from the zona reticularis ; although it is impossible to rule out entirely the presence of an occasional cell from the inner part of the fasciculata, these must at the most be few in number. The technique used and method of introduction into the eye precludes the possibility of transferring detached cells of the outer fasciculata or glomerulosa.

The proliferation of these inner-zone cortical cells is in keeping with the views of Blackman[4], Yoffey[5] and others, who regard the zona reticularis as a functionally active zone, while the formation of a peripheral glomerulosa in the absence of subcapsular cells supports the views of Jones and Wright[6], who consider that mechanical factors are responsible for the existence of this zone in the Eutherian adrenal cortex. Cortical cells in the graft multiply (as evidenced by mitotic figures), even when the right adrenal is intact ; mitotic figures have not, however, been observed in chromaffin cells, and it appears that, although these elements persist, regeneration does not occur.

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A Substance from Eel Serum producing Slow Contractions

Eel serum is well known for its hæmolytic and toxic properties[1] ; but so far no report seems to have been published dealing with its effect on isolated organs. In the course of investigations into the effects and components of various animal venoms, it was noted that eel serum contained a substance which causes a delayed, slow contraction of the isolated guinea pig ileum and bears some resemblance to the substances producing slow contractions present in wasp venom[2] or appearing in cat plasma after intravenous injection of thalassine[3].

The material possessing this property proved so potent that in most cases it was found impossible to test its effect by applying undiluted serum to a sensitive preparation like the isolated guinea pig ileum suspended in magnesium-free Tyrode solution. When eel serum was diluted with 0·75 per cent saline to make a final dilution of 1 : 10 to 1 : 50, doses corresponding to as little as 0·002–0·01 ml. of the original serum contracted the gut suspended in an organ-bath of 18 ml. capacity. The contraction began after a typical latency of 10–45 sec. and reached its maximum after 60–90 sec. It persisted following treatment of the gut with atropine and neoantergane in doses sufficient to suppress strong acetylcholine or histamine contractions, and also remained unaffected after tryptamine desensitization[4]. In contrast to the substance present in wasp venom causing slow contraction, repeated doses of eel serum were progressively less active even if the interval between the contractions was extended to 15 min. and more ; the gut then still responded to histamine in the usual manner.

If eel serum diluted with 0·75 per cent saline was dialysed against distilled water for up to 72 hr., no appreciable activity could be detected in the dialysate, whereas practically all of it could be recovered in the inner compartment, thus indicating that the active component is most probably a large molecule. The material producing the slow contraction was little affected by boiling diluted serum at neutral *p*H for 5 min. but was destroyed after a few minutes of this procedure in 0·1 *N* sodium hydroxide.

Undiluted eel serum caused a contraction of the isolated rat colon resembling that produced by serotonin in very small amounts. If all the activity detected by using the rat colon as a test organ is taken as representing serotonin, eel serum would contain about 0·03 μgm. serotonin per ml., a figure which is in good agreement with that published by Erspamer and Faustini[5] for extracted eel serum. No evidence for histamine occurring in appreciable amounts in eel serum was obtained. Small doses of eel serum produced vasoconstriction of long duration in the isolated rabbit ear perfused with Ringer–Locke solution at room temperature.

The nature of the material producing slow contraction is being further investigated and a detailed account of this work will be published elsewhere.

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Spontaneous Microscopic Activity in Cardiac Muscle

When the heart stops beating, pulsations in individual cardiac muscle fibres continue. This can be observed by examining thin sections of living cardiac muscle under high-power magnification and transmitted light. The pulsations appear to be due to alternate relaxations and contractions of the fibres, with a concomitant change in the width of the striations. Under low-power magnification and by reflected light, the activity seen in the isolated mammalian heart is that of spontaneous minute contraction waves. Prinzmetal *et al.*[1] have noticed a similar though much faster activity in human and dog fibrillating auricles. It disappeared with the cessation of fibrillation.

These spontaneous minute contraction waves can be observed not only in non-beating hearts but also when the latter beat slowly *in situ*. This fact makes it uncertain if the pulsatile activity seen under high-power magnification and the spontaneous minute waves are one and the same phenomenon, and whether the pulsations are present only after cessation of the co-ordinated beat or whether they are a normal accompaniment of the heart-beat.

The spontaneous minute waves have been seen in all mammalian hearts so far examined, and their survival-times have been found to vary considerably, the longest seen so far being in rabbit auricles (stored at 4° C.) in which they could still be observed six days after isolation.