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Prevention by Cortisone of Histamine-induced Gastric Ulcer in Guinea Pig.

By

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Introduction

For many years histamine has been widely used in the experimental production of gastric ulcer. The drug has been given by various routes and in many species of laboratory animals (3). It is suggested that the ulceration is primarily due to increased acid secretion of the stomach. In addition, the vasospastic effect of histamine has been postulated as playing a contributory part in the histamine-induced ulcer by diminishing the vitality of the mucosal cells (5).

The study of the influence of corticoids on experimental gastric ulcer has been the subject of many interesting works. Andreani (1) has demonstrated the inhibitory effect of ACTH on histamine-induced ulcer by the method of Halpern and Martin. Selye et al. (6) have observed the inhibition of occurrence of 48/80 (a particularly potent histamine liberator) induced ulcer by pretreatment with cortisol.

In the present work the influence of cortisone on histamine-induced gastric ulcer is studied.

Materials and Methods

Studies were made in 75 guinea pigs of both sexes, weighing 220-300 g. The method of experiment was similar to that described by Eagleton and Watt (2). The animals were housed in individual cages and received no food for 24 hours prior to injection. Three groups of 25 guinea pigs were used. The first and second groups were injected intraperitoneally with 200 mg/Kg of cortisone acetate. Two hours later

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the second and third groups were injected with histamine phosphate by the same route at the dose of 2 mg/Kg in a 1/5000 concentration in normal saline. With this dose of histamine there were no acute asphyxial deaths among the guinea pigs. The animals were killed 6 hours after the administration of histamine by a blow on the head. The stomachs were immediately removed, opened along the greater curvature, washed with water, and carefully examined by direct lighting, and without knowledge of the treatment. Necrohemorrhagic spots were considered evidence of ulcer formation.

Results

The results are summarized in table I They show that:

1_ The administration of cortisone alone (at the above dose), does not produce any gastric ulceration in guinea pigs.

2_ The intraperitoneal injection of 2 mg/Kg of histamine produces gastric ulcers in 68% of the animals.

3_ Pretreatment of histamine injected animals with cortisone significantly diminishes the occurrence of gastric ulcers.

$(x^2 = \frac{(bc-ad)^2 k}{efgh}\ ;\ p < 0.005)$

Table 1

The incidence of gastric ulcer in guinea pigs treated with cortisone and histamine.

Drugs and Doses (mg/Kg body weight)	No. of animals	No. showing ulcers	Percentage
Cortisone 200 -	25	0	0
— Histamine 2	25	17	68
Cortisone 200+Histamine 2	25	7	28

Discussion

Our findings are in accord with that of Selye and associates (6). Furthermore, it is of importance that cortisone has antagonized the effect of exogenous histamine on the stomach.

It is known that glucocorticoids can produce acute gastric ulcers, although they actually prevent anaphylactoid inflammation. The mechanism of this anti_inflammatory action has not been elucidated. Also, these compounds are not truly antihistaminics (4).

It is possible that in the present experiment cortisone has prevented the occurrence of histamine_induced gastric ulcer by its anti-inflammatory action.

Summary

The effect of cortisone on the experimental gastric ulcer produced by intraperitoneal injection of histamine in the guinea pig is studied. A reduction in the number of animals showing gastric ulcer in observed. The possible mechanism of this effect of cortisone is discussed.

Résumé

L'effet de la cortisone sur l'ulcère expérimental gastrique produit par l'injection intrapéritonéale de l'histamine, chez le cobaye est étudié. Une réduction dans le nombre des animaux porteurs d'ulcères est observée. Le mécanisme possible de cet effet de la curtisone est discuté.

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The Measurement of Isodose Curves of a Cobalt Unit by a Photographic Technique ✡

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Intoduction

When using X- or gamma-rays in the treatment of cancer, it is necessary to know the distribution of the patient's body, so as to be sure of accuracy of dosage to the tumour and to avoid the possibility of overdosage of normal tissues. This distribution is normally found by the use of isodose curves; these are curves joining points of equal dosage, the intensity of which is related to the maximum dose found in the treated volume. A typical example is shown in Fig. 1, which relates to a cobalt unit; it is seen that the maximum dose occurs at a depth of 0.5cm beneath the surface, and that the curves are symmetrical about the beam axis.

For a cobalt unit, the shape of the curves may depend on the physical dimensions of the source, the design of the beam-limiting diaphragms, and the ratio of source-skin-distance to source-diaphragm distance. Since these all vary from Centre to Centre, it is essential that the curves be measured individually, for all field sizes in common use.

However, it is found the doses alons the central axis depend only on the field size and the source-skin distance, and do not vary with the design of the treatment unit. Here, it is convenient a quantity konwn as the percentage central axis depth dose, which is specified as the

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