

Prevention by Cortisone of Aspirin-Induced Gastric Ulceration in the Rat*

By

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INTRODUCTION

The universal popularity enjoyed by aspirin (acetylsalicylic acid) as an effective antirhumatic and analgesic agent, coupled with its ready availability in countless proprietary medicines, prompts the need for critical evaluation of gastric lesions which might be induced in the usual therapeutic doses¹.

One side effect of aspirin is gastric ulceration which sometimes progresses to severe gastric hemorrhage². Many clinical and experimental research has been done on the mechanism of aspirin-induced gastric ulceration^{3,4,5}.

The study of the influence of corticosteroids on experimental gastric ulcer has been the subject of many interesting studies. Selye and associates⁶ have observed the inhibition of occurrence of 48/80 (a particularly potent histamine liberator) induced gastric ulcer by pretreatment with cortisol. It is also observed that pretreatment of guinea pigs with cortisone has significantly prevented the occurrence of histamine-induced gastric ulcers⁷.

In the present work, the effect of cortisone on aspirin induced gastric ulceration in the rat is studied.

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METHODS

Eighty rats of both sexes, weighing 90-110 g were used in this study. The animals were housed in individual cages and starved for 24 hours. They were divided into 4 groups. The first and second groups (10 and 30 rats respectively) were injected with cortisone 200 mg/Kg body weight. Thirty minutes later the groups two and three (30 and 30 rats) were injected with acetylsalicylic acid 150 mg/Kg, suspended in olive oil. The fourth group (10 rats) were injected with 1 ml/100 g body weight of olive oil. All injections were made by intraperitoneal route. Five hours after the last injection the animals were killed by a blow on the head. The stomachs were immediately removed and opened along the greater curvature. They were washed with water and carefully examined by direct lighting. Any hemorrhagic area 2 mm or greater in its diameter was considered as positive evidence of ulceration. The frequency of these necrohemorrhagic lesions, found in the glandular part of the stomach, ranged from 2 to 12 per stomach.

RESULTS

The results are summarized in table I. The percentage of gastric lesions after the administration of aspirin in the control group (group 3) confirmed that obtained by Brodie and Chase².

The results also show that cortisone, with the dose of 200 mg/Kg body weight, significantly prevented the occurrence of aspirin induced gastric ulceration in the rats. There was no ulceration in the group one and four. The statistical significance was calculated by the X² method.

DISCUSSION

The ability of aspirin to produce acute erosive lesions in the stomach, both in human and experimental animals, has been repeatedly demonstrated. The mechanisms concerned in the production of this lesion have not been elucidated. Of the various possibilities, augmented acid secretion and an increased hemorrhagic tendency do not seem to be of importance—but the relative significance of impaired mucus production, mucosal damage consequent on histamine liberation and increase acid diffusion, and accelerated exfoliation of cells from the gastric mucosa remains

to be assessed³. Thus, although the potential effects of aspirin are now appreciated, as yet there is comparatively little understanding of the fundamental mechanisms involved in the production of gastric mucosal damage. Whatever the mechanism may be, it seems that an excess of the release of histamine, leads to an increase acid diffusion, which is necessary for the production of the aspirin-induced gastric lesions. In the present study it is possible that cortisone has prevented the appearance of ulcerations by reducing the elaboration or release of histamine.

Table I

Drugs and doses (mg/Kg body weight)	Number of rats	Number with ulcers	Percentage
Cortisone 20 + 0	10	0	0
Cortisone 20 + Aspirin 150	30	9	30*
0 + Aspirin 150	30	24	80
Olive oil 1 ml/100 g body weight	10	0	0

Table I - The incidence of gastric ulceration in rats pretreated with cortisone and acetylsalicylic acid.

*) Indicates $p < 0.005$ when compared with the aspirin group value. (group three).

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Summary

The effect of cortisone on the experimental gastric ulceration produced by intraperitoneal injection of aspirin in the rat is studied. Cortisone has significantly prevented the appearance of aspirin-induced gastric ulceration. The possible mechanism of this effect of cortisone is discussed.

Resume

L'effet de la cortisone sur l'ulcère gastrique expérimental produit par l'injection de l'aspirin chez le rat est étudié. La cortisone a significativement prévenu l'apparition des ulcérations gastriques induites par l'aspirin. Le mécanisme possible de la cortisone est discuté.

References

- 1- Weiss, A., Pitman, E.R. and Graham, E.C. and (1961). Aspirin and gastric bleeding. *Amer. J. Med.*, 31, 266.
- 2- Brodie, D. A. and Chase, B. J. (1967). Role of gastric acid in aspirin-induced gastric irritation. *Gastroenterology*, 53,604.
- 3- Salter, R. H. (1968). Aspirin and gastrointestinal bleeding. *Amer. J. Dig. Dis.*, 13,38.
- 4- Levrat, M. et Lambert, R. (1960). Ulcères médicamenteux chez le rat. 3. L'acide acétyl-salicylique. *Gastroenterologia (Basel)*, 94,273.
- 5- Grossman, M. I., Matsumoto, K. K. and Lichter, R. J. (1961). Fecal blood loss produced by oral and intravenous administration of various salicylates. *Gastroenterology*, 40,303.
- 6- Selye, H., Jean, P. and Cantin, M. (1960). Prevention by stress and cortisol of gastric ulcers normally produced by 48/80. *Proc Soc. Exp. Biol. & Med.*, 103,444.
- 7- Djahanguiri, B., Khoyi, M. A. and Sadeghi, Dj. (1968). Prevention by cortisone of histamine-induced gastric ulcer in the guinea pig. *Acta Med. Iranica*, Vol. 11,9.

NEEDS OF THE MIDDLE EAST FOR HEALTH MANPOWER*

By

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Introduction

The purpose of this presentation is to introduce the Health Manpower situation in the Middle East in the context of its socioeconomic and general health conditions to make possible an objective estimate of the present and the future manpower needs of the area.

In order to do so, first the area of the Middle East and some of its historical highlights will be reviewed. Then some demographic and socio-economic conditions will be examined. This will be followed by a short evaluation of the health status of the area. The rest of the paper will be devoted to the problem of health manpower and some of the achievements already made in this respect by various countries of the Middle East.

As the subject of this presentation is so broad in its implications of time and of content, it is proper at the outset to delineate the limits of this discussion. Due to the limitation of the data available on all categories of the health team, only two of those, namely physicians and

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