HORMONES AND THE HEART

Richard J. Robbins, M.D., Steven Petak, M.D., J.D.
Houston Methodist Hospital, Houston, Texas

The heart has many different cell types, each with hormone receptors that are responsive to incremental changes in the endocrine system. In fact, the cellular physiology of cardiac cells is governed minute by minute by hormonal regulation of second messengers and membrane ion channels. In this special issue of the *Methodist DeBakey Cardiovascular Journal*, a talented group of endocrinologists summarize how several hormone systems influence normal cardiac function and structure and how these systems, when out of control, can lead to cardiovascular disease states.

This issue opens with a review of cardiovascular disease in acromegaly by Drs. Morali Sharma, Richard Robbins, and colleagues, who take an in-depth look at the effects of growth hormone (GH) and insulin-like growth factor 1 (IGF-I) on the heart. The authors clearly outline the three stages of cardiac response to chronic elevations of GH and IGF-I that ultimately lead to hypertrophic congestive heart failure. Acromegaly can trigger additional independent phenomena such as acromegalic hypertension, coronary artery changes, and arrhythmias, all contributing to negative cardiovascular outcomes. Fortunately, it appears that the use of somatostatin analogs or direct GH receptor antagonists can reverse cardiac decompensation in those who cannot undergo surgical removal of GH-secreting pituitary tumors.

Next, Drs. Patricia Mejia Osuna, Maja Udovcic, and Morali Sharma provide an update on the cardiovascular effects of chronically elevated thyroid hormone levels. In addition to regulating actin and myosin subtypes, excess thyroid hormone can provoke rapid membrane-level changes in sodium, potassium, and calcium channels. The negative cardiac outcomes induced by hyperthyroidism are indirectly supported by other hormones such as catecholamines, renin, and aldosterone. In this review, the authors address the mechanisms leading to thyrotoxic cardiomyopathy and include discussions on the influence of hyperthyroid arrhythmias and the effects of amiodarone on the thyroid.

Drs. Spandana Brown, Laila Tabatabai, and Mary Ruppe next provide a unique look at the cardiovascular effects of altered calcium homeostasis and the hormonal systems that regulate it, including parathyroid hormone and vitamin D. They describe how primary hyperparathyroidism can lead to cardiac hypertrophy both independently and through the resulting hypertension that is common in this disorder. The frequent elevations of parathyroid hormone secondary to renal failure may also contribute to direct negative cardiac outcomes. Finally, the authors review the relationship between vitamin D deficiency and heart failure and describe the cardiac consequences of hypoparathyroidism seen in a number of hereditary diseases and following surgical destruction of the parathyroid glands. Of note, the parathyroid glands were first described by Sir Richard Owen in a necropsy of an Indian Rhinoceros in 1852 (cover picture used with permission of Joel Sartore/National Geographic Photo Ark).

The article by Drs. Travis Goodale, Steven Petak, and colleagues summarizes a large body of literature on the negative cardiovascular events associated with declining serum testosterone in older men. Testosterone activation of androgen receptors in myocardial cells alters the balance of myocardial second messengers and membrane ion fluxes. Although the connection between low testosterone levels and heart failure is well established, a causal relationship has yet to be determined. Nonetheless, many investigators have examined the cardiovascular events that occur with testosterone replacement in older men. The authors carefully recap these studies and examine both heart failure and coronary artery disease in the setting of testosterone replacement therapy.

Finally, Drs. Maja Udovcic, Abhishek Kansara, and coauthors summarize the changes in cardiac structure and function in subclinical as well as overt hypothyroidism. Untreated hypothyroidism can lead to serious complications that impact cardiac output, vascular resistance, blood pressure, contractility, and heart rhythm. The authors discuss how cardiometabolic changes in hypothyroidism contribute to hypertension and heart failure but appear to be beneficial in patients with angina and acute myocardial infarction.

We hope that this issue provides some clarity on the effects of the endocrine system on cardiovascular metabolism and function and enlightens readers on the many treatment options available for patients with hormone-related cardiovascular disease.