MEGAESOPHAGUS IN THE GSD

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Congenital megaesophagus – inherited, seen in various breeds including the GSD. (possibly an autosomal dominant with incomplete penetration.) These present at around 4-5 weeks of age when solid feeds are started.

The typical picture is one of vomiting within 5-10 minutes of eating; puppies affected are often small and weedy due to lack of adequate food getting through to the stomach. Many have a dilation of the throat after eating and a persistent gurgle.

There are two basic types of congenital megaesophagus

1. **Persistent right aortic arch (PRAA)** – this is actually a vascular abnormality that results in constriction of the oesophagus over the base of the heart, causing a build up of food forward of the obstruction.

   Signs – regurgitation of solid foods almost immediately after eating – seen from 3-4 weeks of age. These are diagnosed by their very characteristic appearance on barium X-ray. The oesophagus is grossly dilated in front of the heart and appears pinched over the top of the heart. Behind the heart, the oesophagus should appear normal. Because of the persistent right aortic arch, the oesophagus gets "pinched" over the top of the heart, resulting in dilation of the proximal oesophagus.

   These can be corrected surgically (usually not before 12-14 weeks of age), however as the operation is intra-thoracic, the puppy usually has to be of a reasonable size and weight before operation.

   The prognosis can be guarded as the puppies are often thin, undersized and there can be secondary pneumonia. Most improve dramatically after the operation, however some can persist in having some dilatation in the 1st 1/3 of the oesophagus. Feeding of these puppies is by giving them a more liquid diet and feeding them with the food in an elevated position so as to provide a straighter passage through the chest to the stomach.

2. **Megaesophagus –** these puppies have a grossly dilated oesophagus affecting the entire length of the oesophagus. Occasionally, the two condtions can occur in the one puppy.
   Signs - Like the type of abnormality above, similar symptoms occur. In this type of abnormality, the actual nerve innovation of the entire length of the oesophagus is abnormal. The muscles cannot contract properly, so the food fails to move through.

   These cases often have secondary chest infections due to the constant vomiting and regurgitation. Again these are diagnosed by barium X-ray. There is gross dilation of the oesophagus the entire length from the top to the entrance to the stomach.
   The prognosis is extremely poor as the major defect in the muscles affects the passage of food to the stomach and the condition is not readily correctable.

3. **Acquired Megaesophagus** – this condition can develop in the older animal (usually over 5-7 years of age) from a variety of reasons.

Inheritance of Megaesophagus in the GSD

There are several are breeds with an increased incidence of congenital megaesophagus. These breeds include the Miniature Schnauzer and Wirehaired Fox Terriers where it is considered inherieted. Familial predispositions have been reported in Irish Setters, GSD, Shar Peis, Great Danes, Greyhounds, Newfoundlands, Pugs and cats.

There are several different methods of inheritance proposed for this condition in different breeds. This condition was considered to be inherited (in 2001) as an autosomal recessive in the Wire Haired Fox Terrier. In the Minature Schnauzer the condition is considered to be an autosomal dominant or 60% penetrance autosomal recessive. This information was updated in 2005 to have both breeds inheriting the condition as an autosomal dominant.

There have been several proposed methods of inheritance of this condition in the GSD.

I myself have had several megaesophagus puppies during our kennel's life time. In one litter there were 3 puppies, as I mated the bitch rather late – at 4 weeks just after I started weaning them, 2 of the three started to develop a pouch in the left side of the throat after feeding. One puppy started vomiting after feeding. I barium x- rayed all 3 puppies – 2 had megaesophagus – very dilated along the whole length of the oesophagus.

I euthanaised both affected puppies and desexed the third puppy. I had a very good idea of where the problem came from and I deliberately re-mated the bitch to a ¾ brother the next time around. The bitch was mated on time, 9 puppies, all normal.

The bitch was from German lines but bred in the UK. She had no other affected puppies to any sire.

In my opinion, the condition is most probably inherited as an autosomal dominant, but with a variable penetrance. In other words it only takes one parent to tango. Not all puppies are affected, some with go on to throw the problem in a variable manner, others will breed clear.

The interesting thing is that some puppies may have a gurgle and grow out of the problem I have seen this occur in a few puppies Minature Schnauzers and Rhodesian Ridgebacks. The incidence can be as high as 50% in a litter, but some may develop better nerve innervervation of the muscles of the oesophagus over time, and turn, or *go normal*.

Nerve development can continue after birth. How much can occur is still poorly understood (at least in dogs as far as I know).