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Reptile Cardiology

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Basic Anatomy

It is easy to imagine that the hearts of reptiles represent an evolutionary stage from fish and amphibians to mammals. However, the success of reptiles in the animal kingdom over millions of years would argue that their cardiovascular systems are equally developed as mammalian or avian systems. The reasons for persisting with a relatively "primitive" heart anatomy are not completely understood. Evolutionary pressures would undoubtedly have resulted in more "advanced" anatomy if it were necessary. In some respects, the reptilian heart can be viewed as substantially more complex than mammalian four-chambered, septated hearts with parallel pulmonary and systemic circulations.

Reptiles exhibit two basic cardiac morphologies or patterns. The first, seen in Chelonia (turtles, tortoises, terrapins) and Squamata (snakes, lizards) comprises a sinus venosus, 2 atria (right and left), a common ventricle that is partially septated ventrally, a single pulmonary artery and 2 aortic arches (left and right). The sinus venosus is separated from the right atrium by a sinoatrial valve; the atria are separated from the ventricle by mono-cuspid atrioventricular valves, while the ventricle has semilunar valves separating it from the major vessels.

The squamate/chelonian heart can be partitioned into several "compartments". The cavum dorsale, just ventral to the atrioventricular orifices accepts blood from both the atria and acts as the outflow tract for the aortae. It can be divided into the cavum arteriosum, which receives oxygenated blood from the left atrium, and the cavum venosum, which receives blood from the right atrium and from which both the aortic arches originate. Below the cavum venosum lies the cavum ventrale or cavum pulmonale. The cavum venosum and cavum arteriosum are connected by an interventricular canal – basically a large VSD.

The second pattern is observed in crocodilia (crocodiles, alligators, caymans) and consists of two atria, two ventricles divided by a complete septum, two aortic arches, and a pulmonary artery. However, the two aortic arches communicate via a foramen, known as foramen Panniza(e), as well as a distal aortic communication (anastamosis). While this anatomy appears somewhat similar to that of mammals, it is important to realize that the left aortic arch arises from the right ventricle along with the pulmonary artery.

At a cellular level, reptilian ventricular cardiomyocytes appear to be substantially different from mammalian myocytes, with a more spindle shape and a lack of T-tubules. Studies of calcium flux in turtle cardiomyocytes suggests that SR calcium release is minimal, and that most calcium involved in excitation-contraction coupling comes from L-type sarcolemmal calcium channels.

Functional cardiac anatomy and physiology

Initial examination of the hearts of reptilia is perplexing to the new-comer. Lack of septation, or origin of a systemic artery from the right ventricle, imply significant admixture of oxygenated and deoxygenated blood. However, several elegant studies have defined both the pattern of hemic circulation and the functional advantages conferred by such anatomy.

Contrast and oxygen saturation studies in the 50s and 60s showed that oxygenated and deoxygenated blood do not generally mix in snakes, but mix to some extent in turtles. These studies (and catheterization studies) showed that blood from the left atrium was almost exclusively ejected into the systemic circulation, while blood from the right atrium was expelled into the pulmonary artery. Several features of cardiac function were observed that explained the maintenance of discrete circulations. First, the AV valves are attached medially, so that when they open the form a functional continuation of the ventricular septum, effectively covering the interventricular canal. This allows blood from each atrium to flow into the respective compartments of the cavum dorsale – venosum and arteriosum. Once ventricular systole begins, the blood within the cavum venosum is ejected into the cavum pulmonale and then into the pulmonary artery. Because of the lower pulmonary vascular resistance, all the blood during early systole (first 25%) is ejected exclusively into the PA. As contraction progresses, the cavum pulmonale becomes functionally separated from the cavum venosum so that during the latter portion of systole, blood from the cavum arteriosum is ejected into the aortae. Intracardiac left-to-right shunting is observed in turtles at rest.. Approximately 40% of the blood in the heart enters the systemic circulation, while 60% enters the pulmonary circulation, because a small percentage of oxygenated blood is recirculated through the lungs.

Similarly, in Crocodilia, the right ventricular content is ejected under normal situations exclusively into the pulmonary artery. Blood from the left ventricle is ejected into the right aortic arch and then passes through the foramen Panniza into the left aortic arch, despite the origin of the left aortic arch from the right ventricle. Circulatory exclusivity is maintained by relative resistances of the pulmonary and systemic circulations – diastolic pressure in the left aortic arch is sufficiently high that right ventricular blood is ejected into the pulmonary circulation. A unique crocodilian feature of right ventricular outflow is the presence of cog-teeth just below the pulmonic valve. These appear to provide a mechanical resistance to ejection into the pulmonary circulation toward the end of the cardiac cycle, resulting in ejection of blood into the left aorta, and a biphasic right ventricular pressure wave. The depolarization of the cog-teeth muscles is delayed from the rest of the right ventricle and affected by vagal innervation.

Two additional features allow separation of circulations in reptiles: a very long and slow systole, and ventricular “non-compaction”. A long slow systole (often twice as long as diastole) allows for ejection of first the pulmonary blood, and then the systemic blood from the ventricles. The trabeculated ventricular myocardium, allows “trapping” of blood into small compartments. Evolutionarily, there must be a reason for this cardiovascular design. It may lie with the aquatic nature of many reptiles. Just as mammals have a “dive reflex” which reduces heart rate (by increasing vagal tone), reptiles also respond to submersion with a profound increase in pulmonary vascular resistance, and a decrease in systemic vascular resistance, resulting in a

right-to-left shunting of blood, so that pulmonary blood flow is reduced (in some species, it stops), and systemic flow is increased. This is most pronounced in some species of water snakes, but is also seen in turtles and alligators. This is logical when one considers that during prolonged submersion, pulmonary circulation is futile – no oxygenation would be anticipated. During breathing periods in these reptiles, blood flow shunts left-to right to allow maximum oxygenation prior to re-submersion. In snakes and turtles, the blood flow is redirected to the aortic arches, while in crocodiles, blood from the right ventricle is ejected into the left aortic arch, while the foramen Panniza contracts to prevent shunting between the arches. Hypoxia induces similar responses.

Recent studies have also documented profound reversible post-prandial cardiac hypertrophy in pythons, thought to be a response to the large increase in metabolic demand that digestion imposes.

Cardiac Imaging and Investigation

Early studies of functional anatomy and physiology used contrast, pressure and oxygenation studies. Electrocardiography has been performed on many species, but standards are not always available, and electrode placement has been variable. All reptilian ECGs show complexes similar to mammals –p-waves, QRS complexes and T- waves, often with a prolonged QT interval, a short Tp interval, (snakes), consistent with the long systole and short diastole. Heart rates vary between species and environmental conditions (as would be expected with poikilotherms), but are generally relatively slow. Radiography can occasionally show cardiomegaly, although reference intervals are not described, and require comparison with unaffected individuals of similar size. Recently, echocardiography has been utilized. Standardized imaging techniques have been described for Ophidians and red slider terrapins. Limited case reports describe cardiac diseases in snakes.

Cardiac Diseases in Reptiles

There are very few reports of cardiac diseases in reptiles. Right AV valve insufficiency has been reported in one carpet python, resulting in congestive heart failure. Due to the lack of septation in squamata and chelonia, elevated diastolic pressures could be shared across all ventricular compartments, resulting in bilateral heart failure.

Murmurs can occasionally be ausculted. Tachycardia has been observed with cardiac insufficiency, and cardiomegaly was evident on physical examination. Dilated cardiomyopathy has been anecdotally reported in snakes treated with a parasitacide, and as spontaneous disease. The exact nature of the cardiac disease was not well detailed.

Aortic aneurism has been reported in a Burmese python; pericardial effusion of unknown etiology in a turtle;. Myocardial Listeriosis in a bearded dragon; myocardial salmonellosis in a boa,

Cardiovascular spirorchid flukes can cause endocarditis, arteritis and thrombosis in green turtles. Treatment with praziquantel eradicated the flukes in affected turtles.

Therapy of cardiac disease is virtually unreported. The case of mitral insufficiency in a snake attempted to use frusemide, but it was subsequently realized that snakes do not have a Loop of Henle on which frusemide would be expected to work. Turtles do demonstrate responses to frusemide, suggesting the presence of appropriate chloride channels within the kidney. Whether other diuretics, such as thiazides or spironolactone, would be effective, is unknown.

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