Hunting Dog Hypoglycemia   
  
(<http://2.bp.blogspot.com/_ZT0UWJhXYro/S7aItxAyVCI/AAAAAAAAAoQ/zToStEKb1nc/s1600/Dr.+Charlie+Hjerpe.jpg>)   
Dr. Charlie Hjerpe with 2X CH Sand Creek Bud   
  
(<http://2.bp.blogspot.com/_ZT0UWJhXYro/S7Z_flx_1JI/AAAAAAAAAn4/15t75jPygAk/s1600/C+Hjerpe.JPG>)   
It has been my privilege, through field trialing, to come to know Dr.   
Charles A. Hjerpe, DVM. If it has been done with bird dogs, Charlie has   
probably done it during his long lifetime. Charlie is a little modest in his   
introduction - he is Professor Emeritus of Veterinary Medicine at the University   
of California School of Veterinary Medicine at Davis.  
  
A couple of months ago, Charlie sent me a copy of a longer article that he   
had written about Hunting Dog Hypoglycemia. Great information, including   
some little known information about nutrition and fitness, but a little too   
long for a blog post. At my request, Charlie boiled it down to the   
following article. A big thank you to Charlie for this very valuable contribution...  
  
Some Personal Observations, Opinions, Hypotheses, and a Little Science   
Concerning Hunting Dog Hypoglycemia (HDH)   
  
by Dr. C.A. Hjerpe, DVM - Davis, California  
  
A. INTRODUCTION:  
  
Before discussing my personal observations of and opinions concerning   
hypoglycemia in hunting dogs, I will first offer some disclaimers, present my   
credentials and provide some definitions. First, I wish to emphasize that I   
am not and have never been a small animal specialist, and my personal   
observations and opinions are based on recollections that are not backed up with   
detailed, written records, and should not be regarded as “research”.   
  
I graduated from Cornell University’s New York State College of Veterinary   
Medicine in 1958, worked in private, mixed species veterinary practices   
for 5 years, and was a professor of large animal medicine at the School of   
Veterinary Medicine, University of California, Davis, for 31 years, until   
retiring in 1994. During the last 14 years of my academic career, I also   
served as Director of the UCD Veterinary Medical Teaching Hospital. During the   
past 48 years, I have owned one Weimaraner, 2 English setters and more   
English pointers than I would be able to recall and enumerate (probably more   
than 100). I have field trialed with most of these pointers, and have always   
hunted with my field trial dogs.  
  
B. DEFINITIONS:  
  
I think it appropriate to begin this presentation with a short discussion   
of hypoglycemia. When veterinarians or physicians say that a dog (or cow or   
person) is hypoglycemic, we are saying that the concentration of glucose   
(a specific type of sugar compound) in the blood of that individual is below   
the normal range for blood glucose values in that species. For a dog, it   
means that the patient’s blood glucose concentration is less than 59.4   
milligrams per deciliter (mg/dl).  
  
Many different diseases of dogs may cause hypoglycemia, so hypoglycemia is   
not a disease or a diagnosis but, rather, a non-specific biochemical   
alteration of body fluids. Clinical hypoglycemia in a dog means that (1) the dog’  
s blood glucose concentration is below normal, and (2) at least some of   
the characteristic clinical manifestations that always accompany marked   
reductions in blood glucose values are also present. Since clinical hypoglycemia   
can be caused by a number of different diseases (especially liver diseases   
and pancreatic B-cell tumors) clinical hypoglycemia is also not a specific   
disease. However, when we specify that we are talking or writing about   
PRIMARY clinical canine hypoglycemia, people will know that we are referring to   
a dog that has:  
  
(1) very low blood glucose values (below 50 mg/dl),   
  
(2) accompanied by typical clinical manifestations (symptoms or signs) of   
low blood glucose values, and   
  
(3) that no other primary diseases capable of causing hypoglycemia are   
present. This condition is usually associated with prolonged, vigorous   
exercise, and is generally referred to as exertional hypoglycemia or hunting dog   
hypoglycemia (HDH). During the remainder of this discussion, I will use only   
the latter term when referring to it.   
  
Glucose is the primary energy source for most cells in the body. Glucose   
is liberated from food by the digestive processes, absorbed into the blood   
stream from the stomach and intestines, and transported to the liver by the   
portal blood circulation. Within the liver, glucose is converted to   
glycogen and stored within specialized cells called hepatocytes. In response to   
falling blood glucose concentrations and/or the metabolic requirements of   
other body cells for glucose, liver glycogen is converted back to glucose, and   
released back into the blood stream.  
  
Current scientific knowledge of basic energy metabolism suggests the   
reason why an otherwise healthy dog develops HDH during hunting: the dog’s liver   
glycogen reserves were insufficient for maintaining normal blood glucose   
concentrations, during the period of vigorous exercise that preceded the   
clinical signs of the disease. This supposition is based mostly on   
extrapolation from basic research conducted in mice, rats, human beings and in cell   
cultures, as little or no actual research has been done using HDH affected   
dogs  
  
C. CLINICAL MANIFESTATIONS OF HDH AND THEIR DEVELOPMENT AND PROGRESSION:  
  
The characteristic progression of typical clinical manifestations of HDH   
is organized, below, into Six Stages, using my own classification system:  
  
Stage 1: Whenever hypoglycemia develops while a dog is being hunted, the   
first physical or behavioral evidence of it will be a gradual onset of   
fatigue. However, it should be emphasized that, in the vast majority of   
instances in which fatigue becomes evident during hunting, hypoglycemia will NOT be   
the cause. In most cases, the dog is simply becoming physically exhausted   
and/or overheated, or is experiencing intestinal or stomach cramping.   
Nevertheless, any dog that appears to become fatigued while hunting should be   
carefully observed for the possible appearance of stage 2 symptoms.  
  
Stage 2: In addition to fatigue, the dog begins to evidence incoordination   
and/or staggering. At this point, it is urgent that all physical activity   
immediately cease, and that either the dog be fed (its regular dog food) or   
a concentrated glucose source, such as:  
  
(1) 50% glucose solution in water or,  
  
(2) corn syrup, be administered by mouth.   
  
If the dog won’t eat, and no of sources of glucose are available, the dog   
should be taken to a veterinary clinic without delay (even though it is   
true that some dogs, in this stage of HDH, if rested, will recover   
spontaneously, without any treatment at all).  
  
Stage 3: In addition to stage 1 and 2 signs, muscular tremors and spasms   
may occur, which may result in abnormal postures. The dog may be unable to   
open its mouth, or chew food that is offered, or may be unable to swallow   
food that is placed in its mouth. At some point during this stage, the dog   
may be unable to walk or stand without assistance. During this stage, it may   
be inadvisable to attempt to force-feed food or oral glucose supplements,   
because (if the patient cannot swallow) they may be inhaled and cause fatal   
inhalation pneumonia. It has been stated that glucose can be absorbed   
through the oral membranes, and that simply rubbing glucose on the gums can be   
effective treatment. This method of treatment seems suspect to me, and needs   
to be evaluated under controlled laboratory conditions, before I could   
recommend it. However, there is no harm in doing this, so long as it is done   
while the dog is being rushed to a veterinary clinic.  
  
Stage 4: In addition to stage 1, 2 and 3 signs, the dog may begin having   
seizures/convulsions. This is very serious, because the dog may not be able   
to breathe during convulsions, may die of asphyxia during prolonged   
convulsions or, at the very least, may sustain permanent brain damage as a result   
of low levels of oxygen in the blood flowing through the brain during   
convulsions. From this stage on, an affected animal can only be effectively   
treated by a skilled and knowledgeable veterinarian in a well-equipped   
veterinary facility. The patient will require continuous intravenous drip infusion   
with 5% glucose in water, periodic monitoring of blood glucose values, and   
specialized equipment for correcting hypothermia and maintaining the body   
temperature within normal limits.  
  
Stage 5: The dog is completely unconscious and unresponsive, a condition   
which is referred to as coma. Either the convulsions have ended, or the dog   
has passed directly from Stage 3 to Stage 5, without stopping in Stage 4.   
Dogs may recover completely when treated in Stage 5, or they may recover but   
have permanent brain damage, either from:  
  
(1) the anoxia accompanying the convulsions in Stage 4, or from  
  
(2) a prolonged period of severe hypoglycemia during stage 5.  
  
Stage 6: This stage is death, which may occur in untreated animals as a   
result of exposure (and hypothermia) or, in either treated or untreated   
animals, as a result of severe damage to the brain and/or other vital organs   
from hypoglycemia and/or anoxia.  
  
D. SOME PERSONAL OBSERVATIONS AND OPINIONS CONCERNING HDH:   
I have personally observed and dealt with approximately 21 cases of HDH,   
all in my own pointers. About 10 cases occurred during foot hunting, one   
case occurred during horseback training, and the remainder occurred while I   
was conditioning dogs by “roading” them in harnesses from an all terrain   
vehicle (ATV). Of these 21 cases, only one terminated fatally, and only 2   
required treatment in a veterinary clinic or hospital. Both of these latter   
cases made rapid and complete recoveries. All 3 of these severe cases were   
precipitated by roading. The remaining 18 cases were successfully managed by   
terminating their physical activity (when Stage 2 signs became evident) and   
either administering oral glucose solutions and/or feeding them.  
  
I do not recall any cases of HDH occurring in my own dogs after they were   
3 years of age. Most of my affected dogs were less than 2 years of age.   
Other authors have reported that dogs affected with HDH at a young age will   
(usually) become less susceptible to it with increasing age. Intact male and   
female pointers appear equally susceptible to HDH.  
  
On 4 different occasions, I have observed HDH occurring (during exercise),   
shortly after my dogs were subjected to extreme chilling, as a result of   
being thoroughly drenched with cold water. My hypothesis is that the   
chilling effect of the cold water may (sometimes) trigger an unidentified   
physiological mechanism that impairs the release of glucose from the liver glycogen   
reserves, and might involve reductions in arterial blood flow to the   
liver. It is well known that vigorous physical exercise can shunt the flow of   
arterial blood away from the digestive tract and into the musculature and   
cardiopulmonary circulation. Cold water chilling might simply facilitate or   
accentuate this physiological phenomenon. Three of these 4 incidents occurred   
during roading. The 4th incident occurred during foot hunting, was my   
first experience with HDH, and is described below:   
  
I was hunting with 3 dogs, during the morning of the opening day of   
pheasant season. After I had been hunting for about an hour, a cold rain began   
falling and, within 15 minutes, all 3 dogs began to stagger. Within a few   
more minutes, 2 of the 3 could no longer stand up. In addition to the chilling   
effect of the rainfall, I now know that these 3 dogs were also predisposed   
to HDH by my nutritional program: I had been feeding the least expensive   
dog food available from my local Safeway supermarket, and I had been   
observing that the less of it I fed to my dogs, the better they would run. So, as   
I progressively fed less to my dogs, they ran progressively better, but   
also became progressively thinner. It is also likely that their liver glycogen   
reserves were being progressively depleted. So when it started to rain   
that Saturday morning, and as my dogs became wet and chilled, all those “  
chickens” suddenly came home to roost.  
  
I have seen no conclusive evidence that HDH is ever inherited, and I have   
owned only 3 dogs that were affected with HDH more than once. One dog that   
I am currently field trialing has been affected twice (to date), each time   
while being roaded. I also recall 2 other dogs that were each affected 3   
times, always while being foot hunted. In virtually every instance in which   
one of my dogs has been affected with HDH, my dog was NOT the problem. I was   
the problem! If I had been a little smarter, a bit more “on the ball”, and   
not so willing to “cut corners” with my feeding and conditioning   
programs, nearly every one of the cases in my own dogs could have been avoided. In   
light of these observations, it makes little sense to me to cull a dog,   
simply because it has been affected with HDH on one or 2 occasions.  
  
E. PREVENTING HDH:   
In order to prevent HDH, the problem areas that need to be addressed (in   
approximate order of importance) are: (1) physical conditioning, (2)   
feeding, and (3) nutrition and ration formulation:   
  
1. Physical Conditioning: Most of the HDH cases in my own dogs have   
occurred when I roaded or foot hunted them for periods of time that were   
excessively long, considering the physical condition that they were in at the   
time. Often these affected dogs had been in top physical condition only 10 to   
20 days previously, but in the interim they had not received sufficient   
exercise to maintain their fitness. I appreciate that most hunters do not have   
the time and facilities required to insure that their dogs will always be in   
top condition when they want to go hunting with them. Consequently, it   
should come as no surprise to them when their dogs develop HDH, and they   
should always be prepared to effectively deal with it. All other things being   
equal, the more vigorously a dog hunts and the longer that dog is hunted, the   
greater will be its risk for developing HDH.  
  
2. Feeding: When hunting dog people sit down together to talk about   
conditioning their dogs, they will almost always be thinking and talking about   
an exercise regimen that will result in the degree of cardiopulmonary fitness   
and muscular strength that their dogs must have in order to be good,   
strong hunters. However, there is another aspect to conditioning that is mostly “  
flying under the radar”, that few people know about, and which is almost   
never discussed. That aspect involves conditioning dogs so as to maintain   
large liver glycogen reserves, and to become primarily dependent upon those   
reserves as an energy source, and as a source of glucose for maintaining   
normal blood glucose concentrations. Ideally, a conditioning and feeding   
program for a hunting dog should seek to achieve the following end point   
objective: The dog should have achieved sufficient physical strength and   
cardiopulmonary and metabolic fitness that it is able run and hunt industriously for   
the entire length of the hunt, and be able accomplish this on an empty   
stomach.   
  
It is generally recommended that hunting dogs be fed once each day, in   
late afternoon or early evening. This practice, which essentially starves your   
dog for 24 hours after each feeding, makes it IMPOSSIBLE for your dog to   
rely entirely on glucose entering the blood stream from the gastrointestinal   
tract for maintenance of normal blood glucose levels. As a result, your   
dog is FORCED to gradually increase its liver glycogen reserves, and adjust   
to using those reserves as the primary source of glucose for maintaining   
normal blood glucose levels.   
  
After a hunting dog has been adequately conditioned for the work that will   
be expected of him/her, it should not be necessary to alter the feeding   
program that is being used, except (possibly) to increase the amounts fed so   
as avoid inordinate weight loss in dogs that are being hunted frequently   
for long periods of time. All other things being equal, a dog that is   
excessively thin will tend to be more prone to develop HDH than a dog that is in   
moderate to moderately thin condition. A fat but well-conditioned dog may be   
less prone to develop HDH than a thin dog, but is also more likely to   
become overheated and fatigued while hunting, especially during warm weather.  
  
If a dog is fed a heavy meal shortly before being hunted, the dog may be   
slower and more sluggish than usual, and may experience gastrointestinal cram  
ping and/or or vomiting during the hunt. However, I sometimes take   
advantage of this phenomenon, by purposely feeding a heavy meal to especially fast   
and wide-ranging dogs, just before I intend to foot hunt with them.   
Feeding dogs immediately before hunting them may even help to prevent HDH, so   
long as you adhere to the practice of feeding no more than one meal per day,   
and observe a 24-hour period between feedings.   
  
In addition, if you have an ordinary hunting dog, one that is not in top   
physical condition and may be at risk to HDH, it is not going to do any harm   
to carry some dry dog food with you, and to feed your dog a bit from time   
to time, while you are actually hunting. This latter practice has been   
widely recommended for preventing HDH when hunting with poorly conditioned dogs.  
  
3. Nutrition and Ration Formulation: Basic biomedical research has   
demonstrated that liver glycogen storage can be greatly increased by feeding   
diets that contain only SMALL proportions of carbohydrates, especially small   
proportions of simple carbohydrates (like simple sugars and starches). In   
contrast, when diets containing LARGE proportions of simple carbohydrates were   
fed, it was found that liver glycogen reserves declined precipitously, and   
the animals came to rely heavily on gastrointestinal absorption of glucose   
for maintenance of normal blood glucose concentrations. Simple   
carbohydrates are rapidly digested to glucose in the gastrointestinal tract, and this   
glucose is rapidly absorbed into the blood stream. When these animals with   
low liver glycogen reserves (on high carbohydrate diets) were fasted and/or   
subjected to exercise, they were much more prone to develop hypoglycemia   
than were animals with high liver glycogen reserves (on low carbohydrate   
diets).   
  
Thus, at least in theory, the risk of developing HDH in hunting dogs   
should be lessened by feeding rations that provide the smallest possible   
fraction of total ration calories in the form of carbohydrates, and the largest   
possible fraction of total ration calories in the form of proteins and fats.   
Since a high proportion of total ration nutrients fall into these 3   
categories (carbohydrates, proteins and fats), the carbohydrate fraction will   
usually be lowest in those rations that contain the largest proportion of the   
other two macronutrient classes combined (protein plus fat).  
  
The combined crude protein, crude fat and carbohydrate content of a “  
performance” dog food will account for approximately 85% of ration ingredients,   
by weight. For example, Purina’s Pro Plan Performance Formula dog food   
contains 30% crude protein, 20% crude fat, 12% moisture, 1.8% oleic acid, 0.9%   
calcium and 0.7% phosphorus. When you add up all these percentages and   
subtract the total from 100, you will have calculated the percentage of   
carbohydrates in the product, which is 34.6%. So, 34.6% (carbohydrates) plus 30%   
(protein) plus 20% (fat) equals 84.6%. However, since we, as consumers, can   
not know the digestibility and biological availability of all of the   
proteins, fats and carbohydrates included in this (or any other currently   
available) dog food product, it is not possible for us to calculate the precise   
proportion of ration calories provided by each of these 3 major nutrient   
classes. Nevertheless, we can probably help to minimize our problems with HDH by   
feeding a premium, dry, performance dog food that contains a high combined   
percentage of crude protein and crude fat.  
  
I cannot recommend any of the dietary carbohydrate supplement products   
that are currently being marketed to dog owners for purposes of “rapidly   
replenishing muscle and liver glycogen reserves following strenuous exercise”.   
The use of these products in dogs (as opposed to use in people and horses)   
would be tantamount to feeding a high carbohydrate diet, which would be   
counterproductive to our objective (which is to reduce carbohydrate intake and   
force our dogs’ bodies to increase glucose synthesis from proteins and fats   
for purposes of increasing liver glycogen reserves, and to rely on those   
reserves as the primary source of glucose for body functions).  
  
F. THE BARE MINIMUM THAT EVERY HUNTER SHOULD KNOW ABOUT HDH:   
Hunters who remember and consistently follow the 2 recommendations listed   
below, should never have to pay a veterinary bill for a dog with HDH, much   
less have to bury one that dies of it:   
  
1. Hunters should always carry a half-pint of corn syrup (or other   
concentrated source of glucose) in a pocket of their hunting coat or vest. Should   
your dog become fatigued while hunting, watch him/her closely, and if   
he/she begins to appear weak or to stagger, stop all physical activity with that   
dog for that day, administer a couple of ounces of corn syrup by mouth,   
and feed him/her heavily as soon as possible (in other words, don’t wait to   
feed your affected dog until you normally feed your other dogs). For all   
practical purposes, (1) 50% glucose solution (in water), (2) corn syrup (which   
contains 100% glucose), (3) high fructose corn syrup (which contains 50%   
glucose and 50% fructose), (4) honey (which contains 50% glucose and 50%   
fructose) and (5) 100% natural fruit juices (with no artificial sweeteners)   
are all equally effective for oral treatment of HDH.  
  
2. Hunters should also be aware that, on rare occasions, a dog that does   
not show either stage 1 or stage 2 clinical signs while hunting, may   
suddenly become severely hypoglycemic, shortly after the end of the hunt.   
Consequently, even though your hunting has ended and your dog is safely in a box,   
on a stakeout or in a kennel, your responsibility for the welfare of your   
dog does not end at that point. You must force yourself to remember to check   
on your dog 3 more times, at 10, 20 and 30 minutes after the end of the   
hunt, and be certain that he/she is behaving normally at those times.  
  
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Addendum by Dr. Hjerpe regarding feeding dogs prior to running or   
hunting...  
  
Hi Mike:  
  
I was rereading my article on your blog this morning, and noticed Joe's   
comment (which is pasted below mine). He has an excellent point! If I had a   
retriever, especially a Lab (or a hunting dog of any breed in which bloat is   
known to occur) I would not do this myself, and certainly would not   
recommend that anyone else to do it either, if they hunt with dogs of such   
breeds. However, I have never known gastric torsion to occur in an English   
pointer or English setter, so I am not going to quit doing this in my own dogs. I   
must say that, when I first started doing this, it was not without some   
trepidation. However, as time has gone by and no problems have resulted, I   
had actually forgotten about the potential for mischief that can be   
associated with this practice. Wikipedia lists Weimeraners and Gordon and Irish   
setters as being among the 5 breeds that are most susceptible. Apparently,   
according to the chart pasted below, the GSHP and English springer spaniel are   
also somewhat at risk. Perhaps we should append some sort of disclaimer to   
this article.  
Charlie  
  
What dogs are more susceptible? Breed There is a definite link between the   
likelihood of occurrence of GDV and the breed and build of the dog. GDV is   
much more likely to occur in large breeds with deep, narrow chests. The   
problem can occur in small dogs, but only rarely. The University of Purdue   
conducted a study of hundreds of dogs that had developed GDV, and they   
calculated a ratio of likelihood of a particular breed developing the problem as   
compared to a mixed breed dog. For example, using the GDV risk ratio, a   
Great Dane is 41.4 times more likely to develop GDV than a mixed breed dog.   
Breed GDV Risk Ratio Risk Rank Great Dane 41.4 1 Saint Bernard 21.8 2   
Weimaraner 19.3 3 Irish Setter 14.2 4 Gordon Setter 12.3 5 Standard   
Poodle 8.8 6 Basset Hound 5.9 7 Doberman Pinscher 5.5 8 Old English Sheepdog   
4.8 9 German Shorthaired Pointer 4.6 10 Newfoundland 4.4 11 German   
Shepherd 4.2 12 Airedale Terrier 4.1 13 Alaskan Malamute 4.1 14 Chesapeake Bay   
Retriever 3.7 15 Boxer 3.7 16 Collie 2.8 17 Labrador Retriever 2 18   
English Springer Spaniel 2 19 Samoyed 1.6 20 Dachshund 1.6 21 Golden   
Retriever 1.2 22 Rottweiler 1.1 23 Mixed 1.0 24 Miniature Poodle 0.3 25  
  
Posted by Mike Spies at \_3:31 PM\_   
(<http://wenaha.blogspot.com/2010/04/hunting-dog-hypoglycemia.html>)   
(<http://www.blogger.com/email-post.g?blogID=5177950893772260581&postID=8320019352125304008>)   
(<http://www.blogger.com/post-edit.g?blogID=5177950893772260581&postID=8320019352125304008&from=pencil>)