

## Effects of Stress in Young Adult Dogs Diagnosed with Diabetes Mellitus Type 1

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### COLUMN ARTICLE

Diabetes mellitus (DM) is a treatable disease requiring veterinarian and client dedicated effort. Due to the multitudinous factors that affect the diabetic state, an animal's condition can fluctuate with variable response to therapy, thus making management of DM perplexing. Each dogs/cats needs are individualised therapy plans, with frequent evaluation and alteration of that plan relying principally on the reaction of that patient.

DM is a syndrome affiliated with prolonged hyperglycemia owing to pancreatic beta cell loss or dysfunction of insulin secretion by these cells, reduced tissue sensitivity to the insulin that is secreted, or a combination of all of the mentioned. Due to the graveness of insulin deficiency at the time of diagnosis, affected animals cannot survive without insulin therapy and will advance from profound hyperglycemia to life-threatening ketoacidosis. The standard therapy for canine diabetes has always centered around dietary management and insulin replacement, usually twice daily insulin injections administered subcutaneously; dosage is

titrated based on blood glucose monitoring, urine glucose monitoring, and by clinical signs as emphasised by the patients owner [1].

After very close surveillance and tracking of a recently diagnosed DM type 1 patient, a key factor that requires more consideration is the effects of stress on DM patients. Quantification of stress induced hyperglycemia was performed in hospital, thus making the ability to distinguish the reaction of glucose between the ordinary animal and diabetic animal possible. The model patient was looked at and meticulously examined for fluctuations in glucose driven by stress and the DM type [2].

As previously narrated stress takes a significant part in etiology and DM therapy. With stress comes a physiological cascade of events that enables the sympathetic nervous system to over release catecholamines, which in turn helps promote the stress induced hyperglycemia. While the circulating catecholamine inhibits insulin secretion from the beta cells, it tends to paradoxically boost the processes of gluconeogenesis and glycogenolysis in the liver. No variation in stress induced hyperglycemia among diabetic and non-diabetic patients or stress induced processes of

hyperglycemia in DM, have been reported [3]. I've found that glucose is more susceptible to psychiatric pressure in diabetic patients than ordinary animals. This analogy is based on close supervision of the hospitalised DM patient. Interestingly the pattern for glucose changes varied depending on the lifestyle to which the patient had been acclimated. In a diabetic patient, significant rises in glucose occurred soon after stress exposure, which was sustained during and even following stress withdrawal. Examples of these stressors include but not restricted to hospital kennel confinement, noise caused by fellow patients, excessive direct human contact i.e. in hospital treatments by staff and reduced human contact alike.

### Basic physiology

In the stress hyperglycemic state stage, an instant reaction of the autonomic nervous system activates the adrenal medulla to release catecholamines. In the post stress stage, I found that the levels of cortisol increase subsequently. In comparison with ordinary glucose conditions, the glucose concentration caused by severe stressors in DM patients was considerably more notable and the enhanced amount of glucose caused by stressors was declining steadily to normal values in the patients after 3 to 4 hours [4]. Lack or disability of physiological compensatory mechanisms in the diabetic patient resulted in high impact on the state of hyperglycemia in these compromised patients. The amount of diabetic dogs/cats reaching destination or suitable glucose concentration stayed small in spite of the improvements and developments in anti-diabetic medicines and prescription veterinary diets. An acute glucose disturbance has therefore become capital priority with regards to monitoring to reduce or prevent problems, thus conclusively meeting the goal of achieving appropriate glucose level at tissue level. Various variables influence the glucose levels at tissue level. These factors must therefore be pointed out, pinned down and meticulously regulated in an attempt to handle DM patients more proficiently. Psychiatric stress, briefly listed above, is a major consideration causing increased glucose levels. The processes of gluconeogenesis and glycogenolysis escalate in the liver under the high influence of stress hormone release

i.e. glucagon and epinephrine. Subsequently there is minor absorption of glucose in muscle, with a consequential hyperglycemic state [5]. Numerous trials have demonstrated that psychiatric stress in DM patients exasperates hyperglycemia, yet the model and scope of hyperglycemia and its magnitude are still ambiguous. Furthermore, the psychiatric stress model and the associated hyperglycemia are confounding because psychiatric stress is problematic to assess and quantify objectively. Thus, our hospitalised patient provided essential cardinal information required to fully ascertain the notable effects of stress on glucose levels in DM patients. This valuable information can thus be used for more forcible management of diabetic patients, appropriately and effectively.

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