

Peri-operative Anesthetic Management of Patients With Suprarenal Gland Diseases

ESSAY

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anesthesia

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List of abbreviations

ANS	-----	Autonomic Nervous System.
APA	-----	Aldosterone-Producing Adenoma.
ACTH	-----	AdrenoCorticoTropic Hormone.
ACE	-----	Angiotensine Converting Enzyme.
AI	-----	Adrenal Insufficiency.
ARR	-----	Aldosterone:Renin Ratio.
DHEA	-----	DeHydroEpiAndrosterone.
ICP	-----	Intra Cranial Presure.
IM	-----	Intra Musculer.
IV	-----	Intra Venous.
LD-DST	---	Low-Dose Dexamethasone Suppression Test.
MEN	-----	Multiple Endocrine Neoplasia.
PAC	-----	Plasma Aldosterone Concentration.
PRA	-----	Plasma Renin Activity.
TRH	-----	Thyroid-Releasing Hormone.
TSH	-----	Thyroid-Stimulating Hormone .
UFC	-----	Urinary-Free Cortisol.

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Introduction

The adrenals compared to the body as a whole are small but they produce some major hormones and as such, medical conditions arising from them often have profound effects on the body(**Haley , 2011**).

The adrenal gland can be divided into two very distinct zones, each of which produces specific hormones: The inner part of the adrenal or medulla produces and secretes amine hormones, adrenaline (epinephrine) and noradrenaline (norepinephrine). The medulla is essentially a sympathetic ganglion where the postganglionic cells have become secretory cells named chromaffin cells (also called pheochromocytes). The outer part of the adrenal gland or cortex makes up the majority of the gland. Three main types of hormones are secreted; mineralocorticoids, glucocorticoids, and androgens. Each is produced in a different part of the cortex the outer zona glomerulosa producing mainly the mineralocorticoid, aldosterone, the middle zona fasciculata producing glucocorticoids e.g. cortisol, the innermost layer the zona reticulosa producing, androgens (**Ganong , 2009**).

Pheochromocytomas arise from chromaffin cells in the adrenal medulla and in other paraganglia of the sympathetic nervous system. They are usually benign tumours and the clinical features depend on the activity of the tumour and the relative amounts of epinephrine and norepinephrine secreted. Typical signs and symptoms may include hypertension, hyperglycaemia, headache, palpitations, sweating, pallor and nausea. Definitive treatment generally involves surgical removal of the tumour (**Farling , 2000**).

Cushing's syndrome is the result of excess corticosteroids, the commonest cause being prolonged treatment with relatively large doses. Apart from the iatrogenic causes this disorder is very rare, other causes being primary tumours of the adrenal gland, adenoma or hyperplasia of the pituitary gland. In addition Cushing's syndrome can be secondary to carcinomas elsewhere, such as oat cell carcinoma of the lung, due to uncontrolled adrenocorticotrophic hormone (ACTH) secretion, the 'ectopic ACTH syndrome'. Conn's syndrome is also very rare, caused by a benign adenoma or hyperplasia of the zona glomerulosa producing excess aldosterone (**Smith M, 2000**).

Acute adrenal insufficiency can occur after trauma, severe hypotension and sepsis. It may follow surgical removal of the adrenals unless there is adequate replacement therapy. Chronic adrenal insufficiency (Addison's disease) occurs when there is destruction of the adrenal gland, caused by autoimmune disease, secondary tumour infiltration, tuberculosis or amyloidosis. Excess androgen secretion causes masculinisation (adrenogenital syndrome). This can result from an androgen secreting adrenocortical tumour or due to a congenital enzyme defect affecting cortisol synthesis. In the latter case, the resulting decrease in circulating cortisol stimulates the overproduction of ACTH, which in turn stimulates the adrenals to produce excess androgenic steroids. Females show signs of virilisation while males show precocious puberty. Extreme feminisation in males can occasionally be due to an estrogen-producing tumour of the adrenal gland (**Hahner , 2010**).

Preoperative preparation of the patients with adrenal dysfunction is based on the careful preoperative evaluation of the type and the severity of the disturbance (**Kalezi N, 2011**).

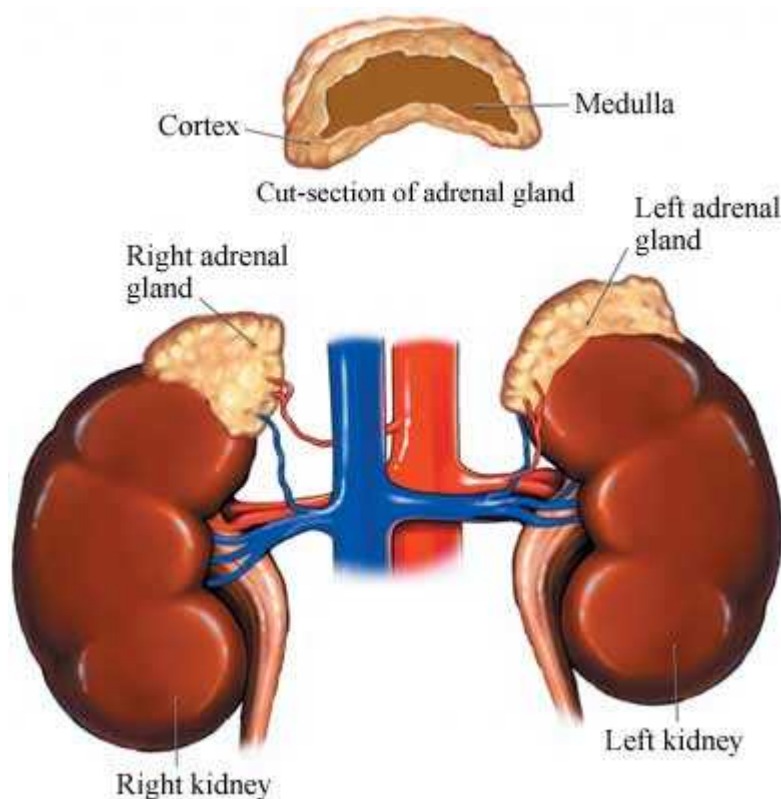
Glucocorticoids regulate protein, fat, carbohydrate and nucleic acid metabolism. Blood glucose is raised by antagonising the secretion and action of insulin. Glucocorticoids cause protein catabolism and fat mobilization. Anti-inflammatory action is due to effects on microvasculature and suppression of inflammatory cytokines (**Kulkarni et al., 2007**).

Chapter 1

Anatomy and physiology of adrenal gland

1) Anatomy of the adrenal gland

The adrenal glands are paired, pyramid-shaped endocrine glands anchored on the superior surface of each kidney. The adrenal glands are embedded in fat and fascia to minimize their movement (Mckinley et al., 2012).



(Figure 1) Adrenal glands quoted from (farling, 2001).

Each of the paired, retroperitoneal adrenal glands (suprarenal glands) contains two endocrine glands of different genetic origin which merged to form compact organ and are surrounded by a common connective tissue capsule. A mesodermal part (coelom lined by epithelium), i.e. the outer adrenal cortex surrounds an ectodermal part (sympathoblasts of the neural crest) that form the adrenal medulla. Each of the adrenal glands, weighing 4.2- 5.0 g. is enclosed in a perirenal fat capsule and rests atop the superior pole of the kidney. On the posterior aspect of each gland exist the hilum which allows veins and lymphatic vessels to exit. Arteries and nerves enter through numerous sites in its surface (**Fritsch et al., 2008**).

The adrenal cortex is partitioned into three separate regions: the zona glomerulosa, the zona fasciculata, and the zona reticularis. The zona glomerulosa is the thin, outer cortical layer composed of dense, spherical clusters of cells. These cells synthesize mineralocorticoids. The zona fasciculata is the middle layer and the largest region of the adrenal cortex. It is composed of parallel cords of lipid-rich cells that have a bubbly, almost pale appearance. Glucocorticoids are synthesized by these cells. The innermost region of the cortex, called the zona reticularis, is a narrow band of small, branching cells. These cells are

capable of secreting minor amounts of sex hormones called gonadocorticoids (**Mckinley et al., 2012**).

The adrenal medulla has a special intermediate place between the autonomic nervous system and the endocrine system. They are supplied by preganglionic sympathetic nerve fibers of the autonomic nervous system. The granule-containing cells lie in clusters between capillaries and larger, muscular veins. They form the two hormones epinephrine (adrenaline) (80 %) and norepinephrine (noradrenaline) (20 %). Epinephrine and norepinephrine are released into the bloodstream during stress and they act on the whole organism by preparing it for increased energy use (**Faller et al., 2004**).

Blood Supply and lymphatic Drainage.

Arteries. Each gland is supplied by an arterial network lying on its surface that is fed by three sources: the superior suprarenal artery arising from the inferior phrenic artery, the middle suprarenal artery arising from the aorta and the inferior suprarenal artery is arising from the rectal artery. The adrenal glands are also supplied by perforating arteries which pass directly to the adrenal medulla (**Mckinley et al., 2012**).

Veins. Venous blood collects in a single central vein located in each adrenal gland, the central veins exit through the hilum of the respective adrenal gland as the left suprarenal vein, which empties into the renal vein or right suprarenal vein emptying into the inferior vena cava (**Ganong, 1999**).

Lymphatic drainage. The majority of lymphatic vessels leaving the adrenal glands follow the course of the arteries, the primary lymphnodes of both adrenal glands are the para aortic and lumbar lymph nodes few lymphatic vessels accompany the thoracic splanchnic nerves, after passing through the diaphragm they reach the posterior mediastinal lymph nodes (**Fritsch et al., 2008**).