ANESTHESIA CONCERNS FOR PATIENTS WITH ADRENAL DYSFUNCTION
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Key points
- Function
- Concerns
- Method
- Monitoring

Why should we be aware of adrenal status? The adrenal cortex produces two major types of steroid hormones. These are the mineralocorticoids and the glucocorticoids. The mineralocorticoids are important in the maintenance of electrolyte balance which in turn also works to control blood pressure. The glucocorticoids work in the regulation of metabolism whether indirectly or directly through interactions with other hormones. Glucocorticoids regulate protein, fat, carbohydrate and nucleic acid metabolism. Blood glucose is raised by antagonizing the secretion and action of insulin. Glucocorticoids cause protein catabolism and fat mobilization. Anti-inflammatory action is due to effects on microvasculature and suppression of inflammatory cytokines.

Adrenocortical disease results in disturbances of body water volume and electrolyte concentrations and intracellular electrolytes may be severe.

Our canine patients with hyperadrenocorticism (Cushing’s syndrome) have physiological changes that include: slow tissue healing, polyuria/polydipsia, hypercoagulability, muscle wasting, skin changes, lethargy, polyphagia and abdominal enlargement.

It has been documented that dogs with this syndrome have alveolar-arterial oxygen gradients above the norm. This presumes that significant resting hypoxemia is present. Muscle wasting leads to muscle weakness which includes the diaphragm. Abdominal enlargement compresses the diaphragm. Hypercoagulability may lead to the development of pulmonary thromboemboli.

Anesthetic management should primarily focus on preoperative assessment and optimization. This may involve the direct effects of excess steroids such as hypertension and end organ damage. Preoperative assessment will also include recognition of associated disease such as pernicious anemia. Intraoperatively, these patients are particularly at risk of cardiovascular instability and appropriate monitoring should be considered. Postoperative management will include adequate pain control and hormone replacement therapy.

The use of an anticholinergic pre-operatively should be based on the patient’s resting heart rate. A combination of an opioid and sedative may be used to decrease the patient’s anxiety level. Pre-oxygenation is preferred if the patient is tolerant.

Induction agents may be varied. Propofol provides for rapid capture and control of the airway. It is expected that ventilation will need to be assisted with the hyperadrenalism patient due to abdominal enlargement leading to diaphragmatic compression when placed in dorsal recumbency for prep and surgery. It is important to remember also that muscular weakness may be present producing resting hypoxemia.

As it is not unusual for these patients to have existing systemic hypertension, I prefer to induce with non-invasive blood pressure and ECG in place for real time information during the prep period. Invasive blood pressure monitoring may be established once in the O.R. suite.
Access to an arterial catheter will also allow you to monitor the patient’s ventilatory and oxygenation status and make adjustments.

Maintenance with the inhalant agent, isoflurane, sevoflurane or desflurane and oxygen may be used post intubation.

In patients who have hypoadrenocortical function, Addison’s, it is important to attempt to stabilize the patient as much as possible prior to anesthesia. Current electrolyte values should be obtained the morning of the surgery or anesthesia for diagnostics in order to support them properly with crystalloid fluid therapy.

Addison's disease, also known as chronic adrenal insufficiency, is a condition in which the adrenal glands do not produce enough hormones. These hormones include cortisol, corticosterone, aldosterone and some steroids. Aldosterone, cortisol and corticosterone play a role in regulating water balance, acid-base balance, and sodium and potassium homeostasis. If at all possible, prevent stress as this patient is physiologically unable to respond. In some cases, patient status dependant, additional glucocorticoids may be given prior to anesthesia. In the postoperative period, mineral corticoid and glucocorticoid supplemental should be considered in Addison’s disease as well as exogenous, steroid induced hyperadrenalism. Premedication may be dictated by the patient’s comorbidities, if any.

Agents for induction should be based on the patient’s ASA status. It is helpful to bear in mind that the hypnotic agent, etomidate, has been shown to depress adrenal function from three to six hours post administration and should be used with caution in these patients.

Intra-operative monitoring of electrolytes may be useful to continue to guide IV fluid administration choice and is recommended in the post-operative period.