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3361

Temperature Characteristics for Heart Rate in Embryos of Limulus.

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For a number of *Limulus* embryos the relationship has been determined between temperature and the frequency of cardiac contractions, during stages of development when the (myogenic) heart is still devoid of a nervous system. A thermostat maintaining any desired temperature between 0.05° and 45° to within ±0.01° or better, and quickly adjustable, was devised to facilitate this work. The temperature characteristics obtained are not the same in all individuals. The values $\mu = 11{,}500$ and 16,400 are usual; but $\mu = 20{,}000$ and $\mu = 25{,}100$ also occur. These are well recognized values in numerous other vital processes, but differ from the magnitude ($\mu = 12{,}200$) which seems typical for the thermal control of frequency of contraction in the heart of the adult (the temperature of the ganglion being altered). This need not be interpreted as corresponding to an essential difference between myogenic and neurogenic rhythm. The critical temperatures for heart rhythm also differ among individuals; the following are of definite occurrence, in different cases: 9°, 20°, 27°, 31°, 41°, 45°.

The results are chiefly important as indicating relatively greater metabolic lability in embryonic than in adult tissues. It is concluded that the apparent inconstancy of μ for contraction rate in explants of chick myocardium (H. Murray) is due to the presence of several functional pacemakers having different temperature characteristics, corresponding to the finding of different μ's for the hearts of similar *Limulus* embryos. This is proved by the fact that in cases giving atypical μ's, the latitude of variation is not a constant fraction of the mean; the marginal heart-beat frequencies yield typical μ's.

It cannot be held that temperature characteristics are determined by the organism as a whole. The movements of heart and of gills, in *Limulus* embryos, show different μ values in the same individual at the same time; and the critical temperatures for the two activities do not coincide. This is also true in *Asellus*. The critical increments or temperature characteristics have, therefore, a specific, local significance.

3362

Measurements of Fatal Doses of Chloroform in the Brains of White Rats.

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Qualitative tests. The aqueous extracts of the brains of frogs, mice, rats, guinea pigs, cats, and dogs, killed by inhaling chloroform, have all shown the presence of chloroform by the pyridine test,[1] while similar extracts from animals killed by electricity, illuminating gas, CO_2, blow on the head, and beheading, have all been negative for chloroform. In a series of 8 brains from rats killed by inhaling chloroform, the tests were made at the end of 1, 1½, 2, 3, 5, 6, 15 and 25 days respectively. In each case chloroform was indicated, in spite of the fact that considerable decay of the tissues had occurred. These results agree with those reported by Luedeking,[2] and Angiolani,[3] to the effect that chloroform in animal tissues persists unchanged for many days after death and decay.

Quantitative tests. The chloroform present in extracts of brains from rats killed by inhaling chloroform has been measured by the pyridine colorimetric method. Into a 1-liter dish containing a rat, a known amount of chloroform was sprayed. At the moment of death (cessation of respiratory movements) the rat was removed and its brain extracted. A dose of 0.1 cc. was sub-lethal in 35 minutes; 0.2 cc. caused death in an average of 11.2 minutes; 0.4 cc. in 5.4 minutes; 0.8 cc. in 4.3 minutes; 1.0 cc. in 3.5 minutes; 2.0 cc. in 2.5 minutes; 4.0 cc. in 1.6 minutes and 5.0 cc. or more in 1.5 minutes. The time of death bore no clear relationship to size of the animal. In Table I are given the amounts of chloroform found in brains of killed and anesthetized rats.