Thus it may be concluded that the main default in the renal handling of electrolytes in adrenal insufficiency in dog was the failure of preserving sodium and especially bicarbonate and that the failure of potassium excretion was merely the result of reduced glomerular filtration rate. That the latter was due to acapnic central deteriorations during the progress of respiratory compensation for the renal acidosis resulting from the excess loss of sodium over chlorine has been fully discussed in a previous report[4]. Glucocorticoids, which can ameliorate this central acapnic deterioration, could prevent the progressive reduction of glomerular filtration rate, as aforementioned (compare the lower limit of glomerular filtration rate in lines *A* and *B* in Fig. 1). Concerning the various kinds of acute experiments showing a short-lived excretion of potassium independent of glomerular filtration rate, care should be taken for leakage of potassium from cells into the tubular urine.

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Peripheral Vascular Effects of Mixtures of Isopropylnoradrenaline and Noradrenaline in Man

IT is well known that the infusion of 0·1–1·0 μg/min adrenaline into the brachial artery in man results in an immediate transient increase in flow of blood in the forearm followed by a fall to below the resting level. Allen, Barcroft and Edholm[1] showed that this biphasic response was not due to dilatation of muscle vessels followed by constriction of skin vessels. Finding that after dibenyline, adrenaline produces only a sustained vasodilatation, Ginsburg and Cobbold[2] suggested that the action of adrenaline when given alone was due to an initial stimulation of inhibitory (β) receptors followed by stronger stimulation of excitatory (α) receptors. The action of adrenaline after dibenyline resembles that of isopropylnoradrenaline. Recently we have found that after dichloroisopropylnoradrenaline, adrenaline causes only sustained vasoconstriction, and under these conditions resembles noradrenaline[3].

If adrenaline stimulates first the inhibitory and then the excitatory receptors, we might expect its action to be matched by a suitable mixture of isopropylnoradrenaline and noradrenaline. We have tested this in a series of experiments, of which Fig. 1 shows a typical example. Forearm blood flow was measured by venous occlusion plethysmography. The typical responses to isopropylnoradrenaline (0·05 μg/min) and to noradrenaline (0·25 μg/min) are shown in the first two panels. When these doses of the two drugs were administered simultaneously (panel 3) the response was an initial vasodilatation followed by a vasoconstriction. This response can be seen to bear a strong resemblance to the response to 0·25 μg/min adrenaline, illustrated in panel 4.

These results indicate that the response of blood vessels of the forearm to adrenaline can be explained

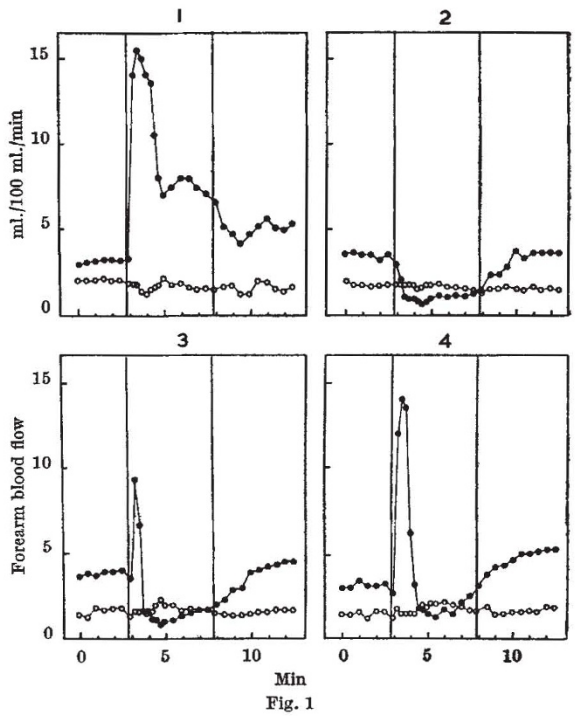

Fig. 1

on the basis of the response of the two types of receptor proposed by Ahlquist[4], and thus provide positive evidence for the theory proposed by Ginsburg and Cobbold.

It would be interesting to know if other actions of adrenaline in man and animals can be reproduced by mixtures of isopropylnoradrenaline and noradrenaline, and whether the proportions needed are always the same.

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Receptive Fields of Single Cells in the Rabbit Lateral Geniculate Body

THE activity of single cells in the lateral geniculate body of lightly urethanized rabbits has been recorded with steel micro-electrodes[1]. The retinal receptive fields of these cells have been explored. The light source, a slit lamp bulb, was incorporated in a multibeam photo-stimulator similar to one previously described[2,3] and mounted on a perimeter arc.

In a proportion of cells the receptive field was 'simple' in the sense that the responses evoked by the exploring light spot were the same throughout the receptive field. In other cases in which the receptive fields were larger the response to light varied across the field. The functional organization of such fields is of interest and a typical result is shown in Fig. 1.