been treated with urinary sulfas and antibiotics only to have the condition recur in a few weeks. The continued administration of 1 gram of ascorbic acid daily has resulted in a complete remission of the cystitis. This has gone on for 18 months.
Ascorbate administration is also useful in urolithiasis. A 4-year-old male cat with recurring urinary calculi was admitted, being unable to urinate. The bladder was emptied with a tom cat catheter. The following morning, the patient was still voiding urine and was released. The client was given a supply of sodium ascorbate and instructions to administer 1-2 grams in the food daily. It has been nine months since the start of this regime and there have been no recurring calculi. It is of interest to note that, in human medicine, as far back as 1946, stone formation has been attributed to a lack of ascorbate (McCormick, 1946).

**Spinal Degeneration:**

This condition is seen with some degree of frequency at this hospital. Some years ago, a 15 pound male, terrier cross was first presented with a case of spinal degeneration. There was a history of anorexia, slight to moderate ataxia and temperature of 105°F. There was also noticeable intense pain in the lumbar area manifested by arching of the vertebral column in the animal’s attempt at relief. Penicillin and streptomycin were administered, 400,000 units I.M., b.i.d. and Dyporone, 3cc I.M. No favorable response was noted in 12 hours. The patient was referred to the local veterinary school that confirmed the diagnosis of degenerative changes of the spinal cord. The dog was euthanized because there was no known successful therapy at the time.

Later a 10-year-old, 80 pound male Labrador retriever was presented with a similar history of anorexia, loss of weight, slight ataxia, 103.5°F temperature and general listlessness. From similar previous cases that had been diagnosed at the veterinary school, using all available laboratory facilities, the diagnosis of spinal degeneration was made. Penicillin with streptomycin, 800,000 units b.i.d. for 48 hours and oral tetracycline, 250 mg every 8 hours, for 48 hours gave no change in the symptoms. Sodium ascorbate solution at 1 gram per pound of body weight was then injected intravenously, b.i.d. After 16 hours, the temperature returned to normal, 102°F, appetite improved and patient’s activity increased, but the ataxia persisted. The injections of ascorbate were continued b.i.d. for three days at which time the ataxia improved. The patient was then put on oral sodium ascorbate, 60 grams daily in his food. The improvement was progressive and gradual; within three months the patient was normal (no ataxia). Apparently this megascorobic regime aided spinal nerve regeneration.

The client was so impressed that he purchases the sodium ascorbate in kilogram quantities and plans to administer it for the remainder of the dog’s life. This is but one of some 10 cases successfully treated in like manner.

**Ligament and Joint Laxity:**

A 2-year-old, 65 pound German Shepherd with a grade #3 hip dysplasia was bred and placed on 2 grams of sodium ascorbate daily. The pregnancy and whelping were very uneventful with eight normal pups whelped. The pups were placed on 50 to 100 milligrams ascorbate a day during early puppyhood. This regime was repeated through four litters. To date there is no dysplasia in the 30 pups. The fourth litter is only four months old and too early for X-ray diagnosis. We realize that definite conclusions cannot be drawn from a single dog, but there are several other pregnant animals on this same harmless regime
and their pups will be carefully followed. These preliminary results may provide the basis for a possible simple procedure for preventing the very frustrating occurrence of canine hip dysplasia.

An 8-week-old, 15 pound pup, recently purchased from a Doberman breeder, was referred with the radiographic diagnosis of “hypertrophic osteodystrophy” and the recommendation for euthanasia. Examination revealed that the carpal joint of the right front leg seemed to be in a constant flexed attitude, although when standing the paw was in normal contact with the floor. This normal contact required the metacarpus to be forcibly extended. There was no pain to touch or to normal movement. The carpal and metacarpal areas appeared normal - no flexion or extension. X-rays exhibited no evidence of abnormalities. It appeared that the carpal ligaments were not holding the carpal joint in normal position and might be related to poor collagen quality in the ligament. Three grams of sodium ascorbate were administered intravenously and 40 grams of sodium ascorbate powder were dispensed with the instructions to give 1/4 teaspoonful in the food daily. In three days the flexed condition had returned to normal. Two other litter mates with the same condition were similarly successfully treated.

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**Discussion**

It is seen that megascorbic therapy is successful and applicable to a wide variety of mammalian pathology. We have always been cautioned to be wary of something that has the appearance of a panacea drug, but this effect is much more than mere drug action; it is a completely new basic modality or rationale for overcoming disease stress. Ascorbate is so basic in the living process, being intimately involved with so many enzymatic reactions, submolecular electron transfers and in the maintenance of optimal oxidative-reductive potential of living tissues, that large amounts of ascorbate are required to maintain biochemical homeostasis under conditions of disease stress. The mammalian liver is geared to react to these stresses with the production of increased daily amounts of ascorbate. But apparently this biological system is overwhelmed by these severe stresses and does not produce enough ascorbate, fast enough to efficiently combat these stresses. All that we are doing is supplying this missing ascorbate and providing the biochemical basis of homeostasis which brings the animal back to normal health. If properly utilized, this concept should help solve many difficult problems in veterinary preventive medicine and therapy and serve as a model for its use in human medicine.

**Summary**

The natural history of the enzyme system for the endogenous production of ascorbate from blood glucose over the past 300 million years shows an increasing requirement and greater production of this vital metabolite in the mammals over earlier vertebrates. This evolutionary evidence provides a logical rationale for the clinical use of “mega” doses of ascorbate in prophylaxis and therapy. Dogs and cats were found to be poor ascorbate producers and suffer from chronic subclinical scurvy, especially when under stress. The full correction of this genetic condition gives dramatic therapeutic results in sick animals and consistent maintenance of full health and mental alertness when used as a prophylactic measure. Ascorbate is a harmless, non-specific orthomolecular virucidal agent. When used intravenously or subcutaneously at about 1 or 2 grams per pound body weight per day it provides rapid relief in viral
diseases like distemper, influenza and rhinotracheitis. In addition to its virucidal action, “mega” doses of ascorbate also relieve stresses and stimulate the animal’s recuperative powers. Successful case histories are given on the use of this orthomolecular treatment in other conditions met in a general veterinary small animal practice such as: paralytic nictitating membranes, jaundice, pregnancy and whelping, hip dysplasia, allergies, pleurisy, epilepsy, cystitis and spinal degeneration. The daily use of a few grams of sodium ascorbate added to the dog’s or cat’s food has given excellent results in maintaining the animal’s continuous good health, well-being and mental alertness. Resistance to disease is improved and many of the present difficulties of pregnancy and whelping are avoided by the simple daily ingestion of 1/2 to 2 teaspoons of the relatively tasteless sodium ascorbate, which is the basis for our Orthomolecular Preventive Medicine Regime.

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References


IRWIN STONE

Irwin Stone is a Professional Chemist Accredited (Life), a Chemical Engineer by training and a Biochemist by profession. He is a Fellow of the American Institute of Chemists, Fellow of the Association of Official Analytical Chemists, Honorary Fellow of the International Academy of Preventive Medicine and a Fellow of the Australiasian College of Biomedical Scientists. In 1962-63 he was President of the American Society of Brewing Chemists. He was one of the early workers on ascorbic acid starting in 1934. In the chemical technology of ascorbic acid he obtained the first patents in 1939 on the stabilization of foodstuffs with ascorbate and a patent on the synthesis of ascorbic acid in 1940. The medical aspects of ascorbate has been his prime research interest since then and in 1965 he started publication of a continuing series of papers describing the potentially fatal human genetic liver-enzyme disease, Hypoascorbemia, pointing out that scurvy is a genetic disease and not a simple dietary disturbance and that ascorbate is a mammalian liver-metabolite rather than a trace vitamin. This provided the basis for “Megascorbic,” a branch of Orthomolecular Medicine. While most of his work has been in human medicine, this paper applies his principles to veterinary practice.

WENDELL O. BELFIELD

Wendell O. Belfield, D.V.M., is the owner and director of the Bel-Mar Orthomolecular Veterinary Hospital in San Jose, California. This is the first veterinary hospital specializing in Orthomolecular Medicine. After graduating from Tuskegee Institute in 1954, he spent five years in the Meat Inspection Division of the Department of Agriculture and two years as Captain in the Veterinary Services of the U.S. Air Force. He is a member of Santa Clara Valley Veterinary Medical Association, California Orthomolecular Medical Society, International Academy of Preventive Medicine and the New York Academy of Sciences. Clinical research conducted at his hospital has resulted in the publication of new and unorthodox surgical procedures and highly successful orthomolecular medical treatments, such as “Canine Orchiopexy,” “The Dysplastic Dog Can Be Helped,” “The Partial Spay,” “The Use of Insulin In Open Wound Healing,” “Vitamin C In Treatment of Canine and Feline Distemper.”
Complex.” Distemper is viral encephalitis.

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(Incomplete journal references to be corrected on later HTML revision)

[Note: Some minor typographical and grammatical errors have been corrected; originals and [corrections] below:

“For a long time the *impression* [impression] has existed…”
“100 ml vials of sterile injectable 25% sodium ascorbate *was* [were] used.”
“…those mammals capable of *synthesizing* [synthesizing] their own ascorbate…”
“…with the radiographic diagnosis of “*hypertrophic* [hypertrophic] …”
“Examination revealed that the *carpel* [carpal] joint…”
“The Use of Vitamin C As An *Antibiotic* [Antibiotic],…”

Poor original editing is marked [??], and additional formatting was added to improve readability. - AscorbateWeb ed.]

HTML Revised 20 November, 2013.
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