The adrenal gland and the use of cortisone in the management of patients with Addison’s disease and Cushing’s syndrome

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In this article, the author addresses the function of the adrenal glands and focuses attention on the therapeutic uses of cortisone, including its use in shock situations. Emphasis is placed on the anesthesia management of patients with Addison’s disease and Cushing’s syndrome.

In reviewing the adrenal gland, it is helpful to consider first the limbic system in the hypothalamus where corticotropin releasing factor (CRF) is secreted. CRF stimulates the anterior pituitary to release adrenocorticotropic hormone (ACTH). ACTH stimulates the synthesis and release of cortisol and androgen precursors by the adrenal gland.

ACTH is normally secreted in a diurnal fashion. High levels are secreted in the morning and by early evening, the plasma levels of ACTH begin to drop. In coordination with this, plasma cortisol levels normally fluctuate in such a diurnal fashion.

ACTH secretion is also affected by the momentary degrees of bodily stress such as cold, fever, muscular exercise, trauma, surgery and low blood sugar. ACTH secretion responds to the renin-angiotensin system and plasma cortisol levels.

Anatomy and physiology of the adrenal gland

The adrenal glands, located atop the kidneys, have two functioning regions, the adrenal medulla and the adrenal cortex. The adrenal medulla secretes epinephrine and norepinephrine, the catecholamines.

The adrenal cortex has three functioning layers. The zona glomerulosa, the outer layer, secretes the mineralocorticoid aldosterone, referred to as the “life saver” hormone. Aldosterone can be secreted without the stimulation of ACTH. The zona fasciculata, the middle layer, secretes the glucocorticoids, especially cortisol. The zona reticularis, the inner layer, secretes the androgens, sex hormones with estrogenic and androgenic potential.

Aldosterone, the major mineralocorticoid, is controlled by the renin-angiotensin system, extracellular sodium and potassium concentrations and ACTH. Aldosterone is known as the “life saver” hormone because alterations in its secretion can lead to severe electrolyte imbalances or hypotension secondary to volume depletion. It acts at the distal convoluted renal tubule to promote sodium and water retention, potassium and acid secretion.

The renin-angiotensin system responds to hypovolemic stress. Low extracellular volume results in renin stimulation, bringing about the synthesis of angiotensin I which is converted to angiotensin II in the lungs. Angiotensin II is an important peripheral vasoconstrictor and a potent stimulator of aldosterone production which, in turn, tends to correct the deficit in extracellular volume.

Hyposecretion of aldosterone results in hyper-
kalemia, hyponatremia, hypochloremia or mild acidosis and hypovolemia.³

Cortisol, the major glucocorticoid, is responsible for the control of carbohydrate, fat and protein metabolism.⁵ Cortisol is a potent stimulator of glucose production from the liver (gluconeogenesis) and an antagonist to the function of insulin.² Cortisol normally raises blood sugar levels.

Cortisol is important for the maintenance of blood pressure. This is related to its effects on sodium elimination and most importantly, because it sensitizes the vascular structures to pressor stimuli. Such a reaction is important in times of stress.

Cortisol is important in the negative feedback of ACTH production by the pituitary. This is referred to as the hypothalamic-pituitary-adrenal axis or the pituitary-adrenal axis. Inadequate levels of cortisol may occur as a result of disease or trauma to the central nervous system or the pituitary.⁴

Cortisol can interact directly with the genetic material of the cell to bring about specific enzyme induction or inhibition.

The normal diurnal variation of cortisol can be 20 μg/cc in the early morning and 5 μg/cc in the late evening. Roughly 15-20 mg of cortisol are secreted each day and are tightly bound by the cortisol binding globulin transcortin. The free cortisol in the plasma is probably physiologically active.

In stress situations such as shock, trauma, blood volume depletion, infection, surgery and general anesthesia, enormous surges of cortisol production should occur. Normal cortisol production during stress is 10 mg/hour or 240-300 mg/day.

### Adrenal insufficiency-Addison’s disease

In 1855, Thomas Addison first described the clinical syndrome of adrenal insufficiency which bears his name. He recognized and demonstrated that with the presence of disease of the adrenal glands, the patient exhibits weakness, fatigue, hyperpigmentation, anemia, feeble heart action, hypotension, and gastro-intestinal irritability.

In 1896, William Osler administered a glycerin extract of fresh hog adrenals to these patients. Striking clinical improvement followed the oral administration of this crude extract. In retrospect, it seems that the potency of this extract could not have been very great. Since many of the patients with Addison’s disease also had active tuberculo-

The status of the patient with adrenocortical insufficiency can vary from the precarious asthenia of untreated severe Addison’s disease to a complete lack of symptoms.²

The Waterhouse-Friderichson syndrome is characterized by acute collapse. It is caused by bilateral hemorrhage into the adrenal glands and can occur in newborns. In adults, it can be caused by anticoagulant therapy, sepsisemia and vasculitis.² Surgical removal of the adrenal glands also induces adrenal insufficiency.

Stress precipitates acute adrenal insufficiency in the patient with latent adrenal insufficiency. Patients suspected of latent adrenal insufficiency include those with urogenital tuberculosis or those diagnosed as having “chronic appendicitis” or “chronic cholecystitis.”⁶

Chronic adrenal insufficiency is characterized by hyperpigmentation due to unopposed ACTH secretion.⁴ A list of symptoms of Addison’s disease compiled in 1980 is in Table I.⁵ Chronic adrenal insufficiency from granulomatous causes include histoplasmosis, tuberculosis and metastatic disease.⁶

Autoimmune adrenalitis is now the most common cause of adrenal insufficiency. When there is production of both adrenal antibodies and thyroid antibodies, this condition is known as Schmidt's disease.

The adrenogenital syndrome in infants is congenital or acquired enzymatic defects of either cortisol or aldosterone synthesis. These infants exhibit volume depletion and signs of excessive androgen production—virilism. Treatment is with suppressive doses of cortisone.²

In adrenocortical insufficiency due to pituitary malfunction and lack of ACTH, hyperpigmentation does not occur and electrolyte disturbances are not as severe because aldosterone production continues.⁴ Patients with this secondary

<table>
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<th>Table I</th>
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<tbody>
<tr>
<td>Symptoms of Addison’s disease</td>
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<tr>
<td>Hypotension</td>
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<tr>
<td>Hyponatremia</td>
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<tr>
<td>Hyperkalemia</td>
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<tr>
<td>Weight loss</td>
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<tr>
<td>Weakness</td>
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<tr>
<td>Anorexia</td>
</tr>
<tr>
<td>Salt craving</td>
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<tr>
<td>Hypoglycemia—more likely seen in children</td>
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<tr>
<td>Hyperpigmentation</td>
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adrenal insufficiency seldom have hyperkalemia as a presenting feature.\textsuperscript{7}

Iatrogenic Addison's disease or secondary adrenal insufficiency is seen in patients who have been on cortisone therapy. Although there is no damage to the adrenal cortex, it fails to respond due to the presence of exogenous cortisol.\textsuperscript{8} A patient who has been on cortisone therapy in the past may have no symptoms of suppression of the pituitary-adrenal axis during daily activity, however, he will not be able to handle a stress situation.\textsuperscript{2}

The higher the dose of cortisone and the longer it is used, the greater is the length of time that the pituitary-adrenal axis will remain suppressed following cessation of therapy. However, there are documented cases in which patients were treated for only two weeks, yet they demonstrated significant pituitary-adrenal axis suppression for 8-12 months following cessation of therapy.

Alternate day cortisone therapy may not depress the pituitary-adrenal axis as much, however, these patients should be treated as Addisonians when coming for surgery.\textsuperscript{2} Surgical patients, especially those with chronic obstructive pulmonary disease, inflammatory bowel disease and arthritis should be questioned for a history of cortisone therapy prior to surgery. Topical cortisone treatment may also lead to suppression of the pituitary-adrenal axis in psoriasis patients.

Physical findings which might suggest use of cortisone include thinning of the skin, hypertension, moon facies, truncal obesity, buffalo hump, plethoric cheeks and proximal muscle weakness.\textsuperscript{5}

Addisonian crises or full blown acute adrenal insufficiency will present a patient who is in shock with hyponatremia, hyperkalemia and hyperbilirubinemia. The hyperkalemia may be masked if vomiting and diarrhea accompany the stress state.\textsuperscript{4} In all of these conditions, cortisone therapy will be necessary. In the literature, all are termed Addisonian.

**Diagnostic tests for adrenal insufficiency:** The Cortrosyn\textsuperscript{®} (cosyntropin) stress test can be used for in-patients or on an out-patient basis. A baseline plasma cortisol is drawn, Cortrosyn (a synthetic ACTH) is given, and plasma cortisol is measured again at 30 and 60 minute intervals. In the normal patient, the plasma cortisol should rise by 8-12 mg/cc from baseline.

For an in-patient, a 24-hour urine collection can be done to measure the 17-ketosteroids.\textsuperscript{2}

When a patient is strongly suspected of having adrenocortical insufficiency in a stress situation, such as a patient under anesthesia in shock, a plasma cortisol level should be drawn and a stress dose of cortisone administered. When the cortisol level result comes back several days later, you can judge if that patient was in a depleted cortisol state. Comparison is made between a natural stress situation as opposed to the simulated stress situation of the Cortrosyn stress test. If the cortisol level comes back as normal (at the time of stress), the patient has adrenocortical insufficiency. In the stressed patient, the cortisol level should be 10-30 mg/cc above the normal range. If a higher result is obtained, rapid cortisone tapering can be done. There will be no endogenous cortisol suppression.\textsuperscript{4}

**Management of the patient with adrenocortical insufficiency**

When surgery is necessary, the patient should be managed as if he is in acute adrenal crisis.\textsuperscript{2} A cortisol deficiency may result in hypotensive demise under anesthesia unless the condition is recognized and treated.\textsuperscript{8} Elective surgery in the febrile Addisonian should be deferred since persistent fever perioperatively raises the possibility of impending crises.\textsuperscript{7}

Preoperative laboratory studies should include blood glucose, electrolyte determinations and assessment of volume depletion. Hyponatremia or hyperkalemia will indicate that the patient is being under-treated with cortisone. Normal potassium levels or hypokalemia may be found in the patient with vomiting and diarrhea.\textsuperscript{2}

The diabetic patients on insulin therapy with Addison's disease or diabetics who have had a transphenoidal hypophysectomy are exquisitely sensitive to insulin. The sliding scale insulin dosage is reduced to half-dosage.\textsuperscript{2}

In patients with pan-hypopituitarism, maintenance cortisone therapy is used because the pituitary controls the adrenals with ACTH, and these patients will require stress replacement dosage in preparation for anesthesia.\textsuperscript{2}

After total adrenalectomy or in primary adrenal insufficiency, fluorohydrocortisone must be given to replace the mineralocorticoid.\textsuperscript{2}

Premedication may be administered according to the individual patient needs. In the normal patient, preoperative apprehension does not seem to elicit activation of the adrenal cortex to produce plasma levels of cortisol much above the upper level of diurnal variation.\textsuperscript{2}

**Intravenous fluids:** The Addisonian patient should be assumed to be sodium depleted and...
treated with isotonic saline until the deficit is corrected. Glucose is needed because of the propensity of the Addisonian to develop hypoglycemia. If kidney function is normal, potassium chloride 20 mEq/L should be added to the intravenous solution to prevent postoperative hypokalemia.

Cortisone dosage: Maintenance dosage of cortisone in the Addisonian is 30 mg/day (see Table II). To mimic the normal diurnal variation, 20 mg is given in the morning and 10 mg is given in the late afternoon. If the patient needs mineralocorticoid, fluorohydrocortisone 0.05-0.1 mg/day is given. Because of irritation of the gastric mucosa from cortisone, patients are advised to take their cortisone with meals, milk or an antacid.

Preoperative stress dosage of cortisone in the Addisonian: Depot forms of cortisone or methylprednisolone are recommended on the evening before surgery. The sustained action can cover administration error or delay on the day of surgery. If a patient is on cortisone therapy for reasons other than Addison's disease (such as connective tissue disorders), he or she may need methylprednisolone at least 2-3 days before surgery to prevent exacerbation of the patient's basic disease at the time of surgery.

Hydrocortisone 300 mg is given on the day of surgery based on the fact that normal cortisol production stress is 10 mg/hour or 240-300 mg/day. A dose of 100 mg of hydrocortisone is given with the premedication and 100 mg is given in the intravenous solution during surgery. Every 6 hours thereafter, a 50 mg dose is administered with gradual tapering in succeeding days until the replacement dosage is reached.

The degree and duration of adrenocortical suppression following cortisol therapy cannot be determined, therefore, the policy of overtreatment persists. When persistent hypotension develops under anesthesia and all other causes of shock have been ruled out, hydrocortisone 100 mg intravenously may be given and an intravenous infusion to run at 10 mg/hour should be started. Following this regimen, a dose of vasopressor which had previously been ineffective may produce the desired results.

Adequate replacement therapy: After a crisis situation and after ACTH testing and maintenance cortisone therapy is underway, adequate replacement is accomplished when there is normal blood sugar, normal blood pressure and normal serum electrolytes.

Anesthesia management: The cornerstone of adequate anesthetic management is adequate cortisone supplementation, otherwise the effects of the anesthetic agents and surgery cause stress and hormonal response in all patients. A recent study on high dose fentanyl anesthesia seems to indicate that this is a good anesthetic choice in patients with adrenocortical insufficiency.

A total of 22 patients for coronary artery bypass graft were selected; 10 patients were given fentanyl 100 μg/kg; and 12 were given halothane. The study showed that high dose fentanyl significantly attenuated the hormonal response to surgical stimulation. There was a decrease in plasma

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**Table II**

A suggested schedule for preoperative and postoperative steroid maintenance in the patient with adrenal insufficiency.

<table>
<thead>
<tr>
<th>Preoperative</th>
<th>Postoperative</th>
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<tbody>
<tr>
<td>Evening before operation:</td>
<td>Day of surgery</td>
</tr>
<tr>
<td>Cortisone acetate 100 mg IM at 9:00 p.m.</td>
<td>Cortisol 50 mg IV or IM every six hours.</td>
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<tr>
<td>Morning of operation:</td>
<td>Postoperative day #1</td>
</tr>
<tr>
<td>Cortisol 100 mg IV in a bolus prior to induction of anesthesia. Cortisol 100 mg IV during surgery.</td>
<td>Cortisol 50 mg IM every six hours.</td>
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<table>
<thead>
<tr>
<th>Postoperative day #2</th>
<th>Postoperative day #3</th>
</tr>
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<tbody>
<tr>
<td>Cortisol 50 mg IM every eight hours.</td>
<td>Cortisol 30 mg IM or orally every eight hours.</td>
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<thead>
<tr>
<th>Postoperative day #4</th>
<th>Postoperative day #5</th>
</tr>
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<tbody>
<tr>
<td>Cortisol IM or orally: 8:00 a.m. 40 mg, 2:00 p.m. 20 mg.</td>
<td>Cortisol IM or orally: 8:00 a.m. 30 mg, 2:00 p.m. 10 mg.</td>
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<tr>
<th>Postoperative day #6</th>
<th></th>
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<tbody>
<tr>
<td>Cortisol orally: 8:00 a.m. 20 mg, and 2:00 p.m. 10 mg.</td>
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catecholamines and cortisol and there were no changes in plasma levels of renin, vasopressin or aldosterone. The patients administered fentanyl were more deeply anesthetized than were the patients given halothane. It is possible that deeper levels of halothane might have attenuated the stress response, but a higher concentration is not well tolerated by patients with coronary artery disease.

Cushing's syndrome

Adrenocortical hyperfunction is due to excessive production of cortisol by the adrenal cortex secondary to excessive stimulation by ACTH or by autonomous secretion from an adrenal tumor. Ectopic ACTH secretion can come from cancer of the lung, especially oat cell cancer, or tumors of the pancreas, kidney or thymus. It is 2-3 times more common in women and is most commonly seen between 20-50 years of age.

Symptoms of Cushing's syndrome are related to the degree and duration of the excess cortisol production (Table III). The most common cause of Cushing's syndrome is the use of pharmacologic doses of cortisone. This iatrogenic Cushing's syndrome is also associated with intercranial hypertension, cataracts, pancreatitis and aseptic necrosis of the hips.

Diagnostic tests: Measurement of plasma cortisol levels will show elevation in Cushing's syndrome. However, the plasma cortisol levels are also elevated in patients on estrogens, in acute stress and in alcoholism. The absence of diurnal variation reveals autonomous production of ACTH or cortisol.

Table III

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<th>Symptoms of Cushing's syndrome</th>
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<tr>
<td>Plethoric facies</td>
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<tr>
<td>Thin skin</td>
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<tr>
<td>Abdominal striae</td>
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<tr>
<td>Easy bruising to hemorrhagic diathesis</td>
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<tr>
<td>Hypertension</td>
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<tr>
<td>Hirsutism</td>
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<tr>
<td>Osteoporosis</td>
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<tr>
<td>Centripetal obesity</td>
</tr>
<tr>
<td>Buffalo hump</td>
</tr>
<tr>
<td>Muscle weakness</td>
</tr>
<tr>
<td>Poor wound healing</td>
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<tr>
<td>Hypokalemia</td>
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<tr>
<td>Alkalosis</td>
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<tr>
<td>Abnormal glucose tolerance and/or diabetes mellitus</td>
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<tr>
<td>Impaired growth in children</td>
</tr>
<tr>
<td>Psychologic difficulties</td>
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</tbody>
</table>

The short dexamethasone suppression study is best in the diagnosis of Cushing's syndrome. Skull films to determine the size of the sella turcica are indicated in patients with bilateral adrenal hyperplasia. CAT scans are useful in determining pituitary size, pituitary tumor and location of adrenal tumor. Metopirone® (metyrapone) may be used as a therapeutic agent as well as a diagnostic test. This blocks the synthesis of cortisol and establishes an euadrenal state prior to surgery in the patient with Cushing's syndrome.

Treatment of the patient with Cushing's syndrome includes transphenoidal hypophysectomy for removal of the pituitary adenoma. In children, pituitary irradiation is used. Bilateral adrenalectomy may be necessary for severe adrenal hyperplasia. In the patient with cancer of the adrenal or unilateral adenoma, surgical excision is indicated. In the patient with an ectopic ACTH secreting tumor, isolated tumor excision is desirable, however, as with oat cell cancer of the lung, diagnosis of Cushing's syndrome is not made before metastasis occurs.

Anesthetic management: With stress, cortisone replacement therapy is indicated, as for the Addisonian patient. Electrolyte balance must be established as hypokalemia is a frequent problem. Hypertension, polycythemia and increased blood volume can be precursors of congestive heart failure, thus furosemide administration may be necessary. Central venous pressure and pulmonary artery wedge pressure monitoring should be used during surgery.

Assessment of the airway and veins preoperatively is important since intubation and venipuncture may present special problems. Considering osteoporosis, x-rays should be reviewed for evidence of subclinical pathological fractures. In addition, careful positioning of the patient is necessary. Enflurane anesthesia has been recommended for the patient with hypercortisolism.

Postoperatively, cortisone maintenance will be necessary after adrenalectomy or hypophysectomy. Potassium will be necessary until oral intake resumes. Pancreatitis from surgical retraction in adrenalectomy is a serious postoperative complication. The incidence of postoperative wound infection and slow healing is also high.

Cortisone

In studying adrenal function and dysfunction and the use of cortisone, the terms physiologic dosage, maintenance dosage, pharmacologic dosage and stress dosage intermingle. Cortisone
dosage can be considered from these various approaches. Six therapeutic principles in the use of cortisone are:

1. For any disease in any patient, the appropriate dose to achieve a given therapeutic effect must be determined by trial and error and must be re-evaluated from time to time as the stage and activity of the disease alter.

2. A single dose of cortisone, even a large one, is virtually without harmful effects.

3. A few days of cortisone therapy in the absence of specific contraindications is unlikely to produce harmful results except at the most extreme dosage.

4. As cortisone therapy is prolonged over periods of months and to the extent that the dose exceeds the equivalent of substitution therapy, the incidence of disabling and potentially lethal effects increases.

5. Except in adrenal insufficiency, the administration of cortisone is neither etiological nor curative therapy but only palliative by virtue of the anti-inflammatory effects.

6. Abrupt cessation of prolonged cortisone therapy is associated with significant risk of adrenal insufficiency, severe enough to be life threatening.

Physiologic, substitution, replacement or maintenance dosages of cortisone are necessary where there is adrenal insufficiency. The dose schedule mimics the normal diurnal variation and must be tailored upwards for the stress situations.

The pharmacologic or therapeutic dosage of cortisone in nonendocrine disease is that which is the smallest, yet will still achieve the desired effect. Therapeutic cortisone may be used in the treatment of a number of diseases (Table IV).11

Cortisone treatment in shock: There is controversy as to cortisone dosage in shock. Two possibilities are physiologic dosage or massive dose therapy. Physiologic doses are indicated in patients in shock who are unresponsive to usual therapy. Because of the possibility that subclinical adrenal insufficiency may be present, all patients in shock who are unresponsive to usual therapy should be given hydrocortisone 200 mg by rapid intravenous injection.11

Massive dose cortisone therapy has been in use in heart surgery for some time. The rationale for cortisone use is maintenance of adequate systemic flow and adequate perfusion pressure. Methylprednisolone at 30 mg/kg has been shown to reduce systemic vasoconstriction associated with low flow states.

A variety of beneficial effects are seen in relation to this effect of the massive dose of methylprednisolone. In vessels, there is the reduction of vasoconstriction in response to the catecholamines. There is improved circulation in the vascular beds rich in alpha receptors, such as the kidneys, intestines, lungs and skin. Platelet adhesiveness, white blood cell clumping and red blood cell delivery of oxygen to the tissues are also reduced. There is improvement in the coronary blood flow with concurrent reduction in myocardial ischemia. There is improvement in cell membrane integrity and a decrease in lysosomal enzyme release.12

All of these effects are desirable in the shock state. A hydrocortisone dosage of 50 mg/kg tends to "normalize" cardiac output and total peripheral resistance. The earlier the steroids are given, the better is the response. Subsequent dosages are repeated every 4, 6 or 8 hours depending on the response and are discontinued after 24-48 hours.13

Pharmacologic interactions with cortisone

Depolarizing muscle relaxants: There is a 50% decrease in plasma butyrycholinesterase activity in patients who receive large doses of cortisone. The cholinesterase enzyme is responsible for the hydrolysis of both succinylcholine and the ester-type local anesthetics (such as procaine and tetracaine).12

In addition, both Addisonian and Cushing's syndrome patients have muscle weakness in their profiles. Both of these factors should alert us to use conservative doses of depolarizing muscle relaxants.

Pancuronium: Pancuronium has an amino steroid structure, therefore, its effect may be antagonized in patients receiving high dose cortisone treatment.14

### Table IV

**Diseases which respond to therapeutic cortisone**

- Rheumatoid arthritis
- Osteoarthritis
- Rheumatic carditis
- Renal disease
- Collagen disease
- Allergic diseases
- Bronchial asthma
- Ocular diseases
- Skin diseases
- Diseases of the intestinal tract
- Cerebral edema
- Shock
In a case report of rapid termination of pancuronium-induced paralysis in a patient receiving high dose cortisone, the possible causes of this effect were considered due to the similarity of their steroid nuclei, competition at the myoneural junction, altered protein binding or induction of hepatic biotransformation.15

Another case report of prolonged action of neuromuscular blockade shows a patient receiving pancuronium in the presence of inadequate cortisone replacement. This prolonged neuromuscular blockade was partially antagonized by hydrocortisone.16

Pancuronium and malignant hyperthermia: Pancuronium and althesin (alphadione), both with steroid structure molecules, are not only safe for patients with malignant hyperthermia but may also be indicated to abort the malignant hyperthermia reaction, as proven in research.14

Narcotic antagonists: In animal studies, cortisone, ACTH, L-dopa and propranolol have been reported to potentiate narcotic antagonists; whereas, atropine and phystostigmine appear to inhibit narcotic antagonists.17

Enzyme induction: Cortisone is listed among barbiturates, alcohol, cigarette smoke, cannabis and dilantin as drugs capable of inducing liver microsomal enzymes. In the clinical situation, this interaction is known as tolerance or cross tolerance. This effect occurs with endogenous cortisol, also as mentioned in the review of the adrenals.14

Conclusion

The adrenocortical function has been reviewed in this article to remind the anesthetist of its importance and interrelationship with the practice of anesthesia. Cortisol, the major glucocorticoid, is important in times of stress because it sensitizes the vascular structures to pressor stimuli.

In Addison’s disease and Cushing’s syndrome, the pivotal point in anesthesia management is adequate cortisone supplementation to cover the period of stress. In both adrenocortical insufficiency and hyperfunction the high dose fentanyl technique is a good choice of anesthetic because it elicits minimal hormonal response. Enflurane can be used in the patient with Cushing’s syndrome as it reduces the plasma cortisol level.

The anesthetist must know when to use cortisone in a variety of clinical situations. The dosage of cortisone can vary from stress supplementation in Addison’s disease and other major medical diseases to massive doses in the patient in extremis. A pharmacological understanding of cortisone can aid the anesthetist in applying its use in special situations, for example, in the patient with malignant hyperthermia.

REFERENCES

(3) Bransome ED, Jr. 1980. Diagnosis and management prior to surgery of adrenal insufficiency and pheochromocytoma. AANA Annual Meeting.

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