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Original Article

Endocrinal complications associated with the treatment of patients with congenital cardiac disease: consensus definitions from the Multi-Societal Database Committee for Pediatric and Congenital Heart Disease

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Abstract A complication is an event or occurrence that is associated with a disease or a healthcare intervention, is a departure from the desired course of events, and may cause, or be associated with, suboptimal outcome. A complication does not necessarily represent a breech in the standard of care that constitutes medical negligence or medical malpractice. An operative or procedural complication is any complication, regardless of cause, occurring (1) within 30 days after surgery or intervention in or out of the hospital, or (2) after 30 days during the same hospitalization subsequent to the operation or intervention. Operative and procedural complications include both intraoperative/intraprocedural complications and postoperative/postprocedural complications in this time interval.

The MultiSocietal Database Committee for Pediatric and Congenital Heart Disease has set forth a comprehensive list of complications associated with the treatment of patients with congenital cardiac disease, related to cardiac, pulmonary, renal, haematological, infectious, neurological, gastrointestinal, and endocrinal systems, as well as those related to the management of anaesthesia and perfusion, and the transplantation of thoracic organs. The objective of this manuscript is to examine the definitions of operative morbidity as they relate specifically to the endocrine system. These specific definitions and terms will be used to track morbidity associated with surgical and transcatheter interventions and other forms of therapy in a common language across many separate databases.

As surgical survival in children with congenital cardiac disease has improved in recent years, focus has necessarily shifted to reducing the morbidity of congenital cardiac malformations and their treatment. A comprehensive list of endocrinal complications is presented. This list is a component of a systems-based compendium of complications that will standardize terminology and thereby allow the study and quantification of morbidity in patients with congenital cardiac malformations. Clinicians caring for patients with congenital cardiac disease will be able to use this list for databases, initiatives to improve quality, reporting of complications, and comparing strategies of treatment.

Keywords: Congenital heart disease; quality improvement; patient safety; outcomes; registry; operative morbidity; paediatric; surgery; congenital abnormalities; cardiac surgical procedures; endocrine complications; thyroid dysfunction; adrenal dysfunction; hyperglycaemia; vasopressin; cardiac post-operative

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Historical background

The fields of cardiac intensive care, cardiac surgery, cardiac anaesthesia and cardiology continue to advance exponentially. The survival of patients with critical congenital heart disease is seldom in question in the modern era. During the past two decades, mortality after surgery for congenital heart disease has decreased dramatically and is now 4% in several large multicentre studies.[1,2] Consequently, the focus of clinical research and improvement efforts has now shifted to that of reduction and eventual elimination of morbidity.

Both mortality[3] and morbidity[4] have been defined for a cardiac surgical registry database. However, complications and death may occur in those with congenitally malformed hearts in the absence of surgical treatment. Additionally, a systematic review and classification of organ-specific complications delineated in a common platform has not been published. These issues prompted The Multi-Societal Database Committee for Pediatric and Congenital Heart Disease to undertake the task of defining organ-specific complications in relation to congenital cardiac disease. Importantly, this compilation of complications can be applied to surgical and non-surgical patients alike, regardless of manner or stage of therapy.

An extensive search of Medline and multiple textbooks was performed to identify the existing literature that provides definitions of the identified endocrinal complications. All participants in the subgroup of The Multi-Societal Database Committee for Pediatric and Congenital Heart Disease responsible for endocrinal complications reviewed the available data and contributed to the consensus definitions. Members of The Multi-Societal Database Committee for Pediatric and Congenital Heart Disease participated in refining all definitions offered in this article by telephone conferences, e-mail correspondence, and meetings; all participating members reviewed and approved the final definitions in this report.

Consensus definitions

The terms in the final list of endocrinal complications developed by The MultiSocietal Database Committee for Pediatric and Congenital Heart Disease, along with their official definitions are listed in Part 4 of this Supplement.

In Part 4 of this Supplement, the following terms are defined:

- Adrenal complication - Absolute adrenal insufficiency (AAI)
- Adrenal complication - Activated adrenal response (AAR)
- Adrenal complication - Insufficient basal cortisol (IBC)
- Adrenal complication - Relative adrenal insufficiency (RAI)
- Calcium complication - Hypocalcemia
- Glucose complication - Hyperglycemia
- Glucose complication - Hypoglycemia
- Relative vasopressin deficiency
- Syndrome of inappropriate antidiuretic hormone secretion
- Thyroid complication - Euthyroid sick syndrome (or Non-thyroidal illness syndrome [NTIS])
- Thyroid complication – Hypothyroidism.

Controversies and interaction with the cardiac system

The hormones of the endocrine system regulate the body's homeostatic mechanisms and include such vast functions as energy production and utilization, fluid and electrolyte balance, and circulatory function. Each cascade of hormones works within a feedback loop, most of which are regulated by the hypothalamus and pituitary gland. Disorders of the endocrine system include problems with overproduction or underproduction of a hormone or problems with receptors for these hormones. Each hormone and hormonal system impacts the function of the cardiac system in different ways. Interactions involving the following hormones and hormonal systems will be discussed:

- adrenal cascade and the Hypothalamic-Pituitary-Adrenal Axis
- thyroid function
- pancreatic function
- the parathyroid gland and calcium metabolism
- arginine vasopressin.

Most endocrinal complications have been defined in the literature previously and were agreed upon without much discussion. It should be noted, however, that these endocrinopathies have also been mostly defined in otherwise healthy patients. Additionally, most of the data for these entities come from critically ill adult populations and may not be directly applicable to paediatric populations. Fortunately there are multiple paediatric studies that are in progress that will help elucidate the impact of these endocrinopathies in the paediatric population. The complication that engendered a fair amount of debate was dysfunction of the hypothalamic-pituitary-adrenal axis in the critically ill cardiac patient.

Hypothalamic-Pituitary-Adrenal Axis

The hypothalamic-pituitary-adrenal axis involves release of catecholamines, sex steroids, glucocorticoids

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and mineralocorticoids from the adrenal gland under control of the hypothalamus and pituitary gland.[5] Many types of stress cause the hypothalamus to secrete corticotropin-releasing hormone. Corticotropin-releasing hormone stimulates the anterior pituitary gland to secrete adrenocorticotropic hormone, often termed "ACTH", which then stimulates the adrenal cortex to secrete glucocorticoids such as cortisol. Adrenocorticotropic hormone has little control over secretion of aldosterone, the other major steroid hormone from the adrenal cortex.

The haemodynamic alterations of adrenal insufficiency, due predominantly to deficiency of cortisol, can present as vasomotor paralysis or shock.[5] Adrenal insufficiency is associated with decreased myocardial contractility, vasodilatation, and capillary leak. Critical illness typically results in increased cortisol levels of up to six times baseline and loss of the diurnal variation.[6–10] The appropriate basal level of cortisol in critically ill patients has been controversial, with proposed values for normal basal cortisol ranging from 6–34 micrograms per decilitre.[6,7,11–13] The discrepant definitions of adrenal insufficiency have yielded highly variable estimates of the incidence of adrenal dysfunction in the critically ill.[6,14] For example, the reported incidence of adrenal dysfunction in sepsis ranges from 17–54%.[6,14–20] Adrenal dysfunction in critical illness is associated with increased duration and requirement for inotropes and/or vasopressors, and worsened clinical outcomes.[14,15] Annane and colleagues classified adult patients with septic shock into three prognostic groups based on adrenal function, with the worst mortality in patients with elevated basal cortisol and a poor response to adrenocorticotropin hormone stimulation.[11,12,21]

Observations in children with septic shock[14] and children with congenital cardiac disease[22] are consistent with these findings. The integrity of the hypothalamic-pituitary-adrenal-axis during critical illness is typically assessed by the cosyntropin stimulation test.[19] In response to cosyntropin stimulation, it is generally accepted that a cortisol increase of less than 9 micrograms per decilitre, is inappropriate and associated with worse outcomes.[12] The appropriate basal level of cortisol in critically ill patients is controversial, however, with proposed values for normal basal cortisol ranging from 6–34 micrograms per decilitre.[6,7,11–13] These discrepant definitions of adrenal insufficiency have yielded highly variable estimates of the incidence of adrenal dysfunction in critical illness. Although the data in children are limited, existing evidence suggests that a basal cortisol level of 16 micrograms per decilitre or more reflects an appropriate stress response in critically ill paediatric patients.[14,22] The "expected" adrenal response to critical illness is thus defined as an adequate basal cortisol, greater than 16 micrograms per decilitre, and an appropriate cosyntropin response of a cortisol level increase of greater than 9 micrograms per decilitre. We have used the term *activated adrenal response* to describe this adrenal response. Using the above reference values, adrenal activity can thus be classified into one of the following four groups:

- *Activated adrenal response*: an adequate basal cortisol of greater than 16 micrograms per decilitre, with an appropriate response to cosyntropin stimulation manifested by a change in cortisol of greater than 9 micrograms per decilitre.
- *Absolute adrenal insufficiency*: an inadequate basal cortisol of less than 16 micrograms per decilitre, with an inappropriate response to cosyntropin stimulation manifested by a change in cortisol of less than 9 micrograms per decilitre.
- *Relative adrenal insufficiency*: an adequate basal cortisol of greater than 16 micrograms per decilitre, with an inappropriate response to cosyntropin stimulation manifested by a change in cortisol of less than 9 micrograms per decilitre.
- *Insufficient basal cortisol*: an inadequate basal cortisol of less than 16 micrograms per decilitre, with an appropriate response to cosyntropin stimulation manifested by a change in cortisol of greater than 9 micrograms per decilitre.

The categories above delineate the various adrenal responses to critical illness. Existing evidence suggests that steroid treatment may be more beneficial in certain subsets of adrenal response than in others. There is uniform agreement that patients manifesting absolute adrenal insufficiency require glucocorticoid supplementation and possibly mineralocorticoid supplementation as well. Although conflicting data exist, previous studies in adult patients with sepsis support steroid treatment in patients with both relative adrenal insufficiency and insufficient basal cortisol.[11,12,21]

Much of the data on the hypothalamic-pituitary-adrenal axis in critical illness stems from research in patients with sepsis. With increasing recognition of adrenal dysfunction in sepsis and/or septic shock in both adult and paediatric patients, it has become evident that adrenal dysfunction has prognostic implications. Of note, the adrenal response may change during the course of an illness.[23] For example, a patient may demonstrate an activated adrenal response early in an illness that subsequently shifts to absolute or relative adrenal insufficiency. In patients treated with hydrocortisone with or without fludrocortisone, studies suggest that inotrope requirements fall most rapidly in patients with an inappropriate cosyntropin response or those with

insufficient basal cortisol.[11,23,24] In other groups of patients requiring inotropes for more than 48 hours, steroid administration may lead to the earlier cessation of inotropes and greater reversal of shock.[25,26] This may explain the correlations between adrenal dysfunction and catecholamine refractory shock in patients with sepsis.[10,15]

Cortisol is secreted from the adrenal cortex regulated by corticotropin and corticotropin-releasing hormone in response to physiologic stress. Cortisol has a plethora of functions:

- it regulates vascular smooth muscle tone[27]
- maintains endothelial integrity[28,29] and
- potentiates the actions of catecholamines.[21,28]

In addition to these vascular effects, cortisol also has the following functions:

- modulates inflammation
- conserves sodium and water, and
- increases glucose levels.[5]

The mineralocorticoid named aldosterone is secreted by the adrenal gland in response to hypotension, hyperkalemia, hyponatremia, and adrenocorticotropin hormone stimulation. Aldosterone induces potassium secretion and sodium and water retention by the kidneys.[5] The classic symptoms of adrenal crisis resulting from mineralocorticoid deficiency, and thus treated with fludrocortisone acetate, include hyponatremia, hyperkalemia and hypovolemia.[5–7] Acidosis, hypercalcemia, and hypoglycemia may also occur. The haemodynamic alterations of adrenal insufficiency, due predominantly to cortisol deficiency, can present as vasomotor paralysis or shock.[5] Adrenal insufficiency is associated with decreased myocardial contractility, vasodilatation, and capillary leak.

Various insults and altered neurohumoral mechanisms may impair the hypothalamic-pituitary-adrenal - axis during critical illness, either resulting from diminished release of corticotropin and corticotropin-releasing hormone or from diminished responsiveness of the adrenal gland to these regulatory hormones. Commonly used pharmacologic agents can alter adrenal function, such as narcotics, ketamine, etomidate, ketoconazole, rifampin and phenytoin.[30,31] Pathophysiologic insults that can alter hypothalamic, pituitary, or adrenal function include cytokines, inflammatory mediators, hypoxia, reperfusion injury, and/or adrenal haemorrhage.[32] Waterhouse-Friderichsen syndrome is massive haemorrhage into the adrenal glands, and is usually bilateral. Various cytokines can suppress the pituitary response to hypothalamic corticotropin-releasing hormone or induce systemic or tissue specific corticosteroid resistance.[6,17] Cortisol levels are reduced after cardiac surgery, which may result from the systemic inflammatory response and cytokine release induced by cardiopulmonary bypass.[33] Finally, developmental factors may modulate adrenal function.

Thyroid function

The hypothalamus secretes thyrotropin-releasing hormone, which stimulates the pituitary gland to secrete thyroid-stimulating hormone, which catalyzes the conversion of thyroglobulin to thyroxine, commonly known as "T_4". Thyroxine is then cleaved to form the active hormone triiodothyronine, commonly known as "T_3". Triiodothyronine increases basal metabolic rate and stimulates growth in tissues by increasing activity of sodium-potassium-adenosine triphosphate pumps.[5] From a cardiac standpoint, thyroid hormones increase contractility, cardiac output, and ejection fraction.[34,35] Although triiodothyronine improves diastolic function and increases intracellular calcium levels via the sodium-calcium exchanger, it may also increase myocardial oxygen consumption.[34–37] In peripheral vasculature, thyroid hormones reduce systemic vascular resistance and increase pulse pressure.[35] Thyroid hormones have been shown to potentiate the effects of catecholamines.[37,38]

Hypothyroidism is associated with decreases in triiodothyronine, thyroxine, and reverse triiodothyronine with high levels of thyroid stimulating hormone. Symptoms of hypothyroidism include bradycardia, pericardial effusions, hypertension and a narrowed pulse pressure, and myxedema.[35] Studies have also shown decreases in cardiac output and cardiac contractility, as well as decreased diastolic relaxation and diastolic filling. There have also been rare incidences of increased QT interval leading to Torsades. Chronic hypothyroidism, including subclinical hypothyroidism, has been associated with accelerated atherosclerosis, hypercholesterolemia, coronary artery disease, and an increased relative risk of death from cardiovascular causes.[39] In those with congestive heart failure, reduction in triiodothyronine levels have been shown to be proportional to New York Heart Association class,[35] mortality,[40,41] morbidity,[37,42] poor haemodynamics, and hyponatremia.[41]

Important in critical illness is *non-thyroidal illness syndrome* or *euthyroid sick syndrome* which presents with a low triiodothyronine level associated with a normal or low thyroxine, high reverse triiodothyronine and normal or high thyroid stimulating hormone.[5,40,43] Euthyroid sick syndrome is associated with abnormal findings on thyroid function tests in the setting of a non-thyroidal illness, without pre-existing hypothalamic-pituitary and thyroid gland dysfunction. After recovery from this non-thyroidal illness, these abnormalities of the thyroid function tests should be completely reversible.

Theories explaining non-thyroidal illness syndrome include:

- the body's attempt to decrease metabolic demands during stress,[34]
- a deficit in converting thyroxine to triiodothyronine,[40,44,45]
- decreased serum thyroid binding capacity,[40,44] and
- decreased hypothalamic activity reflected by decreased thyrotropin-releasing hormone, as sex steroids are also decreased in severe illness.[43]

Non-thyroidal illness syndrome may be exacerbated by glucocorticoids that can decrease pituitary response to thyrotropin-releasing hormone and lead to a lower level of thyroid stimulating hormone.[40,43] Dopamine, which is frequently used in intensive care, confounds assessment of thyroid activity since it can decrease thyroid stimulating hormone[40,43] and induce secondary hypothyroidism.[46,47]

It is important to assess free thyroid hormone levels since critical illness often alters serum protein levels, including thyroid binding globulin and albumin.[43] Reduced levels of triiodothyronine have been observed in as many as 70% of hospitalized patients.[40] Decreased thyroxine levels have been associated with increased mortality, and the combination of low triiodothyronine and thyroxine may carry an even worse prognosis.[40,42,43]

Research is ongoing to determine the thyroid profile of patients undergoing cardiopulmonary bypass procedures, and to assess whether thyroid hormone replacement is beneficial for these patients. Thyroid hormones including thyroid stimulating hormone, thyroxine, free thyroxine, triiodothyronine, free triiodothyronine, and thyroglobulin have been shown to decrease after cardiopulmonary bypass with increases in reverse triiodothyronine.[34,47–53] This decrease in triiodothyronine has been shown to be more profound in children than adults.[34,54] As discussed above, dopamine administration may contribute to these alterations.[46] In children after cardiac surgery, Bettendorf and colleagues reported plasma thyroid hormone concentrations were lowest in patients treated with dopamine, and these patients also took the longest return to normal.[47] Lower levels of thyroid hormones postoperatively have been associated with

- higher scores via the – the Pediatric Risk of Mortality Scoring System (PRISM), which is described in the tenth manuscript in this Supplement titled "Databases for assessing the outcomes of the treatment of patients with congenital and paediatric cardiac disease – the perspective of critical care"
- increased length of mechanical ventilation
- increased time requiring supplemental oxygen
- increased intensive care unit length of stay, and
- increased need for inotropes and Lasix.[34,47,49,55]

Conversely, patients with more favourable outcomes had higher levels of thyroid hormones than those who had complications or that died.[50] The nadir of thyroid hormones corresponds to the decrease in cardiac output after congenital cardiac surgery. This nadir of thyroid hormones begins soon after surgery, and may last for 48 hours. Thyroid hormones may remain at a low level for as long as 5–8 days postoperatively, and longer with more complex surgeries.[47,50,53] The cause of this decrement in thyroid hormones is unknown but may include stress induced depression of the hypothalamic-pituitary-adrenal axis, hemodilution, hypothermia, the use of dopamine or glucocorticoids, or suppression secondary to tumour necrosis factor or interleukin-6.[46,52,54]

Replacement of triiodothyronine in patients with congenital cardiac disease who have undergone cardiac surgery is currently undergoing trials. Initial data suggest that Triostat, triiodothyronine replacement, increases cardiac index, increases systolic blood pressure, and decreases systemic vascular resistance.[45,49,51,56,57] The heart rate in patients increased or was unchanged but there were no serious dysrhythmias. Triiodothyronine replacement corresponded to reduced requirements of inotropes and improved balance of fluids.[49,51,56,57]

Glucose homeostasis

Insulin increases glucose uptake into cells, lowers serum glucose, and stimulates the formation of glycogen, proteins and adipose.[5] As a counter-regulatory hormone, glucagon increases serum glucose by stimulating glycogenolysis and gluconeogenesis. *Hypoglycemia*, defined as a glucose level less than 80 milligrams per decilitre, can be seen in paediatric patients with critical illness secondary to inadequate glycogen stores, adrenal insufficiency, and liver failure. Hypoglycaemia should be avoided as it can lead to neurologic abnormalities[58].

Hyperglycaemia can occur after cardiopulmonary bypass procedures as a result of decreased insulin, insulin resistance, decreased glucose utilization with hypothermia, decreased renal elimination, increased levels of epinephrine or cortisol, and secondary to cytokines and counter-regulatory hormones.[50,59] Potential detrimental effects of hyperglycaemia are well documented; hyperglycaemia alters cellular immune function and is pro-inflammatory, whereas insulin has anti-inflammatory properties.[59,60] Insulin can reduce cytotoxicity by shifting intracellular metabolism from utilization of free fatty acids, which can lead to toxic metabolites, to utilization of glucose.[59] Van den Berghe

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and colleagues first reported an association of hyperglycaemia with increased morbidity and mortality in critically ill adult patients, which improved with tight glycaemic control.[61,62] Findings from other studies contradict these observations, however, so the role of tight glycaemic control in critical care remains controversial, especially in paediatric and neonatal intensive care where the risk of hypoglycaemia is particularly important.[63,64] In children with severe bronchiolitis who required mechanical ventilation, hyperglycaemia was not independently associated with mortality or morbidity.[63] In contradictory observational studies of paediatric cardiac surgical patients, one group reported an association between hyperglycaemia and morbidity and mortality,[65] while another group observed no association between hyperglycaemia and adverse events but increased morbidity with relative hypoglycaemia.[66] No controlled trials exist in paediatrics evaluating the effect of tight glucose control with insulin, but it is evident that the adult data cannot be directly translated to paediatric practice.[67] No consensus exists in the literature at present regarding perioperative glycaemic control in paediatric cardiac surgical patients.

Parathyroid function and calcium metabolism

Although calcium homeostasis is not an issue in most paediatric cardiac surgical patients, *hypocalcaemia* is often important in patients with suspected or known DiGeorge syndrome, which is associated with parathyroid insufficiency. Calcium concentrations are regulated by parathyroid hormone and Vitamin D after conversion to calcitriol. Calcitriol and parathyroid hormone stimulate

- calcium mobilization from bone
- resorption of calcium in the renal tubules, and
- increased absorption of calcium from the gastrointestinal tract.[68,69]

Hypocalcaemia induces secretion by the thyroid gland of calcitonin, which opposes the actions of parathyroid hormone. Critical illness can induce a state of relative hypoparathyroidism, which is exacerbated by hypomagnesaemia.[68] Renal insufficiency can also lead to calcitriol deficiency. To assess calcium homeostasis in the intensive care unit, ionized calcium should be measured, as this is the active fraction which is not affected by alterations in serum protein concentrations. Ionized calcium levels fall with administration of heparin, which is administered in large volumes during cardiopulmonary bypass.[50]

Decreased calcium levels can lead to reversible myocardial dysfunction causing hypotension, increased length of stay and morbidity, and increased mortality in critically ill patients.[70,71] Calcium administration increases blood pressure without affecting heart rate.[69] This effect should be balanced by the risk of destructive intracellular processes that may result from increased extracellular and cytoplasmic calcium, which may be especially relevant in patients with associated ischaemia or sepsis.[69] A retrospective review of paediatric cardiac surgical patients found that postoperative calcium supplementation was associated with mortality and morbidity, including prolonged length of stay, liver dysfunction, neurological complications, infections, and need for extracorporeal support.[72] Studies investigating control of calcium homeostasis in paediatric cardiac surgical patients are lacking, so the role of aggressive calcium replacement in patients with critical cardiac disease remains obscure.

Vasopressin

Activation of either the sympathetic nervous system or the Renin-Angiotensin-Aldosterone system causes increased release of arginine vasopressin in response to low blood pressure, decreased cardiac filling, and/or increased serum osmolarity.[5,73,74] Vasopressin acts through several receptors.[75] Stimulation of V_1 receptors lead to vasoconstriction in skin, skeletal muscle, and mesenteric blood vessels, but vasodilatation at low concentrations in pulmonary, coronary and cerebral vascular beds.[76] Stimulation of V_2 receptors leads to the transport of aquaporin to the apical membrane of the collecting ducts which increases osmotic water permeability and re-absorption without inducing kaliuresis.[5,73,74,76] The V_{1b} or V_3 receptor in the anterior pituitary also regulates hypothalamic-pituitary-adrenal axis activity during stress and resting conditions.[77] Vasopressin thus amplifies the effect of cosyntropin releasing hormone upon adrenocorticotropin hormone release.[77,78] Vasopressin and cosyntropin releasing hormone have synergistic effects upon adrenocorticotropin hormone release by the pituitary.[77]

Disease states most frequently involved with vasopressin in paediatric patients include the following two entities:

- *syndrome of inappropriate antidiuretic hormone secretion*, and
- *relative vasopressin deficiency.*

With the *syndrome of inappropriate antidiuretic hormone secretion*, excess arginine vasopressin is released from the hypothalamus, which mediates free water retention by the kidney, resulting in

- hypervolemia with a low serum osmolarity of less than 280 milliosmoles per litre
- dilutional hyponatremia with a sodium of less than 130 milliequivalents per litre, and
- an increased urine specific gravity.[5]

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Akin to the syndrome of inappropriate antidiuretic hormone secretion, arginine vasopressin levels are elevated in patients with congestive cardiac failure, especially those with hyponatremia.[73] This elevation has been associated with increased mortality, and trials are ongoing to evaluate the therapeutic option of vasopressin-receptor antagonists in heart failure patients to stimulate diuresis.[76]

Relative vasopressin deficiency is defined as an insufficient level of vasopressin for the patient's clinical state. The concept of relative vasopressin deficiency in critically ill cardiac patients has only recently been introduced, so data about this condition are limited. It is diagnosed in patients with hypotension and a normal vasopressin level.[75,79] Vasopressin deficiency can lead to shock and may be associated with other characteristics of diabetes insipidus such as urine output greater than 4 millilitres per kilogram per hour, urine specific gravity less than 1.010 and sodium greater than 145 milliequivalents per litre.[5] As an adjunct for blood pressure support in sepsis, vasopressin can stabilize blood pressure and reduce the need for cathecholamines.[74,79] Vasopressin has been used for postoperative hypotension in paediatric cardiac surgical patients. It has been shown to increase blood pressure and decrease inotrope requirements without causing tachycardia or arrhythmias.[80–82]

Conclusion

The present list represents a comprehensive compilation of endocrine complications occurring before, during, and after congenital cardiac surgery. Those who care for patients with cardiac disease in a critical care setting require a knowledge base about the many feedback loops involved in the hormonal regulation of the body, since these feedback loops may complicate the management of patients with critical cardiac disease, and more importantly, may offer ways to intervene to improve their outcome. Clinicians caring for patients with congenital cardiac disease will be able to use this list for databases, quality improvement initiatives, reporting of complications, and comparing strategies for treatment.

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