LETTERS TO THE EDITORS

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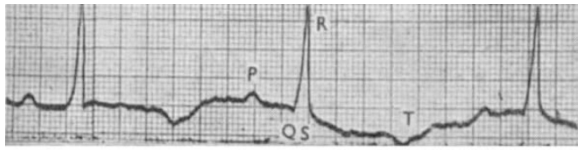
Electrocardiogram of the Crocodilian Heart

As part of an investigation of the histology and functional anatomy of the crocodilian heart, electrocardiograms of the heart in intact animals (Nile crocodiles) were recorded. Part of a tracing, using the standard lead, throat to abdomen, is shown in Fig. 1. While the general form of the electrocardiogram is similar to that of the normal human heart (Fig. 2), except that the *T*-wave is inverted in the crocodile owing to the lead used, significant differences are observed, particularly with reference to the spacing of the several events within the cardiac cycle.

Thus, in the crocodile, the time taken for the wave of negativity to pass from the beginning of the *P*-wave (the onset of atrial systole) to the summit of the *T*-wave (the completion of ventricular systole) is about three-quarters of the entire cardiac cycle (from one *P*-wave to the next), whereas in the human this ratio is reduced to one-half. The heart-rates of the crocodile and human are, of course, different; namely, in these cases, crocodile, 36 beats a minute; human, 72 a minute. In a human heart from a case of sinus bradycardia (Fig. 3), in which the rate (37 beats per min.) happened to be practically the same as that of the heart of the crocodile, it will be noted that the *PQRST* complex occupies about the same time as that in the normal human; this signifies that the wave of negativity passes over the heart as quickly as in the normal human rhythm and that the slowness of the heart in the case of sinus bradycardia is due to the lengthening of the 'silent' period, *T* to *P*, while the ratio of the *P–T* interval to the entire cardiac cycle becomes reduced still further to three-tenths.

Measurements show that the actual time taken for the wave to pass over the ventricles (that is, the *Q–T* interval) in the human in both instances is 0·37 sec., whereas in the crocodile it is 1·0 sec. This difference is still further emphasized when it is remembered that the distance which the wave has to travel across the crocodilian ventricles (the heart of the crocodile in question being about the size of that of the average cat) is very much less than the distance across the human ventricles. The ratio for the above parts of the curves (1·0 to 0·37) is just about the same as the ratio of the *P–R* intervals, that is, for the part of the curve related to the passage of the wave across the atria, which takes 0·40 sec. in the crocodile and 0·15 sec. in the human.

Fig. 1



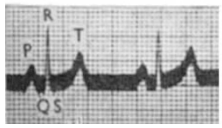


Fig. 2

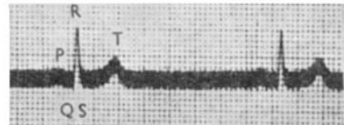


Fig. 3

The crocodilian heart has complete atrial and ventricular septa; but, as our histological investigation has revealed, it possesses no trace of specialized nodal or Purkinje tissues such as are found in the human heart. Thus in the crocodile the impulse for cardiac contraction is initiated and conducted by ordinary cardiac muscle, and the conduction takes very much longer in absolute time and occupies a much greater proportion of the complete cardiac cycle than when Purkinje tissue is provided for its transmission. We submit that in the above phenomena we have further corroboration of the thesis put forward by Davies and Francis[1] that these specialized muscular systems (nodal and Purkinje tissue), found in the hearts of both birds and mammals, are neomorphic structures evolved in conjunction with the higher heart-rates of these homoiothermic vertebrates.

A detailed account of the histology of the hearts of the crocodile and alligator will be published elsewhere.

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[1] Davies, Francis, and Francis, E. T. B., *Phil. Trans.*, B, 231, 99 (1941).

Reversible Poisoning of Nerve Fibres by Heavy-Metal Ions

The activity of enzymes is modified—inhibited in most cases—by heavy-metal ions. Blockage of free sulphhydryl groups of the enzyme protein is held to be the cause of the inhibition[1]. A release of these groups, with subsequent recovery of enzyme activity, can be obtained *in vitro* by thiolic substances such as cystein or glutathione, which combine with the metallic ions. The sulphhydryl groups may be blocked also when the enzyme is in its original locus inside living cells. The inhibition of the enzyme is accompanied in this case by a breakdown of various cellular functions. This biochemical lesion (Peters), produced by a number of "substances thioloprives" (Bacq), can be prevented in many instances by mono- and di-thiols; but only in some cases, particularly in poisoning of the whole organism, can it be reversed.

The effect of heavy-metal ions on peripheral nerve has been already studied[2,3]. Their action (block of excitability and impulse conduction) is described as irreversible; attempts to restore nerve function by washing with Ringer's fluid were not successful.

A series of experiments has been performed in order to investigate the influence of thiolic substances on nerve fibres poisoned with heavy-metal ions. Bundles of motor nerve fibres isolated from the sciatic nerve of *R. temporaria* have been employed. They were free from connective tissue, peri- and endo-neural, and were still attached to the gastrocnemius muscle. Excitability threshold (rheobase) was measured every twenty seconds throughout the experiments. No attempt has yet been made to correlate the observed changes of excitability with variations in other nerve properties. Bufferless Ringer's solution was used. The results so far obtained can be summarized as follows.

Salts of cadmium, copper, mercury, silver and uranium produce an inhibition of nerve excitability. Zinc ions failed to cause any change.