IMMUNE SYSTEM PROBLEMS IN CANINE

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The German Shepherd Dog is a very popular breed. In fact, it is Number One worldwide although in the U.S. it ranks much lower partly because of AKC clubs' non-adherence to the international Standards—usually in the AKC top ten, though. As a result of there being so many GSDs, veterinarians and others typically see more cases of most disorders than they do in other breeds. Popularity has its drawbacks, and undeserved notoriety is one of them. Take the incidence of dog bites, for example. Many breeds have far more of a tendency to bite people than does the GSD, although they don't get the same "press" (publicity). Min-Pins, Skye Terriers, American Cockers, Chows, and many others will sink their fangs in you more readily and with less provocation. But the popularity of Shepherds, Rottweilers, and a couple others is their downfall.

Of course, not all popular breeds are involved in as many biting incidents. You have to travel a long road to find a Golden Retriever that has ever bitten a person, and Labs have an intermediate incidence compared to others of its size. Nor do big dogs bite more (although perhaps with more damage)—the mastino types are usually placid, while the feists like Chihuahua/terrier-types and mixes give credence to their name and the word "feisty."

That was just an example of generalizations that abound in the dog world. Some are unfounded; others have a basis in statistical facts. But I want to concentrate more, in this article, on non-behavioral problems. Another example of those generalizations is the incidence of hip dysplasia. Rottweilers and American Pit-Bull Terriers may have nearly the same percentage and typical severity of HD as in the GSD, but the Shepherd is somewhat different than most breeds, in that HD causes more discomfort and lameness than does the same degree of looseness in the joints of the more stoic breeds (many with some bulldog heritage).

Another problem seen quite frequently in the GSD is the deficient immune system syndrome (a syndrome is a collection of symptoms). It is characteristic of this problem to manifest itself in one dog in a certain way, and in another dog in a different way, a little like a pleiotropic trait. Some evidences are so slight that many owners and vets miss or don't guess at the underlying

cause. This can lead the doctor to wrongly prescribe a certain medicine or none at all, and the breeder to go ahead and breed a covertly defective dog that should not be mated. Further complicating the matter, and preventing as much progress as could be had, is the subclinical nature (a lack of, or hardly-noticeable, signs). Many intermittent or mild complaints that owners have are not identified as related to the dog's immune system, and others have taken years of badgering by breeders before the veterinary community has acknowledged what breeders had known all along. So you will find some disagreement in some of what I will present in the following material. I don't want to just list a table of disorders under the subject heading, but you might want to do that for yourself. Let's take a look at some of those immune-mediated disorders after a few more words about the general subject.

The GSD has more than its "fair share" of immune-related problems, and the clues usually appear in the intestines, eyes, skin, and other places. The breed has many individuals with a deficiency of a particular immunoglobulin called IgA, and this genetic defect may be very close on the chromosome to genes controlling general immune problems. However, we must remember that environmental "events" such as exposure to certain chemicals, and especially to multi-component and frequent vaccines, can be and probably is the greatest factor in the many cases of autoimmune disorders we have seen increase in the GSD and in many other breeds.

Autoimmune disorders

The dog's "Defense Department" has a number of soldiers: antibodies, immunoglobulins, specialized cells, and more. Some vaccine ingredients, adjuvants, or carriers have been known to cause an over-reaction by this army and result in an autoimmune situation in which the body also attacks its own cells. A recent vaccination development called "recombinant vaccine," helps avoid bad reactions to vaccines or the medium in which they are cultured or carried into the blood system, but is not effective against parvo. Nowadays, the over-reaction is more often a matter of the interplay of genetics with stimulus from vaccine components. The more vaccines that are given to the dog, the more frequently administered, and the number that are in the same combined "shot," means that the part environment plays in reactions is much larger.

The German Shepherd Dog breed seems to be at relatively higher risk for a number of systemic abnormalities, and while not all have been directly linked to immune suppression, with most of them there is great suspicion of some connection. We have seen such problems in the breed as pannus (chronic superficial keratitis), corneal dystrophies, and plasmacytic conjunctivitis in the eye; lupus and anal furunculosis (perianal fistulas) in the outer integument; and plasmacytic colitis in the gastrointestinal tract; these we suspect are related to autoimmunity. The various

components and functions of many glands and chemicals in the normal body are lumped together to refer to their joint action of protection, and given the name "immune response" or "immune system." It involves such things as T cells, phagocytes, white blood cells, antibodies (immunoglobulins), complement proteins, and others. Together, their job is that of a second line of defense against antigens and other threatening "foreigners"; the skin, mucous membranes, and stomach acid are some of the first-line defense mechanisms that bodies have, and if something harmful gets past the frontline troops, the interior guard must go to work.

Invading organisms may be viral, bacterial, parasitic, fungal, and may come into the body via puncture, swallowed foreign objects, impurities in eaten material, absorption through the skin, intake by the lungs, or other routes. This second line of defense even goes after waste generated in the cells, or abnormal cells which if left alone could become cancerous. To do this, the soldiers must be able to recognize the enemy, and do so by chemical means, such as "seeing" if the projections on the suspects fit like jigsaw pieces into receptors carried by the troops. If so, they attempt to neutralize by putting chemical handcuffs or a half-nelson on those invaders until they can dispose of them. Some of the home guard, macrophages, actually "eat" bacteria; some poke holes in the invaders or mark them for other cells to eat or destroy. When such interactions occur, the guard is stimulated to call for reinforcements (multiply rapidly) to search for more of the invaders' ilk. B-lymphocytes have antibody protein molecules on their cell surfaces that recognize the foreign molecule called an antigen. One result of this encounter is that these B "white blood cells" become antibody factories called plasma cells, and can turn out their product for many years to come. Thus, some diseases are warded off the rest of our lives because our bodies continue to have patrolling soldiers that can recognize them. The other type of defender lymphocyte is the T cell. They directly kill "bad" cells without using antibodies. Besides killer T cells there are helper T cells and suppressor T cells; the latter call off the attack so the white blood cells (lymphocytes) don't get carried away over-multiplying (hopefully!) or that would present other problems.

One hitch in the war machinery is that some lymphocytes get confused and mistake normal body (called "self") proteins for the "bad guys." If "self" doesn't kill off these errant traitors, as normally happens in the embryo and very young individual, we have what is called an "autoimmune" situation. An oversimplification would be to say that the body is "allergic to itself." In any case, the body uses the home guard to attack and possibly destroy part of itself (its self). Such a defect can affect immune response all through the body. It is possible that this is happening in pannus, "allergies," lick granuloma, and a number of other problems in our dogs. In human AIDS, all the active helper T cells are destroyed, so the body no longer has adequate defenses against any and all antigens. In most disorders, only one or two pathways are affected, so a problem may show up as an itch, skin blisters, hemolytic anemia, a corneal defect, rheumatoid arthritis, or something else in one or more organs.

Demodex — One of the most easily identified immune-mediated problems is demodectic mange. To distinguish between this and the purely contagious sarcoptic mange, see some of my other articles or buy my book on the GSD. The demodex mite is ever-present on nearly all dogs and humans, but doesn't cause a problem unless the host is weakened by something, especially another immune system related disease or stress. It is widely believed that stress of various kinds, whether of a genetic origin such as a very nervous temperament, or either a genetic or acquired immunodeficiency disease that suppresses T cell function in the immune defense system, may be the major factor in an outbreak of symptoms such as demodecosis in a dog. With lowered cell mediated immunity, the individual reacts adversely not only to the mite and its toxins, but also to the presence of other microbes and antigens.

"Neutering" reduces stress in the individual (as well as putting the brakes on the spread in the gene pool). A bitch in estrus is in the highest state of systemic stress that any dog normally encounters, outside of severe trauma and shock, so spaying is recommended in cases of demodex. Often enough, demodecosis is concurrent with another immune-related defect. If your dog has demodectic mange, look for another condition that should be treated at the same time. Is there a history of subclinical pancreatitis? Has there been recent surgery or other physical or emotional trauma? Any exposure to debilitating diseases? Even the minor stress of teething may be sufficient to tip the balance and encourage sudden proliferation of demodex mites and their symptoms in susceptible "on the borderline" dogs. Very healthy dogs rarely show symptoms even when exposed intentionally by clinical transmission of these mites. Stress (such as illness) seems to "awaken" the mites. Combating demodecosis is largely a matter of curing or controlling the dog's other ills, both physical and psychological. Use of steroids is contraindicated because they compromise the dog's immune defense. See my "Total German Shepherd Dog" book for more detail on this topic.

Pancreatitis: In its chronic, subclinical, or often-undiagnosed mode, exocrine pancreatic insufficiency (EPI or PI) is fairly common in German Shepherd Dogs of certain bloodlines. It has even been identified with demodectic mange, possibly because during the stress of the dog's affected digestion, its body is less able to immunologically suppress the proliferation of the mange mites. Supplementation with vitamin A and pancreatic enzymes should be supervised by a veterinarian who is knowledgeable in this area and has been made aware of the genetic portion of the problem in certain lines of our breed. The occurrence in pancreatic insufficiency among German-line dogs in the U.S. seems to have increased since the 1970s, but I believe there are a couple of different reasons for this, if it is an accurate observation.

Malabsorption (poor digestion and poor stool condition) are frequently seen in the GSD, and in my experience, has been more so in the heavily linebred typical lines in American-bred dogs

since the 1970s. EPI is one of the conditions that can contribute to the malabsorption syndrome. The symptoms can be exacerbated by physical or emotional stress, change of food, and other things. I suspect that dogs with subclinical weakness in immune systems or pancreatic function may be most likely to show these reactions. I have also seen an apparent connection with temperament weakness, as those dogs with especially "hard" character have a much lower incidence, and there is a higher percentage of digestive disorders among American-line GSDs which customarily are not trained in, tested for, and eliminated by the Schutzhund (protection) sport.

With **EPI**, the fur often becomes dry and brittle, and even lost to some extent, and Staphylococcus infection scabs may appear on the skin because the compromised immune system doesn't allow the dog to fight off the infection. The symptoms of EPI mostly show up when the TLi value is down (Trypsin like immunoreactivity test). So there seems to be a possible connection, with insufficient pancreatic function and other "resistance" all being tied to the immune system.

A dog with the sub-acute form of pancreatitis may exhibit coprophagy, which means he eats his own (or others') stools. It may be that he smells the undigested fats and carbohydrates and instinctively consumes it as food to give those nutrients "another chance." Remember, though, this can also be a habit arising from boredom, lack of owner clean-up, or diet. Often, the addition of liver to a low fat diet and daily administration of enzyme powder or capsules, or regular supplementation with ground or chopped pancreas if you are lucky enough to get some from a nearby slaughterhouse, will bring the condition under control or at least improve it. Researchers at Tulane University found that a commercially available enzyme supplement could improve blood analysis, neonatal vitality, digestion, and general health. The manufacturers of Viokase™, one dried raw pancreatic enzyme brand, have shown supplements/medicine to be effective in combating nonspecific diarrhea as well as subclinical pancreatitis. The juvenile-onset generalized demodecosis often has a spontaneous semi-remission because of better stress management.

Dogs that exhibit symptoms such as much flatulence, or intermittent diarrhea or pasty light-brown/yellow to clay-colored stool, perhaps should be tested for levels of Lipase, Protease and Amylase, or just fed the recommended preparations without testing. But keep an eye out for other immune-system signs, too. Some people perceive a probable connection or coincidence between anal furunculosis (perianal fistulas) and EPI. Both of these problems may show up in the same dog, strengthening the suspicion that they are manifestations of the same underlying immune system weakness. A great deal of the digestive tract functions and stages may be affected one way or another by the same genes governing immune response.

Pancreatic insufficiency is an abnormality that suggests removal from the gene pool, whether the dog has a severe or a mild case or is asymptomatic most of the time. Most vets take very few hours of nutrition and practical genetics classes in vet school, and then forget most of it because they don't use it every day. Breeders are sometimes more reliable sources of information. Unfortunately, many people who offer their EPI males at stud do not admit or declare any cautions about their dogs. As one observer quipped, "It's funny isn't it, that those who deny all those things have Viokase-V on the shelf in their back rooms?" Although it is good for various unspecified causes of diarrhea, the product is so much more expensive than kaolin or changing the diet, that it makes you wonder if you should try one of the other "remedies" first.

Bloat/Torsion/Volvulus: Is there a connection between PI and GDV (gastric dilation and volvulus)? There have been reports from dog owners indicating that many episodes of EPI begin with a bloating incident, or with gastroenteritis, marked by vomiting and blood tinged diarrhea. One observer said, "From the general info collected, the dog first bloats, which often leads to torsion of the gut, which of course requires surgery for a tacking of the stomach, and this is usually followed by a full blown episode of EPI within a few months of the surgery." I suspect that there is, at least sometimes, a common root cause if these two problems. Dogs with a history of bloating/torsion and/or bouts of unexplained diarrhea are reported by several of my correspondents to be quite likely to be EPI-carrier suspects, although this observation is admittedly anecdotal.

Megaesophagus: German Shepherds have over thirteen times the incidence of esophageal disorders of all other breeds combined, although PRAA, an unrelated heart defect that causes similar symptoms as caused may be part of this statistic. Dr. Labato at Tufts U. School of Veterinary Medicine says, "It [sometimes] may be secondary to ... myasthenia gravis, systemic lupus erythematosus (SLE is an immune mediated disease), ...[and possibly others]." Breeds susceptible to the juvenile-onset, inherited type of megaesophagus include Irish Setters, German Shepherd Dogs, and a few others. Frequently, large dogs are diagnosed with the idiopathic form, which means the cause is unknown. "In most cases we don't know the causes," said Dr. Twedt in the vet school at Colorado State University.

The characteristic loss of peristaltic action is probably due to a disorder of the afferent nerves, which is why there is no successful medical, pharmaceutical, or surgical treatment. There may be a connection with other nerve disorders, even giant axonal neuropathy, which mimics HD and GSD myelopathy. Because of these similarities, some have hinted that a general immune system deficiency is at the root of this problem, as it appears to be in so many disorders.

Diagnosis of megaesophagus is confirmed by means of various tests, some of which are intended to discover the underlying cause, and may include the acetylcholine receptor antibody titer that is used to diagnose myasthenia gravis. An antibody titer is a blood test that looks for immune-mediated disease—one in which the body attacks itself. One source I detail in my GSD book states that the incidence of symptomatic mega-esophagus in the GSD population in the US is at least 1%, but about 18% of U.S. (AKC-lines) GSDs are carriers of the altered gene (assuming autosomal-recessive inheritance). With 18%, the [risk], even if you avoid linebreeding and stay completely away from all the [known] lines, is extremely high. The pedigree study in "The Total German Shepherd Dog" (published by Hoflin) indicates that both Bernd Kallengarten and Lance of Fran-Jo were suspects in carrying the recessive for megaesophagus, and the latter was known to produce a considerable number of descendants with various other manifestations of immune defects.

Intussusception: In very young pups (and other animals including humans) the intestine can invaginate (one part slips inside another). The condition, also referred to as "telescoping intestines," also occurs in adults, but not as frequently. Most common immediate causes include worms, obstruction by indigestible materials, garbage, or toxic substances. However, since the German Shepherd Dog seems to experience a relatively high incidence of this disorder, I have to suspect the possibility that (other than those above causes) there is a genetic propensity in certain bloodlines, and perhaps interrelated to other "GSD disorders"—those more common to this breed than most others.

Pannus: The GSD has more than its "fair share" of immune-related problems, and they appear in the intestines, eyes, skin, and other places. The breed has many individuals with a deficiency of a particular immunoglobulin called IgA, and this genetic defect may be very close on the chromosome to genes controlling general immune problems. The GSD, after the West Highland White Terrier, probably also presents most of the cases of pannus, an eye disorder caused by lymphocytes migrating into the cornea and causing blindness unless treated. More and more vets are referring to it as chronic superficial keratitis; CSK for those addicted to abbreviations. I have watched quite a few eyeballs "peeled" in the surgical part of the therapy, in the days when steroid drops in the eye on a frequent basis (several times a day) for the rest of the dog's life was the post-surgical treatment of choice. Peeling was the more heroic procedure, when injection of cortisone under the conjunctiva as a first step is not effective. Today the drug cyclosporine is used to best advantage in pannus, although a steroid such as dexamethasone is still effective. This cyclosporine is the same drug, originally found as a component of a Norwegian soil fungus, that is given to counteract the body's tendency to reject donor organ transplants. The drug is

given as an ointment or in food twice a day until the cornea is free of the lymphocytes, then there is a once-daily administration; it seems to work partly by causing the tear ducts to operate almost full time. Enough is absorbed by the tissues of the eye to get into the far reaches of the circulatory system where it does the other part of its job, fighting those wayward lymphocytes. Tacrolimus is 10 times as potent as cyclosporine but is minimally absorbed through the skin.

No cure is in sight, since it is highly likely that pannus is an inherited autoimmune disorder, and people who have dogs with pannus will have to deal with the frustration and regimentation of daily treatment for the dog's lifetime. The same situation has been nagging sufferers from human autoimmune disorders for a long time. MS, multiple sclerosis, is considered by most to be such a disease, in which T-cells attack components of the central nervous system (brain, spinal cord and some major nerves). One of those components, myelin basic protein (MBP) has been experimentally fed to lab animals, and later to human MS patients, and it was discovered that the severity of symptoms was considerably reduced. Rheumatoid arthritis, dealt with more in my book on orthopedic disorders, is another supposed autoimmune pathology in which the T-cells act against parts of the joints, especially Type-II collagen. Again, oral-dietary administration of this type of collagen was fed to RA patients with significant improvement in managing the disease symptoms such as number and severity of swollen joints, gripping strength in the hands, and subjective descriptions of pain or stiffness after rest or sleep. Experimental treatments involving feeding normal cornea extracts to dogs with pannus may have similar results.

Pannus was previously called "keratitis superficialis vasculosa pannosa pigmentosa chronica," "German Shepherd Dog Keratitis," and "Keratitis Überreiter" after its Austrian discoverer, is an inflammation and pigmentation of the cornea and sometimes involves the conjunctiva. It is rare in almost every other breed, and in the GSD it usually appears around 3-5 years of age and in both eyes. By that time, many affected dogs will have already been bred. Besides the hereditary, breed-dependent predisposition there is an environmental component that brings it on earlier and more certainly: ultraviolet radiation. UV rays in sunlight trigger the onset of symptoms, explaining why an increased incidence is observed anywhere during the sunnier months of the year, and more cases are presented in higher elevations such as Denver. This means that to avoid outbreaks of the acute phase, the owner should not only keep up with the medication schedule, but also make sure his dog is protected from exposure to strong sunlight, even if reflected off snow or water, and especially at high altitudes. Some dogs are kept indoors (glass windows filter out most UV rays) and are walked in darker hours; some wear fitted sunglasses.

Corneal dystrophy: While I do not draw any definite connecting lines between pannus and this disorder, I mention it because I think there may be an immune system relationship here, also.

Small opacities may appear on the cornea over the pupil or slightly off-center, and the novice might think the dog has cataracts. It may be triggered by an allergic reaction or some other suspected cause, and show up as a small spot, varying from slightly translucent to cloudy-white. The size is usually less than 5mm across, round, oval, or horseshoe-shaped. Most eye specialists refer to this type of opacity as "corneal dystrophy"; the spots do not interfere with vision. In my experience the spots have faded away in a few years after reaching maximum size. Corneal dystrophy appears to be genetic, but is not serious. Probably less than one percent of the breed is affected. I once had a bitch who developed very small oval opacities, one on each cornea, after she was about four or five years old. They finally and gradually disappeared (shrunk to nothingness) by the time she was about ten years or more. This bitch also developed atopic (allergic-type) problems marked by itching feet and sometimes parts of the skin, but most noticeably by an assumed feeling of excess phlegm in the throat. One of her many sons developed the same transient and minor corneal defects, appearing in maturity and going away without treatment by old age. While it is possible that some of these opacities may be immune system related, I think most are largely if not fully environmental.

Pemphigus: Uncommonly seen in dogs and more found in humans, this group of related autoimmune disorders involves mostly the mucous membranes and skin. You may never notice the spider-web mucosal condition in the mouth or purplish, fragile splotches of skin in some forms. In some forms it can produce ulcers in mucous membranes. Very high doses of corticosteroids for life may control the disease, but this is a controversial approach because steroids are generally contraindicated in autoimmune diseases, and usually cause a great deal of capillary rupture and bleeding. It may be best to just leave these alone and see if they will "go away" on their own.

Primary Seborrhea: I strongly suspect seborrhea may sometimes be a sign of an acquired autoimmune disorder. I would bet on it. This disorder is a condition in which there are scaly patches and a greasy feel to parts of the dog's skin. You will probably notice great difficulty in keeping the ears clean and free of dark wax and yeast or fungus. Often, the older, long-affected dog will have an over-all rancid odor. Many of these cases are related to thyroid hormone imbalance, and such an immune- and general endocrine-related disturbance may become chronic and in need of very frequent bathing and/or ear cleaning with little or no hope of remission. Soloxine (thyroxine, or levothyroxine) might be indicated and recommended by your veterinarian. It would not hurt to try, as this drug has very few minor side-effects. The disease most probably is centered in the thyroid.

I have seen many cases that have been brought on (triggered) after extended or repeated exposure to fleas and other factors. The flea allergy or exposure may be the prime cause of the

skin condition, with the flea antigens weakening the dog's immune response and thyroid function, resulting in severe seborrhea. Or, the immune defense weakness may be the prime cause of the dog not being able to withstand fleas. Ask a veterinary dermatologist to try to find the underlying cause as well as give you ideas on how to treat for the symptoms. If the dog shows evidence of much itching, it is usually called secondary seborrhea, which refers to a primary cause being mange, fleabite allergy, or other trigger influences, involves relatively large reddish-skin patches with hair loss, and is often more scaly and less greasy than is the primary form. Primary seborrhea is something GSDs seem to be more predisposed to than are most other breeds, and it is this type that more affects the ear with fungus growth, and sometimes an increase in bacterial colonies on the skin. It is a chronic condition that requires constant or renewed treatment regimens with no hope of eventual cure, just some control.

Most owners of dogs so affected report considerable success in managing or partially suppressing the symptoms by attacking them on several fronts: get rid of fleas (and the cats and carpets they rode in on!), clean the ears daily or several times a week with a 50/50 mix of vinegar and water, and temporary regimens of Soloxine for perhaps two weeks initially. Your vet can suggest a dosage level to try, of this quite safe internal medication, and by adjusting the dosage, you can find an acceptable maintenance level.

Degenerative Myelopathy: DM was once known as "GSD myelopathy" because most cases in the early days of investigation involved that breed. It is the first disorder that comes to mind when German Shepherd Dogs and spinal lesions are spoken of together. "Degenerative" means that it is chronic and progressive, and "Myelopathy" means spinal cord disease. The first symptoms are usually seen at about six to eight years of age and have a duration of five to twenty-four months, perhaps a bit longer if aggressive measures are taken, but who knows if they are really effective? Initially, the dog does not seem to realize what position his rear legs are in; soon he will begin to drag his toenails and the top part of his paws, and later may tremble as if palsied. Eventually, he will be unable to get up on all four legs, and by this time most owners will have decided upon euthanasia. Symptoms and histological changes are very similar to those in multiple sclerosis (MS).

It is also seen (though rarely) in the Belgian Shepherd and the Old English Sheepdog, and some authorities feel that other breeds' degenerative myelopathies are probably not caused by the same immune-system deficiency as we have in the GSD. Autopsy shows demyelination (loss of the insulating sheath) of the spinal cord, destruction of some large axons (nerve cells leading from the cord to smaller branch nerves), and abnormal cells (or certain cells in abnormal locations). Similar signs may be seen in the brain, kidneys, and intestines, giving further hints of the immune system failure being at the root of this disease.

It may be that relatively high vitamin E dosages may be helpful, but it is difficult to compare a particular dog's disease progression with a "what-if" situation. We have a good idea that this vitamin is very helpful in general immune response improvement, so it is natural to assume a probable direct benefit in this immune-related disease. 800 units (IU) a day may be enough, although some years ago one researcher claimed that 2000 IU of vitamin E daily, 500 mg of vitamin C twice a day, and a high-strength vitamin B complex twice a day was the best dosage. While his results were not duplicated by others, I mention it just in case you come across his claim. In DM dogs, low serum and tissue concentrations of vitamin E have been observed. I recommend that vitamin E be given to all older German Shepherd Dogs for general resistance and health. It can't hurt—they will excrete anything they don't need, within reason—and it is synergistic with other vitamins and nutrients. Chemical-pharmacological treatment has largely been via the use of aminocaproic acid, and more recently, acetylcysteine three times a day found some acceptance, although conventional drug therapy (medicines) has been of little lasting help to patients with DM. The combination of exercise, vitamins and certain drugs seem to have delayed the progression of DM in many dogs a little. Treatment has been directed at suppression of symptoms and the multi-pronged approach may slightly prolong the day you have to face euthanasia because of debilitation and inability to stand to defecate or to walk.

Lick Granulomas: Dogs with GSD myelopathy often develop lick granulomas on the extremities, especially the hind feet; these are non-healing ulcerations or, if you are lucky, merely callous-like reactions of the skin to extremely frequent licking, sometimes chewing, at the location of a supposed itch. It is probably a case of the limb feeling as if it has "fallen asleep," to put it into terms familiar to human experience. The tingling sensation caused by incomplete and erroneous signals by the nerves serving that place is much like the irritation caused by an ant bite, or hairs out of place, or anything in between. In trying to lick it away, the dog actually softens and wears away the hair and skin. Lick granulomas are not restricted to dogs with DM, but often occur on the pasterns or toes in dogs that have atopic allergies, another clue to the presence of a general immune system deficiency.

Other problems: Keep in mind that the various parts and systems of the body are all inter-related, that a disruption in the process of one may have an origin or an effect in another. The endocrine system is a prime example, with hormones being produced in more than one gland and greatly influencing some or all of the other glands. Something that has not yet been thoroughly explored in veterinary schools or with research grants is the collection and inter-relation of problems very common to GSDs, with yeast/fungal infections, flea saliva allergens, and general autoimmune system weakness. I have observed countless cases of dogs in this breed with a combination of seborrhea, low resistance to fleas, thyroid insufficiency, nagging ear infections, interdigital pyoderma, and other "complaints." The lines between these dots, I hope, will someday be drawn with more clarity.

More about Vaccines: There is good reason to suspect over-vaccination to be a big contributing factor, if not possibly the sole reason for what many believe to be a rise in incidence of auto-immune disorders. Kris Christine of The Rabies Challenge Fund says, "Please educate yourself to make sure you don't needlessly over-vaccinate your companion animals." She gives links to data and news articles about pet vaccinations on http://www.RabiesChallengeFund.org and manages a vaccine e-mail list.

Following a presentation at the AKC Canine Health Foundation 2007 National Parent Club Canine Health Conference in St. Louis Missouri, October 19-21, 2007, entitled "What Everyone Needs to Know about Canine Vaccines and Vaccination Programs" Dr. Ron Schultz of the University of Wisconsin-Madison answered questions.

To "Is there a risk of over-vaccinating a pet (i.e., injecting it too often, or using vaccines that are not required for the specific pet)?" he replied, "Yes. Vaccines should not be given needlessly, as they may cause adverse reactions. Vaccines are medical products that should be tailored to the needs of the individual animal."

My personal experience as a breeder, show handler, and judge, and other observation of thousands of dogs belonging to others have convinced me that there is an extremely high probability of cause-and-effect between the plethora of vaccinations and inoculations and the rise in autoimmune disorders I have witnessed in more than seven decades of living and working with dogs. I am a scientist, and based on cautious conclusions following careful observations, I believe that most of the immune system disorders I have described above result largely from too many vaccines, too many at once, and too often given.