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Neurohormonal Activation in Cardiorenal Syndrome

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Introduction

Cardiac and renal functioning are highly related as each system is interdependent on the other, and subsequently, dysfunction of one system can potentiate the development of dysfunction of the other, a conditioned referred to as Cardiorenal Syndrome, or CRS. (Scully & Goldsmith, 2013). In CRS, heart failure and renal failure exist together. Heart failure is the result of decreased heart efficiency and results in hemodynamic and neurohormonal changes (Nicholson, 2014). A decrease in cardiac function can cause dysfunction of other body systems, including the renal system, and at the same time, impaired renal functioning can result in increased fluid retention and increased systemic vascular resistance and can prove to be detrimental to cardiac function. According to Giam, et al., (2015), the prevalence of heart failure is expected to rise nearly 46% from 2012 to 2030 secondary to the increase in obesity and associated comorbid conditions. With the prevalence of heart failure expected to rise drastically, renal dysfunction associated with heart failure is expected to rise as well, with heart failure being responsible for nearly 44% of deaths in patients with end-stage renal disease (Giam, et al., 2015). Cardiorenal syndrome can be divided into five separate sub-classes.

A significant portion of the interdependence of cardiac and renal functioning is related to neurohormonal mechanisms, most notably the renin angiotensin aldosterone system, or RAAS. For this reason, angiotensin converting enzyme and inhibitors (ACE-I) have become a mainstay for the treatment of heart failure, however, ACE-I's can potentially have detrimental renal effects (Valika & Gheorghiade, 2012). This can make the management of CRS difficult for practitioners and special considerations must be made. Cardiorenal Syndrome can present a complex challenge for practitioners and with new evidence-based research being released, there exists the possibility for growth and understanding.

Signs and Symptoms

The signs and symptoms associated with CRS are similar to those experienced in heart failure. The signs and symptoms are the result of cardiac and renal dysfunction compounded together. The New York Heart Association classification scale can be used to describe symptoms.

Class I heart failure is associated with no limitations to ordinary activity and can progress to class IV, in which symptoms are present even at rest and activity only exacerbates discomfort.

Other signs and symptoms include:

- Orthopnea
- Paroxysmal Nocturnal Dyspnea (PND)
- Reduced Activity Tolerance
- Chest pain
- Edema
- 2 to 3 kilogram weight gain per week
- Pulmonary edema and crackles
- Arrhythmias

Patients should be educated on warning signs of complications and what should be done when acute issues and complications arise. Quick intervention can reduce the likelihood of exacerbations

Classification	Description
Class I	acute cardiac injury results in renal injury
Class II	heart failure leading to chronic renal failure
Class III	acute kidney injury resulting in cardiac injury
Class IV	chronic renal failure leading to heart failure
Class V	cardiac and renal dysfunction secondary to a systemic disease

Table 1. Classifications of CRS (House, et al., 2010).

Nursing Implications

Nearly five million people in the United States are diagnosed with heart failure, and another half a million each year. There is a significant economic burden as well, with nearly \$39 billion associated costs in 2010 (Coons, McGraw, & Murali, 2011). Lifestyle modifications should include the elimination of behaviors that can further decrease cardiac function. Modifiable lifestyle factors include:

- Smoking
 - Diet
 - Exercise
 - Alcohol use
- Education is essential to reduce the risks associated with medications used in CRS management, especially hypotension. Patients should be educated to:
- Eliminate the use of NSAID's which can decrease renal function and should be taught to
 - limit salt substitutes (often low sodium diets are implemented), as the replacements can be high in potassium (O'Donovan, 2014).
 - Serum potassium and creatinine levels should be monitored by practitioners for the assessment of renal function. At the initiation of an ACE-I, renal function should be monitored, and as dosing is adjusted according to O'Donovan (2014).

Underlying Pathophysiology

The common pathological mechanism of CRS is the chronic activation of the RAAS resulting in oxidative stress leading to impaired cardiac and renal functioning. When cardiac output is decreased, hypoperfusion of the kidneys and a decrease in glomerular filtration rate (GFR) can result. This decrease in perfusion results in the stimulation of the RAA system, a normal mechanism of compensation. Activation of the RAA system results in several changes. First, conversion of angiotensin to angiotensin II results in vasoconstriction, in turn, increased systemic vascular resistance and blood pressure. Second, Aldosterone is stimulated to be released from the adrenal cortex. Aldosterone results in the retention of fluid and sodium from the kidneys. The net effects of the RAA system is increased blood volume and systemic vascular resistance to increase renal perfusion. A negative feedback system then inhibits the RAA system once homeostasis is returned (Klabunde, n.d.).

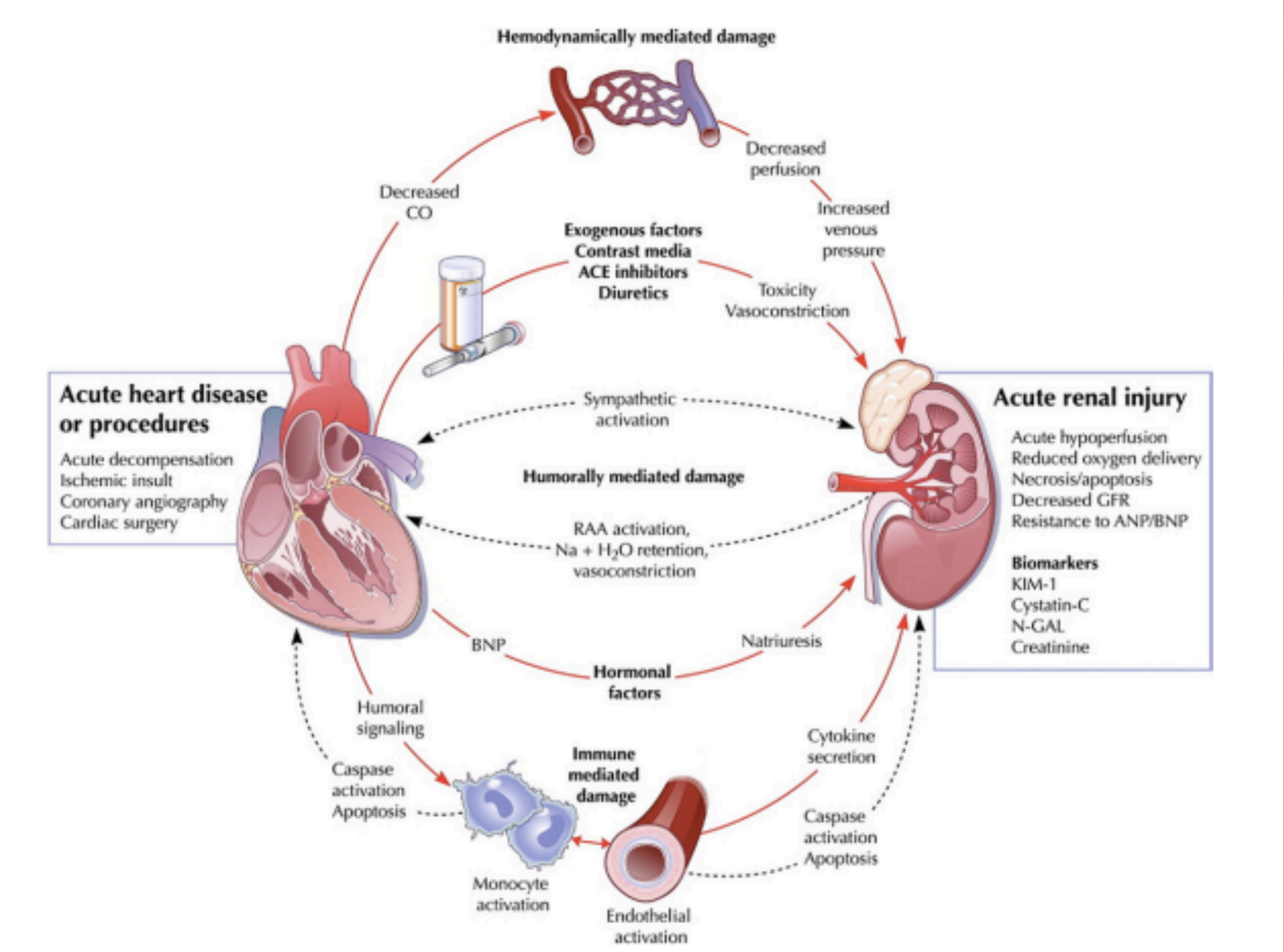


Figure 1. Cardiorenal Syndrome (House, et al., 2010).

In Cardiorenal syndrome however, a normal compensatory mechanism becomes chronically activated, resulting in deleterious effects that can potentiate the failure of both cardiac and renal functioning. Chronic activation of the RAA system can result in hypertrophy, inflammation, apoptosis, and oxidative stress. Angiotensin II appears to be a significant contributor in CRS. A chronic elevation of angiotensin II has been shown to induce apoptosis in renal and cardiac cells as well as activation of NADPH oxidase. NADPH leads to increased reactive oxygen species (ROS) which in turn reduces the availability of nitric oxide (vasodilating effects in normal circumstances) and increased vasoconstriction. The increased levels of ROS also stimulate the production of pro-inflammatory mediators. The best understood mediators are interleukin-6 and transforming growth factor-beta, which contribute to fibrosis in both the heart and kidneys. Fibrotic tissue results in a change of structure, producing a change of function. Angiotensin II is also responsible for the stimulation and release of aldosterone, which when chronically activated, can cause increased collagen formation and further increasing fibrosis (Giam, et al., 2016). According to Koniari, et al. (2010), the cytokines produced also have a negative inotropic effect, which causes a further reduction in cardiac output and renal perfusion. When combined together, all components lead to decreased functioning of cardiac and renal systems. Other deleterious effects of chronic activation of RAA system includes an increased preload and afterload, which can complicate heart failure further.

Significance

Cardiorenal Syndrome is a complex condition. Normal compensatory mechanisms used to maintain homeostasis actually become deleterious for both cardiac and renal functioning. The significance of the pathophysiology lies in the management of CRS.

- Treatment of CRS involves choices that are contradictory
- Cornerstone of management is ACE-I, but use can worsen renal functioning as indicated by elevated serum creatinine and BUN levels (House, et al., 2010).
- Diuretics are used to reduce preload, have been shown to be beneficial in heart failure
- Similar to ACE-I, diuretics can have negative renal effects and further complicate CRS.

Conclusion

Cardiorenal Syndrome is a highly complex pathophysiological processes that involves several mechanisms. The RAA system acts normally as a compensatory neurohormonal mechanism to maintain GFR and renal perfusion. In heart failure however, chronic activation of the RAA system due to decreased renal perfusion can lead the commitment development of renal and cardiac dysfunction, deemed Cardiorenal Syndrome (Giam, et al. 2016). The associated pathophysiological process of CRS guides therapies, which can be directly contradictory, meaning that the management of heart failure can worsen renal function and vice versa (Koniari, et al., 2010). Nursing has several implications in the management of CRS, specifically related to education. As evidence continues to emerge related to CRS, the management should continue to advance, but at the current time, CRS remains a complex process that can lead to poor outcomes.

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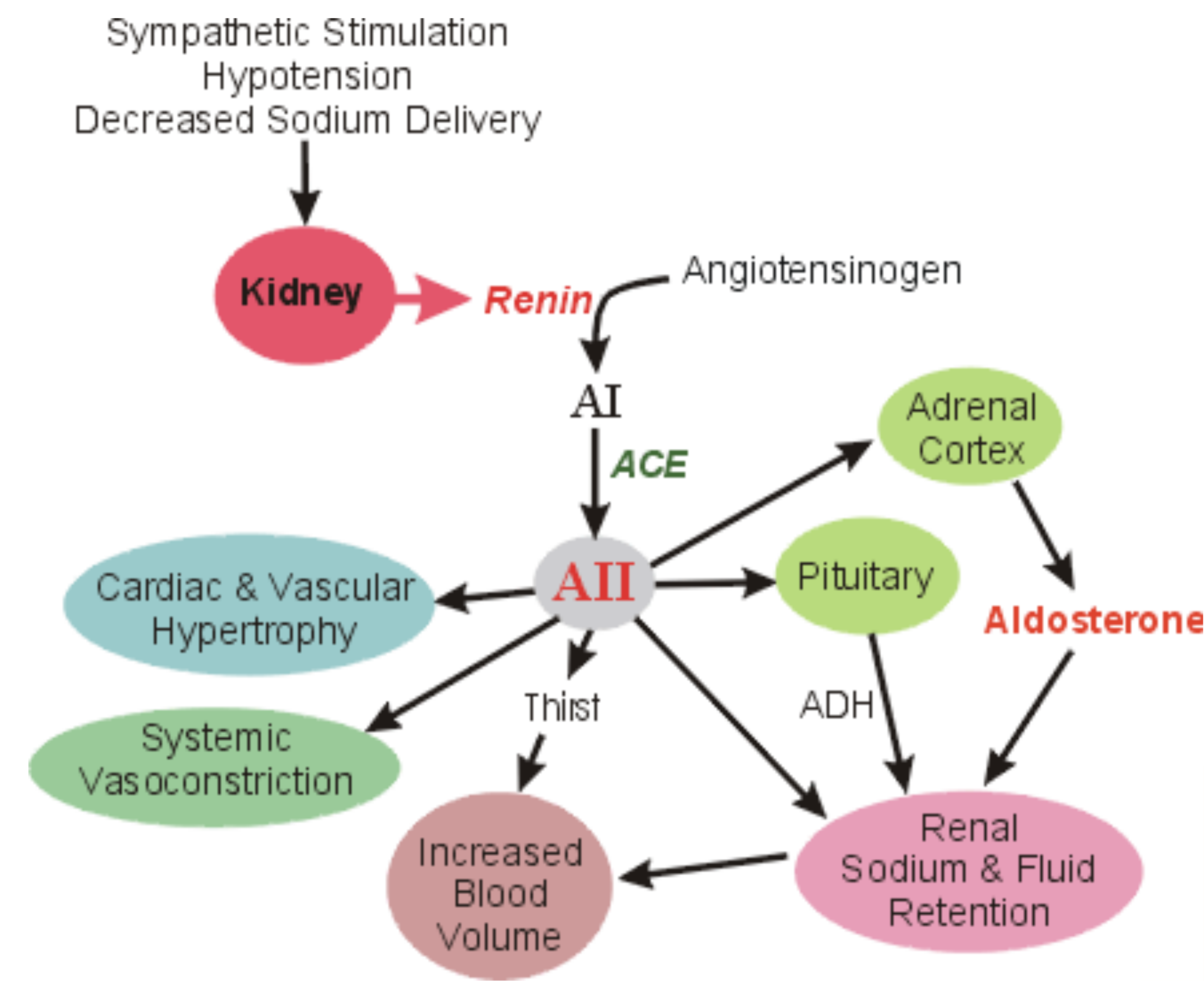


Figure 2. RAA System (Klabunde, 2014).