Heart: Contraction, Conduction, and Electrocardiography

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THE CARDIAC CYCLE

The sequence of events occurring in a complete heartbeat, a cardiac cycle, includes mechanical contraction of the atria and ventricles (systole) and relaxation of the heart muscle (diastole). This sequence is followed by filling of the ventricles (diastasis). Accompanying these events are changes in volume and pressure in the atria and ventricles.

The contraction phase in mammals is normally the shorter phase and varies little with heart rate, but ventricular relaxation varies greatly and inversely with heart rate. Very little work has been conducted on these events in the avian cardiac cycle, but there is no reason to expect major differences between birds and mammals; the pressure-flow curve of the left ventricle of a duck (Jones and Johansen, 1972) is much like the mammalian one in appearance. The actual pressures reached have been discussed in Chapter 4. Langille and Jones (1975) reported that in the Pekin duck contraction occurred synchronously in the right and left ventricles. At a mean heart rate of 219 beats/min, ventricular systole comprised 44 percent of the cardiac cycle, but the duration of systole in the right ventricular was 30 percent greater than in the left ventricle.

Purton (1971), who recorded atrial and ventricular pressures in chickens, reported no principal differences in the left ventricular pressures curves of birds and mammals, but he revealed significant differences in the atrial pressure waves which he divided into four stages (Figure 5-1). In stage 1 (points 1 – 2) pressure rises during atrial systole. In stage 2 points (2 – 3), pressure drops during atrial relaxation and there is a continued flow of blood into ventricles followed by closure of the AV (atrioventricular) valves and ventricular systole. Stage 3 (points 3 – 4) is atrial filling, in which the pressure shows two peaks; the first may be associated with the bulging of AV valve into atrium and the second represents maximal atrial filling (Purton, 1971). In stage 4, (points 4 – 1), the AV valves open and atrial pressure falls to its lowest level as blood enters ventricle.

FACTORS AFFECTING HEART RATE

Small birds and mammals usually have higher heart rates than large ones, but there are exceptions.

Actually most heart rates previously determined (see Table 5-1) have been on birds restrained in different ways and degrees. It is now known that restraint influences heart rate considerably, probably in two ways. The initial excitement attending restraint increases heart rate and sympathetic discharge directly (Cain and Abbott, 1970; Cogger et al., 1974) and continued restraint up to 3 hr causes a progressive decrease in blood pressure and an increase in heart rate (reflexly; Whittow et al., 1965). When birds are allowed to move around (telemetry) or are only partially restrained, heart rates are much lower and probably represent the normal resting rates.

Normal Heart Rates

The heart rates of several adult species are shown in Table 5-1; those for embryos are given in Table 4-2 (Chapter 4).

In embryos. Embryonic chick heart rates have been determined by Girard (1973) (see Table 4-2), by Evans (1972), and by Soliman and Huston (1972), who have described methods of recording embryonic rates directly through the egg shell. These rates have been recorded from day 3 to hatching time and afterwards and range from 138 per minute at 3 days to 221 per minute on the twentieth day (Girard, 1973). The figures found by Soliman and Huston (through the egg shell) range from 218 to 324 per minute during the same periods. Differences in the handling temperature of egg and embryos may have accounted for much of this difference.

In adults. It is apparent that adult heart rates vary considerably between and among species. Much of this variation is attributed to variation in tone or restraint of the cardioaccelerator (CA) and cardioinhibitor nerves (vagus) to the heart; the factors influencing the variation are discussed below.

Neural Control

Center for heart rate. There is evidence for a cardioinhibitory center in the medulla (Cohen et al., 1970). The cells involved have their greatest