number in a particular tissue. They are: (i) mitotic age of the tissue; and (ii) frequency of mitotic abnormalities originally present in the tissue. Gopal-Ayengar[3] has reported a very significant observation which supports this hypothesis. He finds, in his study of transplanted tumours in several strains of mice and rats and in the Chinese and golden hamsters, that the cell population of early transplanted generations of tumours are almost entirely diploid, whereas later transplants have diploid, triploid and intermediate types exhibiting a gradual shift to tetraploidy.

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Physiology of the Heart of *Asellus aquaticus* L.

WORK on this subject was carried out some years ago, on nearly three hundred intact individuals and heart preparations, the latter consisting of heart, pericardium and dorsal body-wall of the thoracic region. The heart-rate of an average (10 mm.) individual at summer temperature was 180–200 per min., but heart-beat was interrupted for short periods, for example, at the onset of a burst of locomotor activity. Dissection of the preparation always slowed and sometimes arrested the beat for periods up to 30 min. On recovery, the beat was regular and incessant, often at the maximal normal rate.

Mechanical and electrical stimulation revealed an accelerator nerve following the anterior aorta, undoubtedly the anterior nerve of Alexandrowicz[1]. Transection of the ventral nerve cord just behind the head regularly slowed the heart *in situ*. Transection of the anterior aorta in this region had little effect, and transection of the ventral nerve cord farther back had progressively less effect. Section of the anterior thoracic segmental nerves also had little effect; but cutting those of posterior segments, or damage to the abdominal ganglia, often (not always) caused temporary arrest—probably an injury-stimulation of cardio-inhibitory fibres.

Faradic stimulation of the preparation accelerated the beat, which synchronized with the stimulus at moderate rates of acceleration. Increasing rate progressively shortened systole, until complete diastolic 'amplitude-arrest'—probably tetanization of the dilator muscles[2]. On switching off, such an arrested heart passed back through the stage of rapid flutter to its normal beat. Sensitivity of the heart to faradic stimulation when applied in the aortic region was greater than when applied in the heart region itself, so that a rate of stimulation which arrested when applied in the former region, accelerated when applied in the latter. Sensitivity was much lower to faradic currents applied transversely than to those along the heart.

Relatively light pressure, localized or diffuse, to the ventral face of the floor of the pericardium caused arrest, its duration proportional to intensity of stimulation; a local reflex inhibition seems indicated. Moderate traction dorsalwards of the roof of the pericardium (that is, the body-wall) usually accelerated the heart-rate, and the volume of the pericardium may be the critical factor. Excessive traction inhibited heart-beat.

Asynchrony between the two ends of the heart was observed after transection of the dorsal heart-wall[3], and often in moribund preparations. Sometimes asynchrony between the lateral halves was observed; on occasion one half, longitudinal or lateral, was completely quiescent. Complete asynchrony of all muscle fibres—the 'bag of worms' type of fibrillation—was produced by excess sodium chloride and possibly by other conditions. Activity of aortic valves and ostia showed some independence of that of the myocardium, as expected from their innervation[1]; but in recovery from arrest, activity often began in the ostia and spread to the myocardium.

The optimal concentration of sodium chloride for survival appeared to be 0·6 per cent, that is, lower than that used by Kawaguti[4] for *A. nipponensis*. Preparations survived up to 48 hours in 0·6 per cent sodium chloride, 0·02 per cent potassium chloride, 0·02 per cent calcium chloride, 0·02 per cent sodium bicarbonate, and 0·2 per cent glucose. Excess calcium and deficit of potassium ions slowed or arrested the heart in diastole. Excess potassium ions accelerated, with diastolic amplitude-arrest as the limit; exoskeletal calcium ions probably prevented effective calcium-deficit, and calcium-free Ringer's solution had little effect. Magnesium, beryllium and hydrogen ions slowed, to arrest in diastole. Acetyl-choline accelerated in relatively high concentration[5]. Adrenalin had no consistent effect. Inhibition by the animal's own digestive fluid[2] was confirmed; mammalian lipase and trypsin similarly inhibited.

The curve relating temperature centigrade (T) (changed gradually) to beat-interval (t), that is, to the reciprocal of rate, was approximately a rectangular hyperbola $(t + a)\ (T + b) = c$, where a, b, c are constants. Thus there was an upper limiting rate and a lower limiting temperature, a 'physiological zero', at which the beat virtually ceased. This varied between + 4° and − 4° C. (obtained by supercooling), in different individuals, but was usually below 0° C. Maximal rate was attained at 25–35° C., that is, again variable between individuals; above this temperature there were irregularities and slowing of the beat. Since a was small (less than 0·5 sec.) a virtually linear relationship between rate and T was often obtained, and it follows also that $(\log t)/\{\log (T + b)\}$ was virtually linear. Preparations frozen by seeding the supercooled medium recovered their beat on thawing.

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