Angell-Boston’s Neurology Specialty Service Offers State-Of-The-Art Treatment

The Neurology group at Angell-Boston is uniquely qualified to offer diagnostic evaluation and treatment in the specialty of small animal neurology.

Our staff is comprised of two board-certified staff neurologists and a neurology resident who work collaboratively to provide optimal care in the diagnosis and treatment of small animal neurological diseases, both medical and surgical, with state-of-the-art equipment and facilities.

In keeping up with technology, the Neurology group at Angell recently implemented a new, ventrally-placed locking bone plate technology for surgical cervical stabilization of large breed dogs affected with dynamic and static stenosis forms of cervical vertebral instability (Wobbler Syndrome). This condition most commonly occurs in Doberman, Rottweiler and Great Dane dogs. However, older Dalmatians are also commonly affected. The success rate of correcting a dog’s abnormal gait with this method has been quite high. Most dogs are able to go home to the owners within 24 hours of surgery with improvement in clinical signs becoming evident within just a few days after surgery.

Angell’s neurologists diagnose and treat a wide range of both medically and surgically treated neurological diseases affecting veterinary patients including seizure disorders, vestibular disease, intervertebral disc disease, encephalitis, vertebral malformations and instability, brain tumors and neuromuscular diseases like myasthenia gravis. Commonly used diagnostic aids include evaluation of radiographs, cerebrospinal fluid analysis, MRI and CT scans, myelograms and electrodiagnostic testing which includes evaluating nerve conduction velocities as well as other electrophysiologic parameters.

For more information or to refer clients to the Neurology Service at Angell-Boston, please call Natasha Bureau at 617 541-5140, email neurology@mspca.org or visit www.mspca.org/neurology.

Update: Amplatz Canine Ductal Occluder

For over ten years, the closure of a patent ductus arteriosus (PDA) could be accomplished in some patients using catheter-based delivery of embolization coils. However, many patients still required thoracotomy for ductal ligation as only smaller PDAs with an appropriate tapering morphology were good candidates for coil embolization. In addition, the placement of coils could be tricky and sometimes resulted in embolization of a coil into the pulmonary arterial circulation. Patients who had unsuccessful procedures required a thoracotomy.

A variety of occlusion devices are available for use in human patients for PDA closure. Although these systems can sometimes be used successfully in canine patients, their shape and design are not ideal for the canine ductus and the pricing of the devices remains high.

Early this year, after a large clinical series was successfully completed on dogs, Infiniti Medical commercially released the Amplatz Canine Ductal Occluder (ADO). This device was specifically designed for the canine species. It comes in a variety of sizes ranging from 3 mm to 14 mm. The ADO has facilitated closure of most PDA sizes and shapes in the cardiac catheterization lab. Although the device is not inexpensive ($650.00 USD), the pricing was made as veterinary-friendly as possible. It is clear based on our initial experience with the device that any increase in equipment cost will be offset by the decrease in anesthesia time and expense. Obviously, avoiding thoracotomy in many patients will significantly reduce recovery time and intensity of post operative management and expense.

When presented with a patient with a PDA, the ductal size and shape is determined echocardiographically, paying close attention to measurements of the pulmonic ostium (where the ductus connects with the main pulmonary artery). This allows approximate determination of appropriate occluder size and size of associated delivery catheters. A variety of device sizes are stocked as the angiographic measurements do not always correlate perfectly with those taken from the echocardiogram. Once the patient is under general anesthesia, vascular entry is achieved from the femoral artery. A calibration catheter is positioned into the ascending aorta and a contrast injection is made to evaluate the size and shape of the ductus. This allows precise measurement and selection of the proper device size, the most critical part of the procedure. The ADO is delivered through a catheter directed through the ductus.
“Syndrome X”: Some Still Do Not See It as a Disease
Rebecca L. Remillard, PhD, DVM, MS, DACVN

There is an increased production of pro-inflammatory and a decrease in anti-inflammatory proteins by adipose tissue as the fat mass increases. This dysregulation between pro- and anti-inflammatory mediators from adipocytes may be the result of a local hypoxia that occurs within adipose as the fat mass increases. Subsequently, there is an increased plasma level of acute phase proteins and inflammatory cytokines which has led to the view that the obese patient is in a chronic state of low-grade inflammation. As abdominal fat stores increase, levels of circulating adipokines increase with increased levels of circulating free fatty acids (FFAs) and there is a concomitant decrease in insulin sensitivity (type 2 diabetes) in people and mice models. TNF-alpha and IGF-I have been shown to disrupt the intracellular transduction signals of insulin. These patterns are also in a chronic state of oxidative stress with increased circulating ROS (reactive oxygen species) levels which play a critical role in the pathogenesis of vascular and glomerular diseases.

Information published in the last 3-4 years would support that ‘Syndrome X’ is occurring in our obese patients as well. Plasma TNF-alpha and IGF-I levels were significantly higher when ideal weight dogs were made obese. (Blundard et al 2004) (Gayet et al 2003). Dogs made obese had increasing levels of TNF-alpha and became progressively insulin resistant as fat mass increased (Gayet et al 2009). Plasma TNF-alpha and IL-1 were significantly higher and insulin sensitivity decreased in males with a higher percent of body fat (Vick et al 2007).

Again, the good news is that these trends are reversible with weight loss. Obese cats placed on a 12 week weight loss diet had significantly lower plasma markers of oxidative stress and inflammation (Takeda et al 2004). Weight gain in dogs resulted in decreased insulin sensitivity; however, during weight loss, insulin sensitivity returned to pre-obese state (Gayet et al 2003). TNF-alpha and IGF-I levels were significantly lower and insulin sensitivity increased when obese dogs lost weight to ideal (Blundard et al 2004)

I believe features of the Metabolic Syndrome ‘Syndrome X’ as described in people in our overweight/obese patients suggest obesity is causing a chronic low grade highly oxidative pro-inflammatory state in our patients with a newly recognized overweight condition (renal insufficiency, pancreas, degenerative diseases and advanced aging) to obesity. Therefore, from every opportunity, from the first puppy wellness to the geriatric cat, we are obligated to speak with owners about weight control.

Update

The primary artery in the main pulmonary artery. The ADO is advanced until the proximal phalange is deployed. The catheter is then pulled back into the ductus until the retention device engages. The second phalange is then deployed into the ductal ampulla where, if sized and positioned appropriately, it will be in a stable, secure position. Completion of closure of the ductus is generally noted within fifteen minutes of placement. The main limitation for the use of the ADO is the size of the patient, as the delivery device range in size from 8 – 10 French (quite large related to the small vessel size in some patients). Patients ranging in size from 20 kg to 32 kg have been successfully treated using the ADO thus far, making this the preferred therapeutic option for most patients with patent ductus arteriosus.

For more information or to refer cases to the cardiology service at Angell-Boston please refer to Susan Grammer at 617 541-5038, email cardiology@mspca.org or visit www.mspca.org/cardiology.